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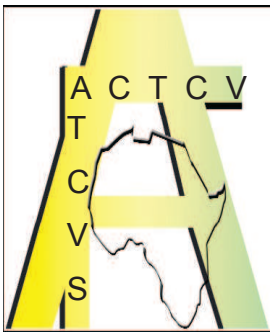


CHIRURGIE CARDIAQUE/CARDIAC SURGERY

BLUNT THORACIC AORTIC INJURY (BTAI) : ADVANCES IN THE ERA OF INNOVATION. A REVIEW (PART 2)

A. THOMAS PEZZELLA, MD[1], ANASTASIOS C. POLIMENAKOS, MD[2]

Treatment

The treatment strategy of the BTAI patient must consider a number of factors: time interval from injury to diagnosis, age, clinical status (emergent, unstable, stable), associated injuries, and underlying comorbidity.

A major therapeutic challenge is treatment of the asymptomatic minimal aortic injury (MAI) (defined as an intimal flap < 1cm and no periaortic hematoma or pseudoaneurysm), and especially those with delayed referral or dignosis[6,11]. Given the sophistication of the aforementioned diagnostic modalities, these small lesions are being increasingly identified[11]. The natural history of MAI is unknown. Malhotra et al.[11] reported a 10% (9 patients) incidence of MAI in a series of 87 patients. Of 6 discharged patients 2 had resolution of the MAI, and 3 developed small pseudoaneurysms. Delayed medical management and surveillance offer a safe and prudent approach[18]. However, EVSG has gained increased support in this scenario.

The larger series of more extensive lesions are the present concern. Mattox et al.[4,13] stress the three objectives of surgical treatment :

1- to prevent exsanguinations from a partial tear with a contained perivascular hematoma;

2- to control blood loss from vessel wall rupture;

3- and to restore vessel continuity.

Symbas[46] proposed an early algorithm (figure 19). Downing et al[49] has proposed an updated algorithm for diagnostic evaluation and treatment (figure 20). A contemporary algorithm features CT scan and EVSG playing prominent roles (figure 21). This has been hightlighted in the recent AAST report (table 1)[8], as well as the updated ATLS changes with level 3 and 4 evidence (table 2)[101].

The ATLS protocol is mandated in evaluation of suspected BTAI[102]. Globally, in low and middle income countries (LMIC), the Essential Trauma Care Project, and its publication Guidelines for Essential Trauma Care, has addressed the need and value of early coordinated, standardized care of the trauma patient[2]. Both stress the ABC's, and primary/secondary survey.

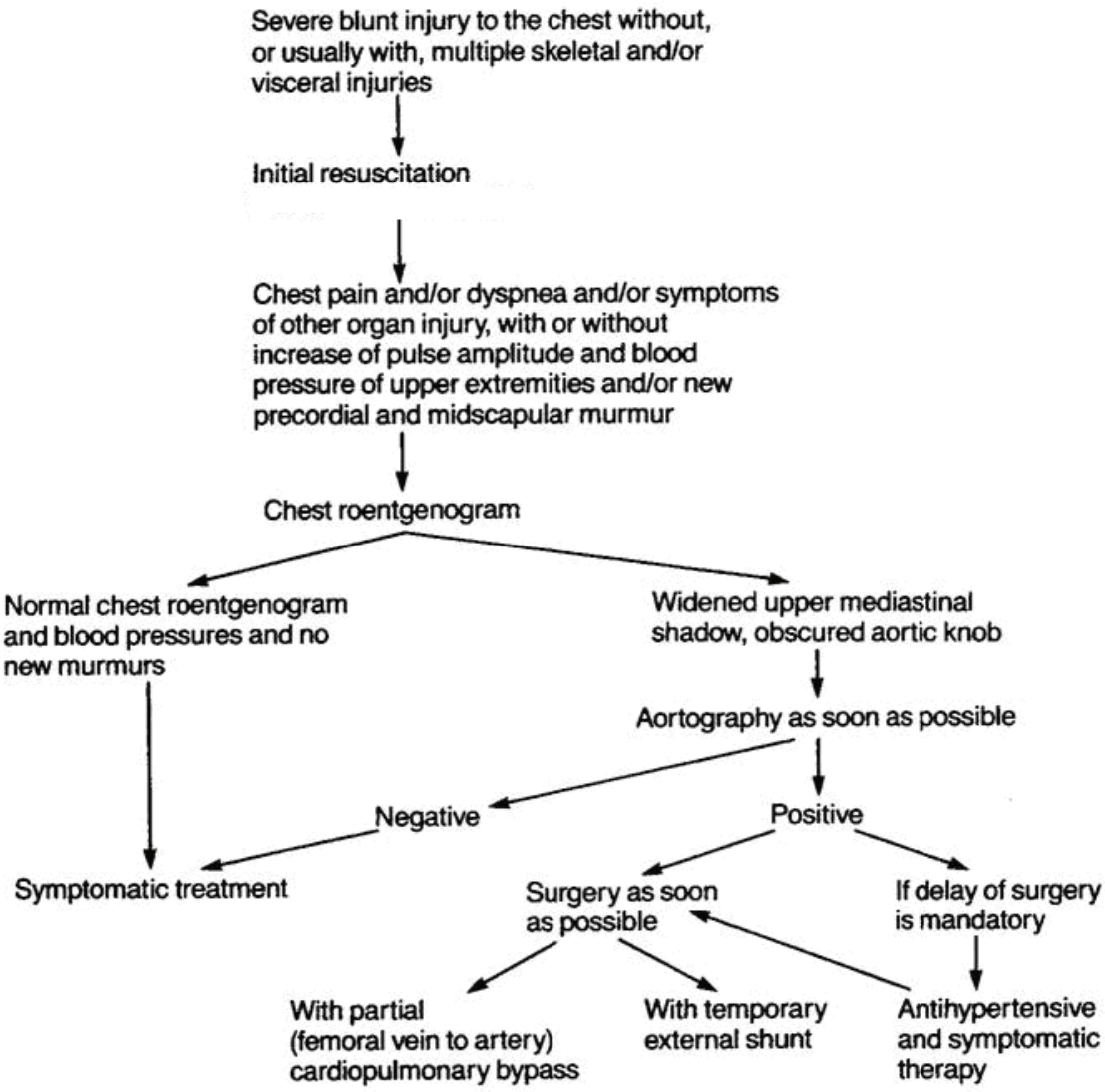
Tension pneumothorax, cardiac tamponade, and massive hemothorax warrant an early response in the primary survey. This requires ready availability of chest tube thoracostomy, subxyphid pericardial window, or emergency/urgent thoracotomy. The patient's clinical status ultimately dictates the subsequent evolution of action.

There are eight thoracic lethal injuries following blunt, penetrating or blast injury: progressive pneumothorax, increasing hemothorax, pulmonary contusion with ARDS, tracheobronchial injury with large air leak, blunt cardiac injury, BATI, diaphragmatic rupture, and mediastinal traverse injury[102]. Diagnostic studies can be precluded in the light of extreme scenarios. With few exceptions a definitive sequence of intervention is mandated[103].

A major decision to be made in the triage period for suspected or diagnosed BTAI is whether the accepting facility can accommodate and treat the injury, or requires transfer to a higher echelon trauma facility (Level I or II). Undertriage places the victim at higher risk, whereas overtriage increases transfer to higher level centers, with resultant greater cost and utilization of resources. The availability of advanced diagnostic studies, cardiopulmonary bypass, and endovascular stent-graft capability are important considerations.

Regarding blunt thoracic trauma, emergency or resuscitative thoracotomy for cardiac arrest, when vital signs are present at the scene, and neurologically intact, has a documented survival rate ranging from 0.6 – 4.5 %[23,64,65]. The role in blunt trauma has not been effective, as compared to penetrating trauma[101,104,105]. The ATLS revised guidelines are noted[101]: " Patients

sustaining blunt injuries who arrive pulseless but with myocardial electrical activity (PEA) are not candidates for resuscitative thoracotomy (RT)". It must be stressed that closed compressions for traumatic cardiac arrest is generally ineffective[23]. The goal of emergency thoracotomy is to gain more effective internal cardiac massage, control bleeding, relieve cardiac tamponade, control air embolism, and cross clamp the descending aorta. All of these goals do not help in the presence of massive hemothorax from aortic rupture, especially if performed outside the operating room.



Algorithm for diagnosis and management of rupture of the aorta.

Fig. 19 : Early historical algorithm with CXR, aortogram, and early open surgery options. (From Symbas PN. Cardiothoracic Trauma. WB Saunders, Philadelphia. 1989. P.190.)

The concept of damage control in thoracic trauma is limited and not well understood. Simpler techniques, including crycothyroidotomy, subxyphoid window, and chest tube thoracostomy are well know and practiced[105]. Yet, unlike packing for abdominal wounds, this is not an option for thoracic wounds. Chest closure may be difficult, though use of delayed chest closure (eg Bogota bag) has been utilized. Thoracoscopy has not been widely utilized in diagnosis or treatment strategies for BTAI.

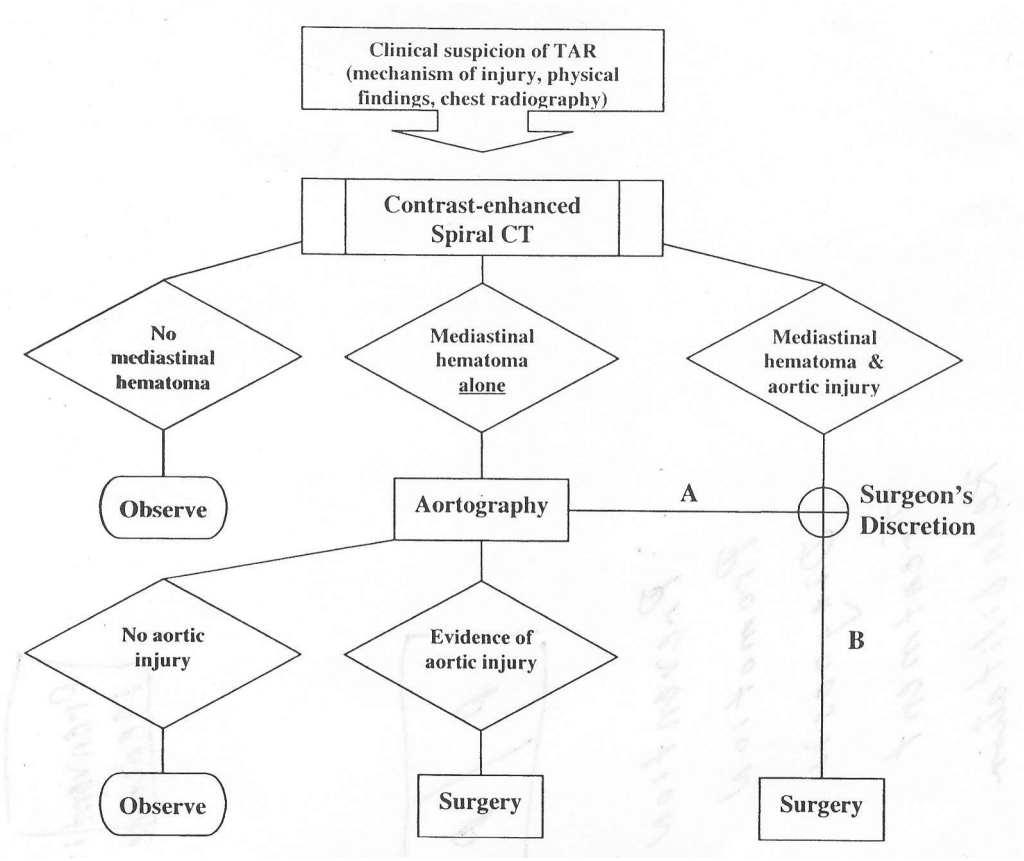


Fig. 20 : Evolving algorithm with CT scan and delayed medical management options.
Diagnostic algorithm for traumatic aortic rupture. Patients with no mediastinal hematoma are observed. Patients with mediastinal hematoma but no definitive sign of aortic injury (a "nonnegative" spiral computed tomograph [CT]) undergo aortography. Patients with mediastinal hematoma and signs of aortic injury proceed either down path A to aortography or path B directly to operation at the surgeon's discretion. (From Downing et al[49])

Addressing massive hemothorax with tube thoracostomy, obtaining two large bore intravenous access and invasive monitoring represent the first line of action. Emergency or urgent thoracotomy is indicated in the initial blood loss of 1,500cc., or 200cc/hour for 2-4 hours[102]. This is better accomplished in the operating room.

Blood products and crystalloid infusion are paramount, but excessive use should be avoided. Massive volume replacement in the multiple injured patient has been temporized by the concept of permissive hypotension, in the absence of significant head injury. A minimum systolic pressure of 90 mmHg is the goal. This is achieved with crystalloid, colloid products, or blood. Alam et al.[106] has reviewed new developments in fluid resuscitation. Delaying or restricting fluids did not increase mortality. They noted 6 randomized clinical studies reviewed by the Cochrane Database. These studies failed to show increased mortality in restricted large volume fluid administration. The major volume sources include : 5% hypertonic saline (HTS), isotonic crystalloids, artificial colloids, fresh frozen plasma, fresh whole blood, and artificial blood.

The principle of anti-impulse therapy, permissive hypertension, or "intended reduction of aortic wall stress (dP/dT)" by keeping mean arterial pressure between 60-80 mmHg and heart rate <100, principally with β-blocker and vasodilator drugs, should be instituted on all BTAI patients, and continued throughout the clinical course, especially perioperatively on open

surgery or EVSG repairs, in both the adult and pediatric populations[6,48,99,100]. The effects of therapy are illustrated in figure 22[31]. Blood pressure control has become well accepted in the early management of suspected or documented BATI[8]. Yet, caution is warranted with sudden or progressive hypotension, since it may be difficult to distinguish drug effect from volume vasodilation, or bleeding.

and anesthesia induction can increase systemic pressure.

For endovascular techniques, the principle of arterial pressure reduction is not unanimous, and maintaining a mean BP > 70, or systolic pressure of 100-120 is the goal during deployment of the device[110,111,112]. With associated head injuries higher systemic pressures should also be maintained[112,113].

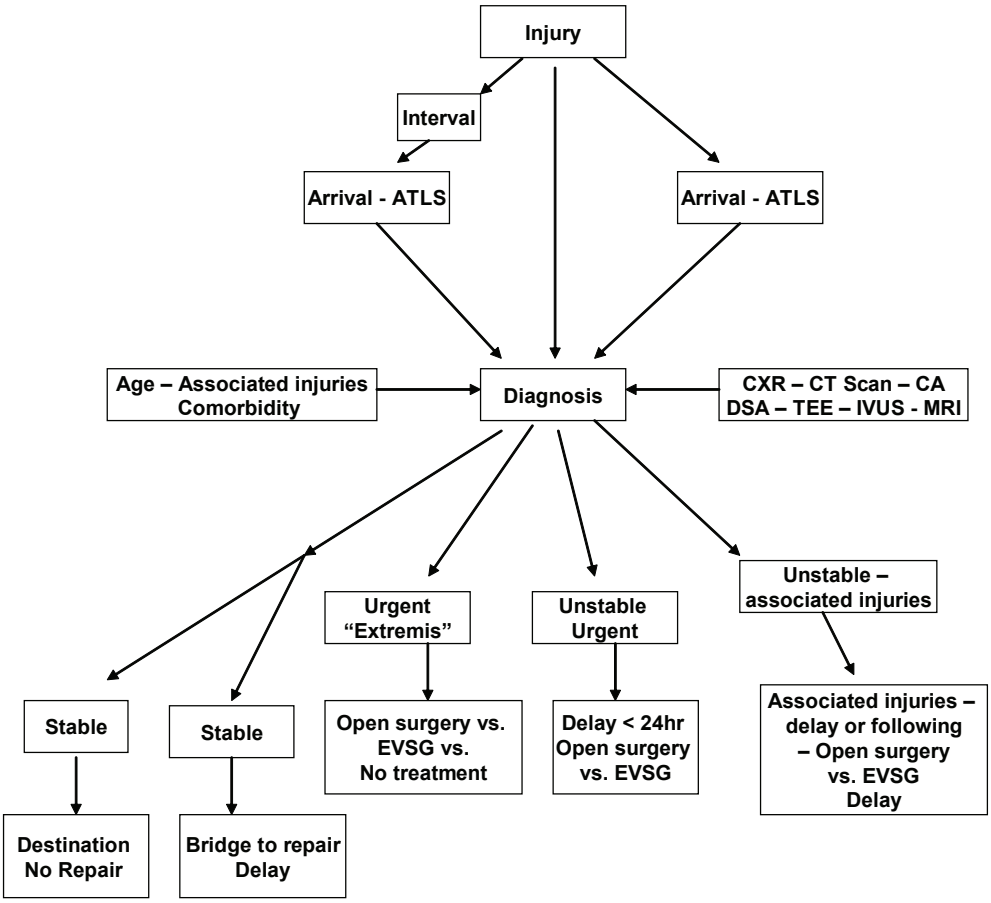


Fig. 21 : Contemporary Algorithm with more diagnostic and EVSG options

The major therapeutic intravenous agents include: esmolol, propranolol, labetalol, sodium nitroprusside, enalaprilat, nicardipine, fenldopam, and nitroglycerin (table 7). Caution re. afterlaod reduction is warranted, since these agents, especially nitroprusside have been implicated in causing spinal cord ischemia[107,108]. Pate et al[109] also cautioned that anti-hypertensive treatment, as well as volume restriction, should be initiated in patients with initial adequate blood pressure and urinary output. Further, treatment should be maintained throughout the diagnostic process since increased stress during transportation, manipulation,

Prioritization of treatment must be considered in patients with BTAI, as multiple associated injuries are frequently present. Camp et al.[70] in 1997 evaluated 395 cases over 11 years from 14 regional trauma centers. 102 cases were categorized as extremis. 99 died shortly after admission, and 3 died after reaching the operating room. BTAI was the primary cause of death in 56 (55%). This means 45% died from associated injuries, thus highlighting the fact that sicker patients are reaching the hospital.

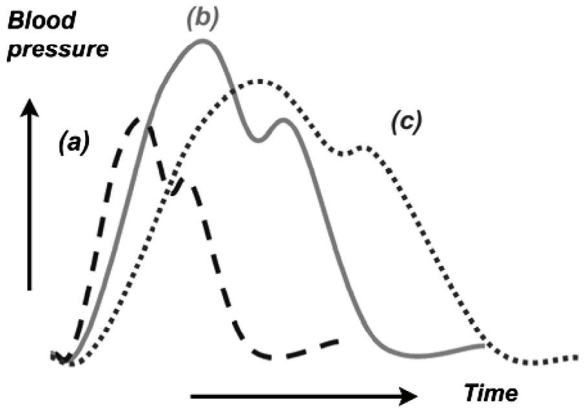
able 7[*#] : Parental antithypertensive drugs

Drug	Dose	Onset/ Duration
Esmolol (Brevibloc)	Bolus: 500mcg/kg IV, repeat after 5 minutes. Infusion:50-100mcg/kg mn,upto300mcg/kg/min. Titrate to systolic pressure of 100mmHg and heart rate <100 beats/minute	Onset:1-5min Duration:15-30-min
Labetalol (Normodyne)	Bolus : 20mg IV x1,then 20-80mgq10 in to maximum dose 300mg. Infusion : 0.5-2mg/min.	Onset: 5-10 min. Duration: 3-6 hr.
Nitroglycerin	Initial: 5mcg/min. IV Maintenance: titrate q 3-5 min to 100mcg/min	Onset: 2-5 min. Duration: 5-15 min.up
Sodium nitro-prusside (SNP)	Initial: 0.2-0.5 mcg/kg/min continuous IV infusion. Maintenance: titrate to goal BP (systolic 90-100); Up to 8-10 mcg/kg/min continuous infusion	Onset : Seconds Duration : 2-3 min
Enalaprilat (Vasotec)	Initial : 1.25 mg IV over 2-5 min. q 6 hours, then increase up to 5 mgs. q 6 hours.	Onset : 15 min. Duration: 11 hrs.
Nicardipine (Cardene)	Initial: 5mg. /hour continuous infusion, up to 15mgs./hour.	Onset: 10 min. Duration: 2-4 hrs.
Fenoldopam (Corlopam)	Initial: 0.01 mcg./ kg./ min IV, titrated up to 0.3 mcg./kg./min.	Onset: 2-3 min. Duration: 30 min.

*Cooper DH. Hypertensive Emergencies. In: Kollef MH, Bedient TJ, Isakow W, Witt CA.The Washington Manual of Critical Care. Woltors Kluwer/ Lippincott Williams & Wilkins 2008; page 149-150.

Brenner M. Critical Care Medicine, 2007 edition. Current Clinical Strategies Publishing. Laguna Hills, CA. 2007. P. 55.

Major long bone and pelvic fractures are common, with 49% requiring early operative intervention. Pelvic fractures, especially with associated retroperitoneal hematomas, have a high mortality[114]. This injury is particularly significant since operative positioning is hazardous. With respect to concomitant intra-abdominal solid organs injury, the FAST (focused abdominal sonography for trauma) evaluation is extremely valuable in ruling out abdominal injury, as well as pericardial effusions/tamponade. Treatment can be delivered in one of three ways: exploratory laparotomy with delayed repair of the aorta; exploratory laparotomy with subsequent repair of the aorta during the same operation; or aortic repair prior to addressing concomitant solid abdominal organ injuries. Only for hemodynamically stable patients with known BTAI and associated solid organ intraabdominal injury, without ongoing hemorrhage, should repair of the aortic injury take precedence. A positive FAST for pericardial effusion warrants immediate evaluation. This requires a subxyphoid window. If positive, then conversion to a median sternotomy is warranted. Medical anti-shock trousers (MAST) should not be used in patients with suspected BTAI, since increased afterload, and systemic pressure may aggravate the injured aorta[4].



Pharmacologic anti-impulse therapy. Diagram of aortic pressure curves under various conditions. The continuous line (B) represents the baseline state. Administration of a vasodilator agent such as nitroprusside is represented by the dashed curve (A). There is significant decrease in pressure levels and acceleration in heart rate, but this is accompanied by a steepest slope of the ascending portion of the curve (increased dp/dt_{max}). Beta-blockade administration is represented by the dotted line (C). Although the degree of pressure lowering is usually smaller, the drug's negative inotropic and chronotropic effects result in decreased impulse and dp/dt_{max}

Fig. 22 : Pharmacology antipulse therapy diagram of aortic pressure curves

Tatou et al.[115] noted an incidence of 21.3% of BTAI having an abdominal procedure prior to aortic repair. Santaniello et al.[116] concluded that nonoperative management of grade I and II liver and spleen lacerations who undergo systemic anticoagulation for aortic repair (utilizing partial left heart bypass) is a safe approach and associated with no statistically significant impact on transfusion rates, length of stay (LOS), or mortality. Fabian[117] has nicely outlined laparotomy damage control for acute and chronic trauma. He emphasizes the concept of the compartment syndrome where ischemia and soft tissue injury leads to edema and increasing tissue pressures. This requires open abdomen techniques and more required time perioperatively for stabilization.

Cardiac contusion has decreased from 62% to < 9%% of patients with BTAI, yet the role of TEE has been invaluable in determining the extent of cardiac dysfunction, or presence of anatomical injury[7,48,74]. This subgroup of patients suffers higher rates of perioperative arrhythmias, cardiac arrest, ARDS and mortality[118]. The AAST has classified blunt cardiac

injury (BCI) into scales I-VI. Scale I is minor ECG changes (non-specific ST or T wave changes, PAC, PVC, or persistant tachycardia). The extreme scale VI is lethal blunt avulsion of the heart[23].

25% have sustained severe closed head injuries and require intracranial pressure monitoring[7,15.] Closed head injuries accompanying BTAI represent a challenging task, with often increased mortality, and surgical management of BTAI should be addressed after a neurosurgical procedure. Chestnut[113] has nicely reviewed traumatic brain injuries. He points out that cerebral perfusion pressure (CPP) equals mean systemic arterial pressure (MAP) minus intracranial pressure (ICP). ICP is directly proportional to central venous pressure (CVP). Cerebral pressure autoregulation maintains cerebral blood flow (CBF) over a CPP range of 50-150 mm Hg. This highlights the need to maintain higher MAP and lower total body volume. Neurological non-invasive monitoring is helpful in tracking trends and sudden changes (e.g. Cerebral oxygen saturation—Invos Cerebral Oximeter, Somanetics Corp., Troy, Michigan, USA). The goal is to not to precipitate increases in intra-cranial pressure (ICP), particulary with increased volume loading.

If indicated, heparinless or decreased heparin (ACT<150 with heparin coated bypass systems with centrifugal pumps, or endovascular stent-graft techniques) are recommended[113,119]. A shortened delay in the setting of severe CNS injuries may not be sufficient enough for prognostication. Delaying the aortic repair and applying the principle of "intended reduction of aortic wall stress (dP/dT)", with maintaining mean arterial pressure between 60-80 mmHg, may complicate the management of patients with head injuries and impaired cerebrovascular regulatory mechanisms[113].

Delayed (bridge) or Non operative (destination) Treatment

Though urgent intervention of well documented BTAI is the recommended therapeutic approach for unstable patients without associated injuries, there is a subgroup of selected patients who may benefit from delayed aortic repair (defined here as planned delay in intervention any time 14-18 hours from admission to operation or non-operative treatment)[7,8,109]. This also includes patients with delayed admission or transfer >24 hours after injury. As noted, patients with associated injuries, especially severe head injury, cardiac injury, unstable intra-abdominal hemorrhage or extensive pelvic fracture, ongoing sepsis and major burns, severe multi-organ trauma (high ISS) with poor physiologic reserve, massive lung contusion with ARDS and respiratory compromise, coagulopathy and no signs of impending aortic rupture or rapid growth of aortic pseudoaneurysm, are all relative indications for expectant or delayed approaches[100,109,120-122]. In recents years patients with increased ISS, as well as patients with minimal aortic injury (MAI) are being seen. This is a reflection of faster and more effective treatment at the scene, more rapid transfer, improved initial treatment (ATLS), and improved/ rapid diagnostic screening.

The history and evolution of delayed management was empiric and anecdotal. Rice[123] in 1951 recognized hypertension as the cause of death in a pregnant woman with a distant chest injury. The medical treatment (Wheat regimen of antihypertensive therapy) was shown to be effect in type B thoracic dissections in 1965[124]. Aronstam[125] in 1970 treated 2 acute traumatic aortic injuries initially medically, followed by elective repair. Fox et al.[126] in 1979 noted the occurrence of acute hypertension in BTAI. Two theories emerged. The first was the pseudocoarctation syndrome of upper body hypertension. The second and most plausible was the positive feedback spinal reflex mechanism related to thoracic aortic stretch. The presence of sympathetic afferent nerve fibers in the aortic isthmus area responding to stretch with reflex hypertension was postulated as the cause.

Akins et al.[120] from Massachusetts General Hospital popularized the` concept of delayed repair in patients with severe associated injuries as noted above. These injuries were more qualitative than specific[100]. Maggisano et al.[121] developed more quantitative associated injuries. These included :

1- Head injury- GCS <6 or intracranial bleeding on CT scan;

2- Pulmonary injury- PaO2/FiO2 <200, or inability to tolerate one lung ventilation;

3- Cardiac injury requiring pharmacological support;

4- PT and PTT >1.5 normal, despite attempted correction. Holmes et al[122] included an INR >1.5, Platelet count <100, 000, and age > 55 years.

As noted, since that time, individualized approaches have evolved with early repair for both stable and unstable patients, with no prohibitive associated injuries or comorbidities, and delayed repair for unstable patients with severe associated injuries[109,121,127-130]. Through 2000, it is estimated that more than 500 patients have been treated with a delayed management protocol[13]. It is estimated that more than 20% of patients may not be candidates for early repair[122].

The cornerstone of delayed management is the principle of permissive hypotension and "intended reduction of aortic wall stress (dP/dT)" by intravenous β-blockers and the selective use of vasodilatory agents

when necessary (figure 22) (table 7)[31]. Intensive care unit and invasive cardiovascular monitoring (arterial and central venous pressure) for adequate resuscitation and monitoring of adequate tissue perfusion status are fundamental components of delayed repair or non-operative management[109].

Results/ Complications

In a ten-year follow-up of delayed management, Pacini et al.[128] proposed that all trauma patients with BTAI who arrive alive in the ED, without signs of impending aortic rupture or rapid growth of pseudoaneurysm present, should be considered for delayed planned aortic repair after the resolution of all other significant associated injuries. Less than 10 % of their patients required conversion to urgent repair, with decreased mortality to 4.2 % and no recorded spinal cord ischemia, regardless of the type of repair (open with total cardiopulmonary bypass, partial left heart bypass, or endovascular graft stenting).

Wahl et al[85], in their multicenter retrospective review, found no statistically significant mortality benefit to delayed repair, but highlighted a significant increase in length of ICU stay and a two-fold increase in cost-analysis. Both Langanay[129], and Hemmila[130] stress the importance of delayed management in the setting of severe associated injuries, with no resultant danger of interval rupture and death. The recent AAST study revealed increased time interval from diagnosis to treatment (16.5 hours to 54.6 hours). This is related to increased ISS, associated injuries, and planned delayed medical management, including permissive hypotension[7,8].

Pate et al.[109] has championed the delayed approach. In 15 patients surgical repair was performed at 2 days to 6 months. The mortality was 13.3%.11 patients had non operative treatment with 54.5% mortality from associated injuries. In neither group was there spontaneous aortic rupture. There was no pseudoaneurysm enlargement in 4 non operated patients at 18-49 months follow up. Holmes et al.[122] reported 10 nonoperative patients at a median survival of 2.5 years without progression of injury, or need for operation.

A subset of BTAI (10%), involving only an intimal flap (MAI), has been considered an indication for nonoperative treatment with no delayed repair. Kepros et al.[99] found no complications related to the aortic injuries during a mean follow-up of 16.8 months. Malhorta et al.[11] had > 90 % success rate with nonoperative management, no recorded complications, and complete resolution of MAI in 3 weeks for 50 % of patients, and stable pseudoaneurysm formation in 40 %. Hemmila et al.[130] in 2004 compared their series of delayed repair (DR) with early repair (ER), and compared the results with the National Trauma Data Bank (NTDB). DR was established to be 16 hours. Beta blockers were used in the DR group (goal of systolic pressure <120 and heart rate < 100). They noted a decrease in rupture in the treated group to 1.5%, compared to the earlier AAST study of 12% in patients not treated with antihypertension strategy[7]. They also noted increased complications, higher hospital and ICU length of stay (LOS), as well as ventilator time. This was reflective of higher GCS and Head/Neck AIS in DR vs ER groups.

However, the results of initial delayed approach or the long-term natural history of BTAI treated with delayed or nonoperative technique is not known and a cautious surveillance plan should be exercised with serial TEE, MRI, CTA or helical CT during hospitalization and following discharge. Fattori et al.[18] used surveillance MRI. As noted, they found MRI more accurate in assessing the evolution of the periaortic hematoma. Long-term complications include : 1) formation, enlargement or rupture of pseudoaneurysm, 2) thomboembolic events by loose intima or thrombus, 3) progressive dissection of the aortic wall and, 4) fistulization with formation of aorto-bronchial, aorto-esophageal or aorto-pleural fistula. Mortality with this approach ranges from 0 % to 14%[48,71,109,122,128].

(To be continued)

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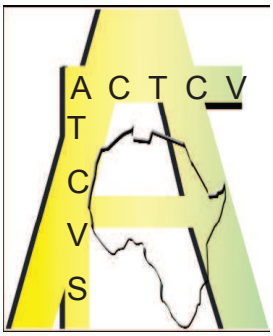

CHIRURGIE CARDIAQUE/CARDIAC SURGERY

MODIFIED DE VEGA ANNULOPLASTY FOR FUNCTIONAL TRICUSPID VALVE REGURGITATION

M. TETTEY, L. SEREBOE, F. EDWIN, E. ANITEYE, D. KOTEI, M. TAMATEY, K. ENTSUA-MENSAH, Y. ADAMU, K. FRIMPONG-BOATENG

Correspondence : Dr M. TETTEY, National Cardiothoracic Centre.
Box 77 Korle Bu, Accra, Ghana
E-mail : mawu @hotmail.com

ABSTRACT

Objectives: Tricuspid valve regurgitation is mostly functional and secondary to mitral valve and or without aortic valve diseases. Modified De Vega annuloplasty is one of the effective methods used in the surgical correction of functional tricuspid valve regurgitation. This study evaluates the long term results of modified De Vega annuloplasty for functional tricuspid valve regurgitation at the National Cardiothoracic Centre.

Patients and Methods: From March 1993 to July 2005, 64 consecutive patients who had modified De Vega annuloplasty for tricuspid valve regurgitation (TVR) were retrospectively selected for the study. The preoperative echocardiographic records of all patients with TVR were retrieved. The age, sex and the specific heart diseases were recorded. All the patients had follow up echocardiography at least once every two years and the tricuspid valve function reported from the latest post-operative echocardiographic reports were recorded.

Results: The average follow-up period after tricuspid valve repair was 61.3 months (5-133 months). Six (9.4%) had grade II post-operative tricuspid valve regurgitation and 17 (26.5%) had grade I post-operative tricuspid valve regurgitation. No post-operative regurgitation was seen in 41 (64.1%) of those who had modified De Vega annuloplasty. Mitral valve incompetence mostly secondary to rheumatic heart disease accounted for 81.3% (n= 52) of the causes of functional tricuspid valve regurgitation. Combined mitral and aortic valve regurgitation accounted for 9.4% (n = 6), mitral valve stenosis 7.8% (n = 5) and one (1.6%) case of functional tricuspid valve incompetence was associated with atrial septal defect. There was no post-operative heart block and no suture tear was observed in the 64 cases that had the modified De Vega annuloplasty. There were three deaths during the period of review. One died 9 years after surgery from arrhythmia and two died from post-operative dilated cardiomyopathy in their second year after the surgery.

Conclusion : Modified De Vega annuloplasty for tricuspid valve regurgitation is an effective, safe and a simple procedure to perform. In a developing country like Ghana, where most of our patients are poor and can not easily afford ring annuloplasty, De Vega annuloplasty remains the procedure of choice in the management of functional tricuspid valve incompetence.

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RÉSUMÉ

Objectif : *L'insuffisance tricuspidienne est le plus souvent fonctionnelle et secondaire à une valvulopathie mitrale avec ou sans une valvulopathie aortique associée. L'annuloplastie tricuspidienne de type De Vega modifiée est une des corrections efficaces de toute insuffisance tricuspidienne fonctionnelle.*

Le but de cette étude est l'évaluation à long terme de l'intervention de De Vega modifiée en cas d'insuffisance tricuspidienne fonctionnelle au Centre National Cardio-thoracique d'Accra.

Patients et Méthodes : *De Mars 1993 à Juillet 2005, 64 patients consécutifs ayant bénéficié d'une annuloplastie de De Vega modifiée ont été rétrospectivement étudiés. Les données pré-opératoires échocardiographiques ont été recencées. L'âge, le sexe, le type de valvulopathies ont été enregistrés. Tous les patients ont eu un suivi échocardiographique au moins 1 fois tous les 2 ans.*

Résultats : *Le suivi moyen de nos patients était de 61.3 mois (5 – 133 mois). Six patients (9.4%) avaient une régurgitation tricuspidienne post-opératoire grade II, 17 (26.5%) un grade I et 41 patients (64.1%) ne présentaient aucune régurgitation*

tricuspidienne post-opératoire. L'insuffisance mitrale rhumatismale était la cause de l'insuffisance tricuspidienne fonctionnelle dans 81.3% des cas (n = 52), la sténose mitrale dans 7.8% des cas (n = 5) et dans un cas (1.6%) une communication inter-atriale était associée à l'insuffisance tricuspidienne fonctionnelle.
Aucun bloc auriculo-ventriculaire complet post-opératoire et aucun lâchage de l'annuloplastie tricuspidienne n'ont été observés chez nos 64 malades. Trois décès post-opératoires lointains ont été notés ; la cause de décès a été une arythmie, 9 ans après la chirurgie (1 cas) et une cardiomypathie dilatée post-opératoire, 2 ans après la chirurgie (2 cas).
***Conclusion :** L'annuloplastie tricuspidienne de De Vega modifiée est efficace, simple à réaliser et sans danger. Elle s'avère utile dans les pays en voie de développement comme le Ghana où les patients sont économiquement faibles ; elle reste, chez nous, la procédure de choix dans le traitement de toute insuffisance tricuspidienne fonctionnelle.*

Introduction

Isolated tricuspid valve regurgitation is rare [1]. This is mostly organic tricuspid valve regurgitation and is usually due to trauma, endocarditis, carcinoid heart disease or rheumatic heart disease. However, 80% of patients with TVR are functional and mostly secondary to mitral and/or without aortic valve diseases[2] . Functional TVR is a progressive disease[3] and mainly due to dilatation of the annulus and always reflect some degree of right ventricular failure with elevated pulmonary vascular resistance. Fonctional TVR with left side valve disease mosthy signals also, a right ventricular dilatation which is a marker for late neglected valvular disease[4]. This is not an uncommon situation in a developing nation like Ghana where sourcing for funding for open heart surgery delays early surgical intervention. Other causes of functional tricuspid valve regurgitation include cor pulmonale, myocardial infarction, dilated cardiomyopathy, endomyocardial fibrosis and pulmonary hypertention.

Surgical management of functional TVR remains an important procedure as part of the management of some cases of mitral and aortic valve disease.

There are many techniques described in the management of functional TVR. These include annular plication, suture annuloplasty, ring annuloplasty and rarely tricuspid valve replacement. Annuloplasty is feasible in most patients with functional TVR. Annular plication is used in patients who have functional tricuspid valve with organic change in the posterior valve leaflet. Valve replacement is considered only for those patients whose tricuspid valve has severe organic change.

Several tricuspid annuloplasty suture techniques have been developed with variable outcomes. The aim of this study is to review our experience with modified De Vega annuloplasty and the follow up results after 12 years.

Patients and Methods

During a twelve year period from 1993 to 2005 at the National Cardiothoracic Centre, 64 patients had modified De Vega annuloplasty done for functional TVR. A retrospective review of patient records, echocardiographic reports and clinical outcome was carried out to ascertain the efficacy of the procedure.

Preoperative echocardiographic records of all the patients with TVR were retrieved. The age, sex and the specific heart diseases were recorded. The post-operative echocardiographic reports for the same patients and the tricuspid valve function after modified De Vega annuloplasty were reviewed. All the patients had follow- up echocardiography at least once every two years and the tricuspid valve function reported from the latest post-operative echocardiographic reports were recorded.

Procedure

A cardiopulmonary bypass was established. A right atriotomy was done and procedure was performed using 3-0 or 4-0 pledgeted prolene sutures with double needle. The initial suturing was from the antero-septal commissure to the postero-septal commissure and was similar to the original De Vega procedure. From the antero-septal commissure the second suturing was carried out in the same direction but in a spiral fashion around the annulus and the first suture. The two ends of the suture through a pledget were adjusted and the valve tested by injecting saline into the right ventricle. The suture was tied when leakage from the tricuspid valve is negligible.

Results

Sixty four patients were included in the study. Thirty five (55%) were females and twenty nine (45%) males. The age distribution is shown in table 1.

Table 1 : Age Distribution

Age (years)	No of patients
0-10	7
11-20	30
21-30	7
31-40	10
41-50	5
51-60	4
61-70	1
TOTAL	64

Within the period of review, 589 cases of valve surgery were carried out. Of these, 64 were associated with functional TVR. The average follow-up period after the tricuspid valve repair was 61.3 months (5-133 months). Forty one patients (64.1%) had no TVR, seventeen patients (26.5%) had grade I post-operative TVR and six patients (9.4%) had grade II TVR. Mitral valve regurgitation secondary to rheumatic heart disease accounted for 81.3% (n = 52) of the cases of functional tricuspid valve regurgitation. Combined mitral valve and aortic valve incompetence and isolated mitral valve stenosis accounted for 9.4% (n = 6) and 7.8% (n = 5) respectively. There was one case of functional TVR associated with atrial septal defect. Three deaths occurred during this period. One died 9 years after surgery from arrhythmia and two died from post-operative dilated cardiomyopathy in their second year after the surgery.

Discussion

Functional TVR is a progressive disease and the incidence is not well known. Some researchers have questioned the functional nature of TVR accompanying left side valve disease[3,5]. It is now becoming clear that even moderate degrees of TVR are unlikely to regress spontaneously after correcting a left heart valvular lesion[2,5]. As suggested by some studies, it is possible that the annular dilatation is at least partially organic[2,5].

Tricuspid valve regurgitation remains a challenge in terms of its precise diagnosis, indications and appropriate surgical treatment[2]. It has been found that preoperative echocardiography based on tricuspid valve grading test does not compare well with tricuspid dilatation found at surgery[6]. Under general anaesthesia intra-operative echocardiography is not very useful to quantify the tricuspid valve regurgitation in order to indicate valve repair or to assume valve competence after correction[6]. In this study, the indication of tricuspid valve repair was based on clinical, echocardiographic and surgical findings. Intra-operative filling of the right ventricle with cold Ringer's lactate was used to test the severity of TVR. Intra-operatively, some authors[6] recommended surgical treatment of annulus > 70mm between the antero-septal and postero-septal commissures or surgically treated the TVR when indexed annulus dimension is more than 21mm/ m^2. These have been effective in terms of clinical improvement and late functional results[6]. Patients with moderate and severe TVR should have repair since it is widely demonstrated that in these patients, tricuspid annuloplasty provides better symptomatic results and may improve survival[6]. Despite correction of a left sided pathologic condition, TVR may persist or recur and produce persistent continuous mobidity[4].

There are three general techniques for tricuspid annuloplasty proposed to correct functional TVR[7,8]. These are annular plication, annular ring insertion and semi-circular annuloplasty by Kay an Reed[1]. Others techniques are the use of plantaris tendon graft for atrio-ventricular valve repair[14] and fixed pericardial C- shaped strip for tricuspid valve repair[4].

Disappointed with the results of annular plication and basing their technique on the pathologic anatomic studies, semi-rigid and flexible Carpentier-Edwards annuloplasty rings or flexible Cosgrove-Edwards annuloplasty rings or modified semi-circular annuloplasty were developed[1,9,10,11,12,13]. Goksin I and al[13] has noted that the results of ring annuloplasty are superior to annular plication and semi-circular and ring annuloplasty[13].

The De Vega technique was first introduced by De Vega in 1972 in Madrid[13]. It reduces the amount of intra-cardiac prosthetic material, maintain annular flexibility and reduce potential for conduction system injury[1,13]. It is one of the most effective methods used in surgical correction of functional TVR and has since been modified by others. Revuelta and Garcia –Rinaldi technique[6] was described in 1989, and consisted of the use of interrupted sutures bolstered in Teflon pledgets and placed around the posterior and anterior segments of the tricuspid annulus. Modified De Vega annuloplasty (Sagban's Annuloplasty[13])

uses spiral sutures after the first suture around the posterior and the anterior segments of the tricuspid annulus. The aim of this annuloplasty was to prevent recurrent tricuspid valve regurgitation secondary to bowstring (guitar string) phenomenon seen in the original De Vega annuloplasty[13]. The modified De Vega annuloplasty carried out in this study is similar to the Sagban's annuloplasty[13].

The results of modified De Vega annuloplasty varies in different hospitals. In this study, 90.6% of patients who underwent modified De Vega annuloplasty for TVR had grade I or no regurgitation at all after surgery. None of our patients needed reoperation or replacement of the tricuspid valve. With the average follow-up period of 17.8 months, a study done elsewhere showed that after modified De Vega annuloplasty, 66.7% had no regurgitation after the procedure [7,13]. This is comparable to our results where 64.1% of our patients had no TVR after repair. In another study after 20 months follow-up, none of the 63 patients who underwent De Vega annuloplasty had moderate or severe TVR[15]. Using the modified De Vega annuloplasty in 399 patients with functional TVR associated with left heart valve disease, only three (0.75%) had severe recurrent TVR which necessitated tricuspid valve replacement[2]. Our results compare favourably with these findings. In a 25 year clinical experience with repair of tricuspid insufficiency, Carrier et al[10] showed that De Vega annuloplasty, Bex linear reducer and Carpentier-Edwards prosthetic ring annuloplasty resulted in low rate failure and in good patient survival at long term follow-up.

However, in an article by McCarthy et al[9], looking at the durability and risk factors for failure in tricuspid valve repair, it was found out that risk factors for worsening regurgitation included repair type other than ring annuloplasty. This study included 790 patients over 9 year period who underwent tricuspid valve annuloplasty for functional regurgitation using four techniques: Carpentier-Edwards semi-rigid ring, Cosgrove-Edwards flexible band, De Vega procedure and customized semi-circular Peri-Guard annuloplasty. Gilbert et al[16] also made a similar observation where annuloplasty ring in patients undergoing tricuspid valve repair is associated with improved survival as compaired to semicircular annuloplasty.

The tricuspid valve reoperation-free survival rate varies from different hospitals. In a study mentioned by Mac Cathy[9], the survival rate at 5 years was 90±2% and 84±2% at 8 years[9]. The 15-year freedom from reoperation rate was 91.6% in a study looking at the long term results of De Vega annuloplasty[17]. Tricuspid valve reoperation rate of < 1% and 3% were recorded at some centers[3,9]. In this study there was no reoperation. None of our patients developed severe regurgitation to necessitate a reoperation. The hospital mortality for modified De Vega annuloplasty in some centers varies from 1.6 to 8.9% [2,3,13,17]. In our study their was no hospital mortality. The death of three patients that occurred after they were discharged from hospital was unrelated to the procedure.

Conclusion

Modified De Vega annuloplasty is a simple, cost effective and reliable procedure in the management of tricuspid valve regurgitation. In our environment where most of our patients cannot afford ring annuloplasty, modified De Vega annuloplasty could be the procedure of choice.

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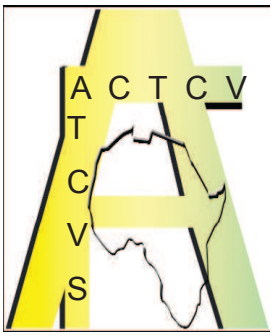

CHIRURGIE CARDIAQUE/CARDIAC SURGERY

THROMBOLYSIS FOR PROSTHETIC VALVE THROMBOSIS: A REPORT OF 6 CASES AND REVIEW OF THE LITERATURE

*E.A. ANITEYE, M. TETTEY, L. SEREBOE, D. KOTEI, F. EDWIN, M. TAMATEY, A. DOKU, K. ENTSUAH-MENSAH, H. BAPTISTA, K. FRIMPONG-BOATENG

Correspondence : Dr. Ernest A. Aniteye, MB CHB, FWACS, FRCA, FGCS
Department of Anaesthesia
University of Ghana Medical School
P. O BOX 4236, Accra
E Mail: aniteyeernest@yahoo.com

Summary
Objectives: To determine the outcome of thrombolysis in patients with Prosthetic valve thrombosis (PVT).
Design: A retrospective descriptive study.
Setting: The intensive care unit of the National Cardiothoracic centre, Korle-bu Teaching Hospital, Accra, Ghana.
Subjects: 5 consecutive patients who were thrombolysed for 6 episodes of prosthetic valve thrombosis.
Patients and Methods : Over a 3- year period 5 patients underwent a total of 6 thrombolytic sessions. All the patients were symptomatic and diagnosis had been confirmed by echocardiography. Streptokinase was used in 5 of the session. 1.5 million International units (IU) was used in the adults and 750,000IU in the 13 year old. One patient had 2.0 million units of urokinase. The infusion was by the short course thrombolytic method over 90 minutes.
Results: There were 6 episodes of thrombosis out of 142 (5.0%) valve replacements during the study period. The mean age was 29.5 $\pm$ 11.2 years (range 13-48years). The time from insertion of prosthetic valve to thrombosis was 15.5 months (range 1 week – 2 years). The INR was sub-therapeutic in 5 (83.3%) of the patients. Streptokinase was used in 5 (83.3%) and urokinase in 1 (16.7%) of the patients. The overall success was 83.3%. Thombolysis was completely successful in 3 (50.0%) and partially in 2 (33.3%). There was no response to thrombolysis in one patient who died after 14 hours.
Conclusion: Thrombosis of prosthetic heart valves is not common from our series. Thrombolysis using streptokinase should be the first line management as it is cheap and relatively safe in the management of such cases.

Key words: Thrombosis, thrombolysis, Prosthetic valve.

Introduction

Prosthetic heart valve disease may be rarely complicated by thromboembolism, bleeding, endocarditis and valve dysfunction from pannus formation[1]. Of these thromboembolism of a mechanical prosthetic valve is the most serious as it leads to severe haemodynamic decompensation including shock and acute heart failure[1-3]. Thrombosis may also complicate pannus formation. Until recently the management prosthetic valve thrombosis (PVT) was mainly by re-operation where a thrombectomy or replacement of the valve is done[2]. Re-operation is usually by cardiopulmonary bypass and because most of the patients are in intractable heart failure there is a high mortality [4-5]. Many workers have advocated thrombolysis as the first line management PVT using the rapid infusion or the slow infusion method[6].

The mortality of the PVT is related to NYHA class of heart failure at the time of presentation, with NYHA IV usually having a poor prognosis [6-7].

The intensive care unit of the Cardiothoracic Centre has for the past 3 years treated 5 patients who had 6 episodes of PVT. This study therefore looks at management of these cases as well the outcome in terms of morbidity and mortality.

Method

Using the intensive care, admissions and discharge register, the report books and the patients case notes, patients who had thrombolysis for prosthetic valve thrombosis between 1st January 2003 and 31st December 2006 were studied. The clinical presentation, NYHA Class of heart failure, the initial INR, and echocardiographic information were also looked for.

The patients were all thrombolysed in the intensive care unit of the Cardiothoracic Intensive Care Unit. All the patients had invasive monitoring through a radial arterial and a central venous line. Inotropic support by dopamine and adrenaline infusions was started as part of the protocol for management of such cases. After pre-thrombolytic therapy of intravenous methylprednidolone 250mg and Promethazine 12.5mg, 5 patients were administered streptokinase in and 1 urokinase. After a test dose of 20,000 IU units, each patient was administered 1.5 million units of streptokinase in the adults and 750,000 units in the adolescent. Two million units of urokinase was administered to one patient who had previously been administered streptokinase. All the thrombolytics were infused over a 90 minute period.

Complete hemodynamic success was defined as return of the transvalvular gradient to normal. Partial success was defined as partial improvement in gradient without complete normalization of the valve movements.

The data was analysed using SSPS (Microsoft 2003).

Results

There were 6 episodes of PVT in 6 patients out of a total of 142 valve replacements during the study period. The age range was 13-48 years (mean 29.5±11.2 years). There was a male to female ratio of 2:1. The mitral valve was involved in 5 (83.3%) of the episodes with the aortic valve being involved in 1 episode. Five (83.3%) of the patients had sub-therapeutic INR. These are depicted in table 1 below.

All the patients presented with pulmonary oedema, 3 (50.0%) were hypotensive and 1 (16.7%) was in shock with multi-organ dysfunction. The mean time from insertion of the valve till thrombosis was 15.5±11.2 months with a range of 7 days to 24 months. Three (50.0%) of the patients were in NYHA IV and 3 (50.0%) in NYHA III.

Table 1: Showing age, sex, age of valve, INR, clinical signs and NYHA class.

Age	Sex	Valve thrombolysied	Age of valve mouths	INR	Clinical signs	NYHA Class
13	M	Mitral bileaflet	2	1,5	Pulmonary oedema	III
29	M	Mitral bileaflet	21	1,3	Pulmonary oedema hypotension	II
29	M	Mitral bileaflet	24	1,7	Pulmonary oedema hypotension	IV
31	F	Mitral bileaflet	22	1,2	Pulmonary oedema hypotension	IV
27	F	Mitral bileaflet	24	2,0	Pulmonary oedema	III
48	M	Mitral bileaflet	0,25 (7 days)	1,5	Pulmonary oedema shock Multi organ dysfunction	IV

Streptokinase was used in 5 (83.3%) of with urokinase in 1 (16.7%) for thrombolysis.

Thrombolysis was successful in 3 (50.0%) of the patients with a partial success in 2 (33.3%). The overall success rate was 83.3%. The patients with partial success later had re-operation. The average time to improvement of haemodynamic signs was 4.4 ± 2.2 hours with a range of 2-8 hours. These are seen in table 2.

Table 2 : Thrombolytic, time of improved function, success and outcome of thrombosis.

Valve throbosed	Thrombolytic used	Time of improved Function/Hrs	Success of thromboysis	NYHA Class	Complications of thrombolylis	Outcome
Mitral	Streptokinase	2	Complete	III	Allery Hypotension	Alive 3 years
Mitral	Streptokinase	6	Complete	III	Allery	Rethrombosed In 3 months
Mitral	Streptokinase	5	Complete	IV	Allery	Alive 4 years

Meanage 29.5 ± 11.2 years, mean valve age 15.5 ± 11.2

Streptokinase was used in 5 (83.3%) of with urokinase in 1 (16.7%) for thrombolysis.

Thrombolysis was successful in 3 (50.0%) of the patients with a partial success in 2 (33.3%). The overall success rate was 83.3%. The patients with partial success later had re-operation. The average

time to improvement of haemodynamic signs was 4.4 ± 2.2 hours with a range of 2-8 hours. These are seen in table 2.

One of the patients who had a partial success from thrombolysis from use of urokinase died after redo-surgery. The commonest complication was allergy (66.7%) and this was from the use of streptokinase. The two patients who died were in NYHA class IV.

Four of the patients who survived the management of their PVT are still alive 39-48 months after the thrombotic events.

Discussion

Prosthetic valve thrombosis though infrequent is usually dreaded by most physicians because to the severe haemodynamic complications. After PVT patients can present with hypotension, pulmonary oedema, embolic phenomenon or more seriously cardiogenic shock [1-3]. The incidence of left sided PVT is reported to be between 0.5 – 8% but this increases to 20% in right sided valves especially in prosthetic tricuspid valves [7]. The institutional incidence of PVT in our study (5.0%) is within this range. Another study by Sivasubramanian who use the same Sorin bileaflet valves as our institution had an incidence of 6.7% [8].

Renzulli cited the most significant risk as tilting disc prostheses, prostheses without pyrocarbon coating, large prostheses, tilting disc prostheses with a small orifice posteriorly oriented, atrial fibrillation, enlarged left atrium and time from implant greater than 4 years. The mitral valve from previous studies has been found to be more commonly involved in left sided PVT and this agrees with our finding of 83.3% [1,4-6] . The patient with the aortic valve had a cage-ball valve all the other patients had Sorin bileaflet valves. Rizzoli et al in their study demonstrated that the relative risk of thrombosis was 12 times higher for the tricuspid prosthesis and seven times higher for the mitral prosthesis [10]. Rizzoli and his colleague also showed that a 69% risk reduction if Sorin tilting valves were used and this risk reduced further to 83% with Sorin bileaflet valves, the common valve used in our institution.

Many studies have shown a correlation between PVT and sub-therapeutic INR. Most of the patients with PVT in those studies had INR below 2.0[1,4-6]. Of the 6 episodes of PVT 5 (83.3%) had INR less 2.0. The main cause of sub-therapeutic INR in these patients was non-compliance in the taking of their coumarin drugs. The patient with the aortic PVT who had an INR of 2.0 had in addition extensive pannus formation around and in the cavity of the valve. Pannus formation, in addition to having an obstructive effect may also predispose to the formation of extensive thrombi which was present in this particular patient[10]. Other causes of thombotic events are associated coagulation disorders including protein C, Protein S and antithrombin III deficiencies[11].

Kontos, while investigating the clinical signs of PVT listed exertional dypnoea, from pulmonary oedema as one of the main features [2]. He indicated that the presence of shock usually indicated a poor prognosis during management. This finding has also been confirmed in other studies [1,4-5]. All the patients in the present study had pulmonary oedema at presentation. Although hypotension was present in 66.7% of the cases only one was in shock with multi-organ dysfunction. All the patients were in NYHA class III-IV at the time of presentation. Roudant et al in their study of 127 cases had patients 90% of their cases in NYHA III-IV[12]. It has been categorically proven that a NYHA class of III-IV is associated with a high mortality rate no matter the mode of management. However workers have advocated thrombolysis for these groups of patients[12-13].

Thrombosis can occur if the administration of heparin is not done early. There was an early thrombosis in our study of 7 days postoperatively. Talwar and his colleagues in their study found out that 6.1% of their patients developed significant thrombosis in 9 days if heparin therapy was not aggressive enough while warfarin was sub-therapeutic which has been confirmed by other workers[13-15].

Streptokinase (SK), urokinase (Uk) and tissue plasminogen activator (rTPA) have all been used for thrombolysis with relatively good results[11-17]. Roudaut et al in their study found out that SK and rTPA were more effective than Uk for thrombolysis. The other factors that may affect the choice of thrombylytic would be, the side-effects of streptokinase, the non-availability of urokinase and the expense of rTPA [6,7,12, 16,17]. Some workers have used the prolonged or short course infusion protocols for thombolysis depending on the haemodynamic condition of the patients. However there is no clear advantage of one protocol over the other in terms of results and the protocol adopted may depend on individual or institutional preferences[6,12,14,16-18]. Our institution uses the short course protocol which is much cheaper than the prolonged course infusion technique. The short course protocol has the advantage in that clinical improvement is seen early in the cases.

Overall success rates of thrombolysis cited in the literature have been between 70-90% and these have been independent of the thrombolytic used[12,16-18]. Our overall success rate of 83.3% falls within range. However thrombolysis in patients

presenting in NYHA class III-IV is less successful than in patients in class I-II.
It has become evident that transoesophageal echocardiography (TEE) has become invaluable in the diagnosis and the proper management of patients with PVT. Many workers use TEE to follow the progress of thrombolysis in these patients to determine the risk of emboli and also to assess the success to thrombolysis [12,13,16-19].

Complications cited in the literature include embolic phenomenon, strokes, transient ischaemic attacks, bleeding and allergy especially to SK [2,12,14,16-18]. Our study had a high proportion of allergy to SK because of the suspected high incidence of streptococcal sore throats in developing countries. Surprisingly there were no embolic phenomenon and also no strokes in our study.

It is now evident that thrombolysis has a lower mortality for all classes of NYHA definition of heart failure from PVT and the ACC/AHA current recommendations advice thrombolysis for most cases of PVT. There is also a high mortality in patients presenting with PVT and shock. Gupta and his colleagues recorded a mortality of 78% of patients who presented with PVT and shock [17]. One patient in out study died during thrombolysis and his presentation was shock and multiorgan dysfunction.

Conclusion

Thrombosis of prosthetic heart valves is not common from out series. Thrombolysis using streptokinase should be the first line management as it is cheap and relatively safe in the management of such cases.

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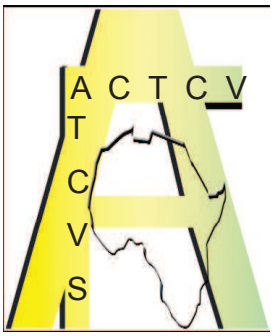

CHIRURGIE THORACIQUE/ THORACIQUE SURGERY

INFECTED BRONCHOGENIC CYST SIMULATING ACUTE SEVERE EXACERBATION OF BRONCHIAL ASTHMA

B. O. OGUNBOSI, MBBS[1], ESE E. EBRUKE, MBBS[1], ADENIJI T. SOFOLUWE, MBBS[2], F. OLUKAYODE. AKINBAMI, MBBS, FWACP, FMCPAED[1] , SALAMI, MBBS (IB)[3], MILLICENT O. OBAJIMI, MBBS, FMCR, FWCR[2], ADEGOKE G. FALADE, MBBS, MD, FMCPAED[1], AND OLUWOLE A. ADEBO, MBBS, FRCSC, FWACS, FMCS [3]

Departments of Paediatrics[1], Radiology[2] and Surgery, Cardiothoracic Surgery Unit[3], College of Medicine, University of Ibadan, and University College Hospital, Ibadan, Nigeria

Correspondence : Professor AG Falade
Department of Paediatrics University. College Hospital
PMB 5116 Ibadan-Nigeria
E-mail addresses: afalade33@hotmail.com, and afalade33@gmail.com
Telephone: 234 2 241 00088 Ext. 2213, 2420
Mobile: 234-803- 353-6410

SUMMARY

Upper airway obstruction classically produces stridor. However, wheezes can be a feature of upper airway obstruction under certain conditions in which case it becomes a diagnostic challenge. A case of infected mediastinal bronchogenic cyst earlier managed as bronchial asthma is presented to highlight such feature. A seven year old African boy presented with a 3-day history of cough and fever; and difficulty in breathing of a day's duration. He has had past episodes of cough and difficulty in breathing more than 6 years with occasional fever and which responded to Franol® and Paracetamol® given *per oris.* During this acute attack, he failed to respond to standard treatment for acute severe exacerbation of bronchial asthma. Radiologic examinations revealed features compatible with posterior mediastinal mass. A right postero-lateral thoracotomy revealed a tense cystic mass in the posterior mediastinum and 100ml of pus was drained and the cyst was completely excised. Bronchogenic cyst though, a rare cause of wheezing should be considered when a mediastinal mass is suggested on chest radiography.

Key words : Bronchogenic cyst, Sumulating, Bronchial asthma, Childhood

Introduction

Sounds emanating from the airway obstruction give a clue as to the possible origin, whether upper or lower airway. Upper airway obstruction classically produces stridor because of the wide diameter of the airway involved and turbulent airflow generated, whereas in the lower airway the smaller diameter causes a whistling musical sound termed wheeze[1]. However, wheezes can be a feature of upper airway obstruction under certain conditions[2]. When this happens, upper airway obstruction becomes a diagnostic challenge due to confusion with the more common cause of airflow obstruction, such as asthma and chronic obstructive pulmonary disease. It is however imperative to make the distinction between upper airway obstruction and lower airway obstruction because as an important cause of airflow limitation, it has the potential to produce acute airway compromise and respiratory failure which may be amenable to curative surgery.

The purpose of this study is to underscore the fact that not all that wheezes is asthma, although most does. We, therefore report a case of infected bronchogenic cyst earlier managed as a case of bronchial asthma to highlight such feature.

Case report

A seven-year-old black African boy presented with a 3-day history of cough and fever; and difficulty in breathing of a day's duration. Cough was paroxysmal, worse in the night and with occasional post-tussive vomiting and exhaustion. The fever was high grade but there were no chills or rigors. He developed difficulty in breathing on the second day of illness, which was associated with significant orthopnea and limitation of speech to monosyllables.

He has had past episodes of cough and difficulty in breathing with occasional fever from age 3 months, once or twice every 2 months; and later on weekly from age 3yrs. Symptoms were relieved by Franol® and Paracetamol® given *per oris,* which were obtained from a neighbourhood patent medicine store. A similar episode 2 years earlier was treated at a peripheral hospital with an intravenous drug suspected to be aminophylline with prompt relief. There were no identified precipitating factors, history suggestive of atopy or family history of asthma.

Physical examination revealed an acutely ill-looking and sweaty boy. The temperature was normal (T=36.9^{0}C), and he was pink in room air. Hydration status was optimal. The major positive findings were in the respiratory system which revealed the following: a loud expiratory sound which was characterized as a wheeze, a barrel shaped chest, severe respiratory distress as shown by flaring ala nasae, and intercostals, subcostal, lower chest wall, supraclavicular and suprasternal recessions. He was tachypneic with respiratory rate of 44 breaths/min. The percussion notes were resonant, and there were widespread inspiratory and expiratory wheezes with a few coarse crackles at the lung bases. The pulse rate was 120 beats/min, regular and of good volume. The blood pressure was 120/70-60 mmHg, and the heart sounds were normal with no murmurs. He had a non-tender hepatomegaly, 5cm below the right costal margin. The neurological examination was normal.

The diagnosis was severe acute exacerbation of bronchial asthma. The treatment included supplemental oxygen through a nasal catheter at a flow rate of 2 liters/min., salbutamol nebulisation (5mg), and intravenous hydrocortisone sodium succinate (100mg every 6 hours). When he was reviewed at 4hours after commencement of the steroid, he had deteriorated as evidenced by a mild central cyanosis; he was therefore commenced on aminophylline drip at a rate of 0.9mg/kg/hour taking cognizance of the fact that he had had a dose of ephedrine® at home before presentation in the hospital. In addition, erythromycin estolate®, 250 mg every 8 hours was administered. Radiographic findings before thoracotomy (Figures 1-3) were compatible with a mass in the posterior mediastinum with possibilities of paratracheal lymph nodes, thymoma, teratoma, retrosternal goiter and a mediastinal bronchogenic cyst. Review of the patient by the Cardiothoracic Surgical Unit, suggested a diagnosis of posterior mediastinal tumor most likely of neurogenic or bronchogenic origin.

A right postero-lateral thoracotomy was done with finding of a tense cystic mass located between the oesophagus and trachea extending across the midline to the left side; 100ml of purulent material was initially drained and the cyst was completely excised. Two drains were left, one located in the cyst bed and the other in the pleural cavity. The postoperative chest radiograph (Fig 4), showed absence of the previously described mediastinal mass. Associated atelectatic changes were present in the lung bases.

The pus aspirate was sterile on culture. He developed post operative pneumonia, which was effectively treated with ceftazidime after a failure of response to Ciprotab® He was discharged well after 14 days on admission.

Fig. 1 : The antero-posterior radiograph of the chest showing superior mediastinal widening by a soft tissue mass which extends from the root of the neck into the superior mediastinum. This mass is lobulated and is seen to displace the upper airway to the right No underlying calcification or bony destruction seen.

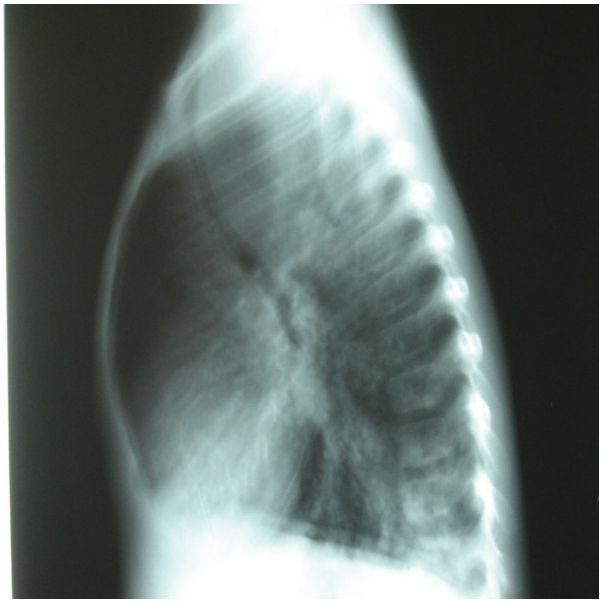

Fig. 2 : Lateral chest radiograph showing a localized mediastinal mass to the posterior compartment with associated anterior bowing and displacement of the trachea suggestive of an upper airway obstruction from a posterior mediastinal mass.

Fig. 3 : The lateral projection of the neck showing normal cervical curvature, vertebral bodies, pedicles and disc spaces. The outlined airway also appears within normal limits.

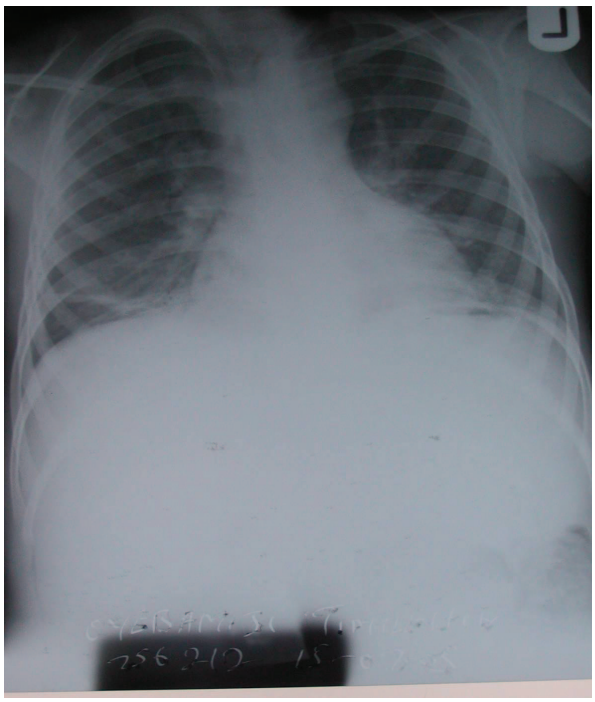

Figure 4. The post-operative chest radiograph showing absence of the previously described mediastinal mass

Discussion

Bronchogenic cyst is a congenital malformation of the respiratory system derived from the primitive foregut[3], which varies in size and location. Common locations include mediastinal, paratracheal, paraesophageal and intrapulmonary, and often on the right. Cough is the predominant symptom. Complications include infection, and also increase in size which can then lead to pressure symptoms and signs on the adjacent structures.

Although pulmonary function testing can aid in the diagnosis of bronchial asthma, this has not been found to show any pathognomic features. Demonstration of bronchial hyperresponsiveness although more specific than pulmonary function testing also suffers from the same shortcoming. Hence, the diagnosis of bronchial asthma is essentially clinical as there are no biochemical markers yet for the disease. The clues to the diagnosis being repeated wheezes, recurrent or persistent cough, night time disturbance by wheeze or cough, and these symptoms being incited by viral upper respiratory infections, exercise or excitement, potential allergens such as those associated with pets, pollens, dust, or feathers and cigarette smoke. Our patient presented with many of these features, and the diagnosis of asthma was inadvertently strengthened with the history of therapeutic response to bronchodilators on previous occasions of breathlessness. During admission into our hospital, initial chest radiograph and poor response to β-2 agonist and glucocorticoid treatments heightened suspicion of a superior mediastinal mass which was confirmed by a lateral film. A CT-scan or MRI would have enabled a preoperative diagnosis; however these investigational procedures are very expensive and not within the reach of most patients in our environment. The place of radiologic evaluation is again highlighted by this case, when there is poor response to usual management lines of apparent bronchial asthma.

Bronchogenic cyst presenting with wheezes is uncommon. Diagnosis is often by radiologic evaluation following complaints as in our patient or during investigations for other cardiorespiratory conditions. Surgical excision through thoracotomy is the treatment of choice because it permits complete excision of the cyst and therefore forecloses recurrence[4] However, there is an associated morbidity due to long muscle-cutting incision. Hence, thoracoscopic resection of mediastinal bronchogenic cyst has been advocated because it provides less operative pain, shorter hospital stay and better cosmetic outcome than the standard thoracotomy. Pneumonia is a common complication following surgery as seen in this case, often from spillage of infected cyst contents into the tracheobronchial tree.

In general, wheezes are produced by intrathoracic (lower airway) obstructive processes. However, fixed monophonic wheezing is produced by opening and closure of one airway at a time. It is usually caused by obstruction of a large, central airway by a process such as external compression, e.g., vascular ring, adenopathy, and bronchogenic cysts among others. Sometimes these processes will also lead to inspiratory stridor[1].

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CHIRURGIE THORACIQUE/ THORACIC SURGERY

CERVICO-THORACIC IMPALEMENT INJURY : CASE REPORT AND LITERATURE REVIEW

A. ETIUMA[1], O. BASSEY[1], A. UDOSEN[2], A. ESHIET[3]

Cardio-thoracic surgical unit[1], Department of Surgery[2], Department of anesthesia[3],
University of Calabar Teaching Hospital, Calabar, NIGERIA

Correspondence : Dr. Anietimfon U. Etiuma,
P.O.Box 3702, Unical Post Office, Calabar – Nigeria.
E-mail : aetiuma@yahoo.co.uk
Phone : +234(0)8033573255, 08057717838

ABSTRACT

Impalement injury especially of the cervico-thoracic region of the body is very rare. It is a very significant injury and usually require special consideration in it management. We report a 50 year old man who fell from palm wine tree and sustained cervico-thoracic impalement of a branch of tree which carried a piece of plastic bag material such that the branch of the tee and the plastic bag were driven into him. He was successfully managed in a hospital with only basic facilities. Important peculiarities on the management of this rare form of severe trauma especially in Africa is discussed. Also shown is the special occupational hazard and problem of poverty among rural farmers Nigeria.

Key words: Cervico-thoracic, Impalement, Injury.

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RÉSUMÉ

La blessure interne, spécialement celle de la région cervico-thoracique du corps est très rare. Cela demande habituellement une considération spéciale pour la gérer. On a enregistré un vieil homme de 50 ans qui est tombé d'un arbre qui portrait un morceau d'un sac en plastique de sorte que la branche de l'arbre et le sac en plastique sont entrés dans lui. Il a été soigné dans un hôpital qui n'avait que les facilités de base. Les particularités importantes sur la gestion de cette forme rare de traumatisme spécialement en Afrique sont mentionnées et discutées ainsi que les métiers à risque et le problème de la pauvreté parmi les agriculteurs ruraux au Nigeria.

Mots-clés: Cervico-Thoracique, Empalement, Blessure.

INTRODUCTION

Impalement injury refers to a special class of penetrating injuries where piercing object of significant dimension remains sticking out from the patient. Patient may be moved with the object. Transfixing injury refers to situations where the patients remain fixed to immobile structure e.g. fence post. These groups of injury are very severe especially when it involves the cervical and thoracic regions of the body due to compact arrangement of vital structures in such regions.

CASE REPORT

Mr. E.U.M, a 50 year old palm wine taper fell from a tall palm tree while trying to harvest palm wine because his climbing robe snapped. He landed on a cut branch of tree on the muddy floor which pierced his right posterior triangle of the neck and traversed the posterior chest wall to point medial to the medio-inferior border of the right scapular. (Figure I). The impaling stick carried with it the sleeve of a plastic bag which the taper hung on the neck such that the stick and the plastic bag were driven into his neck and chest. (figure II).

Despite the frightening nature of the injury, he managed to drag himself home and was subsequently brought by his wife about six hours after the incidence to the casualty unit of the University of Calabar Teaching Hospital, Calabar, Nigeria at night. Though he was covered with mud, he was conscious and the cardiovascular system was stable. His pulse rate was 86 per minute, regular and of full volume. His blood pressure was 130/80 mmHg, respiratory rate was 26 cycles per minute, temperature was 37.4° Celsius and $PaSO_2$ of 96% on room air. There was no significant blood loss and other organs were normal except old cataract and a squint involving the right eye.

He was received in a wheel chair as he could not lie down on bed because of the projecting branch of tree from his neck (fig. 1). He was given a good bath, intravenous fluids antibiotics and analgesics. Chest x-ray, full blood count, urea, electrolyte and creatinine and urinalysis were done. A plain postero-anterior and lateral chest films showed soft tissue shadow representing the foreign body from the neck to the posterior chest wall grazing the posterior right pleura. Extensive lung contusion and dense patchy opacities of the entire right lung fields were noted on the x-ray. All other tests were normal. Blood was grouped and cross-matched for him. All investigations and drugs were sponsored by the attending unit to avoid delay in his management as the patient did not have any funds for his treatment.

Fig. 1 : Patient before surgery after trimming the tree branch and the bag.

Fig. 2 : The impaling stick and bag after extraction.

He had emergency right postero-lateral thoracotomy within six hours of arrival in the Hospital. Impalement objects were removed after full access had been gained into the thoracic cavity. Severe lung contusion had occurred from blunt impact of the fall. There was moderate haemothorax (about 350 ml) and dense fibrinoid exudates involving the entire pleural cavity with tendency to fibrous encasement of the lung. There was no serious neurovascular injury and no rib fracture. Adhesiolysis and release of the lung was done. Lavage and underwater-sealed thoracostomy tube drainage was instituted. Debridement, irrigation lavage and drainage of the impalement tract were carried out and the wound was left open for dressing. Minimum intravenous fluid requirement was given to prevent worsening pulmonary edema which followed lung contusion. He was placed on ceftriazone, metronidazole, gentamicin and analgesics. Antitetanus prophylaxis and haematinics and other supportive care were given. He was initially managed in the intensive care unit for two days before being transferred to the ward, and subsequently recovered without complications. Complete healing took place within three weeks. Patient however absconded in order to avoid payment of hospital bill.

DISCUSSION

Major impalement injuries of the thoracic region are uncommon and reports in literature are infrequent[1-12]. They present as dramatic and frightening cases. Our patient attracted a crowd in the hospital. Literature search shows mostly cases published as case reports as individual surgeons do not see many cases in their series. Most of such cases have extremely high mortality especially when vital mediastinal structures are involved. Mortality occurs at site of injury or within few minutes to hours after. Common cause of immediate death include: major cardiac or vascular penetration, airway obstruction, tension pneumothorax, massive haemothorax, flail chest (representing major chest wall disruption), cardiac tamponade and massive exsanguinating haemorrhage. Parenchymal injuries to the lung, liver, spleen or gut are compatible with survival with expert management. Damage to major vessels and nerves in the thoracic inlet may accompany cervical injury especially zone I (from clavicle to cricoid cartilage). Other associated injuries should be carefully evaluated and managed as they may contribute to mortality. In the case presented, there was lung contusion from blunt deceleration chest trauma. Stable cases that last up to few hours could be successfully salvaged when they present in the hospital. Majority of impalement or transfixing injuries are low velocity in nature so that the outcome depends on the size of the offending instrument and the anatomical structures involved.

Etiologically, these injuries may broadly be classified into three categories: (a) Road traffic accidents: Results from motor vehicle occupant or cyclist being thrown out to collide with projectiles such as fence post, or tree stump or metal. (b) Aggression: This may follow low velocity implement including knife, spear, or arrow. In combat situations, projectiles traveling on speed may meet a stationary victim resulting in high velocity impacts. Domestic violence and industrial assaults with electric drill, dagger, furniture parts, glass or machinery have been reported.[4,11,12]. Falls from a height: This, as shown in our patient could result in transfixion or impalement injury. Victims commonly land on projectiles, stump of trees, sticks, fence post, building materials etc. This is a specific occupational hazard of many African rural dwellers who engage in palm wine tapping or

palm fruit and coconut harvesting. Sport injuries especially victims being thrown out from horse riding and other high velocity or motorized sports.

Transfixion often involves more than one body cavity, usually chest and abdomen, neck and chest as in this case, both thoracic cavities. Apart from chest wall and visceral injury, contamination by clothes, soil, debris, together with bacteria such as Clostridia, Streptococci and Staphylococci are common.

At the site of injury objects should be manipulated as little as possible in other to prevent sudden massive haemorrhage since impaling objects may tamponade damaged (traversed) blood vessels. No attempt should be made to remove the object at the site. However such objects may be shortened or detached from massive immobile unit to enhance patient transport. This may require heavy cutting equipment to cut through wooden fence or metal. Foot and Naidoo[13] reported an exceptional case where this rule was broken to save the patient from fire explosion. Pre-operative bath was important as our patient was covered with mud, to reduce the chances of infection and enhance acceptability of patient for care. This may not be required in all cases. Grouping and x-matching should be done in anticipation of possible severe intra-operative haemorrhage. Resuscitation should follow basic principles of airway, breathing and circulation. When the patient is haemodynamically stable, plain radiographs, endoscopy and/or angiography could be done as indicated. Occasionally, the patient may be moved directly from ambulance to the operating theatre. Antibiotic prophylaxis is always required in this group of patients.

When the impaling object is long and traverses the cervico-thoracic region as in our case, induction of anaethesia could be very challenging as proper supine positioning and neck extension for induction and intubation is difficult. This there requires the services of an experienced anaesthetist. Any disruption in the chest wall with open wound, haemothorax or pneumothorax should necessitate thoracostomy tube drainage under local anaesthesia before commencement of general anaesthesia. This will prevent sudden lung collapse during induction of general anaesthesia. Pre-operative pleural drainage was not indicated in our index patient.

Indication for thoracotomy in our patient was the possibility of gross pleural contamination and possible massive intrapleural bleeding after removal of the foreign body. Preoperatively, it was not certain whether the impaling object had traversed the pleural cavity or not. Thoracotomy was therefore part of the caution, especially in the absence of preoperative CT scan which should give a definitive anatomical diagnosis. Thoracotomy was to enable us effectively handle major intrathoracic haemorrhage if it occurred and toilet the pleural cavity. This turned out to be a very necessary procedure as it also allowed extensive adhesiolysis to be carried out which otherwise could have resulted in severe fibrothorax in the future. We therefore recommend thoracotomy in similar circumstances especially in the absence of thoracoscopic facilities and CT scan. Median sternotomy should be preferred for better access to the mediastinum in circumstances where the impaling object traverses both sides of the chest. Sterno-laparotomy or thoraco-abdominal incision may be required to repair associated abdominal injury. In extensive chest wall mutilation, the plastic surgeon should be involved.

No matter the grotesque nature of chest impalement or transfixation injuries, the prognosis depends so much on the degree of damage to vital structures. Even very frightening injuries may end up with good outcome when appropriate assessment, resuscitation, investigation and surgical principles are promptly and properly employed in the management. An emergency fund for this group of patients could enhance prompt treatment.

In Africa and West Africa in particular, an important major contribution to morbidity and mortality is poverty. Patients may hesitate to go to hospital empty handed, inaccessible health facility distribution, non- existent medical insurance or policies with inadequate coverage of the poor unemployed rural dwellers who commonly present with these injuries, absence of efficient blood bank service that can promptly provide blood for massive blood transfusion in catastrophic emergencies. Many patients depend on the good will of medical personnel to survive as was the case in our patient.

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