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ANNALES 1er SEMESTRE 2017/ ANNALS 1st SEMESTER 2017

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CHIRURGIE CARDIAQUE / CARDIAC SURGERY

CONSTRICTIVE PERICARDITIS MIMICKING RESTRICTIVE CARDIOMYOPATHY IN AN ADULT, A CASE REPORT.

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Summary

Heart diastolic dysfunctions are dominated by two categories of etiologies: constrictive pericarditis and restrictive cardiomyopathy. Constrictive pericarditis clinical diagnosis may be difficult to establish; therefore, Doppler tissue imaging can be useful. We report a case of constrictive pericarditis in an adult living in Sub-Saharan Africa who has been treated successfully via surgery.

Keywords: Constrictive pericarditis, restrictive cardiomyopathy, adult.

Résumé

Les dysfonctions diastoliques sont dominées par deux types d'affections: les péricardites constrictives et les myocardiopathies restrictives. Cliniquement, le diagnostic différentiel peut s'avérer difficile à établir ; d'où l'intérêt du Doppler tissulaire permettant de le facilter ; de plus,dans certaines situations, le scanner thoracique, ou l'imagerie par résonance magnétique nucléaire peuvent être nécessaires.Nous rapportons le cas d'une péricardite constrictive chez un adulte, traitée chirurgicalement avec succès.

Mots clés : Péricardite constrictive, myocardiopathie restrictive, adulte.

Introduction

Constrictive pericarditis is a clinical feature characterized by an impaired cardiac diastolic function. Usually, it is difficult to make a distintion between a restrictive cardiomyopathy and a constrictive pericarditis.Since last decade, Doppler tissue imaging, especially the E' determination may clearly distinguish constrictive pericarditis from restrictive cardiomyopathy [1, 2] .We report a case of constrictive pericarditis mimicking restrictive cardiomyopathy in an adult patient who has been successfully treated by a pericardiectomy.

Case report

A 48-year-old man, who lived in Republic of Congo (Central Africa), was admitted to our unit for a right heart failure. His has a medical history of recurrent right heart failure in the past. He has been treated by Diuretics, vitamin K antagonist. He has no medical history of tuberculosis. Clinical examination revealed blood pressure at 128/80 mmHg, irregular heart rhythm, with mean cardiac rate at 89 bpm. Peripheral signs of right heart failure, spontaneous internal jugular veins dilatation, edema,and hepatomegaly were present. There was no cardiac murmur. Recent biological data were normal.Electrocardiogram, revealed an atrialfibrillation.Transthoracic echocardiography showed:bi atrial dilatation, inferior vena cava dilatation, and a small left ventricle , the ratio E/A of mitral valve was up to 1.5 with a short deceleration time, and preserved ejection fraction.

(figure1a). Chest computed tomography(figure 1b) and resonance magnetic imaging revealed veritable pericardium thickness and calcifications (figure 1c).After written consent, the patient is fully monitored in order to evaluate the hemodynamic impact of the pericardiectomy intraoperatively. We performed a median sternotomy, realized pericardiectomy from phrenic nerve to phrenic nerve (figure 2a), without cardiopulmonary bypass. Central venous pressure decreased from 24 to 11 mmHg. We have been totally liberated the heart, and restore its normal relaxation(figure 2b). Pathology revealed a pericardial fibrosis. No inotropic drugs were used in intensive care unit. Postoperative period was uneventful and the patient was discharged after six days of hospital stay.

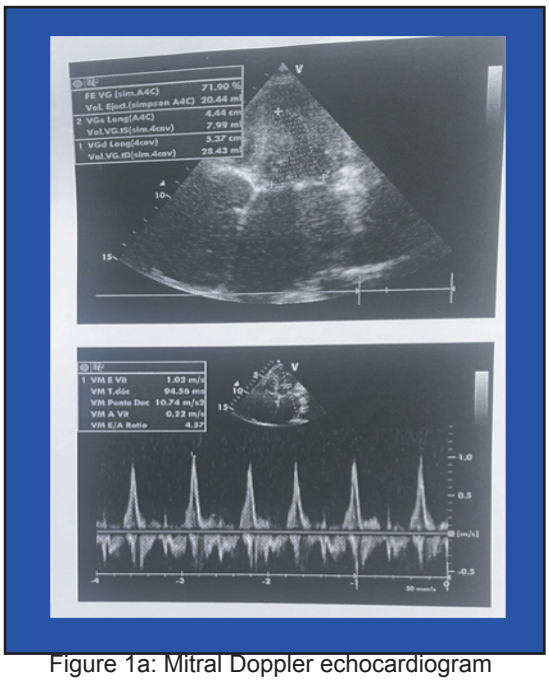

Figure 1a: Mitral Doppler echocardiogram
Restrictive filling (E/A>1.5) and short deceleration

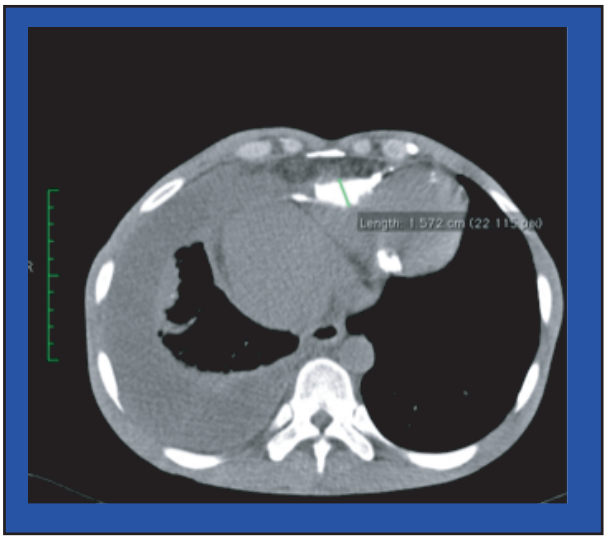

Figure 1b: Chest computed tomography, axial view:
Calcifications in atrio ventricular groove.

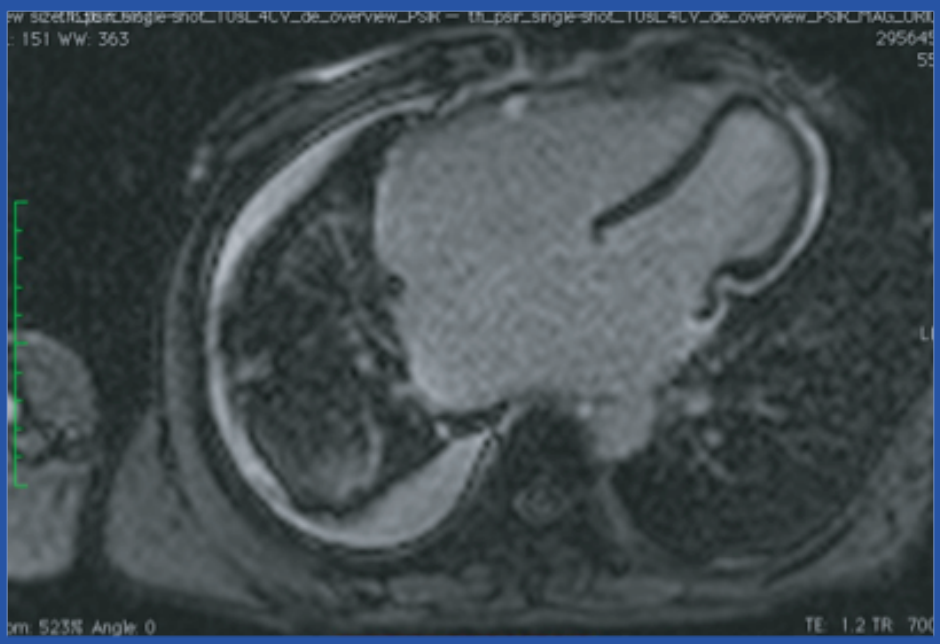

Figure 1c: Resonance magnetic imaging, after gadolinium injection:
Pericardial thickness and calcifications of the left ventricle.

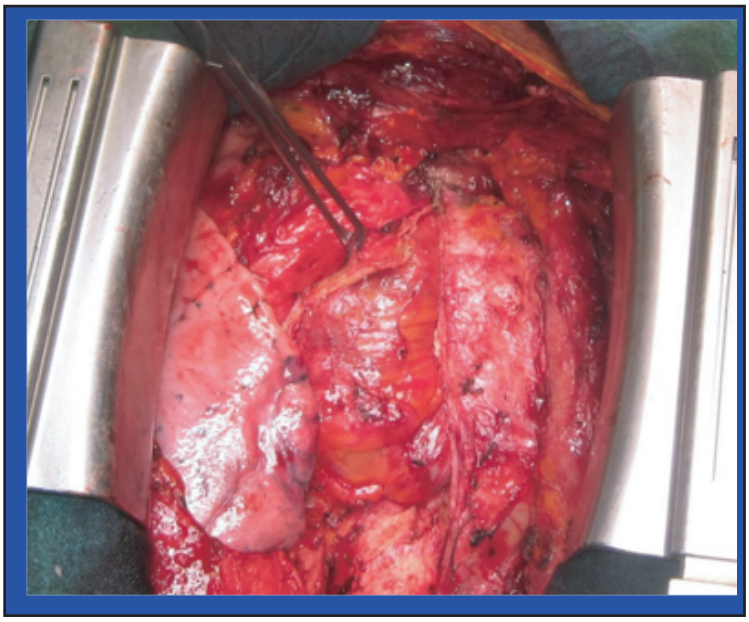

Figure 2a: Intraoperative view:
Anterior mediastinal dissection.

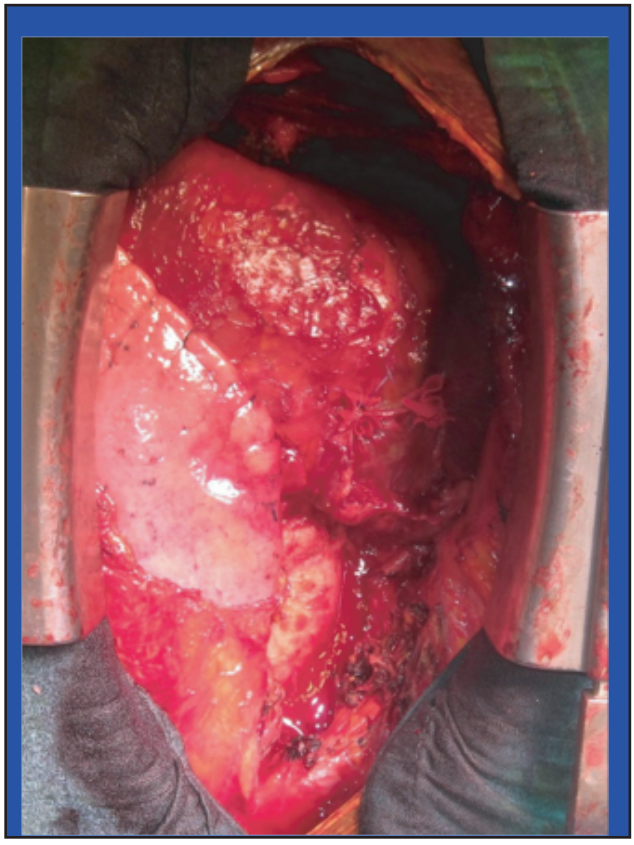

Figure 2b: Intraoperative view:
better diastolic relaxation of the heart after removal of the fibrotic parietal pericardium.

Discussion

Anomalies of diastolic cardiac function assemble constrictive pericarditis and restrictive cardiomyopathy Clinical features of these entities are often similar, dominated by right heart failure. Doppler tissue imaging has brought a distinctive sign: the “E’wave”, which may clearly distinguish constrictive pericarditis to restrictive cardiomyopathy [1-3]..E’ wave is normal or accelerated in constrictive pericarditis, but decelerated in restrictive cardiomyopathy.

The greatest problem in developing countries is the lack of complementary data as E’ wave in Doppler tissue imaging .Despite, the elevation of E/A mitral valve, short deceleration time in continuous wave Doppler; the key data scientifically accepted for differentiation between constrictive pericarditis and restrictive cardiomyopathy is the E’wave of Doppler tissue imaging. Generally, our surgical patients are at NYHA stage III-IV, with sometimes, hepatic and renal dysfunction .

Tuberculosis in developing countries represents most often the first aetiology of constrictive pericarditis while in developed countries, post cardiotomy and post radiotherapy represent the first etiologies [4].The long delay before surgery may be explained by the lack of imaging data in our context.

Most surgical teams in the world perform pericar- diectomy through a median sternotomy without cardiopulmonary bypass [5]. Median sternotomy provides a good heart access with the possibility of a total pericardiectomy and an entire heart relaxation.Median sternotomy offers better access than left postero-lateral thoracotomy. Access to right heart cavities may be difficult through left postero-lateral thoracotomy making a complete pericardiectomy uneasy to achieve.

Generally, cardiopulmonary bypass is used in the case of constrictive pericarditis with an uncontrolled haemorrhage, or in case of an intracardiac anomaly which requires total correction.

Conclusion

Constrictive pericarditis is one of the most frequent etiology of cardiac diastolic dysfunction Challenge for praticers in developing countries, is the lack of complementary Doppler tissue imaging data,which can establish a differentiation betwween constrictive pericarditis and restrictive cardiomyopathy;constrictive pericarditis may be successfully treated by pericardiectomy.

References

1.Jong-Won Ha, MD, PhD.,**Steve R**. **Ommen,** MD.,**Jamil Tajik A**, MD.,**Barnes, Marion E**., MSc., et al.Differentiation of Constrictive Pericarditis from Restrictive Cardiomyopathy Using Mitral Annular Velocity by Tissue Doppler Echocardiography. Am J Cardiol 2004; 94:316–319

2. **Butz Th** .,**Piper C**.,**Langer M**.,**Wie- Mer T**.,**Kottmann A,** et al. Diagnostic superiority of a combined assessment of the systolic and early diastolic mitral annular velocities by tissue Doppler imaging for the differentiation of restrictive cardiomyopathy from constrictive pericarditis.Clin Res Cardiol 2010;99:207–215

3. Avgerinos D, M.D., **Rabitnokov Y**, M.D.,**Worku B**, M.D.,**Neragi-Miandoab S**, M.D., et al. Fifteen-Year Experience and Outcomes of Pericardi\ectomy for Constrictive Pericarditis. J Card Surg 2014;29:434–438

4. **Mutyaba Arthur K**., MBChB,**Sarvesh- Balkaran** Robert **A**.,**Cloete R**.,et al .Constrictive pericarditis requiring pericardiectomy at Groote Schuur Hospital, Cape Town, South Africa: Causes and perioperative outcomes in the HIV era (1990-2012).J Thorac-CardiovascSurg 2014; 148:3058-65

5.**Sang Yoon Kim**, M.D., **Kwon Joong Na**., M.D.,**Kyung-Hwan Kim**., M.D., Ph.D. The Importance of Complete Pericardiectomy and the Role of the Apical Suction Device in Chronic Constrictive Pericarditis. Korean J ThoracCardiovascSurg 2017;50:22-29.

CHIRURGIE CARDIAQUE / CARDIAC SURGERY

DRAINING PERICARDIAL EFFUSION IN PATIENTS WITH CONSTRICTIVE PERICARDITIS: A NEED FOR CAUTION.

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Summary

Background: Constrictive pericarditis is usually associated with ascites, hepatomegaly and pleural effusion. The usually practice is to relieve the respiratory distress caused by the pleural effusion, before surgical management of the pericardial constriction. Renal failure can complicate this protocol **Objective:** To report the occurrence of perioperative renal failure in patients with constrictive pericarditis, who got tube thoracostomy before pericardiectomy. **Methodology:** This is a retrospective report of constrictive pericarditis patients who had post tube thoracostomy renal failure. **Results:** 3 patients had renal failure post tube thoracostomy: 1. S.D, a 28-year-old female who had a left tube thoracostomy that drained 3.7 liters of pus over 48 hours. She developed acute renal failure, and she died on the 6th day post tube thoracostomy; 2. H.M, a 40-year-old female who had a right tube thoracostomy that drained 2.3 of effusion over 72hrs. She went into acute renal failure, but she was successfully managed; 3. U.M, a 37-year-old male, who had subxyphoid tube pericardiostomy and right tube thoracostomy simultaneously. Tube thoracostomy was slowly drained; he however went into acute renal failure, which was successfully managed. He eventually had a successful pericardiectomy. **Conclusion:** Drainage of pleural effusion in patients with constrictive pericarditis can be complicated by acute renal failure. Caution should be exercised in the management of such patients.

Key-words: Pleural effusion,Constrictive pericarditis, Renal failure

Résumé

Contexte: Le drainage de l'épanchement pleural chez les patients atteints péricardite constrictive peut entraîner une insuffisance rénale **Objectif:** Attirer l'attention des praticiens sur la possibilité de survenue d'une insuffisance rénale périopératoire chez les patients atteints péricardite constrictive, ayant bénéficié d'un drainage pleural avant la péricardectomie.
Méthodologie : Etude rétrospective portant sur tous les cas de patients présentant une insuffisance rénale après la pose d'un drain thoracique. **Résultats:** Nous avons retenus 3 patients ; 1. SD, une femme de 28 ans qui avait eu un drainage pleural gauche ramenant 3,7 L de pus en 48 heures. Elle est décédée 6 jours plus tard dans un tableau d'insuffisance rénale aigue.

2. HM, patiente de 40 ans, chez qui avait été posé un drain pleural droit ramenant 2.3L en 72heures. Elle avait présenté une insuffisance rénale aiguë, qui a été pris en charge avec succès. 3. UM, un homme de 37 ans, qui avait bénéficié simultanément d'une pericardiostomie et d'un drainage pleural à droite. L'épanchement pleural a été lentement vidé; néanmoins il a présenté une insuffisance rénale aiguë, qui a été managée avec succès. La péricardectomie avait été réalisée avec des suites simples. **Conclusion:** Le drainage de l'épanchement pleural chez les patients présentant une péricardite constrictive , devrait être faite avec prudence.

Mots clés: Épanchement pleural, Péricardite constrictive, L'insuffisance rénale

Introduction

Constrictive pericarditis is an uncommon disease and can be associated with pleural effusion, ascites and hepatomegaly. Diagnosis of constrictive pericarditis is usually made late, as the pleural effusion or ascites may have attracted more attention. Pericardiectomy is done in many patients following the drainage of the pleural effusion. This approach is undertaken to allow the patient achieve optimal pulmonary function before general anaesthesia. However performing a pericardiectomy without first draining the pleural effusion has been reported(1). Renal insufficiency can occur when the pleural effusion is drained first in a patient with background pericardial constriction.

Objectives

To review the occurrence of perioperative renal insufficiency in patients with constrictive pericarditis, who had tube thoracostomy before pericardiectomy.

Materials and Methods

This is a case series review of 3 patients managed between 2011 and 2015 The clinical records of patients with constrictive pericarditis who underwent pleural drainage and developed renal insufficiency were reviewed

Results

A total of 12 patients within the study period had constrictive pericardial disease, with ages ranging from 18- 62 years and male to female ratio of 2.7 :1. Five patients had pleural effusion associated with the constrictive pericardial disease. Among these 5 patients with tube thoracostomy, 3 of them developed renal failure after tube thoracostomy.

Case No. 1 S.D, 28-year old female. She presented with cough, exertional dyspnea, orthopnea, abdominal distension of 9 months and recurrent fever of 2 months duration Physical signs were tachycardia, hypotension, distended jugular veins. The tracheal was deviated to the right with decreased vocal resonance, stony dull percussion notes and diminished breath sounds over the left middle and lower lung zones; with associated ascites Investigations Serum urea and creatinine were normal, chest X-ray revealed homogeneous opacity in the left middle and lower lung field with tracheal deviation to the right Echocardiography showed pericardial effusion of 1 cm, parietal pericardial thickening and diastolic dysfunction. Thoracic computerized tomographic scan showed parietal pericardial thickening of 5mm,pericardial effusion and left pleural effusion Management A left closed tube thoracostomy done drained 3.7 liters of pus over 48 hours. She developed acute renal failure and died on the 6th day post pleural drainage

Case No. 2 H. M,40-year old female,who presented with dyspnea on exertion, orthopnea, abdominal swelling of 5 months duration. Chest examination revealed tachypnea,tracheal deviation to the left,with reduced vocal resonance and stony dullness percussion notes in the right hemithorax. Tachycardia, hypotension and elevated jugularvenous pressure, and laterally deviated apex beat. Investigations Chest X-ray showed homogeneous opacification of the right middle and lower lung fields, and deviation of the trachea to the left.

Echocardiography showed parietal pericardial thickening and diastolic dysfunction. A chest CT scan revealed a 6mm thickening of the parietal pericardium Management She had a right chest tube inserted which drained 2.3 litres of serous fluid within 72 hours. She also went into acute renal failure post chest tube drainage. The acute renal insufficiency was successfully managed medically. She was however not seen at clinic follow up.

Case No.3 U.M, 37-year old male, who presented with chronic cough, exertional dyspnea, orthopnea and abdominal distension of 4 months duration. He had a past history of pulmonary tuberculosis. Physical examination findings were tachycardia, hypotension, elevated jugular venous pressure, muffled heart sounds. Respiartory system revealed tachypnea, tracheal deviation to the left, reduced vocal resonance, stony dull percussion note and diminished breath sounds in the right middle/lower lung zones and the lower left lung zone. Moderate ascites was also present.

Investigations

He had elevated serum urea , but normal serum creatinine. Chest X-ray revealed homogeneous opacification of the right middle and lower lung field,with tracheal deviation to the left, there was also similar but reduced homogenous opacification of the left lower hemithorax. Echocardiography revealed a 2cm pericardial effusion containing strands,thickening of the parietal pericardium and diastolic dysfunction. A thoracic computerized tomography scan revealed thickened parietal pericardium of 8mm,pericardial effusion of 2cm, and bilateral pleural effusion worse on the right.

Management

He was kept on bed rest, started on diuretics and close monitoring of fluid input and urine output. He had simultaneous right tube thoracostomy and subxyphoid tube pericardiostomy . The tube thoracostomy was gradually drained at the rate of 100 ml per hour, but the pericardial tube drained freely. He however also developed oliguria over 72 hours which was managed with adequate rehydration, diuretics and ionotropes.He recovered normal renal function and eventually had a pericardiectomy and a left tube thoracostomy after 2 weeks with no complications.

Discussion

The prevalence of chronic constrictive pericarditis has been reported as 5% of all cardiovascular diseases in West Africa[2]. Effusive-constrictive pericarditis has been reported in Ibadan, Nigeria as 13% among all patients with pericardial disease[3].

Pleural effusion is a common feature of constrictive pericarditis [4] [5], with various percentages reported from 35%[6] to 96%[4], depending on the series; and it can be unilateral[7] or bilateral[8]. It can be an exudate or a transudate[5]. A previous study in Zaria reported pleural effusion occurring in 19% of patients who had pericardiectomy for chronic constrictive pericarditis[9]. The same study by Mabogunje et al found that the patients had essentially normal blood urea nitrogen, serum electrolytes and liver function tests[9]. The pleural effusion occurring in constrictive pericarditis is usually drained either inadvertently as a result of a missed diagnosis, or deliberately to improve the patient's pulmonary function in preparation for pericardiectomy. This however is not a universal practice, as pleural effusion can resolve if the patient has a successful pericardiectomy[1].

Hemodynamic collapse following the drainage of pleural effusion in a patient with unrecognized constrictive pericarditis has been reported[7]. Our series of patients progressed to acute renal insufficiency, and we propose that hemodynamic factors may be responsible for the renal insufficiency. These patients are ordinarily at risk for cardiorenal syndrome. Cardiorenal syndrome has been defined as a pathophysiologic disorder of the heart and kidneys where an acute or chronic dysfunction of one organ can induce acute or chronic dysfunction of the other organ[10]. The pathophysiologic factors in cardiorenal syndrome include hemodynamic changes, neurohormonal (renin-angiotensin-aldosterone-system, sympathetic hyperactivity), central venous congestion, anemia and oxidative stress[11]. Our series of patients had type 1 cardiorenal syndrome- where a rapid worsening of cardiac function leads to acute renal dysfunction[10]

Considering the above pathophysiologic mechanisms responsible for cardiorenal syndrome, our series of patients seem to have been mainly affected by hemodynamic factors due to drainage of the pleural effusion.

The hemodynamic changes following pleural fluid drainage in constrictive pericarditis maybe due to:

1. Excessive third space fluid losses in a patient with reduced cardiac reserves
2. Increased venous return after drainage of the pleural effusion and reduction in intrathoracic pressure, leading to right ventricular distension shifting the interventricular septum to the left, thus impairing left ventricular filling
3. The re-expansion of the pulmonary vasculature after pleural drainage causes pooling of blood in the lungs and reduces left atrial venous return The cautious approach to management of the thrid patient in this series is notable. Adequate preoperative resuscitation to ensure rehydration and optimal urine output, then post operative intensive care unit admission were helpful.

Despite the gradual drainage of the right pleural effusion he nevertheless went into renal insufficiency, suggesting the high risk these patients represent. Close pre-,intra- and post-operative monitoring and care are therefore the most likely panacea to this dangerous scenario.

Conclusion Pleural drainage in patients with constrictive pericarditis can be complicated by acute renal insufficiency. Close monitoring, judicious use of fluids, inotropes and diuretics are needed in the management of these patients. Prospective studies to investigate the risk factors are needed.

References

1.Cecconi M., Manfrin M., Berrettini U., Ruga O., Di Eusanio G. Constrictive pericarditis presenting as unexplained recurrent pleural effusion: a case report. Cardiol Rome Italy. 1998 Sep;43(9):967–70.

2.Adebonojo SA. The status of cardiovascular surgery in West Africa. J Natl Med Assoc. 1987 Oct;79(10):1077–87.

3.Salami MA., Adeoye PO., Adegboye VO., Adebo OA. Presentation pattern and management of effusive-constrictive pericarditis in Ibadan. Cardiovasc J Afr. 2012 May;23(4):206–11.

4.Marshall A., Ring N., Lewis T. Constrictive pericarditis: lessons from the past five years' experience in the South West Cardiothoracic Centre. Clin Med Lond Engl. 2006 Dec;6(6):592–7.

5.Tomaselli G., Gamsu G., Stulbarg MS. Constrictive pericarditis presenting as pleural effusion of unknown origin. Arch Intern Med. 1989 Jan;149(1):201–3.

6.Ling LH., Oh JK., Schaff HV., Danielson GK., Mahoney DW., Seward JB., et al. Constrictive Pericarditis in the Modern Era Evolving Clinical Spectrum and Impact on Outcome After Pericardiectomy. Circulation. 1999 Sep 28;100(13):1380–6.

7.Kozieradzka A., Kamiński KA., Tycińska AM., Hirnle T., Sobkowicz B. Drainage of pleural effusions in the course of unrecognised constrictive pericarditis: a cause of severe haemodynamic deterioration. Kardiol Pol. 2012;70(6):615–7.

8.Yamamoto N., Noda Y., Miyashita Y. A case of refractory bilateral pleural effusion due to post-irradiation constrictive pericarditis. Respirol Carlton Vic. 2002 Dec;7(4):365–8.

9.Mabogunje OA., Adesanya CO., Khwaja MS., Lawrie JH., Edington GM. Surgical management of pericarditis in Zaria, Nigeria. Thorax. 1981 Aug; 36(8): 590–5.

10. Ronco C., Haapio M., House AA., Anavekar N., Bellomo R. Cardiorenal Syndrome. J Am Coll Cardiol. 2008 Nov 4;52(19):1527–39.

11. Bock JS., Gottlieb SS. Cardiorenal Syndrome New Perspectives. Circulation. 2010 Jun 15;121(23):2592–600.

CHIRURGIE VASCULAIRE / VASCULAR SURGERY

FISTULE ARTERIO-VEINEUSE : EXPERIENCE DU SERVICE DE CHIRURGIE POLYVALENTE AU CHU DE BRAZZAVILLE

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Résumé

L'objectif général de cette étude est de contribuer à l'amélioration de la prise en charge des patients ayant une insuffisance rénale chronique terminale(IRCT).Notre étude a été rétrospective, réalisée dans les services de Chirurgie polyvalente et Néphrologie du centre hospitalier universitaire de Brazzaville, entre Juillet 2016-Juin 2017. Elle a inclus tous les patients ayant bénéficié d'une FAV durant cette période. La chirurgie a été essentiellement ambulatoire, avec anesthésie locale comme technique de référence.Sur les vingt-cinq patients inclus, quinze étaient de sexe masculin. L'âge moyen était de 50 ans. L'hypertension artérielle a représenté la principale étiologie avec 48%. Tous nos patients étaient por teurs d'un cathéter veineux central, avec un cathéter jugulaire tunnellisé. La FAV radio-céphalique représentait 72%, avec comme technique chirurgicale principale l'anastomose termino-latérale. La perméabilité primaire était de 76%. Deux cas (8%) de non maturation de FAV ont été notés après trois mois. Deux décès par œdème aigu de poumon, suite à des écarts de séances d'hémodialyse. La confection de FAV est une nécessité pour mieux réaliser l'épuration extrarénale des patients en IRCT. Depuis un certain temps, elle est devenue une réalité au Congo Brazzaville ; avec une per méabilité primaire proche de celle de la littérature à six mois.

Mots clés : Fistules artério-veineuses, expérience, Brazzaville.

Summary

Aim was to contribute of improvement in treatment of patients with end stage renal failure Patients and methods: we carried out a retrospective study at Brazzaville teaching hospital, in two departments: Nephrology and Polyvalent surgical department. This study took place from July 2016 to June 2017. All patients whom have been beneficiated of arterio venous fistula confection were included. It was an ambulatory surgery, in local anesthesia.Results: Among twenty-five patients, thirteen were male. Average age was 50 years. Blood hypertension was the main etiology in 48%. All patients had jugular venous catheter before surgery, with one tunneled catheter. Distal arterio venous fistulas represented 72%, with end to side the main surgical approach. Primary permeability was 76%. We noted two cases of no maturation, after three months. Two cases of death due to acute lung edema, secondary to lack of renal dialysis.

Conclusion: Arterio-venous fistulas confection is actually real in our teaching hospital, with primary permeability near to those in the literature.

Keywords: Arterio-venous fistula, experience, Brazzaville

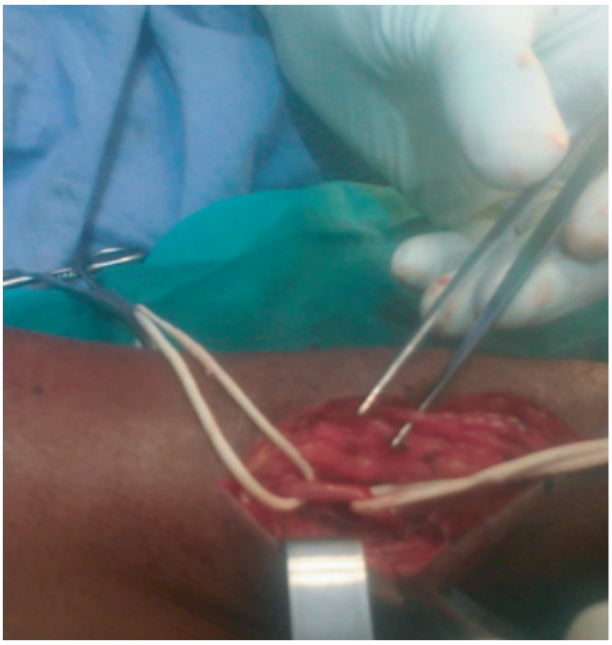
Introduction

La fistule arterio-veineuse(FAV) native est l'accès vasculaire de choix pour l'hémodialyse par rapport aux pontages artério-veineux et aux cathéters. Elle a été réalisée la première fois dans le monde aux USA par Brescia et Cimino (1). Au Congo Brazzaville, la prise en charge des patients insuffisants rénaux chroniques jusqu'en 2012 se faisait exclusivement via la dialyse péritonéale au centre hospitalier universitaire de Brazzaville. Depuis 2013, l'hémodialyse réalisée dans trois centres privés, est faite chez des patients porteurs de cathéters veineux centraux. L'utilisation de cathéters chez les patients hémodialyses est par conséquent associée à une morbi-mortalite supérieure par rapport aux patients hémodialysés avec des fistules (FAV) natives [(2).] Par ailleurs, l'usage des cathéters centraux expose également à des thromboses et sténoses veineuses centrales, compromettant les possibilités de création de futures FAV. L'objectif de cette étude est de contribuer à l'amélioration de la prise en charge des patients ayant une insuffisance rénale chronique terminale.

Patients-Méthodes

Il s'est agi d'une étude rétrospective, réalisée dans les services de chirurgie polyvalente et de Néphrologie au CHU de Brazzaville, entre Juillet 2016 et Juin 2017.Le CHU de Brazzaville dispose d'un service de Néphrologie sans unitéd'hémodialyse ; les seuls centres d'hémodialyse dont dispose le pays sont privés, notamment un dans la capitale politique et deux dans la capitale économique. Le cout direct d'une séance d'hémodialyse en privée varie entre deux cent cinquante mille-cent dix mille francs cfa. Les patients présentant une insuffisance rénale chronique terminale (IRCT) et ayant bénéficié d'une FAV ont été inclus. Au total, vingt-cinq patients ont été opérés durant cette période. L'évaluation des dossiers a été réalisée conjointement par l'équipe médico-chirurgicale. Tous les patients ont été évalués en consultation préopératoire par :

- Un examen clinique du réseau veineux sous garrot - La manœuvre d'Allen - La prise de la pression artérielle aux deux membres supérieurs - Un examen physique standard préopératoire Aucun de nos patients ne disposait d'une échographie doppler préopératoire. L'anesthésie a été soit locale, ou un bloc pléxique. L'antibiothérapie per opératoire a été réalisée systématiquement chez tous les patients avant incision (Oxacilline 2g). Les FAV réalisées ont été soit radio-céphalique distale, brachio-céphalique ou brachio-basilique par une anastomose termino-laterale (end-to-side) au prolène 7/0 (Iconographies1-2). L'héparinisation de la veine et de l'artère a été réalisée localement par injection de 40 cc de sérum physiologique hépariné 1ml/100ml. La chirurgie a été essentiellement ambulatoire. Les variables analysées ont été : épidémiologiques, cliniques, opératoires. Les moyennes ont été calculées à partir d'Excel, Windows 7.



Iconographie1:Aspect préopératoire, après dissection et mise sur lacs de l'artère radiale et libération totale de la veine céphalique

Iconographie 2:

Aspect pre-operatoire final d'une FAV radio-céphalique d'une FAV radio-céphalique

Résultats

1- Données épidémiologiques

Quinze hommes et dix femmes avec un sex-ratio à 1.5. L'âge moyen était de 50 ans (extrêmes 14- 62 ans).La répartition des patients selon l'étiologie, selon l'existence ou non d'un abord vasculaire, selon le type de FAV réalisée ,selon le type d'anesthésie utilisée figurent aux tableaux I,II,II,IV.

Tableau I : Répartition des patients selon l'étiologie

Etiologies	Nombre	Pourcentage
HTA*	12	48
Diabète sucré	2	8
Glomérulopathie	3	12
Indéterminée	8	32
Total	25	100

HTA= Hypertension artérielle

Tableau II. Répartition des patients selon l'existence ou non d'un abord vasculaire

Abord vasculaire	Nombre	Pourcentage
Cathéter central	25	96
FAV	0	0
Autres*	1	4
Total	25	100

Autres : Cathéter tunnellisé, prothèses.

Tableau III. Répartition des patients selon le type de FAV réalisée

FAV	Nombre	Pourcentage
Radio-céphalique	18	72
Brachio-céphalique	5	20
Brachio-basilique	2	8
Autres*	0	0
Total	25	100

Autres : FAV ulno-basilique, prothèse artério-veineuse.

Tableau IV :Répartition des patients selon le type d'anesthésie utilisée

Anesthésie	Nombre	Pourcentage
Xylocaïne 2%	20	80
Bloc plexique	5	20
Autres*	0	0
Total	25	100

Autres : Anesthésie générale, sédation intraveineuse associée à une anesthésie locale.

2- Temps opératoire et thrill

La moyenne du temps opératoire était de 103 min (70-128 min)

Le thrill était présent dans le bloc opératoire chez tous les patients.

3- Perméabilité primaire

La perméabilité primaire était de 76%

4- Complications précoces

Hématome au site opératoire : FAV brachio-basilique, ayant nécessité une reprise au cinquième jour post-opératoire.Thrombose avec non maturation de la FAV à trois mois (deux cas soit 8%)

5-Décès

Deux cas par œdème aigu du poumon à six et neuf mois.

Discussion

1.Les limites de l'étude

Il s'agit d'une étude rétrospective, donc possi- bilité d'un biais de sélection ; en sus, l'absence de centre public d'hémodialyse et le cout élevé de séance d'hémodialyse en privé; ces éléments pourraient expliquer la faible taille de notre échantillon.

2.Interprétation

La confection de FAV se fait le plus souvent en ambulatoire. Il s'agit d'une microchirurgie se réalisant soit en anesthésie locale, locorégionale et rarement générale. L'anesthésie locale en ambulatoire, a été la technique la plus utilisée dans notre série. Certains auteurs , recommandent le bloc plexique, avec comme effet vasodilatateur, potentialisant ainsi le debit de la fistule [3].

3.L'hypertension artérielle (HTA) a été dans notre série la première cause d'insuffisance rénale chronique. Des auteurs africains, rapportent aussi la prédominance de l'HTA comme pre- mière étiologie dans leur étude [2,4]. Fokou et al, au Cameroun, dans leur série trouvent éga- lement l'HTA comme l'une des causes majeures d'IRCT[5]. La deuxième cause dans leur étude en est le diabète sucré ; en revanche, notre étude, après l'HTA viennent les causes indéter- minées, pouvant s'expliquer surtout dans notre contexte parl' insuffisance du plateau technique. La FAV distale radio-céphalique a représenté 72% dans notre série.Les FAV distales sont considérées comme des abords vasculaires natifs de première intention chez les patients hémodialysés chroniques. Récemment, aux USA, l'initiative «fistula first», pré- conise la création d'une FAV chez tout insuffisant rénal chronique avec indication de dialyse avant l'utilisation de cathéter veineux centraux ou de prothèses. Elles présentent des avantages d'un bon débit après le délai de maturation et faible taux de recirculation, si techniquement bien réa- lisée. Alaoui et al, ont dans leur étude rapportée 73% des FAV distales radio-céphaliques contre 27 % de FAV proximales [6].L'alternative au niveau de l'avant-bras est repré- sentée par la FAV ulno-basilique. Le problème avec cet abord est dû d'un côté au diamètre petit de la veine basilique par rapport à la veine céphalique et la profondeur de la veine basilique par rapport à la peau de l'autre côté. Marc Leroy et al au Cameroun, rapportent des FAV ulno-basiliques avec une bonne perméabilité primaire [7].La FAV brachio-céphalique a représenté 20 %. Il sied de préciser que pour tous nos patients le site idéal pour le premier abord vasculaire natif était l'abord radio-céphalique distal. Nous avions été amenés à réaliser la FAV brachio-céphalique, dans les situations où la veine céphalique distale était de petit calibre ou thrombosée avec un test d'Allen positif ; donc impossibilité de réaliser une FAV ulno-basilique.Selon Baktiroglu et al, la FAV brachio-céphalique représente l'alternative de choix quand les FAV distales ne sont pas réalisables, par rapport à sa situation anatomique favorisante à la fois, l'anastomose vasculaire et l'accessibilité lors des séances de dialyse [1].La perméabilité primaire dans notre série était de 76 %. L'étude Rwandaise, trouve une per- méabilité presque similaire de 77,4 % [2].Parmi les complications précoces des FAV, les thromboses y font partie [8]. Nous avions eu deux cas de FAV non matures jusqu'au troisième mois post opératoire. Elles concernaient deux patients diabétiques, dont un était obese. Chez la patiente diabétique, en per opératoire, nous avions eu une artère radiale rigide, calcifiée et de diamètre limite. La difficulté diagnostique que nous avions eue en préopératoire en est l'absence d'échographie doppler chez nos patients ; surtout chez ceux ayant un risque d'athérosclérose, comme les patients diabétiques avec examen artériel limite. Si l'échographie doppler était réalisée, cela aurait peut-être permis de détecter des plaques sténotiques et surtout mesurer le diamètre à la fois veineuse et artérielle, ce qui aurait permis d'adapter la stratégie chirurgicale. Une étude réalisée aux USA en 2012, a permis de comprendre les mécanismes pouvant expliquer une thrombose précoce amenant à une immaturité de la FAV. Selon cette étude, l'erreur technique serait plus en cause que les phénomènes d'hyperplasie in- timale, qui viendraient en second lieu. Ils ont in- diqué la sténose juxta anastomotique comme le«primun movens» de toute thrombose précoce en stipulant que la technique classique d'anas- tomose «end-to-side» serait plus pourvoyeuse de sténose précoce donc thrombose et immaturité de la FAV que la technique «piggyback Slight line On-Lay Technique».

La raison serait que la deuxième n'aurait pas de zone de stress, donc moins de stress pariétal que la première et moins de réaction inflammatoire dans la région juxta anastomotique [9].
Parmi les autres complications précoces, on peut citer le «vol vasculaire», avec comme conséquence, l'hypoperfusion en aval et signes d'ischémie [10-11].L'inexistence de centres publics et le cout élevé d'une séance d'hémodialyse à Brazzaville, peuvent expliquer la quasi-totalité de la présence de cathéters veineux centraux chez tous nos patients. Cela, à pour conséquences, un risque accru notamment thrombotique et surtout infectieux sur ce terrain fragile. La thrombose de certaines grandes veines (Jugulaire, subclaviere, cave), pourrait compromettre la maturation ultérieure d'une FAV. L'étude Rwandaise, rapporte l'intérêt d'une conversion des cathéters veineux centraux en FAV, sur la qualité de vie et le pronostic des patients.Deux décès rapportés dans notre série dans un tableau d'œdème aigu du poumon (OAP), serait probablement liés à l'écart de séances d'hémodialyses, vu le cout exorbitant d'une séance. L'équipe Rwandaise en a eu des cas similaires liés à l'inaccessibilité de l'hémodialyse rénale.

Conclusion

La confection de FAV est mondialement reconnue comme le premier abord vasculaire pour la réalisation d'hémodialyse dans les situations d'insuffisance rénale chronique terminale. Au Congo Brazzaville, elle est devenue une réalité, permettant ainsi d'améliorer le quotidien et la qualité de vie de ces patients.

Références

1.**Baktiroglu S**, **Yanar F**, **Ozturk A**. Brachiocephalic and basilic fistula. J Vasc Access. 2015; 16 Suppl 9:S29-33

2. **Kolb I**, **Twagirumugabe Th**, **Uyisabye I** et al. Conversion des abords vasculaires d'urgence pour hémodialyse en fistules natives au Rwanda : à propos de 37 cas. Néphrologie &Thérapeutique 10; 2014 : 457–462

3.Shemesh D, **Raikhinstein Y, Orkin D**.Anesthesia for vascular access surgery.J Vasc Access. 2014;15 Suppl 7:S38-44

4.Ngatchou W, **Ngbwa Evina A, Halle MP**. Outcome of permanent vascular access for haemodialysis in patients with end-stage renal disease in Cameroon: results from the pilot expe- rience of the Douala general hospital.Acta Chir Belg. 2016 Feb; 116 (1):36-40

5.Fokou M, Ashuntantang G, Teyang A et al.Patients characteristics and outcome of 518 arteriovenous fistulas for hemodialysis in a sub-Saharan African setting.Ann Vasc Surg. 2012 Jul;26(5):674-9

6.**Alaoui H**, **Bourrouhou H**, **Sekkouri K A**. et al. Facteurs de risque d'échec précoce des fistules artérioveineuses. Communication affichée. Société francophone de néphrologie, dialyse et transplantation (SFNDT).Vol 9-N 5-septembre 2013.P :283

7. Guifo M L, **Kaze F F**, **Ndoumbe A** et al. Fistule artério-veineuse ulno-basilique pour hémodialyse au CHU de Yaoundé: à propos de deux cas. Pan African Medical Journal. 2013; 15:103

8 .Alaoui H, **Bourrouhou H**, **Belghiti K A.** et al.Complications des fistules artérioveineuses chez les patients hémodialysés chroniques. Communication affichée. Société francophone de néphrologie, dialyse et transplantation (SFNDT).Vol 9-N 5-septembre 2013.P :283

9.Bharat A, **Jaenicke M**, **Shenoy S**. A novel technique of vascular anastomosis to prevent juxta-anastomotic stenosis following arteriovenous fistula creation. J Vasc Surg. 2012 Jan;55(1): 274-80

10.**Alhassan SU, Adamu B**, **Abdu A** et al.Outcome and complications of permanent hemodialysis vascular access in Nigerians: a single centre experience. Ann Afr Med. 2013 Apr-Jun;12(2):127-30

11.Belli S, **Yabanoglu H**, **Aydogan C** et al.Surgical interventions for late complications of arteriovenous fistulas.2014 Jul-Aug;99(4):467-74.

CHIRURGIE VASCULAIRE / VASCULAR SURGERY

ACUTE LIMB ISCHEMIA: AN 2017 UPDATE

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Summary:

Acute Limb ischemia (ALI) is one of the most treatable and potentially devastating presentations of PAD. It represents one of the toughest challenges encountered by vascular specialists. The diagnosis and initial assessment are largely clinical, and diagnostic errors can cost dear to the patient. Amputation and death rates remain high despite intervention, which is in contrast to major advances in the treatment of many other vascular diseases. Acute ischemia is often an end-of-life condition presented by a patient with multiple medical co-morbidities. This review focuses on the most recent literature in the field of epidemiology, clinical presentation and assessment, treatment and prognosis.
Key words: acute limb ischemia, Anticoagulation, limb salvage, endovascular procedures, vascular surgery.

Introduction

According to the 2016 AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease[1], and to the TASC II Working Group: Inter-Society Consensus for the Management of Peripheral Arterial Disease[2], Acute Limb ischemia (ALI) is any sudden, recent decrease or worsening in limb perfusion less than 14 days causing a potential threat to limb viability.

It represents one of the toughest challenges encountered by vascular specialists. The diagnosis and initial assessment are largely clinical, and diagnostic errors can cost dear to the patient. Amputation and death rates remain high despite intervention, which is in contrast to major advances in the treatment of many other vascular diseases. Acute ischemia is often an end-of-life condition presented in a patient with multiple medical comorbidities. Therefore a careful clinical assessment of the individual is as important as assessment of the limb. Unlike many other vascular con¬ditions, there is not one definitive treatment; a variety of modalities are available, including anticoagulation, operative intervention, thrombolysis, and mechanical thrombectomy. Selection of the most appropriate intervention or combination of interventions can be critical to the eventual outcome [3].

GENERAL CONSIDERATIONS

Epidemiology

Very few published epidemiological data are available to establish the real incidence of ALI in the general population and this incidence varies according to the authors. In the whole population the acute ischemia of limbs and organs happens in 14 cases per 100,000 inhabitants from 10 to 16% in the angiosurgery [4-6]. The incidence of ALI is 9–16 cases per 100,000 persons per year for the lower extremity [7-9]. In the USA, the incidence of hospitalization for ALI decreased from 45.7 per 100,000 to 26.0 per 100,000 [10]. In England, Overall hospital admissions for ALI rose from 60.3 in 1999 to 94.3 in 2011 per 100,000 of the population in those aged ≥60 years. There was a suggestion of a hinge point in 2003 with an average annual rise of 6.2% thereafter [11]

Etiology and Pathogenesis Etiology

Excluding trauma and iatrogenic causes, there are two main reasons to the occurrence of acute limb ischemia: arterial embolism and thrombosis [3]. Factors that cause ALI are numerous and are listed in Table I Distinction between thrombosis and embolism is important in terms of diagnosis and prognosis, but it may not be crucial when deciding on the form of treatment [3]. Nowadays, the major cause of ALI (approximately 85 % of cases) is arterial thrombosis, primarily caused by underlying atherosclerosis. The remaining 10 – 15 % of patients suffers from peripheral embolism (mostly of cardiac origin) [12]

Table 1 : The causes of acute limb ischemia **[6, 13]**

Embolus
Atrial fibrillation
Valvular heart disease
Endocarditis
Myocardial infarction (with mural thrombus)
Aortic and peripheral arterial aneurysms
Ulcerated atherosclerotic plaque with intraplaque hemorrhage
Paradoxical embolus
Atrial myxoma
Cardiomyopathy
Thrombosis
Atherosclerotic occlusive disease
Aortic and peripheral arterial aneurysms
Intraplaque hemorrhage with arterial sthenosis and occlusion
Hypercoagulable states (C ar S protein deficiencies)
Entrapment syndromes
Stasis/low-flow states
Drugs of abuse
Trauma
Penetrating
Blunt
Interventional vascular procedures

Pathogenesis [3]

Embolism

Embolism from the Greek embolos, or “plug” is the result of material passing through the arterial tree and obstructing a peripheral artery. Usually the source of the embolus is the heart, and the material is mural thrombus that has accumulated and detached. The other main cause is atherosclerotic debris from a diseased proximal artery, often the thoracic aorta, in individuals with a heavy burden of atherosclerotic disease. Paradoxical embolism occurs when a clot from the venous system, usually a deep venous thrombosis, travels through a patent foramen ovale into the arterial system. The clinical clue is acute arterial ischemia in a young patient with known deep venous thrombosis. Once the embolus detaches, it passes easily through large arteries and lodges peripherally, usually at an arterial bifurcation, where vessels naturally narrow. Emboli can occlude any artery, but in the legs, the common femoral and popliteal arteries are commonly obstructed. Only large emboli, so-called saddle emboli, occlude the normal aortic bifurcation. In the upper extremity, the brachial artery bifurcation and the brachial artery at the takeoff of the profunda brachialis artery are frequent locations for emboli to stop. Embolic ischemia is usually catastrophic because it often occurs in rather normal arteries, without any established collaterals.

Thrombosis

Thrombosis results from blood clotting within an artery, which can be caused by progressive atherosclerotic obstruction, hypercoagulability, or aortic or arterial dissection. Thrombotic occlusion is most commonly the result of progressive atherosclerotic narrowing in peripheral arteries of the leg. Once a stenosis becomes critical, platelet thrombus develops on the stenotic lesion, leading to an acute arterial occlusion. Another significant cause of acute limb ischemia is the occlusion of an existing patent bypass graft. In situ thrombus formation is usually secondary to underlying disease such as atherosclerosis [14].

Pathophysiology [15]

Irrespective of the underlying etiology, arterial occlusion causes diminished perfusion of the limb [14]. Acute ischemia is the result of a sudden drop in the arterial supply to the limb [3]. It leads to severe lesions, or even irreversible, aggravated during the reperfusion. Data in the literature agree to say that the most vulnerable tissue in ischemia is the nervous tissue, more precisely the neuromuscular junction [16]. Muscle damage becomes significant after six hours of ischemia [17]. The main determinants of severity Ischemic lesions are the pre-existing collateral arteries, duration of ischemia and temperature of limb [18]. Reperfusion injury causes local and systems disorders.

Ischemia

Ischemia leads to a deprivation of the input of substrates exogenous substances such as oxygen and free fatty acids and accumulation of metabolites such as H + ions and lactic products produced by the pathways of anaerobic metabolism. We observed an initial decrease in glycogen and creatine phosphate followed by a secondary decrease in concomitant ATP of the onset of necrosis tissue occurring towards the 4th-6th hour [19].

Reperfusion

It is during reperfusion that the majority of complications appear. During reperfusion, a release of muscle degradation substances (muscle enzymes (aspartate aminotrans-ferase (AST), Lactate dehydrogenase (LDH) creatine phosphokinase (CPK)), myoglobin, lactates, potassium, H + ions) can lead to severe metabolic acidosis and lead to multiviscal failures with the onset of insufficiency renal failure, respiratory distress, or even cardiac arrest. Hyper-kalemia observed after ischemia-reperfusion originates from cell lysis which causes an increase of potassium in the extracellular medium, followed by release in the circulation: it is the wash-out phenomenon.

Clinical Presentation

The symptoms displayed by vascular occlusion depend on the size of the artery occluded and whether collaterals have developed beforehand.

Sudden occlusion of a proximal artery without existing collaterals leads to an acute white leg, whereas occlusion of the superficial femoral artery in the presence of well-established collaterals may be entirely asymptomatic [3].

Symptoms [20]

Embolic occlusions are usually very sudden and of great intensity, such a way that patients often present within a few hours of onset. Acute arterial occlusion is associated with intense spasm in the distal arterial tree, and initially, the limb will appear “marble” white. Over the next few hours, the spasm relaxes and the skin fills with deoxygenated blood leading to mottling that is light blue or purple, has a fine reticular pattern, and blanches on pressure.2 [21]. At this stage, the limb is still salvageable. The classical description of patients with ALI is represented by the “six Ps”: pain, pallor, paralysis, pulse deficit, paresthesia, and poikilothermia [22]. Pallor and the level of coldness (poikilothermia) are important to record to evaluate the progression of ischemia. The pulse deficit is helpful determining the site of occlusion. It should also be remembered that sensory capabilities, such as light touch, two-point tactile discrimination, proprioception, and vibratory perception, are lost early on. Finally, profound paralysis with complete lack of sensation indicates an irreversible state of ischemia, and the patient may be best treated with primary amputation [5] Clinical Assessment

The initial assessment of acute critical ischemia involves an evaluation of both the limb and the patient as a whole.

History

History should be considered in an attempt to define the cause of ischemia. Historically, patients with emboli had valvular heart disease but no evidence of peripheral vascular disease or other atherosclerotic conditions; however, the presence of atherosclerosis no longer rules out embolism. Patients with acute-on-chronic thrombosis often give a history of prior intermittent claudication in the ipsilateral or contralateral leg. A full medical history is important because it may reveal other associated diseases such as diabetes mellitus.

Risk factors for atherosclerotic disease should be sought, including smoking, hypertension, hyperlipidemia, and family history [3].

Risk factors

It is often difficult to distinguish an embolus from a thrombosis, but embolic occlusions should be suspected in patients with the following features: (1) acute onset, where the patient is often able to accurately time the moment of the event; (2) a history of embolism; (3) a known embolic source, such as cardiac arrhythmias; (4) no prior history of intermittent claudication; and (5) normal pulse and Doppler examination in the unaffected limb.1 [2] Differential diagnoses of ALI are represented in Table II.

Physical Findings

Examination of the leg is used to define the severity of the ischemia and is therefore fundamental. The well-known rule of pain, pallor, paresis, pulse deficit, paresthesia, and poikilothermia remains a good guide to both symptoms and signs. A full vascular examination reveals the level of the occlusion by the loss of arterial pulsation. A strong pulse can, however, mask an occlusion at that level because of the water-hammer effect. Other possible sources of embolization may become apparent, such as aortic or popliteal aneurysm or cardiac abnormalities such as atrial fibrillation. Patients with acute leg ischemia are often older adults with multiple comorbidities, and a full physical examination should be undertaken because the final outcome may depend as much on associated conditions as on the severity of the leg ischemia. Hand-held Doppler examination is also a basic part of the examination. Pedal arterial signals may be absent or reduced. The presence of normal biphasic signals excludes the diagnosis. Soft monophasic signals are associated with patent distal vessels but proximal arterial occlusion. In total, according to the 2016 AHA/ACC Guideline on the Management of Patients, with Lower Extremity Peripheral Artery Disease [1], ALI is a medical emergency and must be recognized rapidly.

The time constraint is due to the period that skeletal muscle will tolerate ischemia—roughly 4 to 6 hours [23]. A rapid assessment of limb viability and ability to restore arterial blood flow should be performed by a clinician able to either complete the revascularization or triage the patient [24]. Lower extremity symptoms in ALI can include both pain and loss of function. The longer these symptoms are present, the less likely the possibility of limb salvage [25, 26]. Clinical assessment must include symptom duration, pain intensity, and motor and sensory deficit severity to distinguish a threatened from a nonviable extremity. Hand-held Doppler examination is also a basic part of the examination. Pedal arterial signals may be absent or reduced. The presence of normal biphasic signals excludes the diagnosis. Soft monophasic signals are associated with patent distal vessels but proximal arterial occlusion. Absent Doppler signals in the ankle arteries is a poor prognostic sign. The arteries may be patent but with little flow, or they may be occluded with thrombus. In severe ischemia, ankle Doppler pressures

Table II: Rutherford classification of acute limb ischemia [32]

Class	Category	Prognosis	Sensory loss	Muscle weakness	Arterial Doppler	Venous Doppler
I	Viable	No immediate limb threat	None	None	Audible	Audible
IIA	Threatened: marginal	Salvageable if treated promptly	Minimal-none	None	+/-Audible	Audible
IIB	Threatened: Immediate	Salvageable if treated immediately	More than just toes	Mild-moderate	Rare audible	Audible
III	Irreversible	Limb loss or permanent damage	Profound	Profound	None	None

are impossible to measure, partly owing to the lack of signal but also because of muscle tenderness. In less severe ischemia, an ankle pressure of 30 to 50 mmHg can be expected, and an ankle-brachial index of about 0.3 is diagnostic of subcritical acute ischemia. Doppler can also be used to examine the extremity veins. In particular, the lack of a venous signal in the popliteal fossa suggests popliteal venous occlusion, which is a particularly poor prognostic sign in a patient with acute arterial ischemia.

Classification of Acute Limb Ischemia

Acute limb ischemia used to be classified according to cause—thrombosis or embolism—because this had implications for treatment and prognosis. Patients with thrombosis tended to be younger but had a higher risk of major amputation. Patients with emboli tended to be older and had a higher risk of dying after treatment [27, 28]. It has become clear that this is not a useful classification because there is no way of proving definitively whether an occlusion is thrombus or embolus. A more valuable method of classification is based on the severity of the arterial ischemia, which is helpful in determining the urgency of intervention and its implications for outcome [29, 30].

The Society for Vascular Surgery and the International Society for Cardiovascular Surgery have published definitions of acute leg ischemia that are valuable for treatment and prognosis (Table II) [31, 32]. These standards were modified in 2007 by a larger group—the Trans-Atlantic Inter-Society Consensus—which defined acute ischemia as any sudden decrease in limb perfusion causing a potential threat to limb viability [2].

Diagnosis

Following clinical assessments and classification, the anatomic location of the arterial occlusion can be diagnosed with a high degree of reliability. The anatomic location can be: aortic, iliac, femoro-popliteal, popliteal and infra-popliteal. In view of the symptoms presented by the patient, it is necessary to eliminate other differential diagnoses of ALI represented in Table 3.

Table III: Differential diagnosis of A[2].

Conditions mimicking ALI

- Systemic shock (especially if associated with chronic occlusive disease)
- Phlegmasia cerulea dolens
- Acute compressive neuropathy

Differential diagnosis of ALI (other than acute PAD)

- Trauma
- Dissection
- Arteritis
- Hypercoagulable states
- Popliteal adventitial cyst
- Popliteal entrapment
- Compartment syndrome

Acute PAD

- Atherosclerotic stenosed artery thrombosis
- Arterial bypass graft thrombosis

Investigation

Investigation may be valuable in confirming the clinical diagnosis and planning the appropriate treatment for patients with acute ischemia. However, when the ischemia is critical, there may be no time for investigation if direct operative intervention is required. It is possible to employ on-table angiography to assist in decision making in the operating room. Depending on the time available, a number of methods can be used to definitively determine the site and nature of the arterial occlusion.

This investigation includes: Computed Tomography, Ultrasound, Transfemoral Arteriography, Magnetic Resonance Angiography, and Echocardiography

Treatment

Initial Management

Once the diagnosis of acute ischemia has been established and its severity classified, a number of immediate interventions are possible.

Anticoagulation and Supportive Measures

Systemic anticoagulation with unfrac-tionated heparin should be initiated to minimize the risk of further clot propagation and to prevent microvascular thrombosis of underperfused distal vessels. An initial weight-based bolus of 100 mg/kg is appropriate for most patients followed by an intravenous infusion of 1000 U/hr. If urgent operation is not undertaken, the heparin dose should be titrated to maintain an activated partial thromboplastin time between 60 and 100 seconds or 2.0 to 3.0 times normal values.

Other measures that may be beneficial in patients with ALI include intravenous hydration, supplemental oxygen, and intravenous analgesia.

Treatment options

Endovascular Treatment

Endovascular procedures offer less invasive revascularization strategies for sick or elderly patients with decreased morbidity and mortality. Currently available percutaneous endovascular procedures include catheter-directed thrombolysis, pharmaco-mechanical thrombolysis, catheter-directed thrombus aspiration, and percutaneous mechanical thrombectomy [8, 33]. These techniques clear the occluding thrombus from a peripheral artery with a minimally invasive approach, restore blood flow to the extremity, and allow the identification of underlying lesions responsible for the occlusive event. Culprit lesions may then be addressed in a directed mode with an endovascular procedure such as angioplasty, stenting, or atherectomy.

Peripheral thrombolytic therapy is administered through a catheter-directed approach to achieve regional thrombus dissolution with minimal systemic fibrinolysis. However, a moderate systemic proteolytic state often results from the use of thrombolytic agents, limiting their use to patients without contraindications to such a state (Table IV).

Table IV: Contraindications to Pharmacologic Thrombolytic Agents [34-36]

Absolute Contraindications

- Active bleeding disorder
- Gastrointestinal bleeding within 10 days
- Cerebrovascular event within 6 months
- Intracranial or spinal surgery within 3 months
- Head injury within 3 months

Relative Contraindications

- Major surgery or trauma within 10 days
- Hypertension (systolic >180 mm Hg or diastolic >110 mm Hg)
- Cardiopulmonary resuscitation within 10 days
- Puncture of noncompressible vessel
- Intracranial tumor
- Pregnancy
- Diabetic hemorrhagic retinopathy
- Recent eye surgery
- Hepatic failure
- Bacterial endocarditis

Open surgery: surgical revascularization

Balloon catheter thrombectomy, first introduced by Fogarty et al [37] in 1963, has been the cornerstone of therapy for the surgical management of ALI [38]. Techniques for salvage of an ischemic limb include: (1) balloon catheter thrombectomy or embolectomy, (2) bypass procedures to direct blood flow beyond the occlusion, (3) endarterectomy with or without patch angioplasty, and (4) hybrid procedures combining open and endovascular techniques.

Indications

Management strategy [39]

Heparin (generally intravenous unfractio-nated heparin) is given to all patients acutely [1, 2, 40]. This can stop thrombus propagation and may provide an anti-inflammatory effect that lessens the ischemia. Patients who have received heparin before the onset of ALI and have a decrease in platelet count may have heparin-induced thrombocytopenia [41, 42]. In this situation, a direct thrombin inhibitor is given, rather than heparin, if heparin-induced thrombocytopenia with thrombosis is suspected [1]. For marginally or immediately threatened limbs (Category IIa and IIb ALI [Figure 1]), revascularizа-tion should be performed emergently (within 6 hours). For viable limbs (Category I ALI [Figure 1]), revascularizа-tion should be performed on an urgent basis (within 6–24 hours).

Revascularization strategy

The revascularization strategy can range from catheter-directed thrombolysis to surgical thromboembolectomy. Available facilities and clinical expertise are factors that should be considered when determining the revascularization strategy. The technique that will provide the most rapid restoration of arterial flow with the least risk to the patient should be selected. For example, catheter-directed thrombolysis can provide rapid restoration of arterial flow to a viable or marginally threatened limb, particularly in the setting of recent occlusion, thrombosis of synthetic grafts, and stent thrombosis.367 If this is not available locally, surgical options for timely revascularization should be considered, along with the feasibility of timely transfer to a facility with the necessary expertise.

Amputation

For patients with Category III ALI, amputation should be performed as the index procedure. Prolonged duration of ischemia is the most common factor in patients requiring amputation for treatment of ALI. The risks associated with reconstruction outweigh the potential benefit in a limb that is already insensate or immobile because of prolonged ischemia. Patients who have an insensate and immobile limb in the setting of prolonged ischemia (>6 to unlikely to have potential for limb salvage. In addition, in this setting the reperfusion and circulation of ischemic metabolites can result in multiorgan failure and cardiovascular collapse. However, if pain can be controlled and there is no evidence of infection, amputation may be deferred if this meets with the patient's goals.

Management of compartment syndrome after revascularization

The lower extremity muscles reside in compartments, surrounded by fascia and bones. Reperfusion to ischemic muscles can cause cellular edema, resulting in increased compartment pressure. When compartment pressure is >30 mm Hg, there is capillary and venule compression that leads to malperfusion of the muscle; this is compartment syndrome. Fasciotomy is indicated when the compartment pressure increases. Measurement of intracompart-ment pressure is not always easily accessible. In such cases, evaluation for fasciotomy is prompted by development of increased pain, tense muscle, or nerve injury. Fasciotomy should be considered for patients with Category IIb ischemia for whom the time to revascularization is >4 hours.

Treatment of the Results

Assessment of the comparative effective-ness of catheter-based thrombolysis versus open surgery is complicated by variable definitions of ALI in this literature [1]. Four RCTs comparing catheter-based thrombolysis to surgery, [43-46] as well as a meta-analysis [47], have demonstrated similar limb salvage rates between the 2 approaches but better survival with catheter-based therapy. The survival advantage of catheter-based therapy may be at least in part attributable to multiple comorbidities found among the population of patients who present with ALI. Increased comorbidities are likely to contribute to increased perioperative risk. Several of the RCTs included patients with relatively chronic ischemia. Acuity and severity are both factors in the decision to consider thrombolysis. [43-46]. Regarding Limb loss and survival, Ashraf G. Taha, the overall 30-day amputation rate was significantly higher with OR (13.5%) than with ER (6.5%; P ¼ .02; Table III and Fig 1). The overall mortality rates (Fig 2) were significantly lower with ER than with OR at 30 days (5.4% vs 13.2%; P ¼ .012), 1 year (12.9% vs 33.8%; P < .001), and 2 years (18.7% vs 40.5%; P < .001).

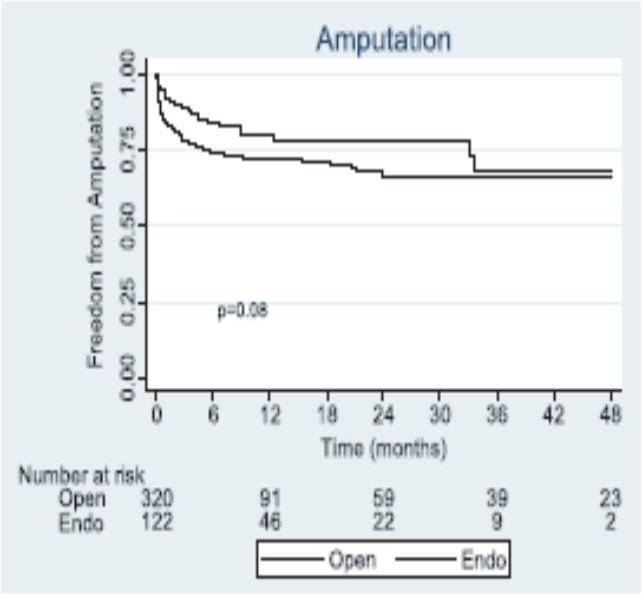

Fig 1. Kaplan-Meier survival curve: Amputation-free survival (limb-based analysis).

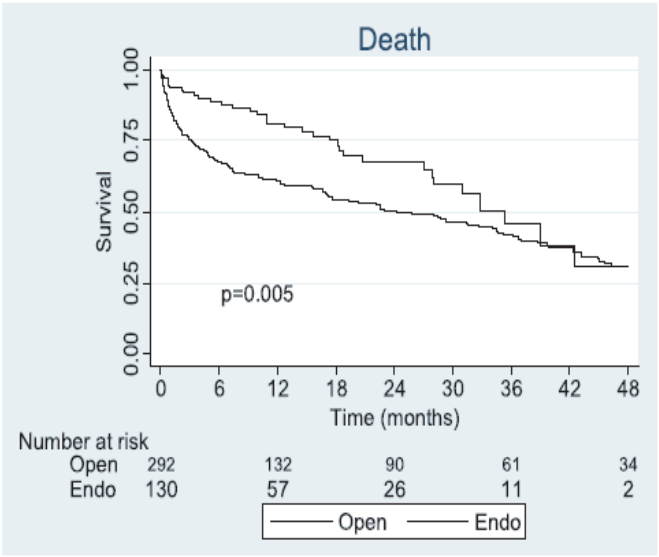

Fig 2. Kaplan-Meier survival curve: Survival analysis (patient-based analysis).

Table V: Outcome of Patients Treated with Initial Thrombolytic Therapy or Primary Operation for Acute Limb Ischemia [3]

Series	Number of Patients	Period (months)	THROMBOLYTIC THERAPY Amputation (%)	Death (%)	PRIMARY OPERATION Amputation (%)	Death (%)
University of Rochester **[43]**	114	12	18	16	18	42
STILE Trial **[44]**	393	6	12	6.5	11	8.5
TOPAS Trial-II **[45]**	544	12	15	20	13.1	17

Table VI: amputation, Mortality, and Long-term Limb Salvage for Open Surgery for Acute Limb Ischemia **[3]**

			RESULTS		
Series	**Year**	**Number of Patients**	**Amputation (%)**	**Mortality (%)**	**Limb Salvage**
Campbell et□al **[34]**	1998	474	16	22	Not reported
Nypaver et□al **[35]**	1998	71	7	10	62% at 1□yr
Pemberton et□al **[36]**	1999	107	12	25	75% at 2□yr

Particular Case: Upper Limb Ischemia[3]

A number of significant differences exist between acute ischemia of the arm and that of the leg. Patients with acute arm ischemia tend to be, on average; about 4 years older than those with acute leg ischemia (mean age, 74 years). Arm ischemia is seldom limb threatening, and treatment decisions are usually less urgent. [48,49].
The main reason for treating arm ischemia is to prevent late complications such as exercise-induced arm fatigue and pain [50]. Most arm ischemia is due to cardiac embolism. Rare causes of arm emboli include thoracic outlet syndrome and proximal subclavian artery aneurysm. Atherosclerosis is rare in upper limb arteries, and collateral vessels usually prevent acute limb ischemia when atherosclerosis is present. Patients with upper limb ischemia often present with a cold feeling and numbness, rather than pain in the arm. The diagnosis of ALI in the upper extremity is clinical and can be confirmed by duplex ultra

sonography. The arm often improves after initial anticoagulation, and decisions about whether to perform embolectomy can be difficult. Up to 50% of patients have late symptoms of arm pain if untreated. Consequently, there should be a low threshold to undertake embolectomy if there is doubt about limb viability [50]. A small number of patients present with class IIb critical ischemia and require urgent surgical intervention [49] Failed surgery in this situation risks ischemic contracture or even arm amputation on occasion. The threat to the arm is generally low, but up to 20% of patients with acute arm ischemia do not survive the acute event, usually owing to cardiac complications[51] As with acute leg ischemia, there is a high attrition rate after successful treatment; only 60% of patients survived 3 to 5 years in one typical series. [51]

Conclusion

ALI is a surgical and radiological emergency. Prompt clinical evaluation and appropriate use of the available management options can result in good limb salvage rates. If the limb demons-trates clinical signs of irreversible ischemia, time should not be wasted at attempted revasculari-zation; saving life over limb is paramount.

References

1-Gerhard-Herman MD., Gornik HL., Barrett C., Barshes NR., Corriere MA, Drachman DE et al.2016 AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Gui-delines. Circulation. 2017; 135(12):726-79.
2-Norgren L., Hiatt WR., Dormandy JA., Nehler MR., Harris KA., Fowkes FG. TASC II Wor-king Group: Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). J Vasc Surg. 45 (Suppl):S5-S67 2007
3-Jonothan J. Earnshaw. Acute ischemia: Evaluation and Decision Making. In: Rutherford's Vascular surgery. 8th ed. Philadelphia: Elsevier; 2014: 2518 – 27.
4-Dormandy J., Heeck L. Acute limb ische-mia. Vig S. Eur J Vasc Surg 1994;8(5):578-83.
5- Costantini V, Lenti M. Treatment of acute occlusion of peripheral arteries. Ann Acad Med Singapore 1994;23(6):844-7
6- Aleksandras Antuševas., Nerijus Aleksynas The surgical treatment of the lower limb acute ischemia MEDICINA 2003 39, No.7
7-Martyn Knowles.,Carlos H. TimaranE-pide-miology of Acute Critical Limb Ischemia. In Cri-tical Limb Ischemia Acute and Chronic. 1st ed. Springler ; 2017 :1-7
8-Creager MA., Kaufman JA., Conte MS. Clinical practice. Acute limb ischemia. N Engl J Med. 2012;366:2198–206. Dryjski M, Sweden-borg J. Acute ischemia of the extremities in a metropolitan area during one year. J Cardiovasc Surg. 1984;25:518–22.
9- Davies B., Braithwaite BD., Birch PA., Poskitt KR., Heather BP., Earnshaw JJ. Acute leg ischaemia in Gloucestershire. Br J Surg. 1997;84:504–8.
10- Donald T. Baril., Kaushik Ghosh., and Al lison B. Rosen, Trends in the incidence, treatment., and outcomes of acute lower extremity is-chemia in the United States Medicare population J Vasc Surg. 2014 September ; 60(3): 669–77.
11- RS von Allmen., A Anjum1., JT Powell1., JJ Earnshaw. Hospital trends of admissions and procedures for acute leg ischaemia in England, 2000–2011 Ann R Coll Surg Engl 2015; 97: 59–62).

12-Lukasiewicz A Treatment of acute lower limb ischaemia.Vasa.2016; 45 (3): 213-21.
13-Hobson RW. Acute limb ischaemia. Se-min Vasc Surg 1992; 5:1-3.
14-S PUPPALA Acute limb ischaemia. Ima-ging, 21 (2009), 109–121.
15-Piriou V., Closon M., Feugier P. Prise en charge en urgence d'un patient en ischémie aiguë des membres inférieurs. EMC (Elsevier Masson SAS, Paris), Médecine d'urgence, 25-190-A-20, 2007
16-Hatzipantelis KP., Natsis K., Albani M. Ef-fect of acute limb ischaemia on neuromus-cular function in rats. Eur J Surg 2001;167:831-8.
17-Belkin M., Brown RD., Wright JG., La Morte WW., Hobson 2nd RW. A new quantitative spec-trophotometric assay of ischemia-reperfu-sion in-jury in skeletal muscle. Am J Surg 1988;156:83-6.
18-Petrasek PF., Homer-Vanniasinkam S., Walker PM. Determinants of ischemic injury to skeletal muscle. J Vasc Surg 1994;19:623-31.
19-Lindsay TF., Liauw S., Romaschin AD., Walker PM. The effect of ischemia/reperfusion on adenine nucleotide metabolism and xanthine oxidase production in skeletal muscle. J Vasc Surg 1990;12:8-15.
20-Rezzan D Acar., Muslum Sahin., and Cevat Kirma. One of the most urgent vascular circums-tances: Acute limb ischemia SAGE Open Med. 2013; 1 XX(X)
21-Callum K and Bradbury A. ABC of arterial and venous disease: acute limb ischaemia. BMJ 2000; 320(7237): 764–767.
22-Mattox KL., Feliciano DV., Burch J., et al. Five thousand seven hundred sixty cardio-vas-cular injuries in 4459 patients. Epidemio-logic evolution 1958 to 1987. Ann Surg 1989; 209(6): 698–705, discussion 706–7
23- Blaisdell FW. The pathophysiology of ske-le-tal muscle ischemia and the reperfusion syn-drome: a review. Cardiovasc Surg. 2002;10:620–30.
24-Londero LS., Nørgaard B., Houlind K. Pa-tient delay is the main cause of treatment delay in acute limb ischemia: an investigation of pre- and in-hospital time delay. World J Emerg Surg. 2014;9:56.
25- Duval S., Keo HH., Oldenburg NC., et al. The impact of prolonged lower limb ischemia on amputation, mortality, and functional status: the FRIENDS registry. Am Heart J. 2014;168:577–87.
26-Morris-Stiff G., D'Souza J., Raman S., et al. Update experience of surgery for acute limb ischaemia in a district general hospitalare we getting any better? Ann R Coll Surg Engl. 2009;91:637–40.)
27-Cambria RP., Cambria RP., Abbott WM: Acute arterial thrombosis of the lower extremity. Its natural history contrasted with arterial embo-lism. Arch Surg. 119:784-787 1984 6732488
28-Dale WA: Differential management of acute peripheral arterial ischaemia. J Vasc Surg.1:269-278 1984 6481876
29-Jivegård L1., Bergqvist D., Holm J.: When is urgent revascularization unnecessary for acute lower limb ischaemia? Eur J Vasc Endovasc Surg. 9:448-453 1995 7633991
30-Earnshaw JJ1., Hopkinson BR., Makin GS. Acute critical ischaemia of the limb: a prospective evaluation. Eur J Vasc Surg. 4:365-368 1990
31-Suggested standards for reports dealing with lower extremity ischemia. Prepared by the Ad Hoc Committee on Reporting Standards, Society for Vascular Surgery/North American Chap-ter, International Society for Cardiovascular Sur-gery. J Vasc Surg. 4:80-94 1986
32-Rutherford RB1, Baker JD, Ernst C, Johnston KW, Porter JM, Ahn S, Jones DN.: Recommended standards for reports dealing with lower extremity ischemia: revised version. J Vasc Surg 1997; 26: 517 – 538
33-Karnabatidis D., Spiliopoulos S., Tsetis D., Siablis D. Quality Improvement guidelines for percutaneous catheter-directed intra-arterial thrombolysis and mechanical thrombectomy for acute lowerlimb ischemia. CardiovascIntervent Radiol 2011; 34:1123-36
34-Campbell WB, et al.: Current manage-ment of acute leg ischemia: results of audit by the Vas-cular Surgical Society of Great Britain and Ireland. Br J Surg 1998; 85:1498-1503 9823910
35-Nypaver TJ., et al.: Nontraumatic lowerex-tre-mity acute arterial ischemia. Am J Surg 1998 ; 176:147-152 9737621
36-Pemberton M., et al.: The surgical mana-ge-ment of acute limb ischemia due to native ves-sel occlusion. Eur J Vasc Endovasc Surg 1999 ; 17:72-76 10071621
37-Fogarty TJ., Cranley JJ., Krause RJ., Stras-ser ES, Hafner CD : A method for extraction of arte-rial emboli and thrombi. Surg Gynecol Obs-tet 1963; 116:241-44.

CHIRURGIE THORACIQUE / THORACIC SURGERY

PEDIATRIC CHEST TRAUMA IN BOUAKE TEACHING HOSPITAL BOUAKE COTE D'IVOIRE

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Objective: Chest trauma in children is rare. However, they remain a source of substantial mortality. The purpose of this study was to highlight the management and outcomes of children received with chest trauma in our institution.**Patients and Methods:** We reviewed retrospectively, from 2012 to 2018, 15 children under 18 years hospitalized for chest trauma. Low chest trauma that were discharged immediately from the hospital for outpatient medical treatment were excluded. There were 13 males and 2 females. Mean age was 11.93±3.97 years. We obtained data on patient demographics, lesions, diagnostic explorations and the clinical results, treatment and results from patients' medical records. **Results:** Pediatric chest trauma represented 12.29 % (15/122) of all chest trauma we received in the study period. We collected 11 (73.33%) blunt injuries and 4 (26.66%) penetrating injuries. Traffic accidents (n=11; 73.33%) were the leading cause of the trauma. Hemothorax (n=8; 53.33%), pneumothorax (n=2; 13.33%), hemopneumothorax (n=4; 26.66%), rib fractures (n=3; 20%), lung contusion (n=2; 13.33%) and diaphragmatic rupture (n=1; 6.66%) were the most common types of thoracic injury. Extra-thoracic injuries were associated in 26.66% (n=4) of cases. Pleural drainage (n=12; 80%) was efficient in most of our cases. Mean hospital length of stay was 7.14 days (range 4-18 days). Hospital mortality was 13.33% (n=2) due to respiratory acidosis and haemorrhagic shock.**Conclusion:** Chest trauma in children is rare. Traffic accidents are the main etiology. Mortality remains high.

Keywords: Chest, Trauma, Bouake, Pediatrics.

Introduction

Chest trauma in children is rare[1]. Because, child is not an adult in miniature[2] and because of the anatomy and mechanisms differences, pediatric chest trauma should be distinguished from adults[3] . Difficulties in accurate diagnosis of chest trauma in children is due to the paucity of clinical symptoms[4]. The purpose of this study was to highlight the management and outcomes of children we received with chest trauma in our institution.

Patients and Methods

We reviewed retrospectively, from 2012 to 2018, 15 children under 18 years hospitalized for chest trauma. Low chest trauma that were discharged immediately from the hospital for outpatient medical treatment were excluded. There were 13 males and 2 females. Mean age was 11.93±3.97 years. We obtained data on patient demographics, lesions, diagnostic explorations and the clinical results, treatment and results from patients' medical records.

Results:

Pediatric chest trauma represented 12.29 % (15/122) of all chest trauma we received in the study period. We collected 11 (73.33%) blunt injuries and 4 (26.66%) penetrating injuries. Causes of chest injury were traffic accidents (n=11; 73.33%), violence (n=2; 13.33%), and do-mestic accident (n=2; 13.33%). (Table1). Table 2 lists types of chest trauma. Thus hemothorax (n=8; 53.33%), pneumothorax (n=2; 13.33%), hemopneumothorax (n=4; 26.66%), rib fractures (n=3; 20%),

lung contusion (n=2; 13.33%) and diaphragmatic rupture (n=1; 6.66%) were the most common types of injury. Extra-thoracic injuries were associated in 26.66% (n=4) of cases.
(Table 3). Pleural drainage (n=12; 80%) was effi cient in most of our cases. Mean hospital length of stay was 7.14 days (range 4-18 days). Hospi-tal mortality was 13.33% (n=2) due to respirato-ry acidosis and haemorrhagic shock.

Table 1 : Causes of chest injury

Variables	Number	%
Traffic accident	11	73.33
Domestic accident	2	13.33
Violence	2	13.33

Table 2 : Types of chest trauma.

Variables	Number	%
Rib fractures	3	20
Hemothorax	8	53.33
Pneumothorax	2	13.33
Hemopneumothorax	4	26.66
Lung contusion	2	13.33
Diaphragm lesion	1	6.66

Table 3 : Types of chest trauma.

Variables	Number	%
Cranial trauma	2	13.33
Abdominal trauma	2	13.33

Discussion

Thoracic trauma is relatively uncommon in children. Okonta[5], in Nigeria, in 2015 repported 12.1% of chest trauma in children among all chest trauma managed in his thoracic units. In our se-ries chest trauma in children represents 12.29 % of all chest trauma we received. Chest trauma in children is not occur frequently as that seen in adults and the aetiology of chest injuries in child-ren is different from that of adults[5]. Althouth ot eud saw cicaroht fo ecneuqesnoc niam ehtthe impairment of oxygen delivery and/or transport, children do not have the same responses to trauma as adults do[6]. Children have reduced functional residual capacity with higher oxygen consumption per unit body mass[7]. Thus, the major complications of chest trauma in the early period is hypoxia. Futhermore, children have a softer chest wall so that an apparently simple chest injury may easily damage the intrathoracic organs[9]. In addition to this anatomic and physiologic considerations, thoracic or pediatric surgeons must also know the most common causes of chest trauma[7]. In fact, blunt trauma is the most frequent cause of chest injuries in children[8-11].

It represents between 60% and 80% of chest injuries in younger children. Theses blunt trauma, were due to impact with motor vehicles[12, 9]. According Ismail MF[13], in 2012, in Egypt, blunt chest trauma
represented 98% of the total cases. In our series, blunt chest trauma represented 73.33% of all chest trauma. According to literature [6, 13,15], the most common thoracic injuries in pediatric trauma are pulmonary contusion, pneumothorax and Rib fractures. According to Nakayama DK [16], hemothorax is less common in blunt trauma. Our finding is contrary to this statement İn fact, ın our sis.

According to Okonta[5], in Nigeria, in 2015 and Balcı, in 2004, in Turkey, hemothorax was the secund and the third common diagnosis in pediatric chest trauma respectively. Penetrating chest trauma are rare in patients under 18 years old. We repport in our series, 26.66%. Okonta [5], in Nigeria, in 2015 repported 22.6%. Ismail MF [13], in 2012, published 2.1%. The most common injury from penetrating chest trauma is a pneumothorax, with or without a hemothorax [6].

Whether blunt or Penetrating chest trauma in children requires early resuscitative measures are to avert mortality. Most thoracic injuries can be managed either non-operatively or by tube thoracostomy[17]. Thoracotomy is reserved for persistent bleeding through chest tube, mediastinal injury or uncontrollable hemorrhage[15,18]. In our series thoracotomy was required in one child who had diaphragmatic rupture occurs by left side abdomino-thoracic blunt trauma.
Futhermore, chest can be associated to extrathoracic injury and head injury is the common cause of death [8]. According to literature, mortality rate of chest trauma in children is from 6.7% up to 25%[14,16,19,20]. Our mortality rate was 13.33%.

Conclusion:

Chest trauma in children is rare.
Traffic accidents are the main etiology.
Mortality remains high.

References

1- Roux P., Fisher RM. Chest injuries in children: an analysis of 100 cases of blunt chest trauma from motor vehicle accidents. J Pediatr Surg 1992;5:551–5.
2- Lippi G., Franchini M., Montagnana M., Guidi GC. Coagulation testing in pediatric patients: the young are not just miniature adults. Semin Thromb Hemost. 2007 ; 33 :816-20.
3- Nakayama DK., Ramenofsky ML., Rowe MI. Chest injuries in childhood. Ann Surg 1989;210–770-5.
4- Sivit CJ., Taylor GA., Eichelberger MR. Chest injuries in children with blunt abdominal trauma: evaluation with CT. Radiology 1989;171: 815–8.
5- Okonta KE.Traumatic chest injury in children: A single thoracic surgeon's experience in two Nigerian tertiary hospitals. Afr J Paediatr Surg. 2015; 12: 181–186.
6- lopez M. Evaluation and Management of Pediatric Chest Trauma. Consulted 2017/03/27 in https://www.ahcmedia.com/articles/10841-evaluation-and-management-of-pediatric-chest-trauma?v=preview
7- Bliss D., Silen M. Pediatric thoracic trauma. Crit Care Med. 2002;30:S409–15.
8- Crankson SJ., Fischer JD., Al-Rabeeah AA., Al-Jaddan SA. Pediatric thoracic trauma. Saudi Med J. 2001;22:117-20.
9- Ceran S., Sunam GS., Aribas OK., Gormus N., Solak H. Chest trauma in children. Eur J Cardiothorac Surg. 2002;21 :57–9.
10- Balci AE., Kazez A., Eren S., Ayan E., Ozalp K., Eren MN. Blunt thoracic trauma in children: Review of 137 cases. Eur J Cardiothorac Surg.
11- Deng X., Yang Y., Deng Z., Huang E. Ex-periences in severe chest trauma of children in 20 years. Interact Cardiovasc Thorac Surg. 2008;7:1031–4.
12- Peterson RJ., Tepas III JJ., Edwards FH, et al: Pediatric and adult thoracic trauma: Agerelated impact on presentation and outcome. Ann Thorac Surg 1994; 58:14–18.
13- Ismail MF., al-Refaie RI. Chest trauma in children, single center experien-ce. Arch Bron-coneumol. 2012; 48:362-6.
14- Balci AE., Kazez A., Eren S., Ayan E, Ozalp K, Eren MN. Blunt thoracic trauma in children: review of 137 cases. Eur J Cardio-thorac Surg. 2004;26:387-92.
15- Meller JL., Little AG., Shermeta DW. Thora-cic trauma in children. Pediatrics 1984;74:813–9.
16- Nakayama DK., Ramenofsky ML., Rowe MI. Chest injuries in childhood. Ann Surg 1989;210–770-5.
17- Inci I., Ozceli KC., Nizam O., Eren N., Ozgen G. Penetrating chest injuries; A review of 94 cases. J Pediatr Surg 1996; 31: 673-676.
18- Al-Saigh A., Fazili FM., Allam AR. Chest trauma in children: A local experience. Annals of Saudi Medicine 1999; 19: 106-109.
19- Peclet MH., Newman KD., Eichelberger MR., Gotschall CS., Garcia VF., Bowman LM. Thoracic trauma in children: an indicator of increased morbidity. J Pediatr Surg 1990;25:961–6.
20- Rielly JP., Brandt ML., Mattox KL., Pokor-ny WJ. Thoracic trauma in children. J Trauma 1993;34:329–31.