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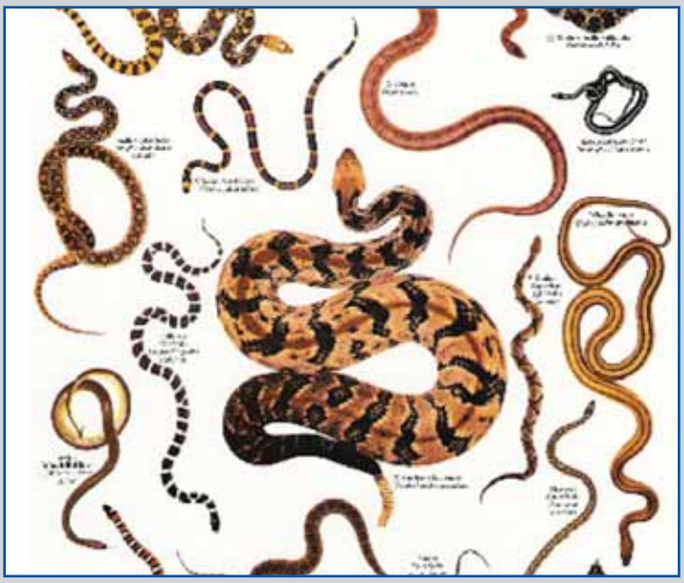

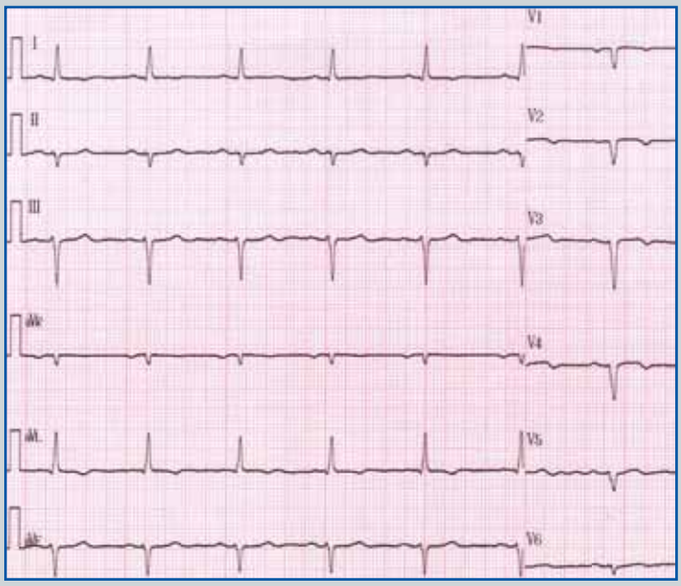

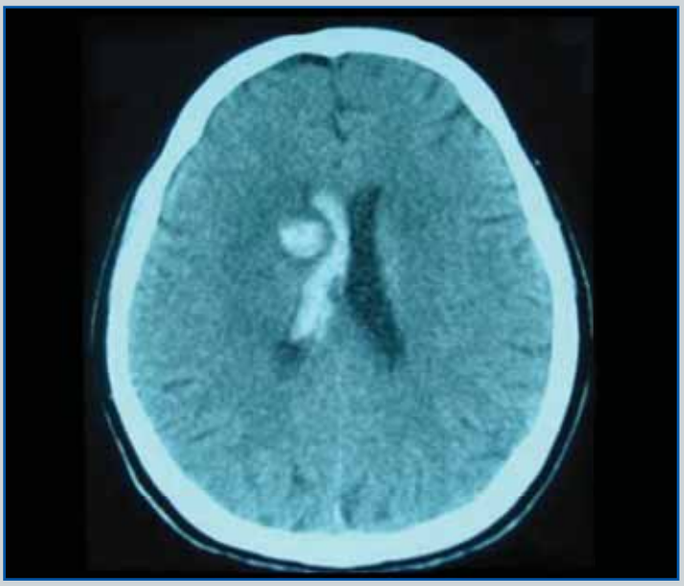

Trimestriel

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December 2011-N°9

The 1st Global Network Conference on Emergency Medicine

Dubai International Convention and Exhibition Centre, Dubai, UAE

13 – 17 January 2012

Pre-Conference Workshops

	Friday 13th January Pre-Conference Courses				Saturday 14th January Pre-Conference Courses				
	WS 1	WS 2	WS 3	WS 4	WS 1	WS 2	WS 3	WS 4	WS 5
09:00 - 18:00	Ultrasound	Simulation	Pediatric Emergency	Metabolic	Ultrasound	Simulation	Pediatric Emergency	Administration	Disaster Medicine

Main Conference

	Sunday 15th January Conference			Monday 16th January Conference			Tuesday 17th January Conference		
	Track 1	Track 2	Track 3	Track 1	Track 2	Track 3	Track 1	Track 2	Track 3
09:00 - 12:30	Intensive Care	Administration	Cardiovascular	Pre-Hospital	Research	Trauma	Toxicology	Sepsis	Education
13:00 - 14:00	Lunch Break			Lunch Break			Lunch Break		
14:00 - 18:00	Ultrasound	Administration	Disaster Medicine	International	Pediatric	Residents Session	Q&A Session	To Be Confirmed	Education

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When there is a will,
there is a way ..

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Emergency medicine organization: A challenge for every health system .....

It makes no doubt that medical practices around the world reflect the people's culture. Amongst all medical specialties, Emergency medicine is the one that witnessed the greatest evolution over the past decade. This evolution necessitates an in-depth reflection over the ideal organization of an emergency chain in any said hospital, country, region or even in the whole world.

Many questions are raised in this regard: how are emergencies handled today? How does the socio-economic environment weigh on the organization of emergencies? What is the best way to organize emergency intervention? Some leading countries have already developed answers to all these questions whilst others are still reflecting over the solutions.

Emergency organization is not restricted to the hospital only. It is rather a whole chain that requires advanced medical resources in terms of personnel and equipment as well as close cooperation with other specialties and medical structures.

Emergency medicine is a global discipline in the sense that it is a cross-cutting specialty that integrates quick access to care, adequate patient transportation and best care by well adapted structures. This specialty is a typical example of secondary prevention and represents an interesting tool that aims at improving public health.

A successful health system is one that takes into account the sound development of Emergency medicine.

In fact, education and research in the field of Emergency medicine are key to its development. This is why Med Emergency publication, is proud to be the official event publication of the first Global Network Conference on Emergency Medicine held on January 13-17, 2012 in Dubai (UAE). As the President of this conference rightly says, this event the first of its kind in the Middle East, aims to promote the specialty of Emergency Medicine and offer a wonderful opportunity for academic/scientific exchange. We strongly encourage you to take part in this event that we hope will be a great success.

Nagi Souaiby, MD, MPH, MHM
Emergency Medicine Specialist
President of the Lebanese Resuscitation Council
Chief Editor

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Those who made a difference in the world of Emergency Medicine

Peter Baskett (1934-2008).....

.....“A Resuscitation Giant”, a “Master”

Born in Belfast, Peter Baskett remained all his life loyal to his homeland and as a stubborn Irish he implemented all his professional projects with determination, overcoming all obstacles with his positive spirit.

Peter Baskett

In 1959, he became an anesthetist and then he started in 1967 one of the first modern reanimation units at Bristol hospital. He was talented enough to know how to extrapolate the fundamental principles of hospital reanimation and implement them in cases of vital distress.

In 1984, he founded the Resuscitation Council (UK) then after four years he founded the *European Resuscitation Council* that he chaired for so many years and for which he remained until his last years a well respected eminent advisor. In 1995, as member of the *International Liaison Committee on Resuscitation*, he established the link between the scientific societies from North America and Europe. He was a founding member of the Club of Mainz and WADEM.

Amongst his notable contributions to pre-hospital medicine, the validation of mix between oxygen and nitrous oxide (Entonox®) an analgesic that he introduced in ambulances towards the end of the sixties. Using his strong anesthetist’s background, he fought for the use of the laryngeal masque in emergencies as an alternative to tracheal intubation.

His commitment to develop and teach Cardio-Pulmonary resuscitation made him deserve recognition by the *American Heart Association* as “Resuscitation Giant” meaning a “Master” and that is a distinction that is rarely granted to a non-American.

Peter Baskett, the Francophile had many friends in the field of Emergency in France with whom he shared many things including medicine. With his strong personality, his right advice, his listening skills, and unshakable conviction, he contributed to the development of a booming specialty: Emergency Medicine.

The Editor.

SUMMARY

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Rim Lakhdhar - Nader Baffoun - Kamel Baccar - Chokri Kadour

Rim Lakhdar

STROKE AND PREGNANCY : Descriptive study of 44 cases

ACCIDENTS VASCULAIRES CEREBRAUX ET GROSSESSE: Etude descriptive de 44 cas

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Abstract

Background: pregnancy and the puerperium are associated with an increased risk of stroke with many specific causes.
Aim of study: evaluate the clinical features, timing, etiology and outcome of stroke occurring during pregnancy.
Patients and Methods: We report a retrospective analysis (1996- 2011) on 54 obstetric patients who have been diagnosed with stroke during pregnancy or were within 8 weeks postpartum. Inclusion criteria Patients were selected based on a coincident diagnosis of pregnancy complicated by preeclampsia and eclampsia and stroke : Arterial ischemic stroke, Cerebral venous thrombosis, Sub arachnoid or Intra cerebral hemorrhage secondary to eclampsia, cerebral vascular rupture of arteriovenous malformation or cerebral aneurysm or Reversible posterior encephalopathy syndrome (RPES) secondary to eclampsia. All patients were investigated with a CT scan of the brain, and MRI and/or cerebral angiography.
Results: We identified forty four patients with a diagnosis of stroke with mean age 29,4 years with extremes 21 to 42 years. Hypertensive disorders of pregnancy were the most common co morbid conditions (32%) and all patients had eclampsia or pre-eclampsia. The pregnancy was well followed in only 12 cases. Delivery was at term in 43% cases and prematurely in 57% of cases with cesarean in 82% of cases. patients were explored by cerebral CT scan in 94,4% of cases, cerebral in 30,6% of cases and venous angio MRI in 27% of cases. Cerebro-vascular complications

INTRODUCTION

Pregnancy and the puerperium are associated with an increased risk of stroke with many specific causes. Stroke is considered an important cause of maternal morbidity and mortality during this time, it accounts for more than 12% of all maternal deaths (1-2). The reported incidences of stroke during pregnancy and the puerperium varied widely, ranging from 5 to 67 per 100,000 deliveries or pregnancies (3,4). Most previous studies on stroke during pregnancy have been mainly focused on incidence and risk factors. Details of etiology and stroke outcome have been fewly reported. In our study, we tried to evaluate the clinical spectrum, timing, etiology and outcome of stroke occurring during pregnancy.

STUDY DESIGN

We report a retrospective analysis (1996- 2010) on all obstetric patients who have been diagnosed with stroke during pregnancy or were within

eight weeks postpartum. Clinical data were compiled retrospectively from database anesthesia and reanimation department of national neurologic institute. Seventy eight patients were included, 34 patients of them were excluded because incomplete investigation or non-stroke diagnosis like reversible leucoencephalopathy or cerebral abcess.

All patients were investigated with a computed tomography (CT) scan of the brain, and MRI and/or cerebral angiography.

Inclusion criteria: Patients were selected based on a coincident diagnosis of pregnancy complicated by preeclampsia and eclampsia wich are defined according to The American college of Obstetricians and gynecologists criteria and stroke:

- Arterial ischemic stroke

- Cerebral venous thrombosis

- Sub arachnoid or Intra cerebral hemorrhage secondary to eclampsia

Or cerebral vascular rupture of arteriovenous malformation or cerebral aneurysm

- Reversible posterior encephalopathy syndrome (RPES) secondary to eclampsia

Non inclusion criteria were embolic cardiopathy, arteriopathy, hematologic affection (Antiphospholipid syndrome, Drepanocytosis..)

Exclusion criteria were neurologic complications of peripartum and surgery or anesthesia

RESULTS

We identified Fifty four patients with a diagnosis of stroke with mean age 29,4 years with extremes 21 to 42 years with a mean gestational age 30 weeks. Hypertensive disorders of pregnancy were the most common co morbid conditions (32%) and all patients had eclampsia or pre-eclampsia. Mean gestity and mean parity were 2. The pregnancy was well followed in only 27 % of cases

(n=12). Delivery was at term in 43 % cases (n=19) and prematurely in 57% of cases (n=25), with cesarean in 36 patients (82%). Complications occured mainly in the post-partum period (86 % of cases). Predominant symptoms of Clinical features were troubles of conscience (agitation, confusion), Headache, convulsions and neurologic deficits as visual changes, aphasia or coma .

Repartition of Cerebro-vascular complications were PRES in 61% cases followed by meningo cerebral hemorrhage in 29,5% and finally TVC 4,5% and arterial ischemia in 4,5% cases. Major causes of cerebral hemorrhage found in this study were pre-eclampsia/eclampsia in 8 patients. Four patients presented hemorrhage secondary to aneurismal rupture, 3 patients presented with bleeding from arterio veinous malformations (AVM). Bleeding as consequences of disseminated intravascular coagulation occurred in 2 patients and 7 patients had hemorrhagic events of unknown origin.

Cerebrovascular modalities included cerebral computed tomography with and without contrast in 95% of cases (n=42), magnetic resonance imaging (MRI) in 17 cases (31%) completed by veinous angio MRI in 12 cases (27%) and angio MRI of willis polygon in 5 cases and by cerebral angiography in 6 patients. Trans cranial Doppler was used in 8 cases.

Control of cerebral imaging data was used in only half of cases.

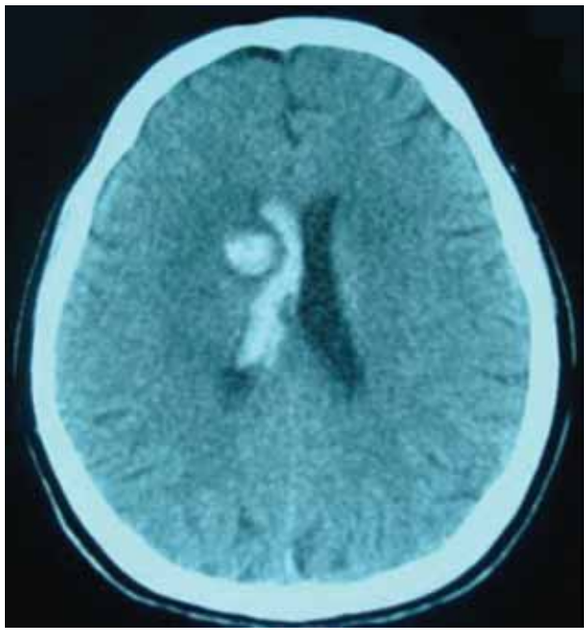

Fig 1: Computed tomography
Right capsulo lenticular hematoma with discrete
Compression of lateral ventricle

Computed tomography scans showed typical lesions of PRES in 23 cases. This exam demonstrated hematoma in 13 patients (fig1) and hypodense lesion in one case of arterial ischemia. It demonstrated a direct sign (delta sign) in cerebral venous thrombosis.

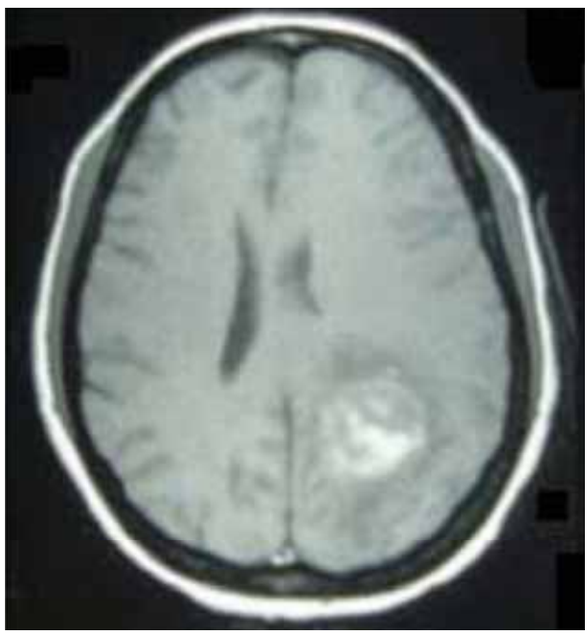

Fig 2: Cerebral MRI (FLAIR sequence)
Left parietal hematoma with edema

MRI confirmed typical lesions of PRES with hyper signal on T2 FLAIR and hypo signal on T1 Flair (fig2) in 10 cases and it was normal in one case. It revealed hemorrhagic lesions (Fig3) in 2 cases, signs of thrombosis in the 2 cases of venous thrombosis and ischemic lesions in 2 cases of cerebral ischemia. Angiography realized in 5 cases of cerebral hemorrhage in the goal to find etiology revealed arteriovenous malformation in one case (Fig4).

occurred mainly in post partum period (86%). Repartition of these complications were PRES in 61% cases followed by cerebral hemorrhage in 29,5% and finally cerebral venous thrombosis in 4,5% and arterial ischemia also in 4,5% cases. Predominant symptoms of Clinical features were troubles of conscience, headache, convulsions and neurologic deficits as visual changes, aphasia or coma. Eight of deaths (18,8%) occurred in our study and total Clinical recuperation was observed in 30 cases.

Conclusion: Stroke is considered an important cause of maternal morbidity and mortality. We found that pre-eclampsia/eclampsia and intracranial malformations were the major causes of stroke in pregnancy. Early diagnosis and adequate treatment cannot be overemphasized. .

Key Words:

Stroke, pregnancy, Cerebro-vascular complications

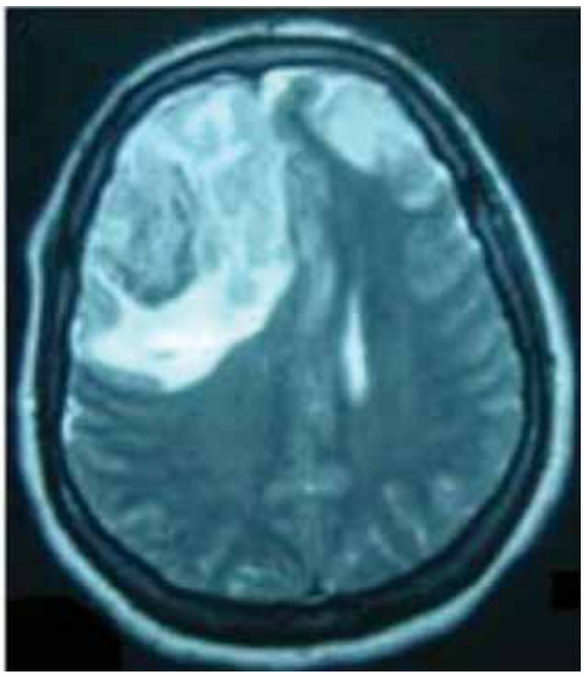

Fig 3: Cerebral MRI
Bi frontal hemorrhagic lesions with heterogenic
Signal in T1 and T2 sequences

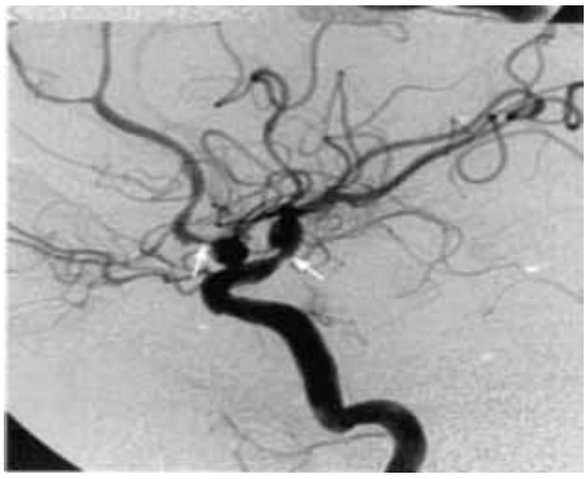

Fig 4: Selective Arteriography
With opacification of left carotid
Aneurysm of anterior artery and spasm of Left intern carotid (distal segment)

Computed tomography and MRI at admission permitted early diagnosis and adequate treatment in all cases. Management of patients was the same as that of nonpregnant patients, with more consideration on maternal and fetal risks.

Outcome: Eight deaths occurred in our study (18,8%), 6 patients with hemorrhage and 2 patients with PRES. Total Clinical recuperation was observed in 68 % of cases (n= 30).

DISCUSSION

Pregnancy and the puerperium have been recognized to increase the risk of stroke, particularly from late pregnancy and through the puerperium. It is quiet hard to find aetiologies concerning strokes during pregnancy or in the post partum period because it remains indeed unknown in 40% of cases (4). The results of our study complement the results of previous studies (4, 5, 6). We found that pre-eclampsia/ eclampsia and intracranial malformations were the major causes of stroke in pregnancy.

The relationship between pre-eclampsia and eclampsia and ischemic stroke and haemorrhagic stroke in pregnancy is unclear. The aetiology of preeclampsia and eclampsia remains mysterious despite intensive research on the subject.

Résumé

Problématique : La grossesse et le post-partum sont considérés comme des situations à risque élevé de développer un accident vasculaire cérébral dont les étiologies sont très variées.

But de l'étude: Analyser les spectres cliniques des AVC durant la grossesse et la période du post partum, la période de prédilection de survenue de cette complication, ses étiologies ainsi que ses aspects évolutifs.

Patientes et Méthodes: Nous rapportons une analyse rétrospective (1996- 2011) de dossiers obstétricaux de 54 patientes consécutives chez qui un diagnostic d'AVC est retenu pendant la grossesse ou pendant les 8 premières semaines du postpartum. Les patientes sont été sélectionnées sur la base d'un diagnostic qui associe au même temps une preeclampsia ou eclampsia à un AVC : ischémie artérielle, thrombose veineuse cérébrale, hémorragie sous arachnoidienne ou intra cérébrale, rupture de malformation artério veineuse ou d'un anevrisme cérébral ou bien un syndrome d'encephalopathie Reversible posterieure (RPES) secondaire à une éclampsie. Toutes les patientes ont été investiguées par un scanner cérébral et une IRM dans 30% des cas avec ou sans angiographie.
Résultats: Nous avons identifiés 44 patientes répondant aux critères d'inclusion ayant un âge moyen de 29,4 ans avec des extrêmes allant de 21 à 42 ans.
Une Hypertension artérielle gravidique a été retrouvée dans 32% des cas et toutes les femmes avaient une éclampsie ou bien une pre-eclampsie. La grossesse était bien suivie dans uniquement 12 cas. L'accouchement était à terme dans 43% des cas et prématuré dans 57% des cas par une césarienne chez 82% des femmes. Les complications cérébro-vasculaires sont survenues essentiellement en post partum (86%). Ces patientes ont été explorées par une TDM cérébrale

Pre-eclampsia is the consequence of placental abnormalities and defective invasion of trophoblastic cells into maternal spiral arterioles through the production of substances that are toxic to endothelial cells, causing the maternal syndrome. Multiple authors have found evidence for altered production of endothelially active mediators, such as endothelin, thromboxane A2, tumor necrosis factor, thrombomodulin (an endothelial cell surface glycoprotein) and active oxygen radicals (5,6)

The presence of abnormal endothelial injury in the placental bed and uterine vessels has been documented in pre-eclamptic patients by Sibai and Shanklin (5,6). The hypercoagulable state of pregnancy, rising osmolarity and underlying damaged endothelium may explain the rise of the risk of stroke for some patients at risk for stroke for other reasons like pre existing cardiopathies, deficit in haemostatics factors and vascularitis. This situation can explain the susceptibility to develop cerebral venous thrombosis.

There is two type of ischemic stroke: thrombotic origin in relation with hypercoagulabitity status and embolic origin in relation with arterial dissection, valvulopathies and rarely with puerperal cardiomyopathies.

Other specific complications in relation with pregnancy can be a cause on ischemic stroke, such choriocarcinoma and amniotic embolism.

The elevation of blood pression causes a dysfunction of cerebral auto regulation and cerebral hyperhemia. This situation leads to a vasogenic oedema and a risk of haemorrhagic Stroke in some cases mainly if pre eclampsia is accompagned with haematologic dysfunction and the rupture of hemato-encephalic barrier. In some situation, we can observe a cerebral “hyper- regulation” accompanied with vasospasm which lead to cytotoxic oedema and tissular ischemia (7,8,9).

Sub arachnoids haemorrhage (SAH) blood seeps into the brain’s surface and merges with ambient cerebrospinal fluid. It represents 1 to 7% of causes of stroke around the world. During pregnancy and post partum, this risk is five time higher than general population at the same age. The essentials causes of SAH are aneurysm rupture and arterio-venous malformations. Gravid hemodynamic changes contribute probably in the instability of aneurysm and the risk of rupture. The risk of rupture probably rises with term but not proved. The risk of rupture during labour is exceptional.

The moment of stroke occurrence during the pregnancy is also a subject of discussion. We found in the present study that stroke is frequent during the post partum period. In recent studies, it was reported that risk is sharply more significant in post partum. This suggests a causal role for the large decrease in blood volume or the rapid changes in hormonal status that follow a live birth or stillbirth, perhaps by means of hemodynamic, coagulative, or vessel-wall changes. A causal role for preeclampsia and eclampsia does not fully explain the much stronger associations with stroke found for the postpartum state than for pregnancy itself (10,11,12,13).

In our sudy, we found the fact that general anesthesia fo cesarian delivery was associated with increased risk of stroke when compared with neuraxial anesthesia in pre eclamptic woman. Mechanisms underlying the impact of general anesthesia and neuraxial anesthesia on the risk of stroke among preeclamptic women who undergo cesarian delivery remain unclear. Huang and Ramanathan (14,15), think that It may be related to different effects on neuroendocrine stress responses to delivery, as the plasma concentrations of adrenocortropic hormone, beta endorphine, epinephrine,norepinephrine, dopamine, and cortisol in preeclamptic women who received general anesthesia were significantly higher than those who received epidural anesthesia.

Management of patients with pregnancy-related stroke is largely the same as that of nonpregnant patients, with more consideration on maternal and fetal risks. The relationship between preeclampsia and stroke involves shared risk factors for both disorders, including chronic endothelial dysfunction and increased risk for long-term hypertension following preeclampsia (one of the major risk factors for stroke). This overlap provides insights into potential preventive strategies for both preeclampsia and stroke. For example, aspirin may prevent both disorders (16).

Anticoagulation during pregnancy is indicated for current arterial or venous thromboembolism,

dans 94,4% des cas, une IRM cérébrale dans 30,6%, une angioIRM veineuse dans 27%. Les complications cérébrovasculaires ont été par ordre décroissant de fréquence: le syndrome d’encéphalopathie postérieure réversible dans 61,4% suivi par l’hémorragie cérébro-méningée dans 29,5% et enfin la thrombose veineuse cérébrale et l’ischémie artérielle dans 4,5% chacune. Les symptômes prédominants étaient les troubles de la conscience, une céphalée, les convulsions et les déficits neurologiques. Le taux de mortalité était de 18,8% et une récupération totale était observée chez 30 patientes.
Conclusion : Les AVC constituent une cause importante de morbi-mortalité maternelle. Selon la présente étude la pre-eclampsie- éclampsie et les malformations vasculaires intracrâniennes constituent les causes majeures. Le pronostic dépend étroitement d’une prise en charge précoce et d’un traitement adéquat.

Mots clés:

AVC, Grossesse, complications cérébro-vasculaires

prior venous thromboembolism on long-term anticoagulation, antiphospholipid syndrome with prior venous thromboembolism and patients with a mechanical heart valve (16).Data from thrombolytic therapy for pregnant women with acute ischemic stroke are limited. It is critical that the risks and benefits of thrombolytic therapy for pregnant women and fetuses are considered cautiously (17,18). Our study shows a mortality rates of 18, 8%, this is can be explained by the facts that our population does not have the same access to medical attention as western populations during pregnancy and after the stroke. Early diagnosis and adequate treatment cannot be overemphasized. The same meticulous care provided during the antepartum and intrapartum periods should be continued into the puerperium.

CONCLUSION

Stroke during pregnancy and puerperium represents a rare occurrence but it could be a serious and stressful event for mothers and also families. Risk is sharply more significant in post partum period. The results of our study complement the results of previous studies that pre-eclampsia-eclampsia and intracranial malformations were the major causes of stroke in pregnancy. The relationship between pre-eclampsia and eclampsia and ischemic stroke and haemorrhagic stroke in pregnancy is unclear despite intensive research on the subject. MRI and CT scans are safe to use for diagnosis of stroke in pregnancy, although use of iodine-based contrast may affect thyroid function of the neonate. Finally, our study shows a high mortality rate wich can be explained by the facts that our population does not have the same access to medical attention as western populations during pregnancy and after the stroke.

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Conflict of interest statement :
There is no conflict of interest to declare

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Test your knowledge

1- Which one of the following is the LEAST consistent with a diagnosis of intussusceptions:
A- Intermittent colicky abdominal pain, interspersed with symptom-free periods.
B- Grossly normal appearing stool.
C- Normal plain films of the abdomen.
D- Previously healthy 9-year-old child.
E- Altered, lethargic appearance.

2- A 26 year old female presented after three successive witness grand mal seizures without recovery of consciousness. Upon arrival she is obtunded with no spontaneous eye opening and withdraws to pain only. Initial ED management could include all of the following EXCEPT:
A- Endotracheal intubation.
B- Barbiturate coma.
C- Phenytoin infusion.
D- Determination of blood glucose.
E- Gastric lavage.

3- Which of the following MOST likely represents vertigo of peripheral origin:
A- Ataxia.
B- Diplopia.
C- Dysphagia.
D- Facial numbness.
E- Unilateral hearing loss.

4- One of the following is INCORRECT about treatment of head trauma:
A- If GCS is >13, pupils are equal, there are no lateralizing deficits, there are no open head injuries, and the patient is neurologically intact with no loss of consciousness, or loss of consciousness less than 5 minutes, and has no major underlying medical problems. The patient may be eligible for discharge to home with instructions.
B- If GCS is > 9, the pupils are equal, no lateralizing deficits are evident, and no open head injuries were sustained, but neurologically is not intact, the condition is probably a contusion or a small mass lesion, such as subdural or epidural hematomas.
C- If GCS is >9 but the patient has either unequal pupils or any lateralizing deficit, a possible mass effect is evident, such as a large subdural or epidural hematomas or an intracerebral bleed. Each of these conditions requires admission after a neurosurgical consultation and head CT scan is obtained.
D- If GCS is <8, with or without unequal pupils, with lateralizing deficits, or with open head injuries, the most likely cause is a large intracerebral mass or a diffuse axonal injury. Obtain a STAT CT scan of head and a STAT neurosurgical consultation. Emergency intubation may be indicated, usually with rapid sequence induction (secondary concern for elevated ICP) for airway control with or without hyperventilation. The intubation should be done with in-line-immobilization, cricothyroidotomy prn.
E- Consider hyperosmolar therapy, such as mannitol 1 mg/kg IV, or hyperventilation by maintaining a pCO2 of 35-45 mm Hg (the normal). And if seizures occur, treat with lorazepam, 4 to 8 mg IVP, repeated until seizures are controlled, followed by phenytoin, 17 mg/kg IV loading at a rate of 50 mg/min.

5- One of the followings is INCORRECT about burn treatment:
A- Parkland formula: 4ml X body weight X %of second or third degree burn divided by 24 hours = hourly fluid requirement for first 24 hours.
B- Administer 50% of total 24 hour requirement over the first 8 hours.
C- IV fluids required for more than 20% or more TBSA burned.
D- Monitor vital signs and urine output 30-50ml/h in adults, 1ml/kg/h in children weighting <30kg.
E- Colloids or plasma 0.5ml/kg/% burn may be given as a part of parkland formula for healthy burned patient in the first 24 hours.

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Correct answers: 1- D, 2-B, 3-E, 4-E, 5-E

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Pneumocephalus following head trauma

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Abstract

Pneumocephalus following head trauma is relatively rare, with tension pneumocephalus occurring in an even smaller group of patients. This review presents a recent case of tension pneumocephalus following the use of a manually operated bag–valve–mask to assist ventilations prior to rapid-sequence intubation. A discussion of this case in terms of other reported cases of pneumocephalus after oxygen therapy follows. A limited number of current case reports identified in the literature indicate a connection between pneumocephalus and positive pressure ventilation following blunt trauma. Continuous positive airway pressure (CPAP) ventilation use in patients with an undiagnosed skull fracture is the most common reported cause of ventilation related pneumocephalus. The case review presented here identifies the use of a bag–valve–mask prior to intubation as a possible contributory cause of the tension pneumocephalus. With only one prior case reported in the literature of pneumocephalus following the use of a bag–valve–mask, this case is unique and may indicate the need for additional awareness for this rare complication. The prehospital diagnosis of pneumocephalus is difficult, as the symptoms and mechanism of injury mimic those associated with intracranial hemorrhage. The use of mannitol in the prehospital treatment of this patient and subsequent improvement in pupillary response may indicate that mannitol has a role in the treatment of tension pneumocephalus when neurosurgical services are not readily available. Additional research is needed to better understand the benefits and risks associated with this treatment modality.

Key words

words emergency medical services; pneumocephalus; accidental falls; mannitol

CASE REPORT

The patient was a 37-year-old man who fell approximately 30 feet from a tree stand while hunting. The fall occurred in a secluded section of the woods and resulted in a delay of between 40 minutes and one hour from the time of the incident to the arrival of helicopter emergency medical services (HEMS). Local fire and rescue services responded after a fellow hunter found the patient an unknown period of time following the fall. According to the second hunter, he had last seen the patient in the tree stand 15 to 30 minutes prior to discovering the patient lying face down in the soft soil beneath the same tree. The hunter noted that the patient was unconscious when he arrived but aroused with a light touch. Fire personnel were familiar with this section of woods and arrived at the patient 20 minutes after dispatch. A recently plowed field adjacent to the wooded lot was utilized as a landing zone and the HEMS flight crew waited approximately 10 minutes after landing for the patient to be carried out of the wooded lot. Fire personnel had performed an initial assessment and secured the patient to a long backboard, but no additional evaluation or treatment had been performed. The flight crew's initial assessment noted the patient to be awake and oriented but unable to recall the events surrounding his fall. Significant facial trauma was present, including periorbital swelling that made assessment of the pupils impossible, along with significant deformity to the nose and frontal bone. Initial vital signs were blood pressure (BP) 138/61 mmHg, heart rate (HR) 44 beats/min, respiratory rate (RR) 24 breaths/min, and oxygen saturation (SpO2) 100%. The patient was placed on oxygen (O2) via a non-rebreather mask (NRB) at 15 L/min. prior to the placement of the NRB, no additional airway therapy or respiratory assistance had been performed. The patient made multiple requests to blow his nose in an attempt to clear the blood draining into his orophyranx from the facial trauma. He was advised against such attempts. Despite verbal coaching to keep the NRB in place and minimize cervical spine movement, the patient became increasingly combative throughout the initial assessment. Suctioning of the oropharynx provided only minimal increase in the patient's ability to protect his airway.

The flight crew decided to intubate the patient utilizing rapid-sequence intubation (RSI) to optimize gas exchange and protect the airway. The patient was premedicated with lidocaine and induced with etomidate and succinylcholine. Prior to induction, the patient had failed to achieve six to eight vital capacity breaths with the NRB in place, despite coaching. The flight crew provided approximately eight vital capacity breaths with a bag–valve–mask (BVM) during induction. An 8.0-mm endotracheal tube (ETT) was placed with an initial end-tidal carbon dioxide (ETCO2) value of 29 mmHg. The SpO2 remained above 99% throughout the procedure. Sedation and paralysis were maintained. The remainder of the secondary survey revealed significant facial and head trauma but no other apparent injuries.

The patient was loaded into the aircraft and placed on the transport ventilator. He tolerated the transfer well, maintaining SpO2 levels above 98% and ETCO2 values between 30 and 35 mmHg with proper waveform. A flight level of 1,000 feet above ground level (AGL)

was provided throughout the transport. Because of massive periorbital swelling, it took both providers to assess the pupils, which were noted to be nonreactive to light and fixed at approximately 8 mm bilaterally. This initial assessment of the pupils occurred at cruise altitude. The flight crew administered a 100-g bolus of mannitol intravenously (IV) per protocol because of concerns of possible herniation. Over the course of the flight, the patient demonstrated persistent bradycardia, with HR values in the 40s and 50s (beats/min) and a continued BP at approximately 130/90 mmHg. The remainder of the flight was uneventful.

Upon arrival at the trauma center, pupillary reassessment revealed an improvement bilaterally with the left pupil at 4 mm and reactive to light and the right pupil at 5 mm and unreactive to light. An urgent head and maxillofacial computed tomography (CT) scan without contrast was performed. Complex facial and skull fractures were diagnosed to include the following: a basilar skull fracture extending across the right inner ear and involving the right petrous temporal bone; comminuted fracture of the greater wing of the sphenoid bone with fracture lines extending into the foramen lacerum and the right internal carotid canal; fracture through the left temporal bone and sphenoid bone, extending through the foramen lacerum and the left carotid canal; fractures of the medial, lateral, and anterior walls of the right maxillary sinus; multiple fractures of the right and left orbits; fractures of the left and right frontal bones; fractures of the anterior and medial walls of the left maxillary sinus; and fractures involving the bilateral lateral walls of the sphenoid sinus and clivus. A significant pneumocephalus extended bilaterally across the frontal lobes, causing mass effect on the left frontal lobe. The patient was also noted to have a small frontal epidural hemorrhage and subarachnoid hemorrhage around the tentorium. Cervical spine CT scan revealed subcutaneous air within the spinal canal and anterior neck. Figure 1 shows the large pneumocephalus. The subcutaneous air within the spinal canal is apparent in Figure 2.

Immediate neurosurgery consultation was obtained and the patient was admitted to the hospital for monitoring and further workup in the neuroscience intensive care unit (ICU). The subcutaneous air within the anterior neck and the fractures along the carotid canals were questionable causes of the persistent bradycardia, which spontaneously resolved within 48 hours after admission. The patient was given antiepileptics and antibiotics, with surgical repair of the facial fractures occurring five days after admission. The patient was discharged home seven days after the incident with no loss of cognitive function, but he continued to complain of bilateral anosmia and limited right-sided hearing loss. At one year after injury, he had complete resolution of these complaints and was no longer being followed by any of the hospital services.

DISCUSSION

Epidemiology

Trapping of air within the cranial vault, also known as pneumocephalus, is a rare but often benign neurologic injury that may result following craniofacial trauma. Gill and van As (1) reported an incidence rate for pneumocephalus of less than 1% in the head trauma population, with a rise to 8% when fractures of either the paranasal sinus or the skull base were present. The rare fracture of the sella turcica dramatically increases the risk of pneumocephalus to 41% (2). Fractures to this region place patients at an increased risk of injury to both the cranial nerves and the carotid arteries. In a review of 14 cases of sella turcica fractures, nine of the 14 patients (64%) suffered paralysis of at least one cranial nerve (3). The dura mater is both thin and tightly attached to the skull in these regions, making sinus or skull base fractures more likely to lacerate the dura and allow the admittance of intracerebral air (2). Isolated epidural pneumocephalus requires a lack of dural defect and is the least common form (1). Tension pneumocephalus may develop if the intracranial air creates a mass effect on the brain, resulting in a possibly life-threatening neurosurgical emergency.

Incidence rate for pneumocephalus < 1% in the head trauma population

Mechanisms

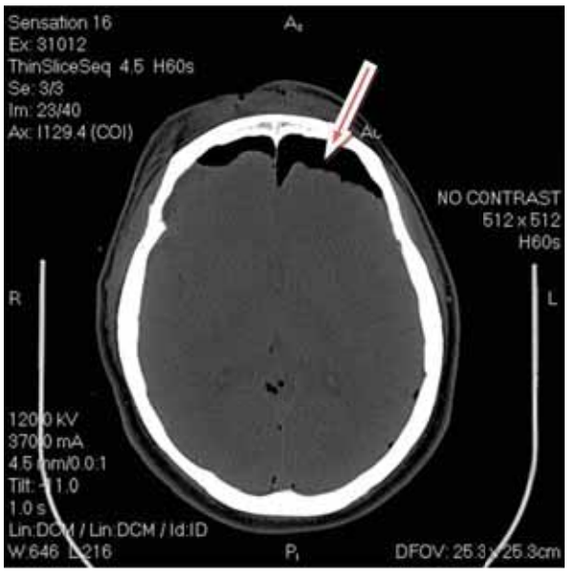
Two mechanisms have been postulated to account for the entrance of air into the cranial vault. The "ball-valve mechanism" has previously been described as a causative agent when a fistula allows ambient air at a pressure above intracranial pressure (ICP) to force itself into the intracranial space (4). This process continues until the ICP overcomes the ambient pressure and the brain and dura mater are forced over the fistula. This process may repeat itself multiple times until the higher-pressure ambient air no longer overcomes the ICP. A second mechanism postulated occurs when a continuous cerebrospinal fluid (CSF) leak from an enclosed space is present. The loss of CSF creates a void space and relative negative pressure, allowing air to bubble in and fill the void. This scenario has previously been termed the "Coke-bottle mechanism."(4) This fluid-gas exchange may continue until no additional loss of CSF is possible. This case demonstrated both mechanisms.

Physiopathology

Pneumocephalus may form either as a large isolated pocket or as a series of smaller diffuse bubbles (2). In this case, the patient showed bilateral frontal lobe pneumocephalus as well as scattered pneumocephalus throughout the brain. Keskil et al. found in a review of pneumocephalus cases that patients with multiple air bubbles had a higher mortality rate and signs of cerebral atrophy following recovery. They postulated that in order for diffuse air bubbles to form, higher accelerations were required to separate air into bubbles throughout the CSF (2). However, this mechanism would require either multiple impacts or pneumocephalus to already be present when the primary injury occurred. The dispersion of air into separate pockets is more likely a result of the mechanism of air admittance or multiple air-admitting avenues. Smaller pneumocephalus bubbles may be more likely to form when the CSF–gas exchange mechanism of air introduction is present. A second possible cause may be the presence of a more diffuse series of skull fractures that allows air to be admitted via a number of separate locations. In a review of 59 cases of otogenic pneumocephalus, Andrews and Canalis (4) found 25 cases resulting from trauma, with a majority of the cases having either subdural or ventricular pneumocephalus and only two reported cases of epidural pneumocephalus. Cerebrospinal fluid otorrhea was present in 14 of the 25 cases, and all 25 patients had fractures of the temporal bone. Keskil et al. (2) identified 21 cases of pneumocephalus during a review of 583 head-injured patients. The authors found associated rhinorrhea

or otorrhea in 47% and basal skull fractures in 57% of the cases as well as a strong association with depressed skull fractures. Intracranial hemorrhage was also common, present in 66% of the cases. However, Keskil et al. found no patients with tension pneumocephalus.

Positive pressure ventilation has been described as a causative agent for pneumocephalus in several case studies, with the majority occurring after an extended period of use such as with continuous positive airway pressure (CPAP) via face mask (5), the administration of oxygen via nasal cannula in pediatrics (6,7), or the use of an oxygen-powered resuscitator face mask (8).



Pneumocephalus with mass effect

Klopfenstein et al. (5) noted a case of pneumocephalus following the use of face mask CPAP in which the patient presented with an unrecognized basal skull fracture and was initially resuscitated with the placement of an ETT and the use of mechanical ventilation. Following stabilization and extubation, the patient was placed on CPAP of 10 cmH2O every 10 minutes for one to two hours. However, diagnosis of pneumocephalus did not occur until seven days following the application of CPAP when diplopia, rhinorrhea, and headache developed. The extended period of time between onset of symptoms and the initiation of CPAP in this case contrasts sharply with the rapid onset of symptoms in the case presented here. A longitudinal fracture extending through the right petrous bone was eventually diagnosed, and 10 days following surgical repair of the associated dural defect the patient was discharged with noted persistent diplopia following paralysis of the right cranial nerve VI.

A single case of pneumocephalus following mouth-to-mouth resuscitation has previously been described (9). According to the case report, a family member provided mouth-to-mouth ventilation for only a few minutes. Spontaneous respirations resumed prior to emergency medical services (EMS) arrival and the patient was maintained on supplemental oxygen via face mask. As in the case presented here, ventilation was limited in duration; however, the patient described by Robbins and Train did not develop signs of tension pneumocephalus (9). A single fracture of the posterior wall of the frontal sinus was eventually diagnosed.

Only one case of tension pneumocephalus following the use of the BVM to provide assisted ventilation during the resuscitation of a patient with head trauma has previously been described in the literature. Dacosta et al. (10) reported a case in which EMS providers utilized the BVM to assist an apneic elderly woman following blunt head trauma secondary to a fall. Manually assisted ventilation was performed for approximately 30 minutes prior to arrival at the trauma facility where intubation was performed, and further use of the BVM was discontinued. This extended time line differs greatly from the eight to 10 breaths given to the patient in our study prior to RSI. Dacosta et al. noted the development of tension pneumocephalus early, approximately 90 minutes following the initial injury, and found that their patient's pupils remained equal and reactive to light (10). During the first three hours after admission, signs of intracranial hypertension were noted with hemodynamic instability. A fracture of the left petrous bone was found with CT, but no fractures of the anterior cranium were noted. While this case demonstrates a faster progression of symptoms than seen with many cases, it is still a far slower time line than that with the patient presented here, who received a much more limited exposure to BVM ventilation.

Case management

Airway control through the placement of an ETT provides a rapid intervention to eliminate the ability of additional air to enter the cranial vault when a one-way valve in the nasopharyngeal structures is the causative agent (11). Mammis et al. (11) described a case in which twist-drill-hole aspiration was employed for the relief of tension pneumocephalus following craniotomy and maxillofacial surgery. In this case, aspiration with a twist-drill hole placed in the subdural space removed some of the initial pneumocephalus; however, additional air continued to enter the intracranial vault because of the presence of a nasopharyngeal defect created during reconstruction, which acted as a one-way valve. A lack of neurologic improvement following aspiration of the intracranial air forced endotracheal intubation in an attempt to bypass the defect. The placement of an ETT provided a rapid means of eliminating further accumulation of air through the nasopharyngeal defect. Mammis et al. (11) noted that while twist-drill-hole aspiration offered a means of escape for the intracranial air, it failed to eliminate the underlying mechanism and persistent accumulation of intracranial air.

The use of mannitol during the resuscitation of the head-injured patients with suspected herniation has been supported in the literature as a bridging intervention to neurosurgical treatment (12). Prehospital use of mannitol in the tension pneumocephalus population has not been identified in previous literature. The mechanism of action of mannitol does not lend itself well to having a direct influence on the resolution of pneumocephalus; however, in this current case, a marked improvement in pupillary reaction to light was noted following mannitol administration. The inability to place an ICP monitor emergently limits the evidence to support a change in ICP following mannitol administration in the initial phases of resuscitation when tension pneumocephalus is present.

There is no consensus in the literature about the use of mannitol during the treatment of either pneumocephalus or tension pneumocephalus. G¨on¨ ul et al. (13) reported a case of continued nasal drainage of CSF following blunt head trauma with no pathologic findings. Treatment for the rhinorrhea included lumbar spinal drainage with ensuing tension pneumocephalus after three days of drainage. Prompt removal of the CSF drainage device and initiation of mannitol at 1 g/kg was performed. The authors noted the resolution of nasal CSF drainage and a gradual decrease in pneumocephalus on serial CT scans. Patient discharge was accomplished with no neurologic deficits.

Pneumocephalus has been reported as a complication of lumbar spinal drainage (14). Pneumocephalus may develop as a result of an improperly sealed lumbar site, which allows a CSF–air exchange to occur. Alternatively, pneumocephalus development may follow the continued decline in intracranial pressure as a result of unchecked CSF drainage. While G¨on¨ ul et al. (13) found no skull fractures on CT scan, continued rhinorrhea suggests that an intracranial–nasopharyngeal fistula was present. This fistula may have provided the avenue for communication between the cranial vault and atmosphere and the subsequent development of pneumocephalus as the intracranial–atmospheric pressure gradient was created.

Following a review of 59 cases of otogenic pneumocephalus, Andrews and Canalis (4) postulated that procedures normally performed to decrease ICP, such as the use of mannitol, elevation of the patient's head, or intrathecal catheter drainage, may allow additional air to enter the cranial vault as the ICP decreases. The authors postulated that procedures contributing to a decrease in brain parenchymal size may create a potential space between the brain and dura. Additionally, as the ventricles become drained of CSF, their ability to compensate against ICP changes decreases (4). The case of pneumocephalus following lumbar spinal drainage supports this proposal that procedures that decrease ICP may allow pneumocephalus to develop (13). However, the cessation of CSF rhinorrhea following the administration of mannitol and the termination of the lumbar spinal drain raise the question as to what role mannitol should play in the management of pneumocephalus.

If the cause of the pneumocephalus was the lumbar spinal drain, then the improvement of symptoms and the reabsorption of air following cessation of lumbar drainage are similar to pneumocephalus resolution following endotracheal intubation in the presence of a one-way valve. A consistent step toward resolution of all pneumocephalus appears to be the identification of the mechanism and point of entry into the intracranial space in order to halt additional air accumulation. The addition of mannitol to the treatment of this case of pneumocephalus suggests that there may be a role for mannitol in the management of specific types of pneumocephalus, though additional studies are needed to identify mannitol's role.

Rapid airway control through ETT placement may prove to be the most effective prevention

Tension pneumocephalus in the critical care transport environment and emergency department setting requires the same basic standards of care provided during all transports and emergency department visits, including rapid triage and assessment, managing airway issues, providing fluid resuscitation as required, and ultimately rapid transport or consultation with neurosurgical services. In the rotor-wing air transport community, flight altitudes that remain as low as practical, generally below 2,000 feet, may help to mitigate any increase in tension pneumocephalus volume.

The Transfer

The use of aeromedical transport for patients with known or suspected pneumocephalus has received additional attention in recent years, as the wars in Iraq and Afghanistan have necessitated the use of such services. The primary transport concern of intracranial air expanding with the decrease in ambient pressure encountered at cruise altitude has been investigated utilizing both computer modeling and small series case reviews. Andersson et al. (15) evaluated ICP changes during ascent and cruise flight inside a pressurized cabin with the use of a computer-based model. The authors evaluated a variety of case parameters, including both normal and elevated ICP values, a variety of initial intracranial air volumes, and three different ascent rates across a full range of altitudes. The results demonstrate a dramatic increase in the volume of intracranial air to as much as a 30% increase above the starting volume as the cabin pressure reaches 8,000 feet. Changes in ICP were dependent on the initial volume of air. The rate of ICP increase was most severe between sea level and 2000–4000 feet. This is of particular interest to many rotor-wing transport services, which routinely utilize this altitude for cruise flight. The authors note that any ICP changes are likely transient, with the patient returning to his or her baseline ICP once the cabin pressure stabilizes. This finding complements the results of infusion studies that found that once the infusion was stopped, ICP rapidly returned to baseline, an event similar to reaching the constant ambient pressure of cruise flight.

However, the more significant finding from this computer modeling was the effect ascent rates had on gas expansion. With ascent rates varying from 250 to 1,000 feet per minute, ICP increased dramatically as the ascent rate was increased. The model predicts that the rate of ascent will produce a more significant increase in ICP compared with the absolute altitude achieved. Previous research evaluating intraocular gas changes with altitude has noted similar trends and provides support for this computer simulation (16). While altitude considerations will play a more important role in the flight-planning process of fixed-wing transport providers, rotor-wing services should note the proposed effects of ascent rates.

Donovan et al. (17) reviewed 21 cases of pneumocephalus aeromedically evacuated from a combat support hospital. The majority of the patients did not have intracranial monitoring devices, making direct comparison with Andersson et al.'s computer modeling difficult. The authors found no neurologic deterioration in flight or within 24 hours following arrival at the receiving facility. While long-term follow-up or comparison of pre- and post transport CT scans was not done, the lack of neurologic changes in flight supports the belief that changes in ICP with altitude will be transient and will stabilize as cabin pressure reaches a constant level for cruise flight. This case series review suggests that the risk of developing tension pneumocephalus during flight is minimal.

Helling and McKinlay (18) report a unique utilization of an IV catheter placed in the frontal sinus of a patient who presented with multiple frontal sinus fractures following a shrapnel injury. The authors utilized a mechanical device to decrease frontal sinus pressure in an attempt to minimize the risk of a hypertensive frontal sinus contributing to pneumocephalus during flight. The patient was noted to have no neurologic deterioration during or following transport as well as a lack of complications resulting from the catheter. As with the proposed use of mannitol, the placement of an intrasinus catheter for pressure management in flight is a new technique and will require reports of additional cases to demonstrate and support its benefit.

CONCLUSION

A nonradiographic diagnosis of tension pneumocephalus

following blunt trauma is difficult as the symptoms may mimic intracranial vascular injuries with mass effect. Many of the common complaints of headache, altered vision, photophobia, or mental status changes are present in the traumatic brain injured population regardless of the presence of pneumocephalus. Symptoms of skull fractures such as rhinorrhea or otorrhea have been strongly associated with pneumocephalus in case reviews (2,4). Prudent use of positive pressure ventilation in these patients and rapid airway control through ETT placement may prove to be the most effective steps to minimize additional introduction of air into the cranial vault. Prior to establishing a definitive airway, steps should be taken to minimize increases in sinus pressure from coughing, nose blowing, vomiting, or any Valsalva maneuver. With case studies finding a link between tension pneumo-orbitus and tension pneumocephalus (19), transport should be to a trauma center that provides both neurosurgical and ophthalmologic services, if possible. With multiple mechanisms and paths of entry, air inside the cranial vault should be a consideration with all head trauma, especially following the use of positive pressure ventilation.

Flight ascent less than 1000 feet.

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The editorial board

Common knowledge:
Some cranial traumas are accompanied by pneumocephalus especially when there are sinus associated fractures. Screening is essentially by radiology. The Emergency related risk is putting them under tension.

What this article tells us :
Two mechanisms are mainly the reason, a valve effect when, in the presence of a dural puncture the intra cranial pressure is lower than the ambient pressure ; the establishment of a relative negative pressure by the Cerebrospinal Fluid leak (CSF). Tracheal intubation plays a protective role in the first case. Injection of Mannitol allows to temporarily reduce the intra-cranial pressure. Aerial Evacuation of those injured must be conducted below 2000 feet.

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Lucie Nader

CARDIAC MRI

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Abstract

Cardiac MRI is a noninvasive imaging technique that has a wide range of clinical applications. Many of these applications are commonly employed in clinical practice as the evaluation of congenital heart disease, of pericardial disease, of cardiac masses, of the right ventricle in right ventricular dysplasia, the analysis of the myocardium (perfusion problem, hibernating myocardium ...), the evaluation of valves and ventricular functions.
While there are modalities for every clinical application of cardiac MRI, there is no diagnostic technique that can provide as comprehensive evaluation as MRI. For this reason, cardiac MRI is known as the «one-stop shop.»

Key Words

Cardiac MRI, Indications and Protocols, Morphology and Function, Myocardial disease, Valve disease, Pericardial disease.

INTRODUCTION

Cardiac MRI is a noninvasive imaging technique that offers useful data related to cardiovascular function and morphology that help in the diagnosis of cardiovascular disease. It doesn't expose patients to ionizing radiations and it is less risky than other invasive diagnostic procedures (1).

Cardiac MRI is indicated for a variety of specific purposes, such as the detection and management of congenital heart disease, thoracic aortic disease and pulmonary vein assessment. It can provide answers to a multitude of questions about cardiac morphology, cardiac function, ischemic diseases and their complications, myocardial viability, infectious disease, myocarditis, cardiac masses, valve and pericardial disease (2).

TECHNICAL ASPECT AND UTILITY

There are several contraindications to MRI. In particular, patients who have implanted medical devices (e.g. pacemakers or defibrillators, cochlear implants, cerebral aneurysm clips), or who may have iron fragments in their eyes, are not suitable for MRI investigation.

In general, cardiac MRI exams are done on 1.5T systems. Sophisticated coil design (up to 32 coil elements for cardiac imaging) that provides increased capability in parallel acquisition techniques, and a good signal-to-noise ratio spatial resolution and image acquisition are used (3).

Electrocardiographic gating is used; it can be performed prospectively or retrospectively. Prospective gating is most common; however the advantage of retrospective gating is the possibility of imaging the entire cardiac cycle (4).

The main cardiac imaging planes are the short axis, the horizontal long axis, and the vertical long axis. The horizontal long-axis view is also known as the 4-chamber view, and the vertical long-axis view is also known as the 2-chamber view. Other imaging planes that may be useful include a left ventricular outflow tract view for ascending aortic pathology, and a 3-chamber view that shows to a better advantage the aortic and mitral valves (4).

INDICATIONS FOR CARDIAC MRI

Ischemic heart disease

There are multiple approaches to detecting coronary artery disease by cardiac MRI. Blood flow can be assessed at rest and under stress to demonstrate significant coronary blockages.

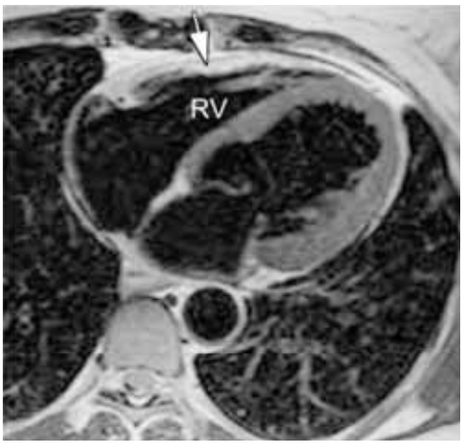
The T2-weighted MRI sequence enables to see edema in the myocardium, a primary sign of acute injury of the heart; it differentiates between patients with acute vs. old myocardial infarction (5). It has the ability to detect myocardial edema and necrosis before the rise in cardiac enzymes when used as an emergency protocol.

Contrast enhanced sequences are useful to study the myocardial infarction: the first-pass perfusion and the delayed enhancement MRI to look for areas of myocardial necrosis.

The agents used in stress test are dobutamine or adenosine/dipyridamole. At low doses, dobutamine can be used to study myocardial viability while adenosine/dipyridamole enhances accuracy in studying myocardial perfusion. At high doses, dobutamine increases myocardial oxygen consumption and demonstrates myocardial dyskenesis. Because there is a risk of severe ischemia and heart rhythm disorder from high doses of dobutamine, these examinations must be monitored in specialized centers.

Cardiomyopathy

Cardiac MRI can be used to demonstrate the abnormalities in heart muscle and function, particularly those associated with cardiomyopathies. In particular, cardiac MRI is useful in the diagnosis and follow-up of arrhythmogenic right ventricular dysplasia (6).



Right ventricular dysplasia: Axial T1-weighted black blood spin-echo images show extensive transmural fatty replacement of the right ventricular myocardium (RV)

Myocarditis

They could be secondary to infectious disease (viral or bacterial), or they could be related to a systemic disease. The affected myocardial areas are hyperintense on T2 weighted sequences, starting in the sub-pericardial and extending throughout the myocardial wall as the disease progresses (1). Sometimes there is a visible muscle hypokinesia on cine sequences.

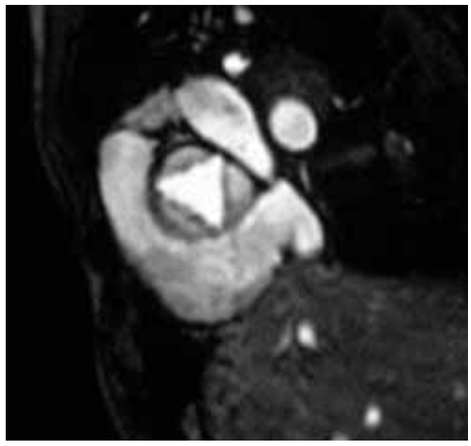
Cardiac masses

Even though the echocardiography remains the first exam for the assessment of cardiac masses, particularly thrombus and tumor, cardiac MRI can easily differentiate between these two diagnoses. It is also useful in the classification of cardiac tumors.

The primitive cardiac tumors are rare but secondary metastatic lesions are not uncommon. The main differential diagnoses that cardiac MRI can identify are thrombus, atrial myxoma, hemangioma, lipoma, lymphangioma, paraganglioma, rhabdomyoma, teratoma, sarcoma, lymphoma, mesothelioma...

Valvular heart diseases

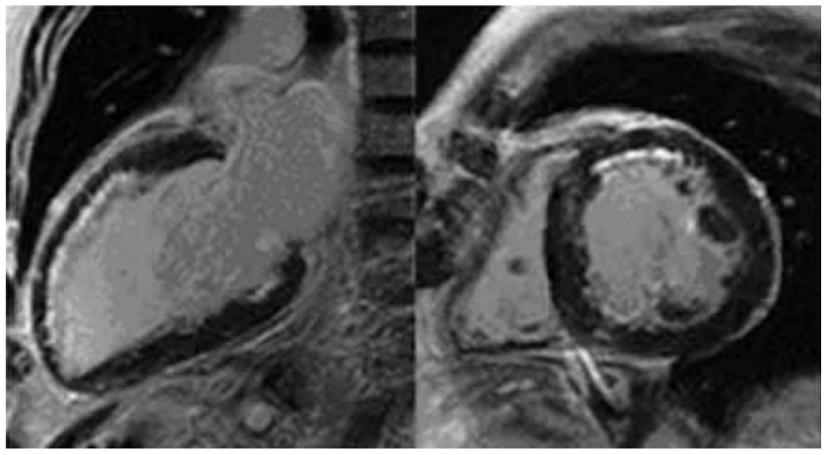
Cardiac MRI can now be used to clearly assess valvular heart diseases (7). Studies have shown that MRI can be an alternative or a complementary technique to echocardiography (7) for the analysis of valvular pathologies. It is reliable and reproducible providing morphological and functional information. It has the distinction of not being limited by the acoustic penetration as the ultrasound.



A three-chamber view showing the aortic valve opened

Cardiac function

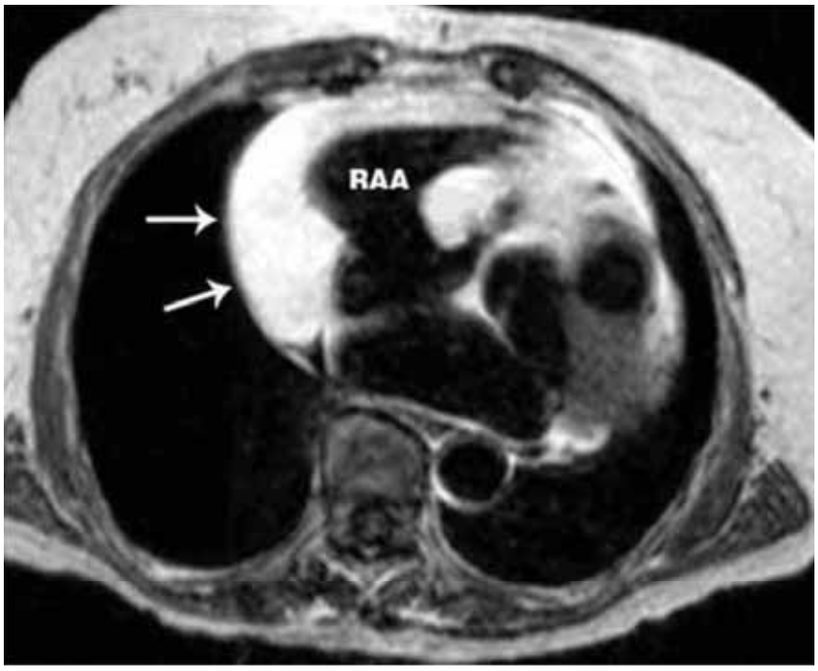
Ultrafast gradient echo sequences have made MRI the technique of choice for the dynamic study of cardiac motion and cardiac contractile function (4). The sequences currently used in the study of cardiac cinetics are of the steady state gradient echo type with balanced gradients. They have the advantage of being very fast, with a high signal-to-noise ratio and T2/T1 contrast clearly differentiating between blood (as a hypersignal), endocardium and epicardium (as an isosignal) and fat (as a hypersignal). These acquisitions are preferably made with retrospective gating to improve temporal resolution. A comparative analysis of the images in telesystole and telediastole, with an estimate of ventricular volume, gives the value of the ventricular ejection fraction. It is also possible to estimate left ventricle mass and the segmented cinetic parameters.



Myocardial infarction: vertical long-axis and short-axis views of left ventricle show extensive subendocardial late enhancement, confirming nontransmural infarction of segments 7, 10, 13, 14, and 16.

Pericardial disease

Cardiac MRI is the modality of choice for studying the pericardium. Diseases of the pericardium include pericarditis, hematoma, pericardial cysts and tumors, whether benign or malignant.



Pericardial lipoma: Axial ECG-gated non–breath-hold T1-weighted SE echo-planar shows a hyperintense pericardial lipoma close to the right atrial appendage (RAA)

Congenital heart diseases

CONCLUSION

When it comes to choosing a modality for diagnosing cardiovascular disease, the question doesn't always involve the best tool to make the diagnosis, it goes to what is the simplest and most accessible tool. While it represents an extremely powerful tool, cardiac MRI requires training and expertise; it is not a simple exam.

Cardiac MRI allows a study of morphological and functional heart disease by assessing myocardial contractility, studying myocardial enhancement after injection of gadolinium chelate in the case of acute or chronic ischemic diseases, identifying myocardial systemic and infectious diseases. It allows analysis of myocardial viability in a doubt about the usefulness of a possible reperfusion (surgery or stent). It may be the procedure of choice for studying the cardiac valves. But could it become, one day, the gold standard for evaluation of heart?

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LEV 124 JA NE (NE) 05-11

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1. NAME AND PRESENTATION: Tablets containing 250mg and 500mg levofloxacin and IV Solution for infusion containing 5mg/ml levofloxacin. **2. THERAPEUTIC INDICATIONS:** All presentations: community-acquired pneumonia (CAP), complicated urinary tract infections including pyelonephritis (CUTI/P), chronic bacterial prostatitis (CBP), skin and soft-tissue infections (SSTI). 250 & 500mg tablets: acute sinusitis (AS), acute exacerbations of chronic bronchitis (AECB). 250 mg tablets: uncomplicated urinary tract infections (UUTI). For possible National guidance on the use of fluoroquinolones see full SmPC. **3. POSOLOGY AND METHOD OF ADMINISTRATION:** CAP: 500mg once or twice daily (7 to 14 days), CUTI/P: 250mg once daily (7 to 10 d), CBP: 500mg once daily (28 d), SSTI: 250mg once daily to 500mg twice daily (7 to 14 d), AS: 500mg once daily (10 to 14 d), AECB: 250 - 500mg once daily (7 to 10 d). UUTI: 250 mg once daily (3 d). Tavanic solution for infusion is only for slow intravenous infusion: 30 minutes per 250mg. It is possible to switch to the oral route at the same dosage. Impaired liver function: no adjustment. Ederly: only consider renal function. Impaired renal function: see full Sumary of Product Characteristics (SmPC) for details. **4. CONTRA-INDICATIONS:** Hypersensitivity to levofloxacin, other quinolones or excipients. Epilepsy. History of tendon disorders related to fluoroquinolone treatment. Children and growing adolescents. Pregnancy and breast-feeding. **5. SPECIAL WARNINGS AND PRECAUTIONS FOR USE:** Nosocomial infections with Pseudomonas aeruginosa: consider combination therapy. Cease treatment immediately if tendinitis is suspected; this risk may be increased in the elderly or by co-administration of corticosteroids. If pseudomembranous colitis is suspected cease therapy. Use with extreme caution in patients pre-disposed to seizures or taking drugs which lower seizure threshold. If renal impairment adjust the dosage (see SmPC). Patients should avoid strong sunlight or artificial UV-rays. **6. DRUG INTERACTIONS:** Antacids containing iron salts, magnesium or aluminium, sucralfate, theophylline, fenbufen and similar NSAI, probenecid and cimetidine, cyclosporine, vitamin K antagonists: see full SmPC. **7. PREGNANCY AND LACTATION:** See contra-indications. **8. EFFECTS ON ABILITY TO DRIVE:** Caution when driving (Possibility of dizziness, vertigo, visual disturbances) **9. UNDESIRABLE EFFECTS:** Common side effect: Gastro-intestinal: nausea, diarrhoea. Liver: increased liver enzymes. Others: pain, reddening of the infusion site and phlebitis. Rare and very rare: Musculo-skeletal: tendon disorders < 0.1%, tendinitis < 0.1%, tendon rupture < 0.01%. For uncommon, others rare and very rare side effects see full SmPC **10. OVERDOSAGE:** No known antidote. Symptomatic treatment with ECG monitoring **11. PHARMACODYNAMIC PROPERTIES:** ATC Code: J01MA12.

Abbreviated Prescribing Information based on the UK SmPC as of September 2007

Always refer to the full Summary of Product Characteristics (SmPC) before prescribing.

1. Hans H. Liu, Drug Safety 2010, vol. 33, No. 5: 353-69, ISNN: 114-5914
2. Peterson J. et al, Current Medical Research and Opinion, Vol. 25, No. 3, 2009, 559-568
3. Tavanic Full prescribing information

Full prescribing information available upon request at:
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SANOFI

Amal Tohmy

Dr. Amal Tohmy

Better Safe Than Die, Dry Bite or Not!!!

Guidelines, Protocols, and Myths are a Grace or a Curse?

This is a sad case report; hopefully it will not be repeated. I do wish that you will read the whole story before you sentence your judgment. The story and its details are made concise to avoid unnecessary repetition, but the main and critical points are listed in sequence without names, because our aim is constructive and not destructive. **" We do pray for God to save us from the temptation "**

Mr Samir M. a 28 year's old married male, a previously healthy cute women hair dresser. He performs necessary daily exercises to preserve his body in good attractive shape. He's no smoker and drinks alcohol occasionally.

On April 1: While he was searching for coal on the shelf of a small cabinet in his garden where a good number of his friends were gathering for dinner, he felt a prick in a finger of his right hand, it was so difficult to identify which finger at that time. The prick precipitated chilliness with goose skin sensation for seconds, after which he glanced a small cylindrical creature, around 20 cm in length, disappearing smoothly between the items on the shelf.

There are hundreds of species of snakes around the world but only a low percentage of these are venomous. However, given the shock, distress and panic that most of us would feel on unexpectedly encountering a snake, it is highly unlikely that we would be able to easily identify whether the snake we were facing was venomous or not! Learning and teaching "Snake awareness "is an important part of protection (WHO: Asian and African program).

" I am bitten by a snake, Help me " Samir started screaming , by the time every body jumped quickly for help , " Don't believe him , it is the 1st of April hoax " shouted one of his friends . Everybody sat back on their chairs gossiping "Oh he almost fooled us".

April Fools' day is celebrated in different countries around the world on April 1 every year. Sometimes referred to as All Fools' day. The day is marked by the commission of good-humored or otherwise jokes , hoaxes , and other practical jokes of varying sophistication on friends , family members , work associates , neighbors , etc…1,2.

During this time, Samir was flat on the ground, pale, cold, and sweating. Few minutes later his wife was shocked by his site and condition on the floor, she started screaming HELP!!!!!

By the time everybody arrived, Samir raised his head slowly with great difficulty and said "Its OK, I am alive, but I feel so weak, look this where I was bitten".
Some people who are bitten by snakes or suspect or imagine that they have been bitten, may develop quite striking symptoms and signs even when no venom has been injected. This results from an understandable fear of the consequences of a real venomous bite 1, 11, 12.

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Abstract

Better safe than die, dry or wet snakebite it does not matter: Some people who are bitten by snakes or suspect or imagine that they have been bitten, may develop quite striking symptoms and signs even when no venom has been injected. This results from an understandable fear of the consequences of a real venomous bite. Unfortunately, there is no simple rule for identifying a dangerous venomous snake. Bites by small snakes should not be ignored or dismissed. The risk of envenoming after bites by venomous snakes varies with the species. Bites in which the fangs pierce the skin but no envenoming results are known as "dry bites". The anti-venom remains the specific treatment for snakebite, yet the reaction to the anti-venom should always be considered.

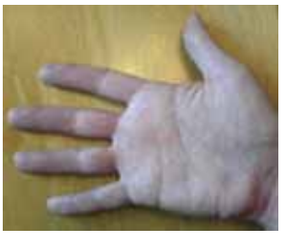
Key Words

snakebite, dry bites, anti-venom

Immediately he was rushed to the nearby Emergency room (ER), where the nurse in charge was surprised and frozen for some time when she heard the wife screaming "Please help us, a snake bitted my husband. In few second, the whole team was ready for help.

The Doctor on duty was surprised and unable to find the site of bite in comparison with the pale, cold sweating, tachycardic and trembling patient with stable cardiovascular status.
"Are you sure you were bitten by a snake and where? Show me".
The Doctor was not impressed and was not convinced. It could be a dry bite he said.

A dry bite is a bite by a venomous animal in which no venom is released. Dry snake bites are called "Venomous snake bite without envenoming" 3. Dry bites can occur from all snakes, but their frequency varies from species to species. For example brown snakes can inflict dry bites 80% of the time , while Australian taipans only inflict it 5% of the time4 .About 25% of snakebite cases can be dry bites and is characterized by fang and tooth marks and the absence of injected poison5.
Dry bites often are confusing for the attending physician and the victim. The phenomenon also is exploited by quack doctors as evidence for the effectiveness of supposed miracle cures 6.



"It is similar to a panic attack" The Doctor said.
"Dear Doctor stop, it is the 1st of April joke, his friends are saying" said one of the nurses that was questioning the group to help the Doctor on duty.
Mr Samir was kept in ER for 1-2 hours, and in view of his stable cardiovascular status and relative recovery of his general well being, he was discharged home on a bunch of instructions and advices. The wife kept asking "What about the all over sweating, cold body, and the pallor???" The doctor convinced her that all of these are manifestation of fear (panic attack).

The wife added "What about an anti-dote for snake bite in his case …?" The Doctor answered "It is not indicated in his case according to the international guidelines. The color of his hand did not change in 2 hours time and his general status is quite good, and we, as many hospitals, we do not have an anti-dote to snake bites, so I do advise you to transfer him to a bigger hospital". After a long conversation, the patient was transferred on the chair to one of the cars of the group.
Guidelines for the indications of use of antivenom clearly indicate that the antivenom is recommended if and when a patient with proven or suspected snakebite develops manifestations of severe local envenomation or one or more signs of systemic envenomation . 10,11,12

The first and important step in the management of snakebites is immobilization of the inflicted site. Antivenom should be given only to patients in whom its benefits are considered likely to exceed its risks .It should not be used indiscriminately since the antivenom is often limited in supply and the risk of reaction to the antivenom should always be taken into consideration. 10,11,12

Later, they were refused in several hospitals the moment they declare about the possibility of snakebite because they do not have an anti-dote to snakebites.
Finally, a hospital accepted to receive them especially that they have an anti-dote with very close expiratory date. The patient and family were made to sign on an
Off-responsibility form. By the time the Doctor on duty finished interviewing and examining Mr Samir he informed them all that there is no evidence of snakebite, there is only one possible prick site, most probably a dry bite, and in the absence of signs of systemic envenomation and even local manifestation, there is no indication for the the use of the anti-venom although they were so willing to use it.

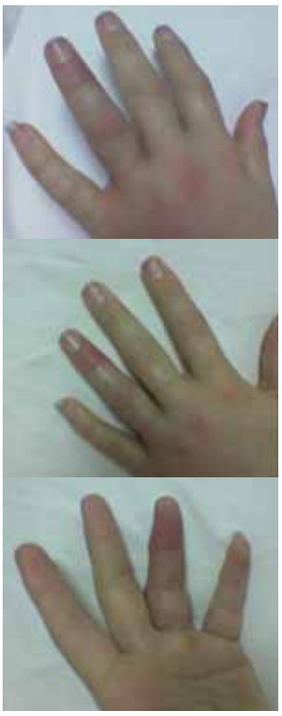
One of the most difficult problems associated with dry or presumed dry bites , is that it is not simply a matter some bites being dry and others venomous ,some species have quite sophisticated mechanisms for controlling the dosage of venom injected , which can vary widely and very unpredictably . Jumping to conclusions on the basis of just a few experiences of a given species or a group of related species, can be fatal. There are many variables; anecdotally, very young venomous snakes for example, tend to have disproportionately dangerous bites—there is reason to suspect that they inflict a full bite routinely. 4,5,10,11,12

« I do not feel well , I am not myself yet , I will sign a paper for you if you inject me with the anti-venom «said Mr Samir in a begging attitude , but no response

The whole group including Samir and his wife left the hospital 1-2 hours after midnight in a long drive away to his home.

Toxicology literature has documented many cases describing the late effect of venom that usually is not harmful to man but the circumstances determines toxicity , one of these cases 2

In her garden with her bare arm in bushes, our lady felt a sting and withdrew hurriedly. Seeing nothing, she thrust her arm, in again and felt a worse sting, inspection revealed a Palystes (Huntsman spider) nest-cocoon, with the female on guard. There was minimal fine redness at the site of the bites, but no other ill effects. Later that evening, however, there was dizziness and nausea, and her arm became swollen and painful, with local discoloration at the sites of the bites. She ended in hospital starting a long trip ….4,8,11,1



On the way back home, Samir noted that his finger is changing in color. He said nothing and we all know why. After 15-20 minutes, his finger became larger with sensation between numbness and burning. He silently directed his wife's vision to his finger and hushed her. She was scared and her immediate response was :

"Quickly please, back to the hospital, Samir's finger is changing in colour".
After 10-15 minutes the wife noted her husband is becoming weak and lethargic ,
she started shouting " Quickly please I am loosing my husband , to the nearest hospital on our way ".
The reception in the nearby hospital that they have visited early in the night yesterday was very bad " We told you , we cannot help him , we do not have an anti-dote for snakebites " said the nurse in charge and the Doctor on duty , But after the insistence of the wife , they accepted to admit him in ER and assess his medical status with one condition , " The outcome is not our responsibility because we have clearly informed you several times that we do not have an anti-dote for snakebites " said the nurse in charge .
During this time, Mr Samir was found in the back seat of the car non-responsive, hardly breathing, very pale, and cold all over. All attempts in ER to help him have failed. Mr Samir passed away.

Too many questions remained unanswered :
- What has happened and why??
- Who is the victim in this story??
- Who is guilty and responsible for this outcome??

N.B. : The pictures of the patient's hand were taken by the patient early and later by the wife using their mobile phone , they represent a sample indicating the wife's approval to write this story and use these pictures .

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Conflict of interest statement :
There is no conflict of interest to declare

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12. Guidelines for the management of snake bites by David A. Warrell 2nd ed released by WHO on May 23 2011

ATLS® at the American University of Beirut: Program Overview

Injured patients present a wide range of complex problems. The ATLS Student Course presents a concise approach to assessing and managing multiply injured patients. The course presents doctors with knowledge and techniques that are comprehensive and easily adapted to fit their needs. The skills described in the manual represent one safe way to perform each technique, and the American College of Surgeons (ACS) recognizes that there are other acceptable approaches. However, the knowledge and skills taught in the course are easily adapted to all venues for the care of patients.

The American College of Surgeons (ACS) and its Committee on Trauma (COT) have developed the Advanced Trauma Life Support (ATLS) program for doctors. This program provides systemic and concise training for the early care of trauma patients. The ATLS program provides participants with a safe, reliable method for immediate management of the injured patient and the basic knowledge necessary to:

1. Assess the patient's condition rapidly and accurately.
2. Resuscitate and stabilize the patient according to priority.
3. Determine if the patient's needs exceed a facility's capacity.
4. Arrange appropriately for the patient's inter-hospital transfer (who, what, when, and how).
5. Assure that optimum care is provided and that the level of care does not deteriorate at any point during the evaluation, resuscitation, or transfer process.

For doctors who infrequently treat trauma, the ATLS course provides an easy to remember method for evaluating and treating the victim of a traumatic event. For doctors who treat traumatic disease on a frequent basis, the ATLS course provides a scaffold for evaluation, treatment, education, and quality assurance. In short, ATLS is a measurable, reproducible, and comprehensive system of trauma care.

The ACS- Lebanon Chapter in association with the American University of Beirut introduced the ATLS course in Lebanon in November 2010, giving them the exclusivity of the program in Lebanon. Since then 5 courses took place, 88 Doctors trained and received their certificates. The ATLS certificate is provided by the ACS and is internationally valid for 4 years.

Course Director	Dr George Abi Saad, Professor of Surgery, FACS and trauma surgeon at the AUBMC	
National Course coordinator	Diana El Skaff, RN, BSN, Administrative coordinator in the Continuing Medical Education office and the External Medical Affairs.	
Registration fee	800$ per student for the provider course	RNs and paramedics are welcome to attend the course as auditors: 50$ per person
Upcoming courses	February 4-6, 2012 March 31- April 2, 2012	May 26-28, 2012 July 14-16, 2012
Information & registration	Diana El Skaff: Mobile: 961-3-385710 Skaff.diana@gmail.com	

C. Hamouda, H. Ghorbel, A. Hedhili, N. Ben Salah, M. Amamou.

An unusual intoxication by Hyoscyamus albus: about two cases

Dr. Chokri Hamouda

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Abstract

We report two cases of intoxication by Hyoscyamus albus, admitted in the toxicology department of the anti-poisoning centre of Tunis after a consumption of boiled vegetables (soup) based on Chinese leaves and Hyoscyamus albus. These two reported cases illustrated the danger of the lack of knowledge of toxic plants and the risk of non controlled product consumption.

Key Words

Intoxication, plants consumption, Hyoscyamus albus

Introduction

In Tunisia, many plants are used culinary and medically more and more frequently (1, 2). Nevertheless, in front of all these increasing interests in natural products, there have emerged an undeniable anxiety related the accidental poisoning by plants (3). Every year, many accidental poisoning cases are noticed at the Tunisian anti-poisoning centre essentially due to the confusion between edible and toxic plants, or overdoses use.
During this work, we report two cases of intoxication by Hyoscyamus albus (fig 1), admitted in the toxicology department of the anti-poisoning centre of Tunis after a consumption of boiled vegetables (soup) based on Chinese leaves and Hyoscyamus albus.

Fig. 1 : Hyoscyamus albus

Clinical cases

This study concerned two masculine patients aged respectively 26 and 61 years old, originated from Tunis. The intoxication diagnosis was based on the anamnesis, clinics and the urinary toxicological analysis. Mr X and Mr Y are without noticed pathological antecedents. They were admitted in the toxicology department for anti-cholinergic syndrome after ingestion of boiled vegetables.
Two hours after the ingestion, the two patients began to show progressively a muscular hypotonia, some dizziness and an asthenia followed by two vomiting episodes. After 8 hours, the patients have faced a visual blur, a mouth drought and a urinating difficulty.
The clinical exam showed patients without conscious disturbances. Pupils were in reactive mydriasis. Osteotendinous reflexes were bright and symmetrical. Temperature was at 37°C. Respiratory rhythm was 20 cycles per minute for the younger patient and 18 for the older one. Pulses were respectively at 120 and 100 per minute. Arterial pressure was at 90/70 mm Hg for the son and 180/100 mm Hg for the father.
The EKG, the arterial blood gases, the blood formula numeration as well as the bio-chemical

exams were normal except hyperglycemia showed for the second patient (11mmol/l). The qualitative toxicological analyses concerning psychotropic and pesticide products research in urines were negatives. The research of alkaloids by thin layer chromatography has revealed the atropine presence (fig 2).
The evolution under symptomatic treatment was marked by the disappearance of anti-cholinergic syndrome and digestive troubles in 24 hours for the two patients.

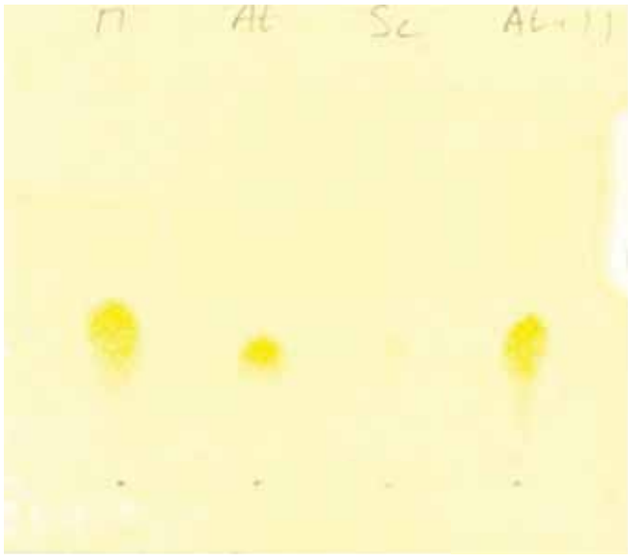

Fig. 2: Research of alkaloids by thin layer chromatography

M = patient; Sc = scopolamine

At = atropine; At + M = atropine + patient

Discussion

The acute intoxications, from which plants intoxications represent 1%, are one of the first causes of hospitalization and represent a major problem of public health (4, 5). The difficulty of taking care and diagnosis is mainly related to the poison nature, the swallowed dose and time between ingestion and hospitalization.
When the toxic product is a plant, the diagnosis must be based on the anamnesis, symptoms, toxicological and biological analysis.
For the two reported cases, the anamnesis has revealed an undoubted confusion between the two plants: the Chinese leaves and the Hyoscyamus albus (fig 3).
The first which is an annual or bi-annual (Chenopodiaceous), largely consumed in the Tunisian kitchen. Its leaves equally used in external usage under the form of powder for its cicatricial characteristics, the decocted leaves are also recommended to omit edema.
The second (Solanaceae family) is a plant known for its toxicity. It's responsible for drunken case and hallucination. It's famous for treating the hemorrhoids. The decoction of the above parts is recommended in the treatment of some dermatosis equally cured by the smoke emitted by the seeds thrown in fire.
Active ingredients responsible for toxicity are Atropine, Scopolamine and Hyosciamine (6, 7).
These two reported cases illustrated the danger of the lack of knowledge of toxic plants and the risk of non controlled product consumption. For these two cases the poison is known from the anamnesis' data of patients considering the absence of parts of the plants. The symptoms agree with the incriminated toxic products. The toxicological analyses were not indispensable to put the diagnosis. So the analytical result has oriented and confirmed it in these two specific cases where symptomatology was delayed to 8 hours. In addition, the qualitative screening has gapped the eventuality of intoxication by other products such as tricycles' anti-depressor and anti-Parkinsonien drugs which are responsible for the same poisoning futures. The atropine presence which is the main alkaloid in Hyoscyamus albus composition clearly accounts for the appearance of the anti-cholinergic syndrome for the two patients (8, 9).
The prognostic of the two intoxication cases were favorable under symptomatic treatment and was marked by the disappearance of the anti-cholinergic syndrome and digestive trouble within 24hours.

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Conclusion

The medical usage of plants is more and more frequent, if certain eatable species are easily recognized, others may be confused and their consumption may be harmful. Nowadays, in Tunisia, there are no data showing any acute accidental intoxication cases owing to their diversity.

Fig. 3: Chinese leaves (left) and Hyoscyamus albus (right)

The diagnosis of such intoxication must be based on many data: anamnesis, clinic and analytic. In most cases, only a symptomatic and/or supervision are enough without forgetting that some preventive measure should be determined in order to reduce the frequency and severity of such intoxications.

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INTERNATIONAL EVENTS

«LEVERAGING DISASTER RESPONSE TO SPUR ONGOING HUMANITARIAN DEVELOPMENT» - Haiti Disaster Relief and Development Conference
January 10, 2012 to January 18, 2012 – Haiti

1ST GLOBAL NETWORK ON EMERGENCY MEDICINE CONFERENCE
January 13, 2012 to January 17, 2012 – Dubai International Convention and Exhibition Centre

III SIMPOSIO NACIONAL DE REANIMACION "MANEJO DEL QUINTO ESLABON EN REANIMACION" "CUIDADOS INTEGRADOS POST-PARO"
February 10, 2012 at 7am to February 11, 2012 at 1pm – Auditorio Ponce de León, Fundación Cardio Infantil, Colombia

GLOBAL EMERGENCY MEDICINE PROGRAM
March 5, 2012 to March 16, 2012 – Weill Cornell Medical College, New York

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Souheil Chamandi, May Helou, Tony Matta

Management of severe headache in awake patients with intracerebral hemorrhage

Dr. Souheil Chamandi

Article history / info:
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Key Words

Cerebral hemorrhage, awake patient, pain management, greater occipital nerve block

Summary

Our intensive care unit has been treating patients with intra cerebral hemorrhage and subarachnoid hemorrhage for many years.

The high majorities of patients with intra cerebral bleed are comatose and require intubation and sedation during their ICU stay, until recovery.

Rare are the cases of the patients with diagnosed intra cerebral hemorrhages who were awake, without the need of intubation.

Between 2007 and 2010, 12 patients (9 males and 3 females) were admitted into our ICU unit with a confirmative imaging (MRI) of intracranial hemorrhage. (Intracerebral and subarachnoid hemorrhage). All 12 patient were awake and did not require intubation.

The chief complaint of those patients was a severe thunderclap headache that responded poorly to classic antalgics such as acetaminophen and Non Steroid Anti Inflammatory drugs.

Opiate such as morphine was also used without a significant relief of their headache.

The pain management of the headaches in this group of awake patient with classic antalgics and morphine was not optimal and the need to establish a new pain management method has led us to perform a new supportive therapy for their headache: nerve block.

A greater occipital nerve block along with the lesser occipital nerve block, using a nerve stimulator was done bilaterally on all twelve patients using Bupivacine on each side.

A dramatic improvement of the headache (according to VAS scale) was noted by the 12 patients after this block.

After having an intra cerebral hemorrhage, patients often experience on going severe headache.

The pain is well documented as a cause of raised intra cranial pressure and it is important to make pain relief for such patients a priority. (Hickay 1996).

Pain is usually unbearable and leads in many cases to agitation. [1]

This pain (headache) with or without agitation must be avoided because it can aggravate the intra cerebral pressure elevation throughout straining (increasing thoracic, jugular venous, and systemic blood pressure), increased cerebral metabolic rate of oxygen, and also may cause uncontrolled hyper/hypo-ventilation[1].

During an ICP spike, pain management of the headache may be all that is necessary to control the intra cranial pressure.

Analgesia is a must to prevent this viscous circle of pain, agitation, increased ICP and increased clinical deterioration (re bleeding); rebleeding risk 4% on first day and 2% per day, up to 30% in the first month [3].

Effect of acetaminophen

Fever is common after SAH and is found in at least one third of patients [2], it is a neurogenic type of fever and treatment with acetaminophen may be needed for hyperthermia. Its antalgic effect alone in this severe headache was poor and its main effect was to decrease the fever without notable improvement of the headache.

Narcotics

IV morphine has been demonstrated to cause significant arterial dilation manifested by a reduction in systemic vascular resistance.

Cerebral vasodilatation may be occured secondary to carbon dioxide retention.

We conclude that bolus injection of opioid should be used with caution on patients with ICP because they can transiently lower Mean Arterial Pressure (MAP) and increase ICP due to autoregulatory vasodilatation of cerebral vessels. [1]

Neurogical assessment is of massive importance in patients with cerebral hemorrhage [4] and the use of opioid is believed to mask the neurological deterioration due to its central inhibiting effect.

Methods

Approval for this study was obtained from the Ethics in Hyman Research Committee (Notre Dame des Secours University hospital). Potential volunteers were given an information sheet and a briefing by a researcher, with an opportunity to ask questions. Written informed consent was obtained from all subjects agreeing to participate in both studies. Data were anonymised and information on the performance of individual participants was not made available to anyone outside of the study team.

All patients were presented with severe headache that responded poorly to Acetaminophen and other pain killers. A greater occipital nerve block along with the lesser occipital nerve block, using a nerve stimulator was done bilaterally on all ten patients using Bupivacine 0.125% 6cc on each side.
The block was performed every 6 hours.
No discomfort was observed no hematoma was seen.

Result

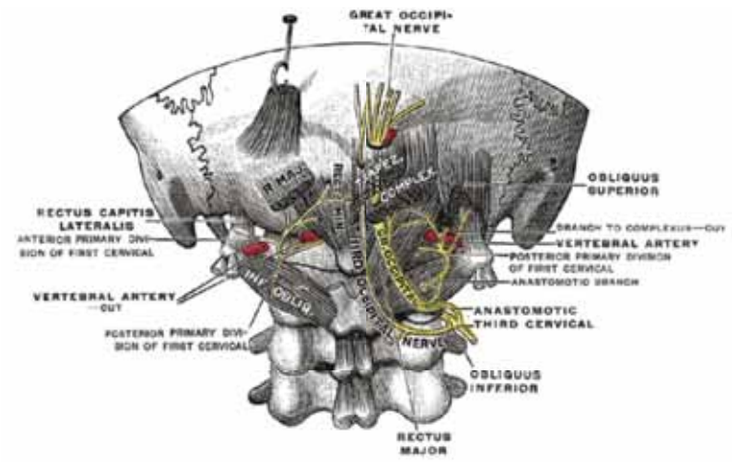
After 30 minutes patient was observed and examined.

Pain was almost subsided in ten patients. Only two patients reported no improvement of their headache.

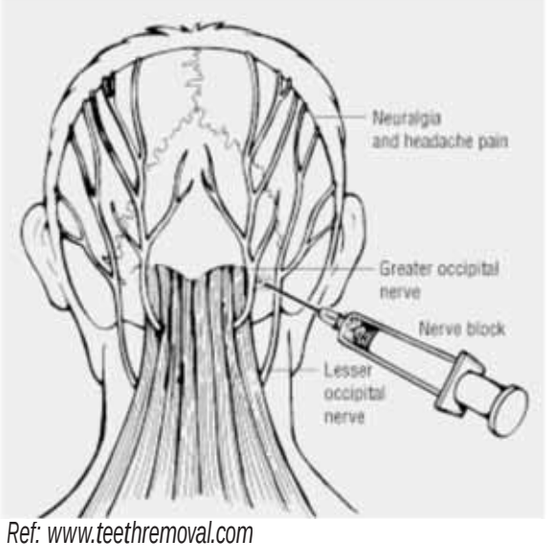
Discussion

Our study suggests that in the hands of experienced anesthesiologist (pain management), the greater occipital nerve block along with the lesser occipital nerve block, in managing the headache after intra cerebral hemorrhage and subarachnoid hemorrhage, is a very safe tool that provides relief and comfort for this group of patients.

As for the two cases that did not respond to this block we assume that either the block was a failure or didn't work at all.



Ref : en.wikipedia.org/wiki/Greater_occipital_nerve



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Dr Cécile Roncoroni

Clinical predictive factors in the treatment of headaches

Headache is a common complaint in the general population and accounts for 1-4% of emergency department visit patterns [1-8]. The management of a headache in the emergency room is a challenge for physicians.

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Abstract

The objective of this study is to assess the ability of physicians to establish the diagnosis before performing additional tests and to determine clinical factors directing them to a secondary etiology where the achievement of these tests is necessary

Method: Prospective study of adult patients presenting to the ER with a headache for ten months (01/07/07 to 31/05/2008). Were excluded patients with post-traumatic headache or presenting after the clinical neurological abnormalities or meningeal stiffness.

Results: One hundred four patients met the inclusion criteria. After clinical examination, emergency physicians ranked as primary headache 37% of cases, secondary 31% of cases and of unknown etiology 32% of cases. The diagnostic tests have concluded definitively: 34% of patients had a secondary headache; with 11.5% carriers of a disease can be life-threatening and 66% a primary headache.

Fever ($p < 0.05$), the location of frontal headache ($p < 0.05$), the presence of aggravating factors such as orthostasis ($p < 0.01$) and vomiting ($p < 0.05$) the notion of improvement after usual analgesic treatments ($p < 0.02$) were significantly associated with the presence of a secondary origin.

INTRODUCTION

The diagnosis of headaches is often underestimated, especially the original primary headache [7, 8, 17], but also secondary extra neurological conditions such as sinusitis or carbon monoxide poisoning that are often overlooked. The management of patients with headache during the hyperalgesic phase of the headache is not easy [17]. And finally, an association between concurrent primary and secondary headache is present in 10% of cases [11]. However, the vital or functional prognosis of the patient is engaged in at least 10% of cases [9].

The international classification of headaches is the classification of the International Headache Society (IHS) developed in 1988 and revised in 2004 [10]. Unfortunately, it is difficult to use it in routine in the emergency rooms (ER) and a recent study in 2007 showed that 36% of headaches are unclassifiable because they do not meet the IHS criteria [11].

Diagnosis underestimated

There is no reference in an emergency context for headaches, hence the need for a simplified decision algorithm for appropriate care in the ER.

The headache is primarily a non-specific symptom that requires a careful and complete physical examination including a neurological exam.

The presence of a clinical neurological abnormality, such as the existence of meningeal stiffness, or abnormal vital signs guides to a secondary etiology. Unfortunately, their absence does not eliminate the diagnosis of secondary headache. The examination, the evolving profile, the background and age are critical to performing further investigations [15, 16].

The main objective of this study is to find predictive clinical factors that would distinguish primary headache in which investigations are unnecessary and secondary headaches where additional tests have their place.

The secondary objective is to evaluate the ability of physicians to diagnose the headaches at the end of their clinical examination and before any further investigations and the adequacy in carrying out these examinations based on the suspected diagnosis.

MATERIALS AND METHODS

Our work is a prospective and observational study concerning adult patients consulting in an emergency department on the basis of headache not related to trauma. The study was conducted at Grenoble University Hospital for a period of 10 months (01/07/07 to 31/05/2008). Patients with post-traumatic headache or who have a neurological abnormality or stiffness meningitis were excluded .The data were collected by the emergency physicians using a questionnaire containing demographic items, history, anamnesis, clinical examination, diagnosis made before any examination or paraclinical test and the paraclinical tests realized. The follow up was realized by phone.

STATISTICAL ANALYSIS

We conducted a descriptive first part, with an analysis of epidemiological and general data. For the statistical analysis of the second part, we used a Stat View software and looked for statistically different parameters between the groups of primary headache and secondary headache. Statistical differences between categorical variables were investigated with the contingency test chi-2 and for the comparison of continuous variables; we used the Student t test. To establish a correlation between continuous variables, we used the Z-test correlation. A value of p less than 0.05 was considered significant.

RESULTS

In total, 158 patients were identified, 104 patients were enrolled and 54 patients were excluded (Figure 1). Of the 104 patients included, there were 52 women and 52 men, aged from 16 to 92 years. The average age was 38 ± 17. The time after which the patients came to consult was 95.5 ± 135 hours. The transport was performed in 20% of cases by an ambulance, SAMU or firemen and in 80% of cases by personal vehicle (Table 1).

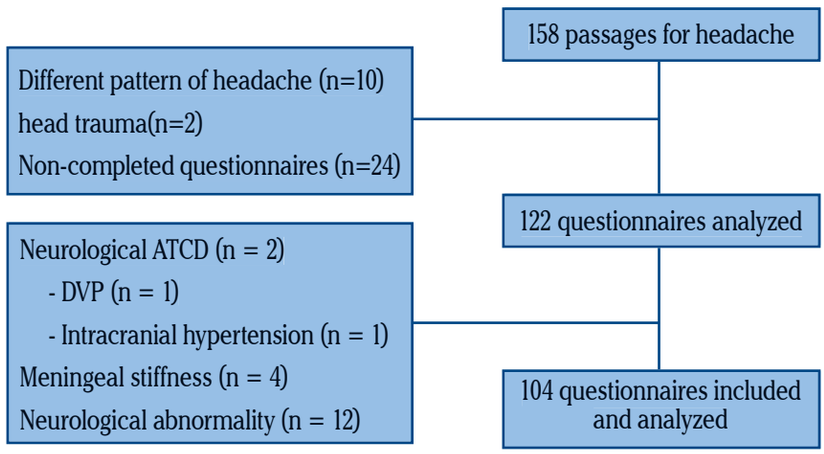


Figure 1: Steps of the inclusion

	Primary headaches (n=69)	Secondary headaches (n=35)	Total (n=104)	P Value
Age (mean ± SD)	37±17	41±17	38±17	0,25
Sex (female/male)	37/32	15/20	52/52	0,30
Duration in hours				
(mean ± SD)	89±143	107,5±119	95,5±135	0,52
Transport				
- Ambulance	15 (22%)	6 (17%)	21 (20%)	0,31
- Personal vehicle	54 (78%)	29 (83%)	83 (80%)	
headache history	27 (39%)	7 (20%)	34 (32,6%)	0,04
family history	20 (29%°	6 (17%)	26 (25%)	0,19
Hypertension	14 (20%)	7 (20%)	21 (20%)	0,97
Tachycardia	11 (16%)	7 (20%)	18 (17%)	0,60
Hyperthermia	2 (3%)	9 (26%)	11 (10,6)	<0,01

Table 1: Univariate analysis of general data and clinical

Abstract(suite)

Conclusion: This study highlights the discomfort of physicians in the treatment of headaches, since in 50% of cases the doctor feels the need to make an imaging in spite of already diagnosing a primary headache. It is necessary to develop clinical decision-making algorithms to optimize the realization of additional tests in the ER.

Key Words

headaches, predictive factors, diagnosis, emergency room (ER)

Complementary Examination	Primary headaches (n=69)	Secondary headaches (n= 35)	Population total (n= 104)
CT scan	47(68%)	23(66%)	70(67%)
Lumbar puncture	11(16%)	12(34%)	23(22%)
Biology	46(67%)	29(83%)	75(72%)
Others			
(MRI, sinus radio)	1(1%)	2(6%)	3(3%)
No exams	13(19%)	4(11%)	17(16%)

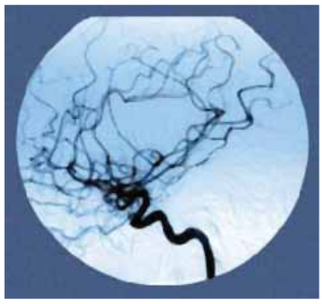
Table 2: Complementary examinations carried out in groups of primary headaches and secondary headaches by number and percentage of patients

ADDITIONAL TESTS

In the group, 70 patients (67.3%) had a head scan with CT angiography for 36 times. The scan was normal in 91% of cases, 1 patient had a brain MRI that was normal, 23 patients (22%) had lumbar puncture, 22 (21%) following a scanner. For one patient, lumbar puncture detected a subarachnoid hemorrhage (SAH) after a normal CT. In the end, 72% received laboratory tests, and 17 patients (16%) had no further examination (Table 2).

ETIOLOGIES OF HEADACHES

It appears from our study that 69 patients (66%) had a primary headache and 35 (34%) a secondary headache. The infectious disease is the most common etiology found for secondary headaches (24% of cases). On analysis, 11.5% of headaches were potentially serious pathologies in which were included 5 viral meningitis, 1 bacterial meningitis,3 subarachnoid hemorrhage, 1 cerebral thrombophlebitis, 1 parenchymal hemorrhage, but also 1 tumor-induced headache, and 1 headache due to a metabolic disorder (dysnatremia).(Table 3).



Carotid angiogram: A suprasylvian meningioma is responsible for a mass effect, and distant the frontal and parietal vessels. (BSIP/NEIL BORDEN)

Final etiologies of headaches	Number and percentage of patients
Secondary headaches	35 (34%)
Infectious causes	24 (24%)
- Sinusitis	6 (7%)
- Other infectious syndromes	12 (11,5%)
- Viral meningitis	5 (5%)
- Bacterial meningitis	1 (1%)
Tumors	1 (1%)
Vascular causes	4 (4%)
- HSA	2 (2%)
- intraparenchymal hemorrhage	1 (1%)
- cerebral thrombophlebitis	1 (1%)
Metabolic causes (hyponatremia)	1 (1%)
Drugs	1 (1%)
Intracranial hypotension	
- Post PL Syndrome	2 (2%)
Arnold's neuralgia	1 (1%)
Trigeminal neuralgia	1 (1%)
Primary headaches	69 (66%)
- Migraine	13 (12,5%)
- Non-specific headaches	55 (53%)
- Cluster headache	1 (1%)

Table 3: The causes of headaches in numbers and percentage of patients

Diagnostic difficulties in 1/3 of the cases

MONITORING

It was conducted by phone.12 patients (11%) were not reachable, and we did not find evidence of second passage of those patients to the ER for headache. Of the patients contacted, 16 (15%) re-consulted their doctor for headache, 8 returned to the ER, including 3 for post lumbar puncture syndrome. Let us add that only 2 patients reported a secondary diagnosis of sinusitis, initially diagnosed as cluster headache and unspecific headache.

UNIVARIATE ANALYSIS

The results of the univariate analysis of predictive clinical factors of secondary conditions are summarized in Table 4. Some variables have a significance as the expected unilateral headache ($p = 0.02$), or the presence of a personal history of headache ($p = 0.04$) in primary headache.

Hyperthermia is a predictor of secondary origin (p = 0.004). The location was significantly associated with frontal headache side (p = 0.04) but not significantly related to the severity of them (p = 0.12).

The existence of aggravating factors of the headache (p = 0.04) was significantly in favor of a secondary etiology, particularly vomiting (p = 0.03) and orthostasis (p =0.01). Similarly, the presence of factors improving the headache (p = 0.04) indicates a secondary origin, such as improved by the usual analgesics (p = 0.02).

The characteristics analyzed with the number and percentage of patients				
	Primary headaches (n=69)	Secondary headaches (n=35)	Total (n=104)	P value
Brutal occurrence	6 (17%)	13 (19%)	19(18%)	0,83
Intensity	7,1±2,2	6,1±1,9	7±2	0,36
Localization :				
- Unilateral	28 (40%)	6 (17%)	34 (32%)	0,02
- Bilateral	41 (60%)	29 (83%)	70 (68%)	
Topography :				
- Frontal	20 (29%)	19 (54%)	39 (37,5%)	0,01
- Occipital	16 (45,7%)	11 (15,9%)	27 (26%)	0,32
- Temporal	21 (30%)	7 (20%)	28 (27%)	0,27
- Retro-orbital	10 (14,5%)	5 (14%)	15 (14%)	0,97
Type :				
- Pulsatile	20 (29%)	8 (23%)	28 (27%)	
- Vice	17 (24,6%)	8 (23%)	25 (24%)	0,79
- throbbing	18 (26%)	9 (26%)	27 (26%)	
Accompanying Signs				
- Vomiting	17 (25%)	13 (37%)	30 (29%)	0,18
- Photophobia	28 (40%)	11 (31%)	39 (38%)	0,38
- Phonophobia	16 (23%)	8 (23%)	24 (23%)	0,96
- Osmophobia	1 (1%)	2 (6%)	3 (3%)	0,21
- asthenia	22 (32%)	15 (43%)	37 (36%)	0,27
Aggravating Factors	18(26%)	21(60%)	39(38%)	0,04
-standing	1(1%)	5(14%)	6(6%)	0,01
-lying down	6(9%)	2(6%)	8(8%)	0,59
-Head position	8(12%)	10(29%)	18(17%)	0,08
- Walking	7(10%)	3(9%)	10(10%)	0,80
- Vomiting	1(1%)	4(11%)	5(5%)	0,03
-Valsalva manœuvre	4(6%)	4(11%)	8(8%)	0,31
Improving factors	21(30%)	27(77%)	48(46%)	0,04
- lying down	17(25%)	14(40%)	31(30%)	0,11
- Standing	3(4%)	1(3%)	4(4%)	0.71
- Head position	2(3%)	2(6%)	4(4%)	0.48
- Walking	1(1%)	0	1(1%)	NA
- Vomiting	1(1%)	2(6%)	3(3%)	0.22
- conventional treatment	10(14%)	12(34%)	22(21%)	0.02
- Valsalva manœuvre	NA	NA	NA	NA

Table 4: NA: not applicable because of insufficient number - n: number of patients - A p value <0.05 is significant

The capacity of doctors to establish the diagnosis of headaches

Emergency physicians have classified 37% of cases as primary headaches , 31% of cases as secondary headaches, and 32% of cases as undetermined etiology . This evaluation shows that in 1/3 of cases, the doctors cannot distinguish if the headache is primary or secondary.

When the doctor diagnosed a primary origin of the headache, the diagnosis was confirmed in 85% of cases. For six of 39 patients (15%), it was actually a secondary headache (1 bacterial meningitis, a viral meningitis, sinusitis, and 1 3 unspecified infectious syndromes). In 50% of cases of presumed primary headache, a CT scan was performed and in four cases, scan and lumbar puncture were performed. In fact, 47% of secondary headaches were diagnosed as primary headaches. A brain scan was performed in 69% of cases where the hypothesis of secondary headache was evoked (Table 5).

	Diagnostic de céphalée primaire (n=69)	Diagnostic de céphalée secondaire (n=35)
Assumed primary etiology (n = 39)	33(85%)	6 (15%)
Etiology supposed secondary(n = 32)	15 (47%)	17(53%)
Undetermined etiology (n = 33)	21 (64%)	12(36%)

Table 5: Doctor's ability to determine the diagnosis of headache

DISCUSSION

Given the difficulty of diagnosing headaches in the ER and the economic impact of their management, it becomes necessary to establish a clinical decision-making algorithms in order to orientate either to a primary origin that requires no further consideration, but appropriate treatment, or to a secondary origin that can be life-threatening

in 10% of cases. Our study was therefore interested in looking for predictive clinical features of secondary origin in order to guide the management of headaches. First of all, we showed that there is a significant relationship between the frontal location and the presence of a secondary origin. Then we wanted to confirm that the presence of aggravating factors and / or increasing headache is suggestive of secondary headache.

Similar studies have been done like Ramirez-Lassepas et al who studied 468 patients presenting to the ER with headache and found that age over 55 years, sudden onset, abnormal neurological examination, occipito-nuquale location and the presence of accompanying signs were predictive of severe intracranial pathology [6]. These same factors were later found in a study of 558 patients conducted by Locker et al in 2006 [9].

The presence of an abnormality on clinical examination, as well as the presence of meningeal stiffness are suggestive of secondary origin in a number of studies and are items identified as requiring further investigation according to a grade B recommendation by the American Association of Emergency Medicine [15].

However a normal neurological examination or absence of meningeal stiffness, do not eliminate a secondary etiology. Studies have shown that in 0.9% to 10% of cases, patients with headache and a normal neurological examination have intracerebral lesions [19, 21].

It turns out that 10% of HSA and 15 to 30% of thrombophlebitis have a normal neurological examination [20, 22].

In addition, the meningeal syndrom in an HSA may appear patient only 12 hours after the onset of the headache [19, 20]. For this reason we included only patients with a normal neurological examination and without meningeal stiffness in this study.

Age is an independent risk factor for a severe disease [15.23]. A first episode of headache in a patient over 65 years is in 15% of cases associated with a secondary origin, while in the youngest patients, in less than 3% of cases it is a mild headache [14]. A first migraine attack often appears after the age of 40 [1]. We did not find any significant difference in age between our two groups.

The sudden onset of a headache has always been a gravitating factor since in 50% of cases it was

a serious condition [6, 15, 16, 20, 23]. It does not appear here as an element of gravity.

The location of the headache is also important. Landblom et al and Liedo et al.studied patients with thunderclap headache and found a significant association between occipital location and the presence of HSA [24, 25] The temporal location would be in favor of a mild headache. In our study, the occipital location does not appear as a predictive factor in contrast to the frontal location that is significantly related to a secondary origin, but not significantly related to the severity of the underlying pathology. One could explain this result by the importance of including infectious causes of sinusitis in the secondary etiologies. Of the 19 patients with frontal headaches of secondary origin, 9 patients (48%) had a headache due to infection, 2 associated with sinusitis.

The intensity of the headache can be as high in a migraine as in an HSA and is not a predictive factor.In a number of studies, the presence of several accompanying signs were predictive of secondary pathology [6]. In our study the aggravating factors such as vomiting or orthostatic and the presence of enhancing factors such as improved by the usual analgesics are predictive of secondary origin. This confirms the American recommendation in 2008 Grade C which eliminates the concept of therapeutic test [15]. A favorable response to analgesics does not eliminate a secondary etiology [26, 27].

Nemer et al have found a link between the severity of the headache and the mode of transport by ambulance, but this was not confirmed by our study [28].

Thus, most predictive clinical factors of secondary headache found previously have not been confirmed by our study and new elements have emerged as the importance of the frontal location and the presence of elements contributing to or aggravating headache.

In the second part of the study, we evaluated the ability of physicians to diagnose a headache before any further investigations. Perry et al studied the 2005 trial of the doctors in determining the diagnosis in patients presenting to the ER for a headache with a normal neurological examination. In 75% of cases, the doctor realized a brain imaging even if he assessed the patient at low risk of intracranial pathology [18].

In our study, doctors judge primary headache in 80% of they feel the need to achieve in 30% of cases, doctors the headache is primary or come from a misunderstanding lack of an interrogation.

Common infectious etiologies of secondary headaches

correctly in their diagnosis of cases. However, in 50% of cases, a brain imaging. In addition, can not determine whether secondary. The explanation may of the diagnostic criteria and

Thus, these results demonstrate the difficulty and discomfort of physicians in the treatment of headaches without conducting further investigations.

The results are interpreted with a number of limitations.

First the existence of a small number of patients, compared to previous studies with insufficient recruitment in an emergency center that has an annual passage of 20,000 patients.

Then there is a lack of rigor for the filling of questionnaires leading to missing data, making the results less reliable. For 18 patients (17%) of which 13 (19%) in the primary headache and 5 (14%) in the headache secondary aggravating factors and improvers are not specified and considered absent. In 6 cases (5%) the vital signs are missing, and in 11.5% of cases, doctors have not written their diagnostic hypothesis and by default the assumed diagnosis was considered indeterminate.

Finally, all those included in the study have not been contacted by telephone. Twelve patients were lost to view. We considered that only the patients who had not benefited from the couple of brain scan and lumbar puncture should be followed.

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CONCLUSION

Studies on a larger scale and methodologically more reliable are needed to develop recommendations and decision-making algorithms that will determine the directions of further investigations in headaches. They allow a reduction in their consumption and would make physicians more comfortable with their care.

Conflict of interest statement :
There is no conflict of interest to declare

The editorial board

What we knew

Headache is a benign symptom. Even if it is, most often in correlation with a primary benign disease, it may be a sign of a major secondary pathology. The diagnostic approach is not always obvious.

What this article brings

this study reflected the difficulties of doctors to choose the appropriate complementary examinations. However, more than 10% of patients have a potentially lethal secondary headache. Some signs may be alerting like the presence of fever, frontal headache location, and / or it's aggravation in an orthostatic position.

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RENAULT

Claude DUSSART, Cécile BERTARD, Frédéric CHAVET, Gilles GRELAUD, Christophe LABLANCHE, David ALMERAS.

Prise en charge du risque tétanigène aux urgences :
Nécessité d'un protocole pour les services d'urgences.

Dans le cadre de la démarche qualité, nous avons voulu savoir si la prise en charge du risque tétanigène semblait homogène dans les différents hôpitaux d'instruction des armées *(HIA)*. Dans un second temps, nous avons élaboré un protocole permettant de positionner les différents moyens prophylactiques et les tests rapides de connaissance du statut immunitaire à destination des services d'urgences.

Claude DUSSART

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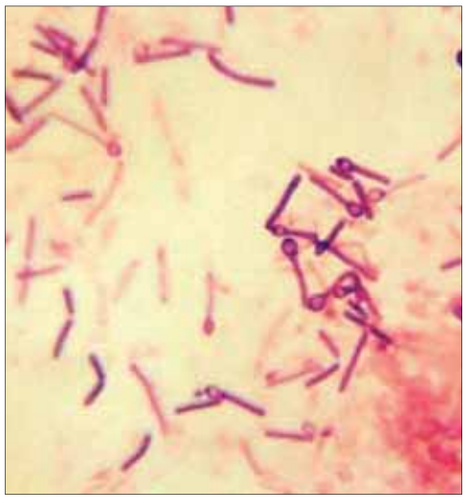
Tétanos, prophylaxie antitétanique, plaie tétanigène, protocole

Résumé

Les recommandations officielles concernant la prise en charge du risque tétanigène lors du traitement de plaies aux urgences ne sont pas très précises. De récentes études ont montré que cette prise en charge semblait être très hétérogène d'un hôpital à l'autre. La couverture vaccinale antitétanique de la population est imparfaite et la preuve d'une telle vaccination est difficile à obtenir, la définition d'une plaie à risque tétanigène ne fait pas l'objet d'un consensus. Par ailleurs, l'utilisation de tests de détection des anticorps antitétaniques n'est pas généralisée et les conditions d'utilisation de ce type de test ne sont pas identiques d'un établissement à un autre. Nous avons donc décidé de proposer un protocole permettant de positionner les différents éléments de la prophylaxie antitétanique et, en particulier, le Tétanos Quick Stick qui est un test de détection rapide des anticorps antitétaniques.

D'après l'Organisation Mondiale de la Santé, le tétanos touche 1 000 000 personnes et en tue 500 000 chaque année dans le monde [(1)]. Dans les pays développés, la prévalence de cette maladie a fortement diminué depuis les années 50. Mais le tétanos existe encore en France puisque quelques dizaines de cas sont déclarés tous les ans. En effet, 28 cas ont été déclarés en 2001 puis 17 en 2002, 30 en 2003 et 25 en 2004 [(2)]. Entre 2005 et 2007, 41 cas de tétanos ont été notifiés : 13 patients sont décédés *(létalité de 32%)* [(3)].
Le tétanos est pourtant une maladie qui peut être facilement prévenue grâce à des moyens prophylactiques très efficaces que sont la vaccination et l'injection d'immunoglobulines. Il est donc nécessaire que les médecins, et en particulier les urgentistes, prennent correctement en charge le risque tétanigène. En effet, ceux-ci sont fréquemment confrontés à cette prise en charge puisque plus de 1 million de patients se rendent chaque année dans les services d'urgences français pour des plaies [(4)].



Clostridium tetani (Gr:x1000). D'après Atlas de bactériologie/Bayer Pharma.

TÉTANOS ET PROPHYLAXIE

QUELQUES RAPPELS SUR LE TÉTANOS :
Le tétanos est une infection aiguë due aux exotoxines d'un bacille nommé *Clostridium tetani*. Ce bacille à Gram positif est anaérobie strict et a la capacité de sporuler ce qui lui permet d'être très résistant dans le milieu extérieur. Les spores sont ubiquitaires : elles sont notamment retrouvées sur le sol et dans les déjections animales et humaines [(5)].
Lorsque les spores pénètrent dans l'organisme à travers une plaie cutanée et que les conditions d'anaérobiose sont réunies, le processus de germination se met en route et aboutit à la forme végétative toxique du bacille tétanique [(1)]. Ce bacille sécrète deux toxines : la tétanolysine et la tétanospasmine. Cette dernière est neurotrope et diffuse vers son site d'action au niveau du système nerveux central grâce à un transport rétrograde le long des nerfs. Elle est ainsi responsable des symptômes cliniques qui apparaissent après une période d'incubation de 3 à 21 jours en moyenne.
La maladie peut se présenter globalement sous quatre formes : généralisée, localisée *(touche une région proche de la plaie)*, céphalique *(atteinte des nerfs crâniens)* ou néonatale. Dans les pays en voie de développement, la forme néonatale est une cause importante de mortalité chez les enfants. Par contre, elle n'existe quasiment plus dans les pays développés où la forme généralisée est la plus fréquente puisqu'elle représente 80% des cas. Cette forme généralisée évolue selon trois phases [(5)] :
- ***phase d'incubation*** de 8 jours en moyenne,
- ***phase d'invasion.*** C'est la période séparant le premier symptôme de la généralisation des contractures. Elle dure en moyenne 48h. Le premier symptôme classique est le trismus *(contracture bilatérale et douloureuse des masséters)*.
- ***phase d'état.*** Cette phase est caractérisée par trois symptômes : contractures généralisées *(avec la classique attitude en opisthotonos)*, spasmes réflexes et dysautonomie.

L'évolution peut être favorable en 4 à 6 semaines sans complications mais dans 6 à 20% des cas, les malades souffrent de séquelles motrices et dans 20% des cas, les patients décèdent [(6)].

> *« la ténospasmine est une toxine neurotrope»*

LES MOYENS PROPHYLACTIQUES :
Deux grands moyens de prophylaxie permettent de prévenir le tétanos : la vaccination et l'utilisation d'immunoglobulines.

Prophylaxie en cas de plaie			
Type de blessure	Patient non immunisé	Patient totalement immunisé Délai depuis le dernier rappel	
		5 à 10 ans	Plus de 10 ans
Mineure, propre	Commencer ou compléter la vaccination : anatoxine tétanique 0,5 ml	Pas d'injection	anatoxine tétanique 0,5 ml
Majeure, propre ou tétanigène	Dans un bras immunoglobuline tétanique humaine 250 UI Dans l'autre bras, anatoxine tétanique* 0,5 ml	Anatoxine tétanique 0,5 ml	Dans un bras immunoglobuline tétanique humaine 250 UI Dans l'autre bras, anatoxine tétanique* 0,5 ml
Tétanigène, débridement retardé ou incomplet, > 20 heures, poids > 80 kg	Dans un bras, immunoglobuline tétanique humaine 500 UI Dans l'autre bras, anatoxine tétanique* 0,5 ml Antibiothérapie	Anatoxine tétanique 0,5 ml Antibiothérapie	Dans un bras, immunoglobuline tétanique humaine 500 UI Dans l'autre bras, anatoxine tétanique* 0,5 ml Antibiothérapie

Tableau 1 : Recommandations de la Haute Autorité de Santé (2) Recommandations de la DGS.
* Mise à jour selon le calendrier vaccinal.

Key Words

Tetarus, tetanus prophylaxis, tetanigeric wound, protoco

Abstract

Management of tetanus risk in emergency departments : on the need for a protocol.

Official guidelines relating to the management of tetanus risk during wounds treatment in emergency departments are not very precise. Recent studies have shown that this management seems very heterogeneous from one hospital to another. The tetanus vaccination coverage of the population is imperfect and the evidence of such vaccination is difficult to obtain, there exists no widely accepted definition of a tetanigenic wound. Furthermore, the use of tetanus antibodies detection tests is not widespread and the conditions of use of such tests are not identical from one institution to another. We have therefore decided to propose a protocol to situate the different elements of tetanus prophylaxis and, in particular, Tetanus Quick Stick which is a test for early detection of tetanus antibodies.

La vaccination antitétanique :

Le vaccin antitétanique *(VAT)* est produit en traitant une préparation de toxines tétaniques par du formaldéhyde ce qui les transforme en anatoxines. Ces anatoxines sont immunogènes, mais ne sont pas toxiques : elles permettent donc de stimuler le système immunitaire du sujet à vacciner sans produire d'effets délétères [2].

Le VAT est obligatoire pour les enfants de moins de 18 mois depuis la loi du 24 novembre 1940 [2]. Le schéma vaccinal n'est pas le même chez l'adulte et chez l'enfant. Dans le cas d'un enfant, il correspond à 3 injections intramusculaires *(IM)* espacées de 1 mois à partir de l'âge de 2 mois puis à un rappel 12 mois après puis à un rappel tous les 5 ans jusqu'à l'âge adulte et tous les 10 ans ensuite. Dans le cas d'un adulte, la primo-vaccination correspond à 2 injections IM espacées de 1 mois et à un rappel 1 an après. Les rappels doivent ensuite être faits tous les 10 ans [7].

Le VAT est très efficace puisqu'il permet d'obtenir une protection du patient de presque 100% [5]. Malheureusement, l'état vaccinal de la population française n'est pas très bon. En effet, d'après l'InVS, seuls 62,3% de la population adulte est couverte contre le tétanos [8]. Ce chiffre est en accord avec celui trouvé dans un service d'urgences belge [9]. D'autre part, l'observation de la proportion de personnes protégées en fonction de l'âge et du sexe montre que les sujets âgés sont moins bien protégés que les sujets jeunes et que les femmes sont moins bien protégées que les hommes. Ainsi, moins de 50% des femmes de plus de plus de 60 ans sont protégées contre le tétanos [10]. La couverture vaccinale varie aussi en fonction du niveau d'étude et de la région de résidence [8].

Les 41 cas de tétanos déclarés étaient non ou mal vaccinés [3].

Les immunoglubulines antitétaniques :

Le deuxième grand moyen de prophylaxie contre le tétanos correspond aux immunoglobulines humaines antitétaniques. Elles sont issues du fractionnement du plasma obtenu lors de don de sang total ou de plasmaphérèse, elles ont à ce titre le statut de médicaments dérivés du sang et font l'objet d'une traçabilité.

© Clap Image.

Elles sont commercialisées sous le nom de Gammatétanos® par le laboratoire LFB Biomédicaments et permettent d'assurer une protection passive du sujet non vacciné en attendant que la vaccination engendre des anticorps protecteurs. En effet, elles permettent d'avoir un taux protecteur d'anticorps en 2 à 3 jours avec une demi-vie de 3 semaines alors que la vaccination ne commence à protéger qu'à partir du 14 à 28 jours. Elles doivent être administrées le plus tôt possible pour s'opposer à l'action de la toxine avant que celle-ci ne se fixe de manière définitive sur le système nerveux central.

Ainsi, ces immunoglobulines sont principalement indiquées dans la prophylaxie du tétanos en cas de plaie chez les sujets dont la vaccination est incomplète, trop ancienne ou inconnue. La dose à administrer est de 250 UI mais elle doit être doublée en cas de plaie avec débridement retardé ou si la plaie a eu lieu plus de 24h auparavant ou pour des adultes de plus de 80 kg [11, 12].

RECOMMANDATIONS OFFICIELLES ET DIFFICULTÉS D'APPLICATION

Les recommandations officielles :

Les recommandations officielles de la Direction Générale de la Santé sont présentées dans le tableau 1. Elles indiquent que la prise en charge du risque tétanigène doit se faire selon le statut vaccinal du sujet et selon le type de blessure.

Mais tous les hôpitaux n'utilisent pas ces recommandations. Certains ont par exemple fait le choix

d'utiliser les recommandations de l'American College of Emergency Physicians, d'autres ont mis en place leur propre protocole.... [1, 13, 14]

Problème concernant la connaissance du statut vaccinal du patient et intérêt du Tétanos Quick Stick :

Le premier problème empêchant l'application correcte de ces recommandations est la difficulté à connaître le statut du patient. En effet, une étude a montré que moins de 2% des blessés accueillis aux urgences pouvaient fournir une preuve écrite de leur situation vaccinale et que l'interrogatoire était peu fiable [1, 15].

Pour connaître rapidement le statut vaccinal d'un sujet, des tests rapides tels que le Tétanos Quick Stick [TQS] ont été récemment commercialisés. Il s'agit de tests basés sur le principe de l'immunochromatographie dont la fiabilité et les intérêts à la fois économiques et médicaux ont été prouvés par diverses études [1, 4, 15, 16, 17, 18]. Il parait donc pertinent de faire apparaître un tel outil dans les protocoles destinés aux services d'urgence. Malgré son coût d'achat, son utilisation associée à un protocole peut ne pas entraîner de surcoût [19].

Plaies à l'origine de cas de tétanos avérés

- Plaie avec choc septique
- Prise charge > 6H
- Intervention chirurgicale gynécologique ou digestive
- Plaie souillée et/ou sale et/ou nécrotique et/ou en contact avec la terre, le sol ou des matières fécales
- Plaie chronique
- Plaie causée par des végétaux
- Plaie avec corps étranger
- Plaie causée par des animaux
- Plaie causée par des aiguilles ou objets perforants
- Plaie causée par des activités extérieures ou de bricolage
- Plaie causée par un accident (de la voie publique, traumatisme, chutes...)
- Gelure, brûlure, gangrène...

Tableau 2.

Problème concernant la détermination du niveau de risque de la plaie :

Le second obstacle empêchant l'application des recommandations officielles est la détermination du *« type de blessure »*. Les recommandations officielles font la distinction entre les plaies *« mineure, propre »*, les plaies *« majeure, propre ou tétanigène »* et les plaies *« tétanigène, débridement retardé ou incomplet »*. Mais elles ne donnent pas de précisions pour permettre de catégoriser les plaies.

L'enquête InVs montre que chez les 41 cas de tétanos déclarés entre 2005-2007, 68% présentaient des blessures, 10% des plaies chroniques et pour 22% la porte d'entrée n'avait pas été identifiée.

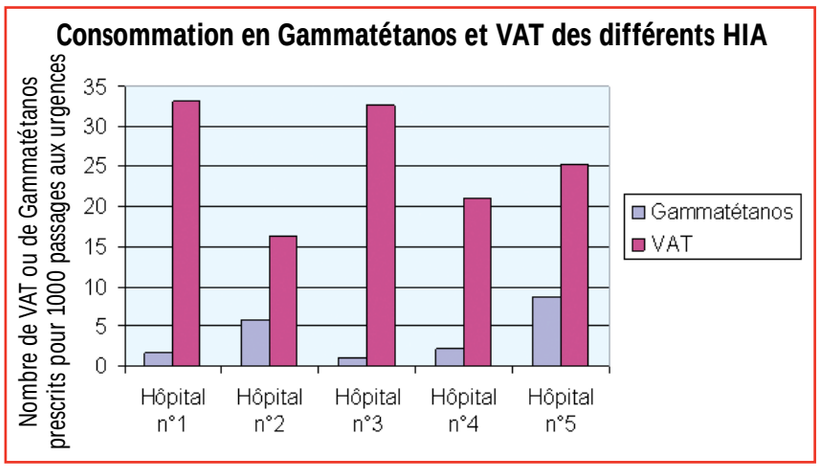


Figure 1.

© Clap Image.

De plus, la définition du caractère tétanigène d'une plaie ne fait pas l'objet d'un consensus univoque [20]. Les plaies listées dans le tableau 2 sont à l'origine des cas publiés de tétanos avérés :

HÉTÉROGÉNÉITÉ DE LA PRISE EN CHARGE DU RISQUE TÉTANIGÈNE

Des études américaines et anglaises ont démontré que les urgentistes connaissaient plutôt mal les recommandations concernant la prise en charge du risque tétanigène et qu'ils les suivaient peu [21, 22]. En France, des études ont montré que la consommation d'immunoglobulines était très variables d'un hôpital à l'autre [13, 16, 23]. Ainsi, la prise en charge du risque tétanigène semble être différente d'un hôpital à l'autre.

Pour avoir une idée de cette hétérogénéité dans les neuf hôpitaux militaires français, nous avons décidé d'envoyer un questionnaire aux pharmacies de ces hôpitaux pour connaître, en particulier, le nombre de passage aux urgences dans leurs hôpitaux et le nombre de Gammatétanos® et de VAT consommés en 2006. Nous considérons que la proportion de plaies parmi les passages aux urgences dans ces différents hôpitaux ainsi que les types de plaies sont semblables d'un hôpital à l'autre. Ainsi, nous rapportons les consommations de VAT et de Gammatétanos® à 1000 passages aux urgences afin de rendre les résultats comparables.

La figure 1 présente les consommations en Gammatétanos® et en VAT rapportés à 1000 passages

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Arbre décisionnel proposé pour la prise en charge de plaie aux urgences

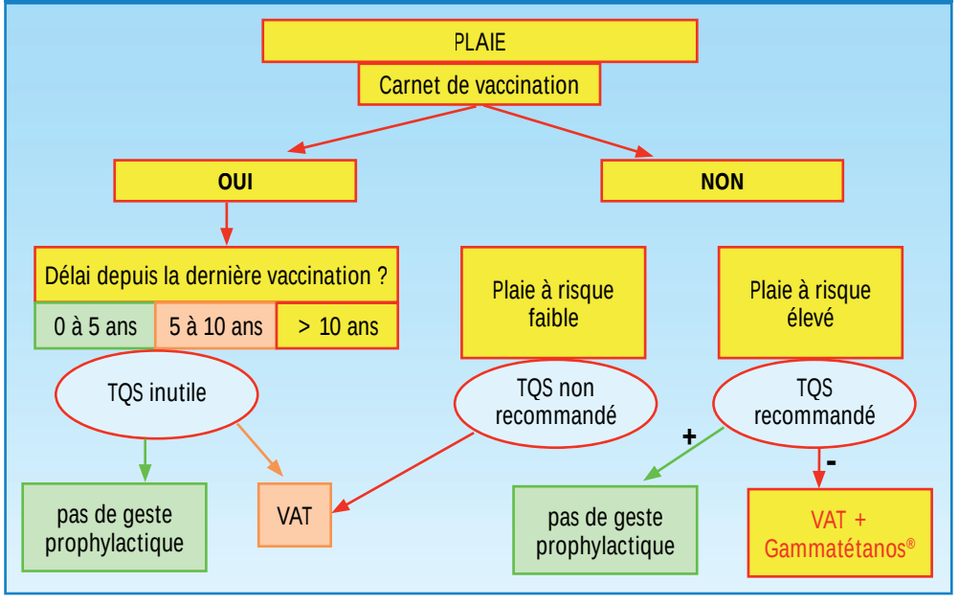

Figure 2.

aux urgences pour les cinq hôpitaux qui ont répondu à notre questionnaire. De façon surprenante, les consommations en Gammatétanos® ne sont pas corrélées aux consommations en VAT. En effet, l'hôpital n°1 consomme 33 VAT et 1 Gammatétanos® alors que l'hôpital n°5 consomme 25 VAT et 9 Gammatétanos® pour 1000 passages aux urgences.
Or, parmi les patients présentant des plaies et n'étant pas protégés contre le tétanos, c'est-à-dire parmi ceux qui doivent être vaccinés, la proportion de plaies devant être traitées avec des immunoglobulines devrait être assez constante.
Ainsi, la prise en charge du risque tétanigène semble être hétérogène dans les services d'urgences des hôpitaux militaires. Par conséquent, il est nécessaire de mettre en place un protocole permettant de situer clairement les différents éléments de prophylaxie.

« prise en charge hétérogène du risque »

PROPOSITION D'UN PROTOCOLE

Le protocole que nous proposons reprend les recommandations de DGS et incite à utiliser le TQS, en particulier lorsque le risque tétanigène de la plaie est élevé et lorsque la vaccination est absente ou incomplète ou inconnue. La figure 2 représente l'arbre décisionnel que nous avons élaboré.
Mais certains points de ce protocole restent critiques. Tout d'abord, il s'agit de déterminer précisément les caractéristiques d'une plaie à risque tétanigène élevé. Comme il n'existe pas à l'heure actuelle de consensus sur ce point, chaque service doit s'entendre sur une liste de plaies considérées comme *« à risque tétanigène élevé »*. Cette liste doit faire partie du protocole.
D'autre part, l'utilisation du TQS fait débat. Alors que certains hôpitaux ne l'utilise toujours pas, d'autres ont choisi de faire un TQS à chaque fois qu'un patient présente une plaie et ne peut pas justifier son statut vaccinal par des documents *(98% des cas)*. Ainsi, ils réalisent le TQS avant même d'évaluer le risque tétanigène de la plaie. Cela revient à considérer que toute plaie est à risque, en effet la liste des plaies proposées est large car provenant des plaies identifiées lors des cas de tétanos publiés. Sans être aussi extrémistes, nous proposons l'utilisation du TQS en cas de plaie à risque tétanigène élevé associée à une vaccination absente ou incomplète. Ceci permettra tout particulièrement de savoir s'il est nécessaire d'injecter des immunoglobulines ou non.
Par ailleurs, le TQS peut être proposé dans le cas d'une plaie à risque faible associée à une vaccination absente ou inconnue. Mais nous ne le recommandons pas puisqu'il ne permettra que d'éviter l'injection de VAT. Or cette injection ne présente pas de véritables risques et, au contraire, elle pourrait permettre de sensibiliser le patient sur la nécessité d'un bon suivi vaccinal tout en lui remettant un document prouvant sa vaccination.
Le troisième point critique de ce protocole correspond au VAT. En effet, lors de la primo-vaccination

La Rédaction

Ce que nous savions :
Le tétanos reste une pathologie d'actualité, même si sa prévalence a diminué. Sa prévention est pourtant facile grâce à la vaccination et à l'apport d'immunoglobulines, en cas de plaie à risque.

Ce que cet article nous apporte :
La prise en charge du risque tétanigène est très hétérogène d'un hôpital à l'autre. Les auteurs proposent d'établir une liste de plaies « à risque tétanigène élevé », qui entrerait dans le cadre d'un protocole reprenant les recommandations de la Direction Générale de la Santé. Ceci permettrait d'optimiser la sécurité de la prise en charge des patients aux urgences.

des patients, le VAT est quasiment toujours associé à d'autres vaccins tels que les vaccins contre la diphtérie et la poliomyélite. Si le patient n'est pas à jour pour le VAT, il y a une très grande probabilité pour qu'il ne soit pas non plus à jour pour d'autres vaccins. Il serait donc peut-être plus judicieux d'injecter à ces patients un vaccin dTPolio *(Diphtérie-Tétanos-Poliomyélite)* par exemple. D'autre part, la vaccination n'étant pas une urgence, certains hôpitaux ont fait le choix de ne pas vacciner [15]. Ils envoient ainsi le patient chez son médecin traitant avec une ordonnance. Ce médecin peut alors réaliser le suivi vaccinal complet du patient.

CONCLUSION

Le tétanos est une maladie qui fait toujours quelques dizaines de victimes en France chaque année. La prophylaxie antitétanique repose sur la vaccination de l'ensemble de la population et sur l'injection d'immunoglobulines aux patients se présentant à l'hôpital pour des plaies et n'étant pas à jour de VAT. Les recommandations officielles concernant la prise en charge des plaies aux urgences ne sont pas assez précises pour permettre de les appliquer sans ambiguïté. Ainsi, des études ont montré qu'il existait une hétérogénéité dans cette prise en charge entre différents hôpitaux. Nous avons montré que cette hétérogénéité existait aussi dans les hôpitaux militaires et pour remédier à ce problème nous avons proposé un protocole permettant d'harmoniser les pratiques. La recherche de la preuve écrite sur le carnet de vaccination constitue la pierre angulaire de notre protocole. Néanmoins, il n'existe pas de consensus sur la définition d'une plaie à risque tétanigène élevé, laissant la place à une appréciation personnelle.

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There is no conflict of interest to declare

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Chantal Khalil

Dialyse péritonéale à domicile, une prise en charge pluridisciplinaire

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Résumé

La dialyse péritonéale à domicile consiste à traiter hors centre lourd des patients atteints d'insuffisance rénale chronique nécessitant un traitement par dialyse, le patient réalisant lui- même, après formation, sa dialyse. C'est un type de dialyse qui a pour objectif d'éliminer les déchets tels que l'urée, la créatinine, l'excès de potassium ou de liquide que les reins ne parviennent pas ou plus à épurer du plasma sanguin. Ce traitement concerne 120 000 patients par an dans le monde, soit 14 % de la population des personnes dialysées. Cette maladie coûteuse, altérant la qualité de vie, exige une prise en charge pluridisciplinaire afin d'améliorer le suivi du patient et la rationalisation des coûts de santé. Une fois acquise par la personne, la technique peut être effectuée à domicile, préservant son autonomie et permettant de conserver une activité professionnelle le jour, les séances étant faites la nuit.

Mots clés

Dialyse péritonéale, insuffisance rénale, prise en charge pluridisciplinaire, modalités de traitement, infection péritonéale.

Dénuées de symptômes, les maladies des reins sont souvent identifiées trop tardivement. Un tiers des insuffisants rénaux ne sont diagnostiqués qu'au stade terminal, au moment où la dialyse s'impose en urgence.
Quand l'insuffisance rénale devient sévère, il est indispensable de programmer sans tarder un traitement pour suppléer le rein : la dialyse ou la greffe rénale. Compte-tenu de la pénurie d'organes disponibles, plus de 30 000 patients sont traités par dialyse.

L'insuffisance rénale, un danger silencieux !

Bien qu'indispensables à notre vie, nos reins brillent souvent par leur discrétion, en dehors des terribles coliques néphrétiques. Pourtant, ces organes sont d'incroyables travailleurs : ils filtrent 180 litres de notre sang par jour et éliminent les déchets de l'organisme. Mais un danger silencieux rode : l'insuffisance rénale. Longtemps dénuée de symptômes, cette réduction progressive du fonctionnement des reins correspond à une destruction progressive et irrémédiable des canaux (les néphrons) qui constituent le rein. Elle apparaît lorsqu'il ne reste plus qu'un tiers de ces canaux en état de marche. Elle peut être détectée par un bilan biologique (dosage de la créatinine) à l'occasion d'un diabète, d'une infection urinaire, d'une hypertension artérielle, de calculs rénaux, d'albuminurie, d'une infection sévère, d'une anémie inexpliquée, d'intoxications médicamenteuses, etc. Ces manifestations peuvent être la cause ou la conséquence d'une insuffisance rénale. Arrivée au stade terminal (90 % des néphrons inefficaces), cette maladie nécessite un traitement urgent (dialyse ou greffe), sinon c'est le coma et la mort en quelques jours(1)…

Une prise en charge trop tardive

Outre les conséquences physiques et psychologiques, cette maladie dont on aurait pu freiner l'évolution a un coût élevé. A titre d'exemple, un patient dialysé traité en centre d'auto dialyse coûte au minimum 35 000 euros par an, tarif qui est supérieur pour les patients traités en centre lourd, public ou privé. Au total, le coût des soins correspond à 2 % des dépenses de l'Assurance maladie(1).

Modalités de traitement (2)

Mode de traitement	Hémodialyse	Dialyse péritonéale
Durée	En moyenne, 4 à 6 heures tous les deux jours, auxquelles il faut ajouter les temps de transport pour se rendre sur le lieu de traitement et les temps de préparation. Au total, les malades consacrent près d'un tiers de leur temps d'éveil à leur traitement…	Quatre échanges par jour (durée globale de 40 à 60 mn chacun) Ou Une séance journalière unique d'une durée de 8h à 10 h, avec la possibilité de la réaliser la nuit, pendant le sommeil, en restant « branché » au générateur.
Avantages	Cette technique permet d'oublier la maladie entre chaque séance, sans avoir de matériel médical à domicile.	Cette technique permet d'avoir la maîtrise de son traitement, de moduler ses horaires de dialyse, de ne pas dépendre d'un centre de dialyse.

Inconvénients	Cette technique nécessite de passer 4 à 6 heures plusieurs fois par semaine dans un centre de dialyse. Certaines personnes supportent mal d'avoir l'avant bras déformé par une fistule car au fil du temps le vaisseau se dilate et n'est pas discret au regard des autres.	Cette technique nécessite chez soi un emplacement suffisant pour stocker le matériel et les solutés nécessaires. Certaines personnes supportent mal l'idée d'un tuyau qui sort de leur ventre mais qui est nécessaire pour cette technique. Le cathéter implanté dans la cavité péritonéale est une porte d'entrée propice aux germes. Les nombreuses manipulations sur le cathéter lors des phases d'infusion et de drainage augmentent le risque d'infection locale ou générale.
Contre-indications	Il existe des difficultés pour la réaliser si le patient présente un mauvais état vasculaire ou si la pression artérielle est très basse du fait d'un état cardiaque perturbé. Ce qui peut être relativement fréquent chez certains patients diabétiques ou obèses.	Les contre-indications « relatives » sont la conséquence d'une obésité majeure, d'antécédents d'interventions chirurgicales abdominales importantes, de hernies abdominales récidivantes, d'une insuffisance respiratoire sévère ou au cours des premières semaines qui suivent un échec de transplantation.

Dialyse péritonéale

La dialyse péritonéale est l'épuration du sang en utilisant le péritoine comme filtre. Elle est indiquée principalement dans le cadre du traitement de l'insuffisance rénale terminale ou aussi de l'insuffisance cardiaque et de l'hypertension réfractaire aux traitements médicamenteux. Une fois acquise par la personne, la technique peut être effectuée à domicile. Un bel exemple de la qualité de vie permise par cette méthode est donné par la traversée de l'Atlantique en solitaire, puis le tour du monde en solitaire, effectués à la voile en 2009-2010 par un navigateur expérimenté, monsieur Jean-Louis Clémendot, sur son ketch Harmattan(3).

1) Principe

La dialyse péritonéale utilise deux principes mis en action grâce à la propriété physiologique de perméabilité du péritoine : l'ultrafiltration de liquide et l'épuration des déchets par diffusion.

Pour effectuer la dialyse, un liquide artificiel, le dialysat, est introduit dans la cavité péritonéale. Ce liquide sera ensuite évacué après un temps de contact déterminé.

Dans la cavité, le dialysat est en contact direct avec les deux feuillets du péritoine. L'échange se faisant grâce à la perméabilité de la membrane, le dialysat va capter les éléments à éliminer présents dans le plasma sanguin ainsi que le surplus d'eau.

Les dialysats les plus couramment utilisés sont composés d'une solution tampon (du lactate ou du bicarbonate) à pH acide (5,2 - 5,5) ou physiologique (7,4) à laquelle sont ajoutés des électrolytes (sodium, calcium, magnésium, chlore) et un agent osmotique (du glucose ou de l'icodextrine). Les électrolytes et l'agent osmotique jouent chacun un rôle dans le mécanisme d'échange, selon leurs propriétés physico-chimiques respectives :

- les déchets du métabolisme (tels que l'urée ou la créatinine) ou autres électrolytes en surabondance que le rein n'élimine plus ou insuffisamment via l'appareil urinaire et les urines, vont s'extraire du plasma sanguin par diffusion des éléments vers le dialysat dont les taux de concentration de ces mêmes éléments sont moindres ;
- l'excédent d'eau, que le rein élimine normalement pour la régulation du volume plasmatique, va être attiré par osmolarité ; les grosses molécules de glucose agissent comme des éponges et attirent l'eau du plasma sanguin : ce processus est nommé ultrafiltration ; le taux d'ultrafiltration varie en fonction de la concentration du dialysat en glucose : plus la solution sera concentrée en sucre, plus l'eau présente dans le corps sera captée par le dialysat(3).

Abstract

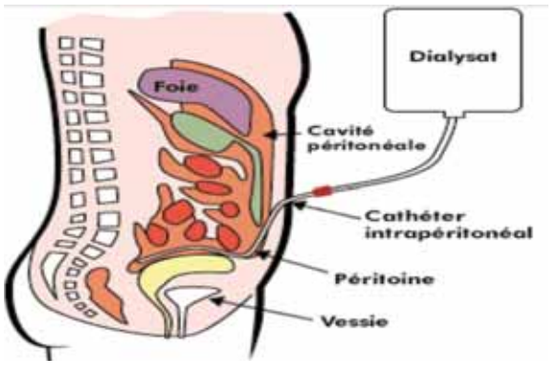
The home peritoneal dialysis is to treat off-center heavy patients with chronic kidney failure requiring dialysis treatment, the patient realizing, after training, its own dialysis. This type of dialysis has to eliminate wastes such as urea, creatinine, and the excess of potassium or liquid that kidneys couldn't eliminate anymore to purify the blood plasma. This treatment concern 120,000 patients a year worldwide, which mean 14% of dialyzed people's population.
This costly disease, altering the quality of life, required multidisciplinary support to improve the prognosis of the patient and the rationalization of health care costs. Once acquired by the person, the technique can be performed at his home, preserving its autonomy while keeping his professional activity the day, having made the sessions at night.

Key Words

Peritoneal dialysis, Kidney failure, multidisciplinary care, treatment modalities, peritoneal infection.

2) Protocole et méthodes

La dialyse péritonéale peut être dispensée selon deux modes opératoires : la *dialyse péritonéale continue ambulatoire* (DPCA) et la dialyse péritonéale automatisée (DPA).



Les deux méthodes utilisent le même procédé : l'introduction d'un dialysat dans la cavité péritonéale via un cathéter (cathéter de Tenckhoff) implanté chirurgicalement au niveau du cul-de-sac de Douglas s'abouchant à la peau dans la zone ombilicale.

Une fois le liquide infusé, débute la phase de stase. Pendant cette phase, les échanges ont lieu entre le plasma sanguin et le dialysat au sein de la cavité durant un laps de temps défini. À l'issue de cette période, le liquide contenant les déchets est drainé par le cathéter.

Le cycle infusion/stase/drainage peut être répété plusieurs fois en vingt quatre heures en fonction des indications médicales et des méthodes utilisées(4).

A/ Méthode par DPCA

C'est la technique « à l'ancienne « : la dialyse s'effectue entièrement manuellement. On connecte un système appelé « double poche « au cathéter du patient. Il est constitué d'une poche de drainage et d'une poche remplie de deux litres de solution de dialyse.

Dans un premier temps, la cavité péritonéale est vidangée dans la poche vide. Il suffit de la disposer sur le sol, le patient reste assis sur une chaise, et le liquide s'écoule sous la seule action de la gravité. Il faut environ 15 à 30 minutes pour vidanger complètement la cavité. Ensuite, on injecte le dialysat en accrochant la poche pleine à un pied à perfusion, et le liquide s'écoule, par gravité, dans le péritoine. L'injection va généralement un peu plus vite, et dure de 10 à 20 minutes. La plupart du temps on effectue quatre échanges dans la journée (matin, midi, soir, coucher), et on garde un liquide à plus longue durée d'action pour la nuit. Il est aussi possible, au moyen d'un appareillage très simple, de réaliser un échange supplémentaire la nuit, sans avoir à se réveiller.

B/ Méthode par DPA

C'est une technique plus moderne, qui est généralement choisie car elle est un peu moins contraignante. Dans ce cas, tous les échanges ont lieu la nuit. Il faut installer suffisamment de poches sur un appareil appelé « cycleur «, de les relier entre elles, puis de connecter le cathéter à l'appareillage. La machine prend ensuite en charge les vidanges et les injections.

Dans ce cas les temps de stagnation sont plus courts, ce qu'on compense en augmentant le nombre des échanges (6 à 8 échanges avec des volumes de 2 à 3 litres, en général le volume total doit se situer entre 15 et 20 litres). La durée totale du traitement (sans compter le temps nécessaire à l'installation et la désinstallation des poches) s'échelonne de 8 à 10 heures. La journée, on restera selon les cas le ventre vide, ou bien rempli d'un liquide à plus longue durée d'action(5).

3- Les solutions de dialyse

Une quantité variable de solution (généralement entre deux et trois litres) est introduite par le biais du cathéter dans la cavité péritonéale. Les échanges s'effectuent alors comme en hémodialyse : les déchets métaboliques, l'eau et les ions en excès migrent du sang vers la solution de dialyse au travers de la membrane péritonéale. Le liquide est assez rapidement saturé, c'est pourquoi il faut le renouveler plusieurs fois par jour.

Il existe principalement deux techniques, qui sont a priori équivalentes, mais dans la pratique on choisira celle qui est la plus adaptée à chaque patient. En effet, à la différence de l'hémodialyse, on ne peut pas choisir la qualité de la membrane échangeuse, et tous les péritoines ne fonctionnent pas de manière équivalente.

On distingue :

- **Le péritoine « hyper-perméable »** : il laisse passer très facilement les molécules que l'on souhaite éliminer ; dans ce cas le liquide est très rapidement saturé, et il faut le renouveler très souvent ; on applique dans ce cas la technique de **(DPA);**
- **Le péritoine « hypo-perméable » :** il laisse moins facilement passer les molécules, et le liquide

doit rester dans la cavité péritonéale plus longtemps pour épurer le sang suffisamment ; dans ce cas on choisit la **(DPCA)**.

- Enfin **le péritoine intermédiaire, dit « normo-perméable »**, qui se contente aussi bien de l'une ou de l'autre technique(5).

Education du patient

L'éducation du patient est une phase essentielle dans la prise en charge, voire un moment privilégié. Ces moments d'échanges (transfert de connaissances et de compétences) sur la technique choisie, voire sur les risques et les complications pouvant être affrontés, vont permettre au patient ou à une tierce personne (infirmier(e) extra-hospitalier(e) ou d'institution) de prendre en charge tout ou partie des soins inhérents à la dialyse(6).

L'installation à domicile

Avant d'installer un patient à domicile, l'équipe soignante, parfois aidée de l'assistant(e) social(e) vérifie que le logement du patient est adapté à un traitement à domicile, grâce à une visite à domicile, ou après interrogation du patient ou de ses proches. Plusieurs points sont à vérifier:

- **L'hygiène et la taille du logement.**
  Les manipulations avant et après la séance de dialyse impliquent une hygiène irréprochable afin d'éviter au maximum toute infection.
  La dialyse à domicile, nécessitant parfois l'installation d'un cycleur et d'un traitement de l'eau, il n'est parfois pas possible de la réaliser dans certains appartements.
- **L'existence d'un point d'eau facilement accessible**
  Pour des raisons d'hygiène, il est préférable d'avoir un robinet mitigeur. Dans certains cas, un déminéralisateur d'eau sera installé.
- **L'installation électrique**
  Il faut aussi vérifier la présence d'une prise de terre pour le branchement du cycleur et un UPS en cas de panne d'électricité(6).

Le jour du retour à domicile, un ou des membres de l'équipe soignante vont accompagner le patient afin de :

- L'aider à gérer sa première dialyse dans son cadre de vie habituel,
- Lui prodiguer des conseils de rangement,
- Contrôler l'adéquation entre le matériel prescrit et le matériel livré,
- Et surtout diminuer son angoisse et celle de son entourage(6).

Surveillance de la dialyse péritonéale

A) Infection

Surveillance du cathéter:

o Soins du cathéter

Il est indispensable de suivre des règles d'hygiène très strictes : douche régulière, sous-vêtements propres, lavage/séchage des mains et port de masque avant chaque manipulation et vérification de la propreté de l'orifice, que l'on peut - mais ce n'est pas obligatoire - protéger par un pansement.

o Surveillance de l'émergence du cathéter :

- Inflammation, rougeur.
- Pas de croûte.
- Eczéma de contact.
- Infection avec le risque d'atteinte du trajet sous cutanée (tunélite) avec écoulement, inflammation, d'où hospitalisation pour traitement afin d'éviter la péritonite.

B) Surveillance de l'hydratation.

- En dialyse péritonéale il est possible de garder le plus longtemps possible l'ultrafiltration (excès d'eau) = diurèse résiduelle.
- Surveillance du poids.
- Se peser tous les jours dans les mêmes conditions avec au départ un poids de base.
- Si augmentation du poids rapidement = risque d'œdème aigu du poumon (OAP) d'où

consultation (deux à trois kilos en 15 jours).
- Si diminution du poids rapidement = consultation car asthénie, déshydratation (un kilo par semaine).
  o La suralimentation entraîne des taux sanguins trop élevés en urée et phosphore.
  o La sous-alimentation entraîne une dénutrition et peut aboutir à une hospitalisation prolongée.
- Surveillance des oedèmes.
- Réajuster le traitement = poche hypertonique, Lasilix*(Diurétique).
- Bilan entrée sortie en dialyse péritonéale = peser les poches à l'entrée et à la sortie(7).

NB : **La solution de dialyse est très sucrée**, ça crée un apport de glucose très important, donc le régime est assez strict au niveau des sucreries, et le **risque de diabète** s'en trouve accru.

C) Surveillance de l'aspect du dialysat.

- Il doit être limpide (jaune, jaune orangé).
- Si liquide trouble = infection du péritoine, appeler immédiatement l'hôpital pour hospitalisation (apporter la poche pour analyse = + 100 éléments/mm3 = infusion).
- Si douleurs abdominales de type colique = suspicion d'une péritonite.
- Traitement : Si l'infection est détectée suffisamment tôt, et si l'agent infectieux n'a pas migré dans le sang (septicémie), le problème se règle en quelques jours d'hospitalisation : lavages péritonéaux à l'aide de solutions de dialyse auxquelles on ajoute un antibiotique puissant, et une ou deux perfusions d'antibiotiques(8).

Tous ces critères de surveillance seront donnés par les infirmières de l'équipe qui poursuit le patient à domicile, ainsi que le rythme de visites de surveillance auquel il devra se plier lorsqu'il sera bien entrainé, en général une visite par mois.

Que faire en cas de problème ?

La prise en charge d'un patient en dialyse à domicile repose sur un partenariat solide : celui d'un patient et de sa famille, de son antenne médicale et de la structure de dialyse hors centre. Le médecin et les infirmiers (es) responsables de la formation restent leurs interlocuteurs privilégiés.

Une permanence téléphonique 24 heures sur 24 heures est prévue pour répondre aux appels des patients à domicile. Une liste de contacts-clé est remise au patient à son retour à domicile lui permettant de savoir comment réagir en cas de problème et vers qui se tourner. Il devra toujours avoir à portée de main la liste des numéros de téléphones à contacter en urgence (technicien, médecin, infirmière)(8).

Affronter la maladie

La plupart des gens n'aiment pas le changement. L'insuffisance rénale est particulièrement stressante, malgré que le traitement par dialyse péritonéale conserve une certaine autonomie de vie. La manière dont le patient fait face à la maladie, émotionnellement et psychologiquement, aura un impact fondamental sur tous les aspects de sa vie, y compris sur son état physique. D'où, une prise en charge pluridisciplinaire s'avère indispensable afin d'accepter de vivre avec des reins défaillants et adapter un mode de vie en conséquence(9).

Conclusion

Les progrès réalisés dans le domaine de la dialyse péritonéale au cours des 20 dernières années ont été importants. Ils concernent notamment la compréhension des phénomènes de transfert à travers la membrane péritonéale, l'amélioration du matériel mis à disposition et les solutions disponibles. Il en résulte une diminution de la fréquence des complications, notamment des infections péritonéales.

Comme tout traitement médical, la méthode de dialyse par dialyse péritonéale à domicile présente avantages et inconvénients. Elle requiert une prise en charge pluridisciplinaire se reposant sur plusieurs interlocuteurs visant à obtenir la meilleure tolérance d'un point de vue médical, tenant compte du bien-être social de la personne.

Conflict of interest statement :
There is no conflict of interest to declare

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L'E.C.G. pour les nuls

Yannick GOTTWALLES

9- Entre clinique et électrique, mon cœur balance

Pour un clinicien, deux interrogations majeures persistent devant la lecture de tout ECG :
- y'a-t-il une ischémie que je ne vois pas ?
- existe-t-il des troubles qui peuvent me faire craindre pour la vie de mon patient à court terme ?

Ces questions restent d'actualité devant toute douleur thoracique et devant tout bilan des sacro-saints « malaises », et leurs réponses doivent permettre de diminuer la mortalité post-consultation.

ABSTRACT: ECG for the hopeless, Chapter 9

Torn between the clinical and the electrical
For a clinician, there are two major questions upon reading an ECG:
- Is there an ischemia that I can't see?
- Are there disorders that make me fear for the life of my patient on the short run?
Those questions remain valid when faced with any chest pain and when reading any result for the very famous « malaises » and their interpretation that must help reduce the post-consultation mortality.
We have seen since issue 1 of Med Emergency / Urgence, the importance of conducting an ECG while watching carefully the positioning of the electrodes, the standardized speed and amplitude of the line that is drawn and the characteristics of large syndromes. But even if the ECG line includes 12 derivations (ideally 18) that analyze 12 lines of different areas, however due to the three dimensional morphology of the cardiac cavities, there are some areas that remain none or ill-explored. Signs appear very less obviously. We shall talk about the indirect signs of ischemic cardiopathy and life threatening rate disorders.

Key Words

ECG, clinical symptoms, electrical signs

Nous avons vu, depuis le numéro 1 de Med Emergency / Urgence, l'importance de la réalisation d'un ECG dans des conditions données de bon emplacement des électrodes, de vitesse et d'amplitude de tracé standardisées, et les caractéristiques des grands syndromes. Mais même si le tracé ECG comporte 12 dérivations (ou 18 dans l'idéal) analysant 12 traces de surface différentes, du fait de la morphologie tridimensionnelle des cavités cardiaques, il persiste des zones peu ou pas exploitables. Les signes n'apparaissent dès lors plus aussi évidents. Nous allons aborder les signes indirects des cardiopathies ischémiques et des troubles du rythme menaçants.

Les SCA (voir aussi Med Emergency / Urgence No 8)
Le diagnostic des SCA ST+ est relativement aisé, hors bloc de branche gauche et stimulateur cardiaque. Cependant, près de 80% des SCA ne rentrent pas dans les ST+. Et si dans la majorité des SCA non ST+, un courant de lésion sous-endocardique (ou sous-décalage) est patent, il est des circonstances non rares où il convient de rechercher des signes mineurs de souffrance myocardique, mineurs dans leur expression, mais tout aussi significatif dans leur implication diagnostique afin de dépister les ECG suspects.

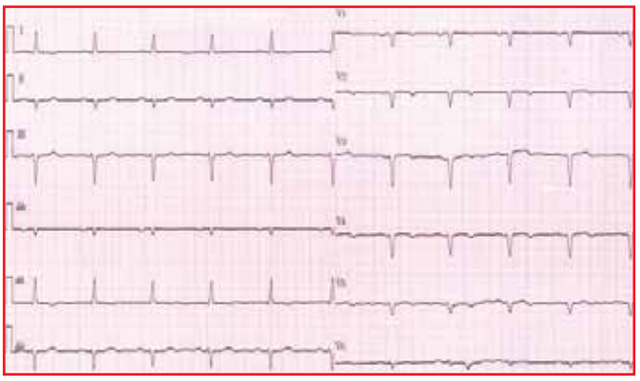
I. NORMALITÉ DE L'ECG (MED EMERGENCY / URGENCE NO 3)

Pour mémoire, la lecture d'un ECG normal c'est la recherche des items suivants :

Items	Traduction de l'item
Il existe une onde P devant chaque complexe QRS et chaque QRS est suivie d'une onde P	Le rythme est sinusal
L'espace PR est constant d'un cycle à l'autre et est inférieur à 1 grand carreau	Et régulier, sans BAV
Les QRS sont fins, sans aspect de M de V1 ou de W en V6, et tous sont égaux	Il n'y a pas de bloc de branche, ni d'extra-systole
Il n'y a pas d'onde Q	Il n'y a pas de séquelles d'infarctus
DI est positif	Il n'y a pas d'hémibloc postérieur gauche
DII est positif	Il n'y a pas d'hémibloc antérieur gauche
ST est iso-électrique (pas de sus-décalage dépassant 0,5 mm)	Il n'y a pas de courant de lésion
Toutes les ondes T sont positives (sauf aVR et V1)	Il n'y a pas d'ischémie

À ces éléments de base, s'ajoutent la progression harmonieuse de l'onde R en précordial, la proportionnalité de l'onde T par rapport à R, l'harmonie des segments ST, et l'absence d'évolutivité dans un temps restreint.

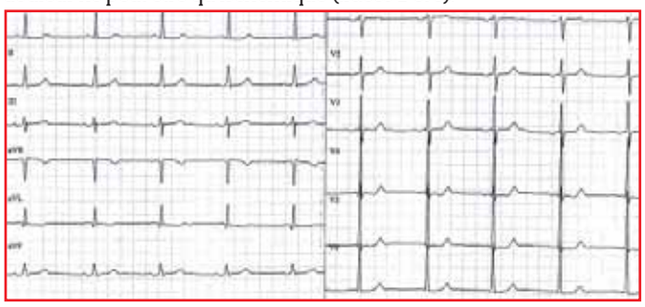
La progression harmonieuse de l'onde R en précordial a été expliquée dans Med Emergency / Urgence No 3. L'absence de cette progression se traduira par la présence d'un rabotage de l'onde R, parfois appelé trou électrique, qui dans sa forme ultime sera une onde Q (tracé 1).



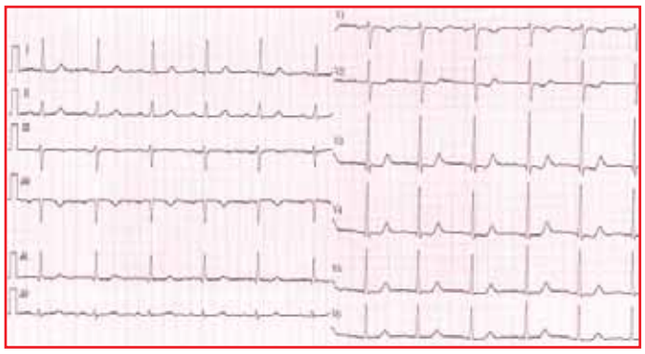
tracé 1 : RSR avec absence de montée de l'onde R en antérieur étendu ; il ne s'agit pas d'une onde Q, car il existe une micro-onde R dès V3 ; à noter également un hémibloc antérieur gauche (DII négatif) et une ischémie sous-épicardique en antérieur et latéral haut (V2 à V5 et DI/aVL)

L'onde T (Med Emergency / Urgence No 5), dans les conditions normales, doit être proportionnelle en taille à l'onde R. Habituellement, l'amplitude de T correspond au tiers de l'amplitude de R, règle très grossière, mais aisément applicable.
Enfin, il existe une harmonie entre les QRS et le segment ST, pour les

dérivations où l'onde R prédomine : si l'un est positif, l'autre est positif, et inversement. Les segments ST sont alors dits « harmonieux ».
Pour terminer, il est parfois utile de rappeler que la pathologie coronaire est une maladie évolutive, qui se signale par une symptomatologie douloureuse, et dont l'expression clinique est variable en fonction des degrés de sténose, des efforts, des besoins en oxygène, de la physiopathologie, ... En conséquence, l'absence de modifications évolutives du tracé ECG alors qu'il y a une évolution de la clinique, est très peu en faveur d'une origine coronaire. Et l'inverse est également vrai : une évolution électrique parallèle à une évolution clinique de la douleur ne peut être qu'ischémique (tracés 2 et 3).



tracé 2 : patient de 57 ans, hypertendu connu, tracé de repos, de référence : RSR avec hypertrophie ventriculaire

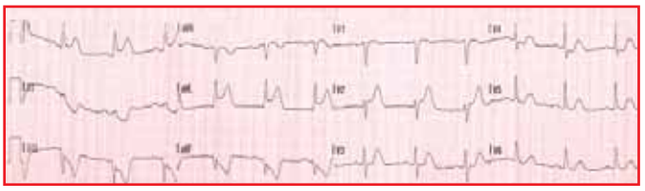


tracé 3 : même patient présentant une symptomatologie douloureuse angineuse, avec apparition d'un courant de lésion sous-endocardique en antérieur ; dès V2, le sous-décalage se majore, et devient plus net en V3 ; à l'arrêt de la douleur, le tracé de repos réapparaît

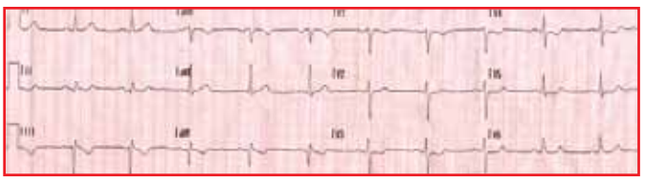
II. POURQUOI ET COMMENT DÉPISTER LES ECG SUSPECTS ?

La recherche et le diagnostic des tracés ECG suspects permettent une prise de décision rapide, thérapeutique ou d'orientation. Si la prise en charge des SCA ST+ est bien codifiée, celle des SCA non ST+ l'est beaucoup moins, avec de surcroît une surmortalité nette dans ce groupe. Or, il est établi que tout traitement est d'autant plus efficace qu'il est mis précocement en place, ce qui est d'autant plus vrai dans une pathologie évolutive. Il est donc essentiel de diagnostiquer précocement ces tableaux afin de diminuer la mortalité et la morbidité des SCA non ST+.
Si un doute existe entre la clinique et le premier tracé, si vous avec un doute sur un signe ECG qui ne vous paraît pas parlant, il convient de répéter l'enregistrement ECG 15 minutes plus tard. Cette stratégie sera bien plus payante que la réalisation d'un cycle de troponines, qui est souvent une « aberration pseudo-scientifique » car :

- Pour que les troponines augmentent, il faut une ischémie (donc des douleurs) d'au moins 20 voire 30 minutes ;
- Une ascension ne sera notée qu'après au minimum 4 heures de temps ;
- Un premier dosage négatif à la 3e heure d'une douleur sera contrôlé 3 à 4 heures plus tard, soit une perte de temps d'au moins 6 heures au total ;
- Et en 6 heures, combien de troubles de rythme ou de mort subite peut-on faire ?
- En se rappelant qu'un trouble ECG apparaît après moins de 2 minutes d'ischémie coronaire, le choix est vite fait.



tracé 4 : patient présentant une douleur thoracique intermittente depuis 24 heures, avec quelques accès de moins de 5 minutes – onde de Pardee en latéral haut avec miroir inférieur, sur sténose serrée de la circonflexe



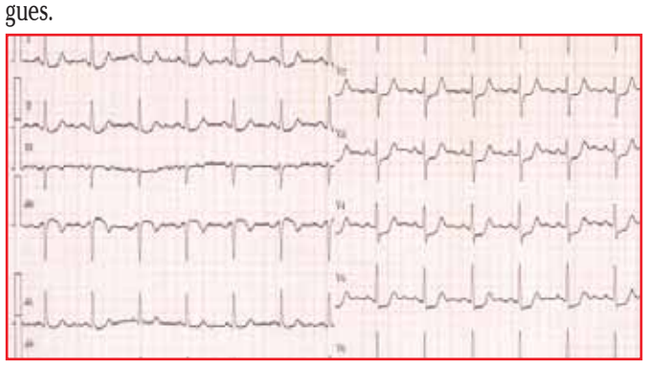
tracé 5 : même patient, 6 minutes après l'enregistrement du tracé 4 : les troubles en V3-V4 sont les plus marquants, et vont disparaître dans les 5 minutes ; pour un tracé fait ultérieurement, celui-ci est normal, et les troponines seront non dosables ; pourtant, en bouchant sa circonflexe, le patient peut parfaitement fibriller et décéder

1. Le segment ST (Med Emergency / Urgence No 5)

Si un sus-décalage est aisément reconnaissable, il en est de même pour les miroirs (ST sous-décalé dans des dérivations opposées à celles où le ST est sus-décalé). Un infarctus inférieur aura un miroir antérieur et inversement, un sous-décalage en V1-V2 est un miroir du postérieur, ... (Med Emergency / Urgence No 3) (tracé 4).

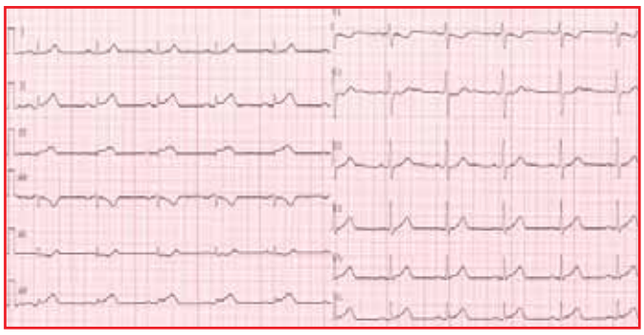
Toujours dans les miroirs, la repolarisation de V1 doit être grossièrement l'inverse de celle de V6 (sauf pace-maker). V6 est le miroir naturel de V1 (tracé 5). En cas de discordance, il y a anomalie

Un courant de lésion sous-endocardique (tracé 6) est toujours un signe de gravité, d'autant plus qu'il se situe en V3, qui par définition ne peut pas avoir de segment ST sous-décalé physiologique. En présence isolément du tracé 5, nous pouvons affirmer sur V3 qu'il s'agit d'une cardiopathie ischémique, et dans un contexte de douleurs thoraciques, il serait criminel de ne pas confier ce patient aux cardiologues.



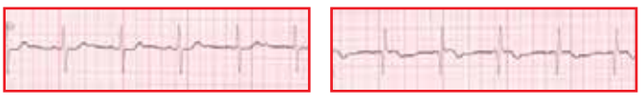
tracé 6 : important courant de lésion en antérieur étendu = prise en charge immédiate et agressive

La localisation du sous-décalage est également contributive. Le ventricule droit est axé sur DIII, à 120° (schéma Emergency / Urgence No 1). Le sus-décalage d'un SCA ST+ inférieur sera donc maximal en DIII, et l'inverse est vrai : le miroir de 120° est -60°, soit aVL, donc le sous-décalage sera maximal en aVL. Cette subtilité est majeure, car si le sus-décalage de DIII est supérieur à celui de DII, ou à l'inverse le sous-décalage de aVL est supérieur à celui de DI, l'origine ischémique est prouvée (tracé 7).



tracé 7 : infarctus inférieur ; les signes maximaux sont en aVL (sous-décalage de ST quasi-inexistant en DI), et en DIII (sus-décalage maximal en DIII, même si l'onde T est plus ample en DII et aVF, c'est le sus-décalage qui prime) ; à noter le miroir en V2

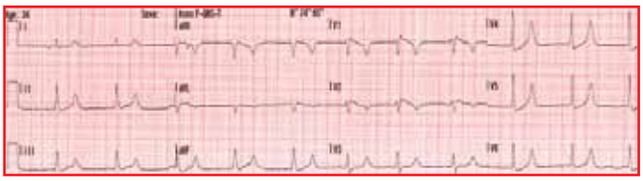
Dernier élément, n'hésitez pas à retourner votre tracé, et à le regarder par transparence, à l'envers et à l'endroit : l'œil visualise beaucoup plus facilement un sus-décalage qu'un sous-décalage ; en retournant le tracé, on découvrira des sous-décalages qui ne nous avaient pas frappés (tracé 8).



Tracé 8 : à gauche, V2 du tracé 7, à droite, V2 du tracé 7 mais vu à l'envers ; autant le sous-décalage de V2 pouvait être considéré par certains comme une trémulation liée à la respiration de la ligne de base, autant en inversant le tracé une onde de Pardee débutante suivie d'une ischémie sous-épicardique est frappante

2. L'onde T *(Med Emergency / Urgence No 5)*

Nous avons évoqué la proportionnalité de l'onde T par rapport à l'onde R. Une autre règle de normalité est celle où R doit écraser T : dans les dérivations où l'onde R prédomine, T est toujours inférieure à R. Dans le cas contraire, il s'agit soit d'une ischémie, soit d'une hyperkaliémie, elles sont toutes deux avec une onde T pointue, mais l'onde T ischémique est symétrique, avec une base large, l'onde T d'hyperkaliémie étant pointue à base fine (tracé 9).



tracé 9 : patient de 34 ans, en insuffisance rénale aiguë, avec hyperkaliémie à 7,8 mEq/l : hormis le bloc sino-auriculaire, l'onde T est pointue, de la taille des QRS, à base fine, avec une cassure nette dans le segment ST

Une trop grande onde T par rapport aux QRS est très suspecte d'ischémie, d'autant plus qu'elle est observée en V3-V4 (tracé 9).
Dernier point concernant l'onde T, sur un tracé normal, cette onde se fond avec la fin du complexe et le segment ST. S'il existe une cassure nette, ou si la détermination du début de l'onde T est possible, il s'agit d'une onde T anormale (tracé 9).

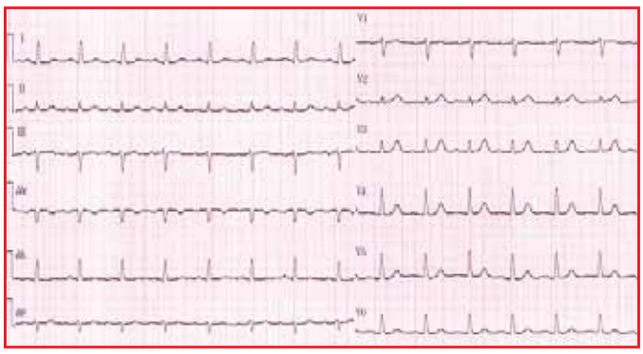
3. L'onde Q

Image de séquelle de nécrose tissulaire myocardique, elle est également évolutive dans le temps, pouvant régresser voire disparaître à distance d'un épisode. Son apparition témoigne d'un délai long d'anoxie cellulaire. Elle reste encore parfois le témoin d'un défaut de prise en charge où le temps n'a pas été donné à une procédure de revascularisation, mais à l'attente de troponines positives, ou pire au contrôle d'une troponine déjà positive.

4. Les QRS fragmentés

En regard de zones ischémiées et nécrosées, une altération de la dépolarisation existe (Med Emergency / Urgence No 3). Ce phénomène implique une hétérogénéité des potentiels d'action, traduite par des QRS fragmentés. Ils sont nommés fQRS. Ils peuvent être évoqués en l'absence de bloc de branche, sur des QRS restant fins, et lorsqu'ils sont visibles sur 2 dérivations contiguës. Classiquement on visualise une onde R', un crochetage de S, puis une onde R".

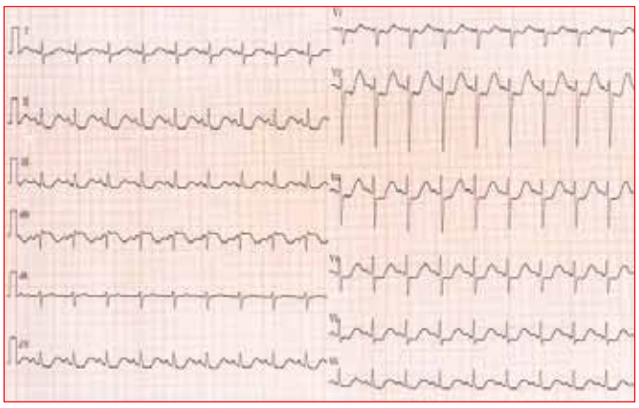
Ils sont le signe d'une cicatrice d'infarctus, avec les complications potentielles notamment rythmiques des cardiopathies ischémiques (tracé 10).



tracé 10 : patient adressé pour troponines positives : si un tracé avait été fait, on note T trop ample par rapport au QRS en V2-V3, T symétrique dans les mêmes dérivations, et un crochetage des QRS (rSr's' en V1 – rsr's' en V2)

5. aVR

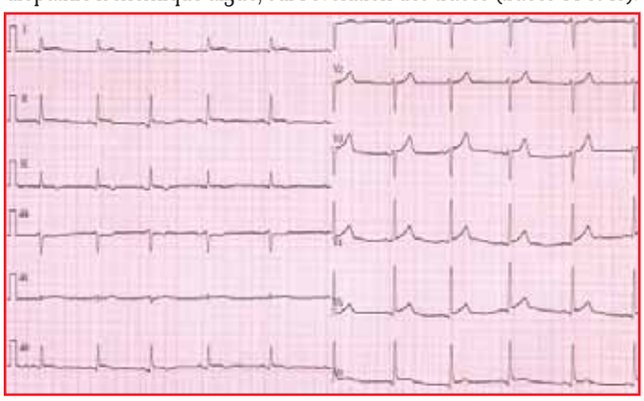
aVR tourne le dos à tout le ventricule gauche, seule cavité réellement efficace au niveau musculaire. Sa positivité est d'une grande aide, car annonce l'origine ventriculaire du complexe. La présence d'un sus-décalage supérieur à 1 mm est le signe d'une lésion serrée du tronc commun ou de l'IVA proximale, d'autant plus qu'il est accompagné d'un courant sous-endocardique en antérieur étendu. C'est un signe de gravité majeur, les patients pouvant présenter une mort subite par occlusion du tronc (tracé 11).



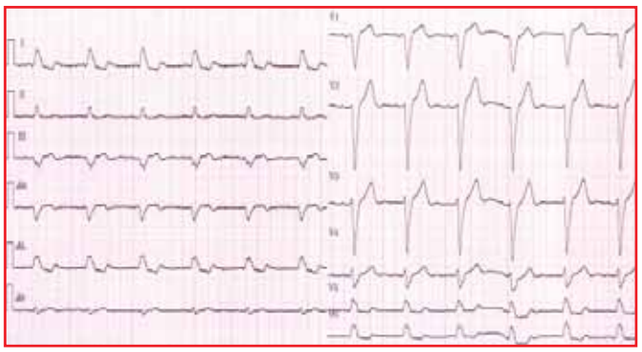
tracé 11 : important courant de lésion sous-endocardique en antérieur et inférieur, avec un aspect net d'onde de Pardee en aVR ; ce patient va mourir dans les minutes qui suivent ou se mettre en état de choc cardiogénique : il existe une sub-occlusion du tronc commun, avec une occlusion ancienne de la coronaire droite, ce qui signifie qu'il vit sur un fil ; les signes en aVR sont moins nets sur le tracé 6, mais sont présents également

6. Diagnostics différentiels d'un sus-décalage du segment ST (Med Emergency / Urgence No7)

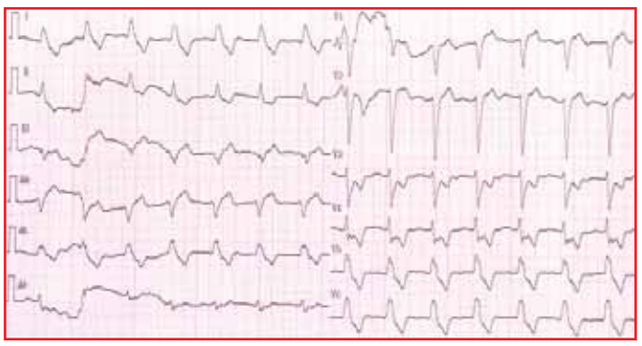
L'hypertrophie ventriculaire gauche (HVG) est parfois de diagnostic difficile. Il existe des critères d'amplitude, le plus connu étant l'indice de Sokolow (somme de R en V5 et de S en V1 ≥ 35 mm) ; un tracé où les complexes se chevauchent est très évocateur d'une HVG. Cependant les anomalies de la repolarisation qu'elle entraîne, peuvent être évocatrices d'autres pathologies : s 'il y a une discordance entre les QRS et les ondes T, s'ils sont soit positifs/négatifs, soit négatifs/positifs, cette discordance est très évocatrice d'HVG. Le tracé 2 enregistre une HVG avec des complexes se chevauchant en l'absence d'erreur d'amplitude, et une discordance entre QRS et segment ST (même si les ondes T restent harmonieuses) (voir aussi tracé 16).
La repolarisation précoce survient préférentiellement chez un sujet jeune, masculin, avec un aspect de ST qui reste concave vers le haut, sans miroir, et sans évolutivité. Cela correspond en fait à la non-possibilité de descente de l'onde R pour rejoindre la ligne iso-électrique, avant d'entamer le segment ST.
Dans une péricardite, il n'y a pas de miroir, et il peut exister un sous-décalage du segment PQ qui va majorer l'impression du sus-décalage de ST. Les troubles prédominent dans les dérivations inférieures (tracé 12).
Un bloc de branche gauche (Med Emergency / Urgence No 3) aura un aspect d'onde de Pardee survenant sur un complexe large (tracé 13).
Mais malgré ce bloc, il est parfois très aisé de faire le diagnostic de cardiopathie ischémique aiguë, sur l'évolution des tracés (tracés 14 et 15).



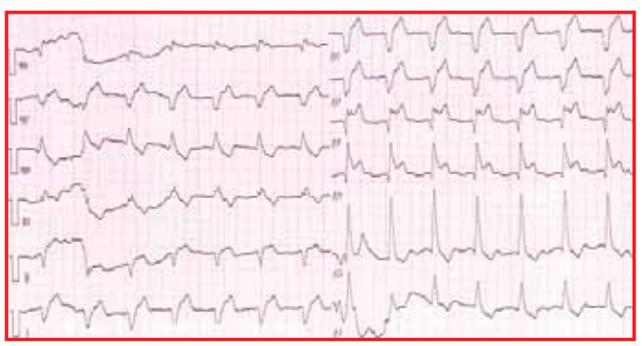
tracé 12 : les signes maximaux sont en DII-aVF en non en DIII, maximaux aussi en DI et non aVL, il y a un sus-décalage sans miroir, bien que l'ensemble soit territorialisé en inféro-latéral : il s'agit typiquement d'une péricardite



tracé 13 : bloc de branche gauche typique



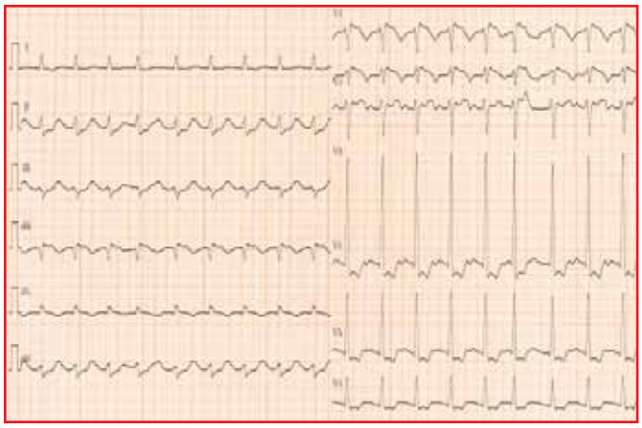
tracé 14 : même patient qu'en 13, avec douleurs thoraciques constrictives : apparition d'un courant de lésion en antérieur étendu



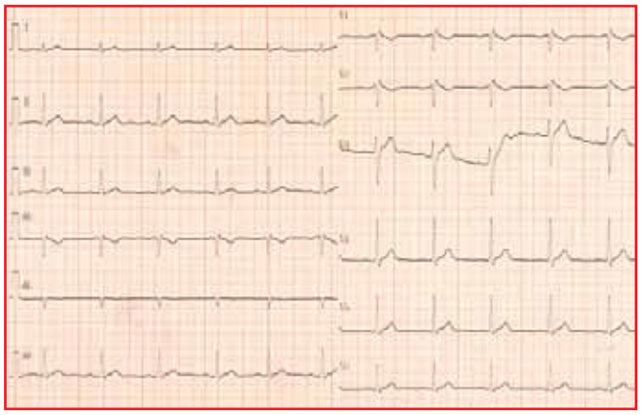
tracé 15 : identique au 14, mais regardé par transparence et à l'envers : l'image en cathédrale en V3 et V4 de l'onde de Pardee est criante, malgré le bloc de branche gauche

Un anévrysme ventriculaire séquellaire d'un infarctus révèlera une onde de Pardee sans miroir, stable dans le temps (Med Emergency / Urgence No 7).

Un syndrome de Brugada-Brugada correspond à un aspect de bloc de branche droit avec sus-décalage de ST en V1-V2. Il existe un haut risque rythmique, avec régulièrement une histoire familiale de mort subite ou de perte de connaissance – voir Syndrome de Brugada – A.TOHMY - Med Emergency / Urgence No 1 (tracés 16 et 17).



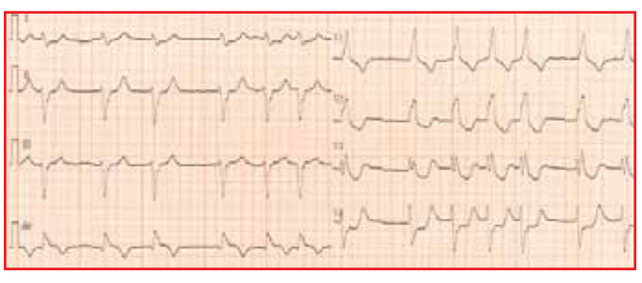
tracé 16 : patient de 23 ans, admis pour perte de connaissance, 2e épisode ; il y a une HVG nette (amplitude, discordance des QRS/T) mais surtout un aspect de BBD avec sus-décalage de ST en V1-V2, typique d'un syndrome de Brugada de type 1



\tracé 17 : patient de 19 ans adressé pour perte de connaissance évoluant dans une histoire de palpitations et de lipothymies depuis 1 an ; l'aspect est moins net, mais reste très évocateur avec un sus-décalage de ST en V1-V2, nous sommes dans un Brugada de type 2

7. Diagnostics différentiels du ST

Les variations physiologiques : il faut se souvenir que l'ECG enregistre sur un même individu 12 dérivations différentes dont la repolarisation peut varier durant le nycthémère, et que ces dérivations ont aussi une variabilité interhumaine nette.
Une cupule digitalique, une hypothermie ou un trouble métabolique (notamment une hypokaliémie) sont responsables de troubles de la repolarisation de ce type (Med Emergency / Urgence No 7) (tracé 18).



tracé 18 : patient de 72 ans, adressé pour hypothermie à 32,8°C, sous digitaliques au long cours du fait de son ACFA, avec BBD et HBAG ; on distingue clairement l'onde J d'Osborne en V4

III. LES ANOMALIES POUVANT FAIRE CRAINDRE UN TROUBLE DU RYTHME POTENTIELLEMENT LÉTAL

Dans la pratique courante, deux cas de figure peuvent se rencontrer.

- Le premier est celui d'un ECG réalisé à titre systématique, chez un sujet apparemment sain (visite d'aptitude par exemple). Les anomalies de l'ECG qui doivent être recherchées sont le syndrome de Brugada, le syndrome de Wolff Parkinson White (WPW), les extra-systoles ventriculaires (ESV), la dysplasie arythmogène du ventricule droit (DAVD), le QT long (tracés 19 et 20), et la fibrillation auriculaire (FA). Les ondes à connaître sont essentielles (figure 1).
- La seconde catégorie sont les ECG réalisés dans le cadre de symptômes, de points d'appel cardiaques ou rythmiques, ou d'un bilan d'une pathologie. Les anomalies à rechercher sont toutes les cardiopathies (ischémiques, valvulaires, post-HTA, ...), en plus des précédentes. Dans ces conditions, une HVG, des troubles du rythme, des anomalies des QRS seront notés.

Chez un sujet dit sain, en routine, il faut donc éliminer

Syndromes	Signes électriques	Références Med Emergency
Brugada	BBD avec ST+ en V1-V2	n° 1 / 7
WPW	Aspect de PR court, onde delta sur la phase ascendante du QRS, visible en fonction du territoire analysé et de sa localisation	
ESV	« Tardive, monomorphe, isolée » versus « précoce, polymorphe, multiple, couplée »	n° 6
DAVD	T négatives en précordial droit et onde epsilon	
QT long	QT mesuré ≥ 0,40	n° 5
FA	Pas d'onde P, rythme irrégulier	n° 6

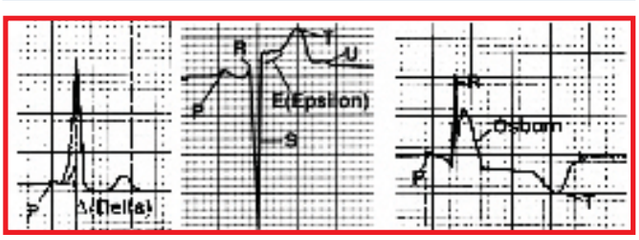
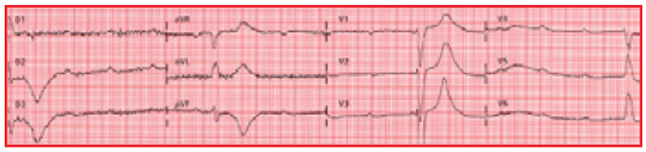
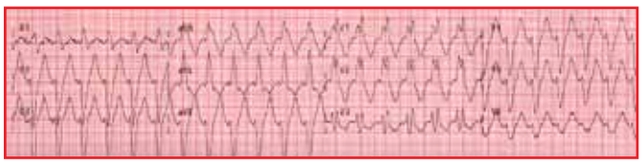


Fig 1 : Les ondes à connaître
l'onde delta = pré-excitation
l'onde epsilon = DAVD
l'onde J d'Osborne = hypothermie



tracé 19 : patient en BAV III, sous amiodarone et béta-bloquants ; les ondes P sont bien visibles en V4, le QRS est large, mais surtout le QT est monstrueux



tracé 20 : même patient qu'en 19, avec survenue d'une ESV dans la période réfractaire du QRS, engendrant un passage en tachycardie ventriculaire à 156 cycles/mn ; le diagnostic de TV est porté sur le tracé comparatif précédent ; en l'absence de celui-ci, il serait porté sur la tachycardie, la fréquence, et la positivité d'aVR, le BBD et l'HBAG n'étant pas contributifs au diagnostic de TV.

CE QU'IL FAUT RETENIR

1. Le premier signe d'un infarctus reste la douleur coronaire
2. Devant tout doute, il faut réitérer les tracés et les comparer
3. Le ST ischémique se caractérise par une évolutivité dans le temps, parallèle à l'évolution de la clinique
4. Le ST ischémique est maximal en DIII (ou aVL), et trouve son miroir
5. Un ST ischémique est totalement anormal en V3 (et V2)
6. R doit progresser, parallèlement à S qui régresse
7. T est proportionnelle à R et R écrase T
8. T pointue à base large = penser ischémie, T pointue à base fine = penser hyperkaliémie
9. En cas d'HVG, il y a une discordance entre les QRS et l'onde T
10. Un ECG doit permettre d'éliminer un Brugada, un WPW, des ESV menaçantes, une DAVD, un QT long et enfin une FA asymptomatique

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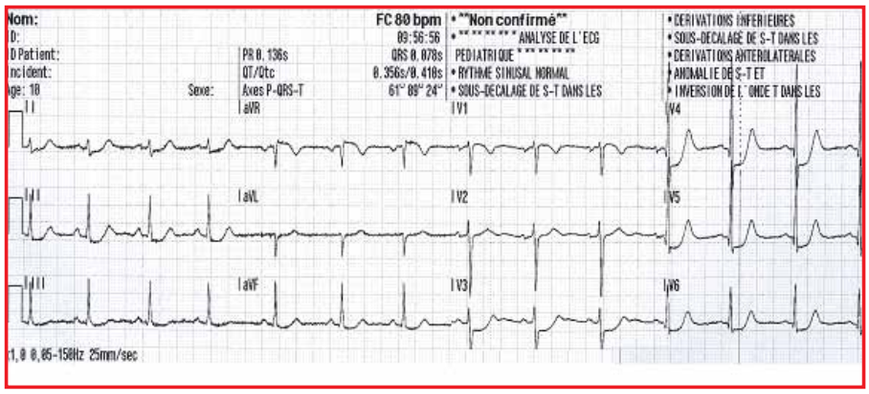
Conflict of interest statement :
There is no conflict of interest to declare

Un tracé pour s'entraîner



Interprétation selon « l'ECG pour les nuls »	Traduction classique
patient de 10 ans	patient de 10 ans
onde P visible, fréquence régulière, avec un QRS après chaque onde P et avant chaque autre P	rythme sinusal régulier
fréquence moyenne à 80 cycles/mn	à 80 cycles/mn
PR constant	pas de BAV du premier degré
les complexes sont fins mais amples dans les précordiales	attention, tracé d'un enfant de 10 ans => les critères d'amplitude ne sont pas valables
QRS fins, sans oreille de lapin en V1 ou V6	pas de bloc de branche
ST sous-décalé de V3 à V6, et en DI, DII, DIII et aVF	courant de lésion sous-endocardique étendu
aspect d'onde de Pardee isolé en aVR	
T positive dans toutes les dérivations sauf aVR et V1	pas d'ischémie
Conclusion : rythme sinusal régulier à 80 cycles / mn, sans troubles conductifs, avec SCA non ST+ chez un enfant de 10 ans – au niveau clinique, il s'agit d'un enfant ayant eu un point de côté puis une douleur thoracique au décours d'un footing – il existe une anomalie de naissance du tronc commun qui est comprimé par l'artère pulmonaire à l'effort, expliquant la symptomatologie	

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