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Case Report

Acute Aortic Dissection at the Douala General Hospital: A Report of Two Cases

Dissection aortique aigue : à propos de deux cas vus en l'intervalle d'un mois à l'hôpital général de Douala

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ABSTRACT

Acute aortic dissection is the most frequent and lethal presentation of acute aortic syndromes with an incidence of 3-4 cases per 100.000 per year. In general, 20% of patients with aortic dissection die before reaching the hospital and 30% die during hospital admissions. We present two cases of acute aortic dissection we received in sequence over a period of one month: A case of Standford type A aortic dissection with extension to renal and iliac arteries initially misdiagnosed as acute myocardial infarction and a case of standford type B aortic dissection. A clinician may not attend to a case of aortic dissection in all his practice. High index of suspicion and initiation of appropriate registries are potential avenues to curb mortality.

RÉSUMÉ

La dissection aortique aiguë est la présentation la plus fréquente et la plus mortelle des syndromes aortiques aigus avec une incidence de 3-4 cas par 100.000 par an. En général, 20% des patients atteints de dissection aortique meurent avant d'atteindre l'hôpital et 30% meurent au cours des hospitalisations. Nous présentons deux cas de dissection aortique aiguë que nous avons reçus en séquence pendant un mois : un cas de dissection aortique de type A de Standford avec extension aux artères rénales et iliaques initialement mal diagnostiquée comme un infarctus aigu du myocarde et un cas de dissection aortique de type B. Un clinicien ne peut pas assister à un cas de dissection aortique dans toute sa pratique. Un indice élevé de suspicion et l'instauration de registres appropriés sont des moyens potentiels de limiter la mortalité.

INTRODUCTION

Acute aortic dissection (AD) is a critical diseases and the most frequent and lethal presentation of acute aortic syndromes. The incidence is about 3-4 cases per 100.000 per year(1). In its natural history without treatment, acute type A aortic dissection has a mortality rate of about 1% per hour initially, 50% by the 3rd day, and almost 80% by the end of the 2nd week. Death rates are lower but still significant in acute type B aortic dissection: 10% minimum at 30 days, and 70% or more in the highest-risk groups (2). In the literature, 20% of patients with AD die before reaching the hospital and 30% die during hospital admissions (3). Common predisposing factors to AD noted in the International Registry of Aortic Dissection (IRAD) were hypertension in 72% of cases, followed by atherosclerosis in 31% and previous cardiac surgery in 18% (4).

The typical presentation of AAD is that of a man in his 5th or 6th decade of life presenting with retrosternal chest pains radiating to the back and on physical examination the blood pressure is asymmetrical in both arms (2). Diagnostic imaging studies in settings of AD are aimed to rapidly confirm or exclude the diagnosis, classify the dissection as proximal (Standford A or Debakey I and II) or distal lesions (Standford B or Debakey III) (5). The most commonly used imaging tool in the diagnosis of AD is thoracic CT angiogram (CTA). sensitivity of CTA is superior to 95% and specificity to 87-100%. Other imaging modalities include echocardiography and MRI (5). The management of AD depends on the type and the classification. AD type A is a surgical emergency whereas AD type B is managed medically except for specific surgical indications. Essentially, the initial objectives are normalization of blood pressure and lowering of left ejection fraction with objective of systolic blood pressure 100-120 mm Hg and heart rate < 60 beats/minute (6).

CASE REPORT

Case No 1: Standford type A Aortic Dissection

A 53-year-old sub-Saharan African man with a poorly controlled hypertension was referred to our cardiac intensive care unit (CICU) by his cardiologist for the management of a sudden onset, severe and intractable retrosternal chest pain of approximately 50 hours duration. The pain was tearing in character, radiating to the back and lumbar regions, non-positional and associated with shortness of breath and headache. The electrocardiogram (ECG) three hours after the onset of pain showed sinus rhythm and nonspecific repolarisation changes (flattened or inverted T waves in I, aVL and lead V3-V6; the diagnosis of acute myocardial infarction (MI) was made. Persistence of pain after initial therapy with low molecular weight heparin (LMWH) and nitrates prompted referral to our centre. On physical examination, he was diaphoretic, anxious, dyspnoeic (NYHA III) with a respiratory rate of 28 breaths/ minute). His temperature was 36.9^{o} C, heart rate 79/min, blood pressure was 187/73 mm Hg (right arm) and 145/56 mm Hg (left arm). Cardiac examination revealed a systolic murmur (grade 3/6) in the aortic area, which radiated to the left carotid, but there were no signs of heart failure. The neurological examination was unremarkable. Chest x-ray (fig. 1) showed a widened mediastinum with cuffing of the aortic knob. Echocardiography showed dilated left atrium, normal left ventricular systolic function (ejection fraction 72%), severe aortic insufficiency and dilatation of the aortic root and ascending aorta (44 mm). Thoracic CTA (fig. 2) showed dissection of the aorta from the ascending aorta to the iliac arteries, including celiac trunk, left renal artery. This was associated to splenic infarction. Doppler ultrasound of the carotid arteries showed no extension to the carotid arteries. We made a working diagnosis of Standford type A acute aortic dissection. Biological investigations (FBC, CRP, urea and creatinine, CK-MB, NT-pro BNP, troponin I, myoglobin, LDH) were within normal ranges. The treatment consisted of high flow oxygen at 5 L/ minute; nicardipine titrated to a maximum of 10 mg/hour, bisoprolol 5 mg / 12 hourly, analgesics and compressive stockings. LMWH was stopped. By day six, blood pressure and heart rate targets were achieved. On day ten, patient developed fever (39.1^{o} C) and there was a sudden onset of dyspnoea at rest. Physical examination showed tachycardia (119 beats per minute), mean blood pressure of 124/76 mm Hg, and oxygen saturation of 98%. Chest examination revealed crepitations in both lung bases, more prominent on the right. We made a presumptive diagnosis of severe pneumonia. A repeat chest x-ray showed bilateral interstitial heterogeneous opacities. The C-reactive protein (CRP) titer was high (310.43 mg/L) and there was leucocytosis (17.7 x 10^{6} cells/L). The result of BNP analysis to exclude heart failure was 122 pg/ml. Blood samples for culture were collected and antibiotics (Amoxicillin-Clavulanic acid 1 g eight hourly and Clarithromycin 1000 mg 12 hourly) were introduced. About three hours later, because of persistent dyspnoea and hypoxemia ($SpO_2 \leq 65$ and $PaO_2 \leq 60$), he was intubated; but during the process, he had a cardiac arrest. Patient later died on day 12 following a cardiac arrest despite life support. Blood culture results (which came after patient's demise) were positive for *Klebsiella pneumonia.*

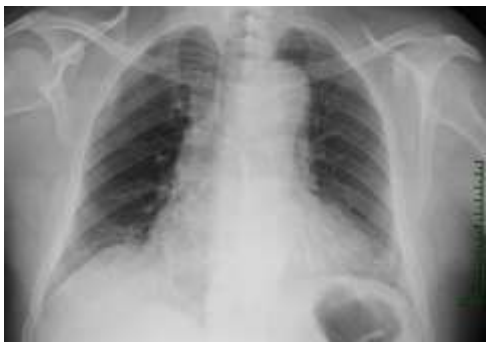

Figure 1: Chest x-ray showing widened mediasternum

Case No 2: Standford type B Aortic Dissection

A 62-year-old poorly controlled hypertensive sub-Sahara African man was referred by his general practitioner to our cardiac intensive care unit (CICU) centre for chest pain of sudden onset. The pain was of 48 hours duration, tearing in character retrosternal radiating to the back and associated with shortness of breath on exertion. On admission, he was anxious and diaphoretic, with asymmetrical blood pressure of 210/110 on right arm and 170/96 on left arm. He was tachycardic (116 beats/minute); first and second heart sounds were heard in all four auscultation areas. There was no murmur and no signs of heart failure. Neurological examination was unremarkable. There was no widened mediastinum at chest x ray. Thoracic CTA showed dissection of the descending thoracic aorta, without involvement of aortic arch or ascending aorta. We made a diagnosis of standford type B aortic dissection. Biological investigations showed that FBC, CRP, urea and creatinine, CK-MB, NT-pro BNP, troponin I, myoglobin, LDH were within normal ranges. ECG and echocardiography were deferred. The management consisted of high flow oxygen 5 L/minute, nicardipine 5 mg/hour, bisoprolol 5 mg tablet 12 hourly, analgesics and compressive stockings. The evolution was marked by stabilization of blood pressure on day five of hospitalization. He was discharged on day six of hospitalisation with Nicardipine 50 mg 12 hourly tablets and Bisoprolol 5 mg tablet 12 hourly. He died at home two days following discharge.

DISCUSSION

AD is characterized by separation of the layers of the aortic wall resulting from the entry of extraluminal blood through an intimal tear producing a false lumen. Tears are commonly seen at areas of high stress, commonly in the anterior aortic wall just above the aortic valve (66%) and

the posterior wall of the proximal descending aorta (33%). When blood enters through an intimal tear it passes longitudinally along the tunica media separating the intima from the adventitia. There are several different classification systems of aortic dissection. The two most commonly used formats are the Debakey and Standford classifications as described in literature (7,8).

The typical presentation of AAD is a sudden, unexpected intense retrosternal pain radiating to the back and or abdomen associated with asymmetrical blood pressure (9). Patients are typically hypertensive, middle aged or elderly and as such, the differential diagnosis would include acute myocardial infarction, acute coronary syndromes, pericarditis, pulmonary embolism, peptic ulcer disease, and acute pancreatitis. Due to the possibility of extension to involve the mesenteric, iliac and renal arteries, other presentations may include intestinal ischemia, stroke, and renal failure(10). A misdiagnosis at presentation may occur in up to 38% of AAD, as well as may be discovered during post-mortem in 28% of cases without any prior identification or suspicion (4).

Our two patients presented with typical features of acute AD. Case N^{o} 1 was misdiagnosed as acute myocardial infarction. Factors that may have contributed to a misdiagnosis of this condition in the presenting centre included the relative rarity of this condition in our setting compared to acute coronary syndrome (ACS), low regional epidemiology, clinical similarity with ACS, and lack of specific diagnostic imaging modalities such as thoracic CTA in the presenting centre. In one study factors that contributed to misdiagnosis of AD included: walk-in mode of admission, severe or worst ever chest pain, anterior chest pain and widened mediastinum (11). In both indexed cases, the diagnosis of acute AD was made relatively late after 50 and 48 hours for case 1 and 2 respectively. Factors leading to delay in the diagnosis of AD in general include: presentation with atypical symptoms, patients with absence of pulse deficit or asymmetry blood pressure, initial presentation to a non-tertiary health care and late presentation (12). In both cases presented above the patients initially presented in non-tertiary hospitals and in case two the patient presented late. The target SBP and heart rate as described was obtained after 6 days and 5 days of controlled for case one and two respectively. The diagnosis of AD type A in case No 1 was an indication for emergency surgery. Surgery for aortic repair was not done because of lack of local centres and expertise and financial constraints in evacuating the patient. Indexed case one developed health care associated pneumonia with positive ***Klebsiella pneumonia*** on day 10 of hospitalization, was intubated and started on antibiotics and died on day 12 of hospitalization. Indexed case two was discharged from the hospital on day 12 of hospitalization and he died two days at home following discharge. The circumstance in which he died at home could not be gotten. Predictors of mortality in patients with AD include advanced age, female sex, abrupt onset and migratory chest pain, hypotension/shock/tamponade on presentation, and evidence of neurological or pulse deficits at presentation. Furthermore, the presence of a widened mediastinum on chest radiography, electrocardiographic evidence of new Q waves and/or ST-segment deviations, or a lack of a normal ECG were associated with higher in-hospital mortality rates(13). Contributors of mortality in our cases could include advanced age, sepsis and shock in case 1, widened mediastinum and abnormal ECG in case 1 and early discharge from the hospital in case 2.

CONCLUSION

Although relatively infrequent, acute aortic dissection is associated with a high mortality contributing to the burden of cardiovascular diseases. Furthermore, limited resources in developing countries contribute to the high burden of aortic dissection. Physicians taking care of cardiac patients should have a high index of suspicion of aortic dissection in all patients presenting with acute chest pain. The initiation of registry may help curb the burden associated with this condition.

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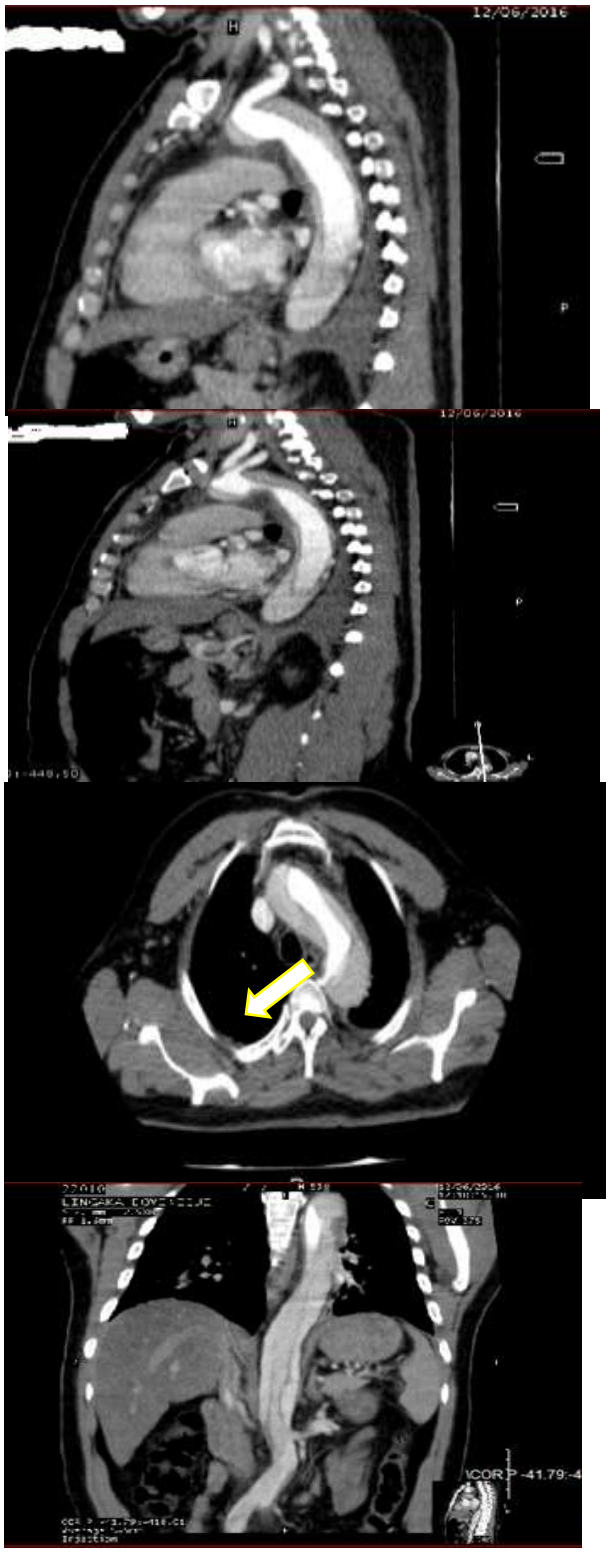

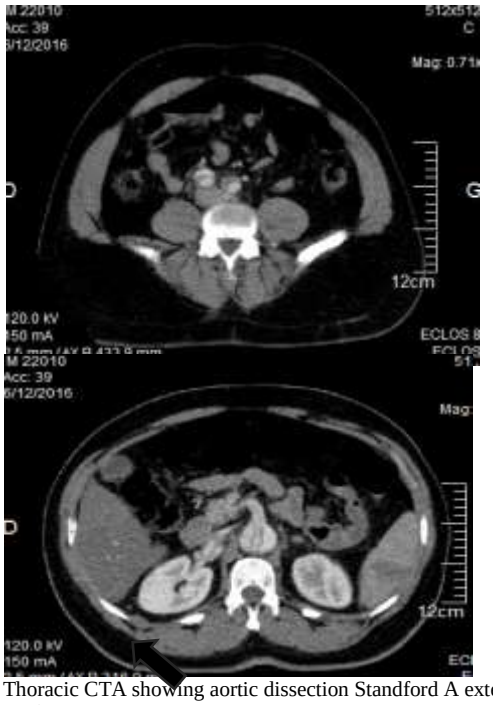

Figure 2: Thoracic CTA showing aortic dissection Standford A extending to the left renal (dark arrow), iliac (yellow arrow), and superior mesenteric (red arrow) arteries and causing splenic infarction (blue arrow).

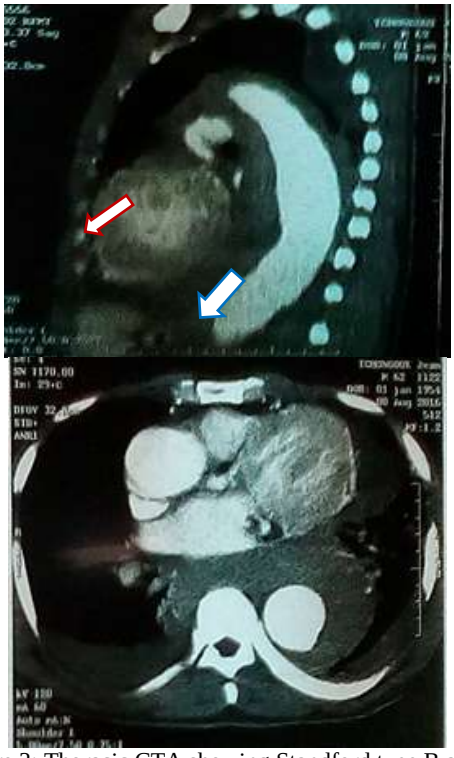

Figure 3: Thoracic CTA showing Standford type B aortic dissection