



CASE REPORT



AN INCIDENTAL FINDING OF DISSECTING AORTIC ANEURYSM IN 63 YEAR OLD MALE; CASE REPORT

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ABSTRACT

Aortic dissection is one of the acute aortic syndromes and a type of arterial dissection. The term Acute Aortic Syndrome (AAS) is used to describe a spectrum of life-threatening aortic emergencies of the thoracic aorta: classic Aortic Dissection (AD), Intramural Hematoma (IMH) and Penetrating Atherosclerotic Ulcer (PAU)^{1,2}.

Aortic dissection starts with a tear in the intima of the aortic lining. The tear allows a column of blood under pressure to enter the aortic wall, forming a haematoma. Hemorrhage in the media (at vasa vasorum) leads to either a tear in the weakened intima which breaks into the lumen or hemorrhage separates media from adventitia^{1,2,3}. The false lumen extends for a variable distance in either direction.

The most common sites for the intimal tear are within 2-3 cm of the aortic valve or distal to the left subclavian artery in the descending aorta².

The incidence of aortic dissection is approximately 3-4 per 100,000 per year^{2,3}. Aortic dissection is the most common emergency affecting the aorta. The incidence is about 3:1 male to female predominance.

It is most common between the ages of 50-70, being rare below the age of 40. The age of the index case falls within this range (63 years). Aortic dissection is very rare in children but it has been reported in association with coarctation of the aorta²

Aortic dissection is a medical emergency and can quickly lead to death, even with optimal treatment, as a result of decreased blood supply to other organs, heart failure, and sometimes rupture of the aorta. Aortic dissection is more common in those with a history of high blood pressure, a known thoracic aortic aneurysm, and in a number of connective tissue diseases that affect blood vessel wall integrity such as Marfan syndrome and the vascular subtype of Ehlers-Danlos syndrome. Other risk factors include structural aortic abnormalities such as bicuspid aortic valve, aortic coarctation, Turner syndrome, pregnancy, intra-aortic balloon pumps, infection (salmonella, typhoid and syphilis), trauma (decelerating injuries), takayasu and giant cell arteritis^{3,4}.

The diagnosis is made with medical imaging (computed tomography, magnetic resonance imaging or echocardiography).

This case is being presented to highlight the essence of prompt request of imaging modality a patient who presents with recurrent chest pain, treated as a chronic case of dyspepsia without due investigations who was eventually seen to suffer from aortic dissection.

Keywords: aortic dissection, false lumen, chest pain
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CASE REPORT

A 63 year old retired teacher presented several times to the medical out-patient department of Calabar teaching hospital (UCTH) with repeated history of dull, retrosternal chest pain of 2 years duration. He occasionally suffered generalized abdominal pain and was treated a case of chronic peptic ulcer disease. Patient is neither a known hypertensive nor diabetic; nil history of fainting attacks during any of such visits. Family and social history was not contributory. Four months prior to the last presentation, he was rushed to the medical emergency with history of sudden onset of tearing retrosternal chest pain. Hematological, biochemical tests and a basic

chest x-ray were requested.

The hematological indices were normal aside from a low packed cell volume of 24%. The biochemical profile including serum lipid profile was also within normal limit.

The Chest X-ray showed a grossly unfolded and dilated aorta with double density shadow, overlying a normal sized heart. The aortic unfolding seems to have begun from the aortic arch with extension to the descending thoracic aorta. No atheromatous plaque was seen within the aortic lumen. The lung fields were clear and pulmonary vasculature appeared within normal limits.

The managing physicians on seeing the chest x-ray report

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admitted the patient, referred him to the cardiothoracic unit for further evaluation; computed tomography was requested ; contrast enhanced and angiography. The patient vehemently refused admission, insisting he was fine and did not require any admission but however agreed to carry out the said investigations on an outpatient basis. This he did, two weeks after.

The computerized tomographic angiogram revealed an antegrade aortic dissection originating from the arch of aorta, propagated all the way to the iliac bifurcation, giving the appearance of a double barrel aorta. The patient was duly counseled as to the implications of the findings, given some medications, educated on lifestyle modification and placed on regular follow up visits. Patient never turned up for any of the appointment and is presumed lost to follow up or worse still feared dead.

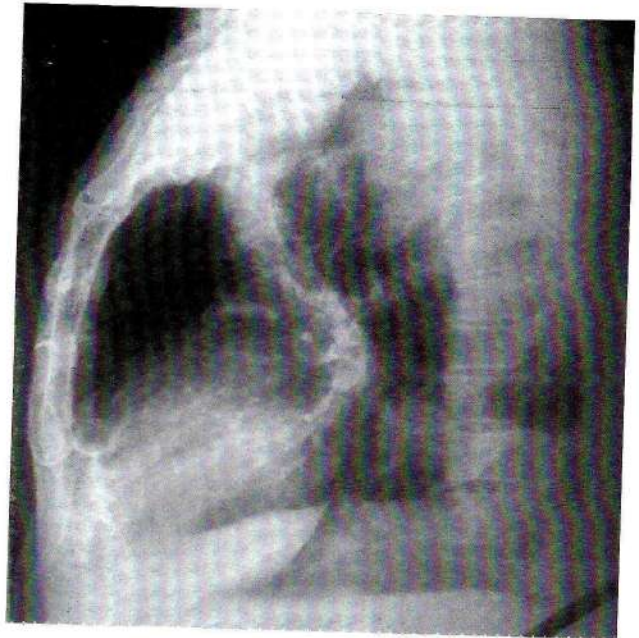


Fig. 2: Right lateral chest x-ray showing reduced retrosternal space superiorly by the dilated aorta as well as thoracic spondylotic changes.

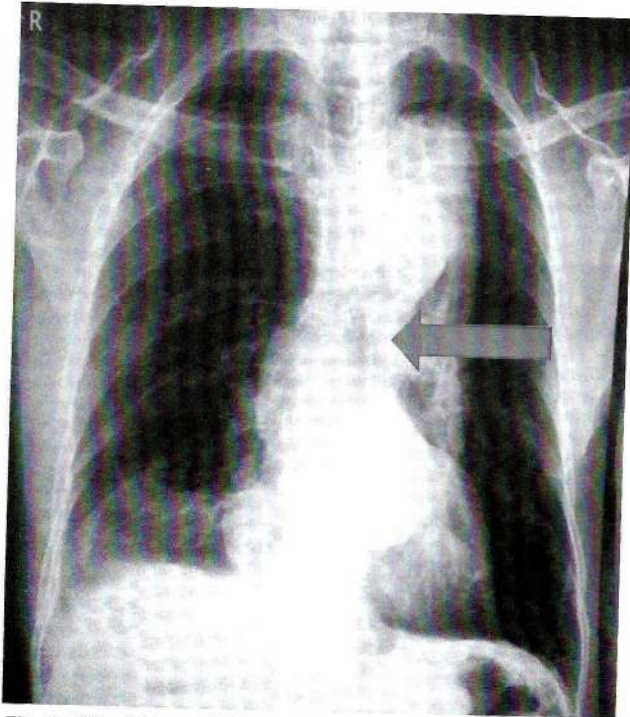


Fig. 1: Chest X-ray (PA) reveals a grossly unfolded aorta with lobulated outline and double density shadow overlying the heart, not silhouetting the left cardiac border. (see arrow)



Fig. 3: Contrast-enhanced axial CT image demonstrates an intimal flap separating the two channels in the descending aorta diagnostic of a Stanford type A dissection.

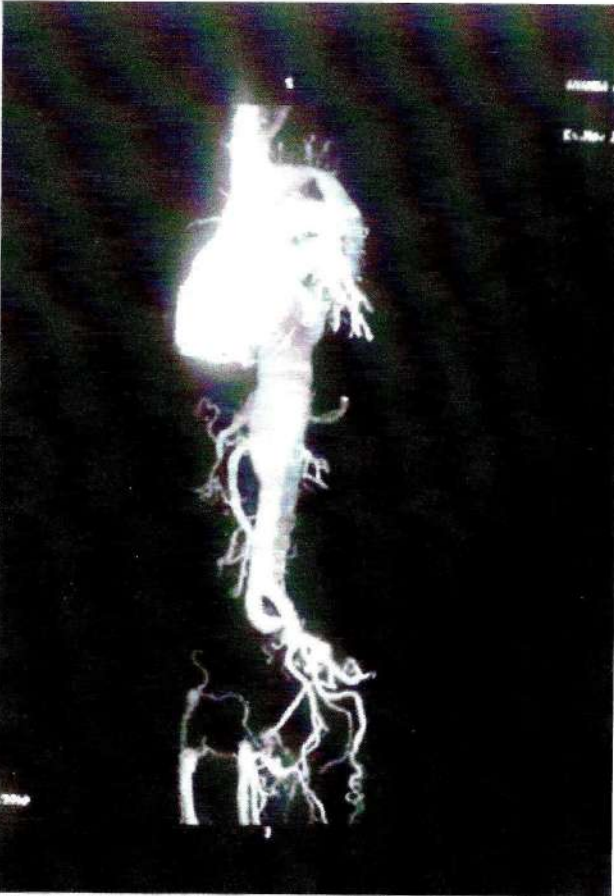


Fig. 4 is a 3D reconstructed computerized angiogram of the aorta, rotated in opposite direction to show the dissection clearly. It reveals Anterograde dissections originating from the aortic arch, propagated all the way to the iliac bifurcation of the aorta, and recanalizing the intravascular lumen leading to a double barrel aorta.

DISCUSSION

Aortic dissections are arbitrarily divided into acute (first 14 days of onset of symptoms) and chronic (after 14 days of onset of symptoms)³. Chronic dissections are seen in individuals who do not present with pain^{2,3}.

The clinical presentation of aortic dissection includes: hypertension with anterior or posterior chest pain and a tearing sensation in the chest. The individual may be normotensive or hypotensive. Our index patient was normotensive. Symptoms that may suggest end organ ischemia may be present. These findings are often dependent on the extent of dissection and occlusion of branches (seen in up to 27% of cases). Commonly reported are abdominal organ ischemia, limb ischemia, ischemic or embolic stroke (involvement of artery of Adamkiewicz). In some cases of aortic rupture, the involvement of coronary arteries may result in collapse and death. Symptoms of cardiac tamponade (Beck's triad) may also be seen^{3,4,5}. The

index case complained of generalized abdominal pain, probably due to abdominal organ ischemia.

About 96% of individuals with aortic dissection present with severe pain that are of sudden onset, often described as tearing, stabbing, or sharp in character⁵. 17% of individual's pain migrates as the dissection extends down the aorta. The location of pain is associated with the location of the dissection^{3, 4}. Anterior chest pain is associated with dissections involving the ascending aorta, while inter-scapular (back) pain is associated with descending aortic dissections. Pleuritic pain may suggest acute pericarditis caused by bleeding into the pericardial sac. This is a particularly dangerous eventuality, suggesting that acute pericardial tamponade may be imminent. The index case complained of dull retrosternal chest pain and occasional generalized abdominal pain, no history of anterior chest pain despite the extensive involvement of the ascending aorta. The severity of the pain was not sufficient enough to incapacitate the patient, which was probably why he trivialized the outcome of the investigations.

The pain due to aortic dissection may be confused with the pain of a myocardial infarction (heart attack), but aortic dissection is usually not associated with the other signs that suggest myocardial infarction, including heart failure and ECG changes. The index case suffered from only recurrent retrosternal chest pains; ECG findings were also normal.

There are a number of important features about the pain; in aortic dissection, pain is abrupt in onset and maximal at the time of onset. In contrast, the pain associated with acute myocardial infarction starts slowly and gains in intensity with time. It is usually more oppressive and dull. The pain is said to migrate as the dissection progresses. In proximal dissections, the pain is usually retrosternal, but with distal dissections the location is between the scapulae and in the back. Although *tearing* is the classical description associated with aortic dissection, the pain is described as *sharp* more often than tearing, ripping, or stabbing.

Pericardial tamponade is the most common cause of death from aortic dissection^{1, 2, 3}. There were no features to suggest pericardial tamponade in the index case.

The pathophysiology of aortic dissection shows that blood penetrates the intima and enters the media layer. The high pressure rips the tissue of the media apart along the laminated plane splitting the inner 2/3 and the outer 1/3 of the media apart^{2,4,6}. This can propagate along the length of the aorta for a variable distance forward or backwards. Dissections that propagate towards the iliac bifurcation (with the flow of blood) are called anterograde dissections and those that propagate towards the aortic root (opposite of the flow of blood) are called retrograde dissections¹⁻⁷. The initial tear is usually within 100 mm of the aortic valve so a retrograde dissection can easily compromise the pericardium

leading to a hemocardium. Anterograde dissections may propagate all the way to the iliac bifurcation of the aorta, rupture the aortic wall, or recanalize into the intravascular lumen leading to a double barrel aorta. The double barrel aorta relieves the pressure of blood flow and reduces the risk of rupture. Rupture leads to hemorrhaging into a body cavity and the prognosis depends on the area of rupture. Retroperitoneal and pericardial ruptures are both possible^{5,7}. The index case is thought to be an anterograde dissection. Hypertension is typically associated with distal aortic dissection; this was not the case with our index patient.

The vast majority of aortic dissections originate with an intimal tear in either the ascending aorta (65%), the aortic arch (10%), or just distal to the ligamentum arteriosum in the descending thoracic aorta (20%).

Imaging is essential in delineating the morphology and extent of the dissection as well as allowing for classification (which dictates management). Two classification systems are in common usage, both of which divide dissections according to involvement of the namely the Stanford classification and DeBakey classification.

Stanford Type A lesions involve the ascending aorta and aortic arch and may or may not involve the descending aorta.

Stanford Type B lesions involve the thoracic aorta distal to the left subclavian artery.

The Stanford classification has replaced the DeBakey classification (type I= ascending, arch and descending aorta especially seen in patients less than 65 years: type II= only ascending aorta: type III= only descending aorta commonly seen in hypertensives and atherosclerotics). The index case appears to be a Stanford type 1 and he is 63 years old.

Treatment options for the 2 subgroups of the acute aortic dissection are very different:

Stanford type A will be treated with surgery or endovascular therapy while Stanford Type B will be treated medically^{7,8}.

The reason for surgical repair of Type A dissections is that ascending aortic dissections often involve the aortic valve, which, having lost its suspensory support, telescopes down into the aortic root, resulting in aortic incompetence. This needs re-suspending to reseat the valve and repair / prevent coronary artery injury. Also the area of dissection is removed and replaced with a Dacron graft to prevent further dissection from occurring. However type B dissections are not improved, from a mortality point of view, by operation, unless there is leaking, rupture or compromise to other organs, example, kidneys, spleen.^{5,7}

Chest radiography may be normal, or demonstrate a number of suggestive findings, including widened mediastinum, double aortic contour, irregular aortic contour, inward displacement of atherosclerotic calcification. The chest x-ray of the index case, revealed a normal size heart, grossly unfolded aorta with a double aortic contour, atheromatous

plaque was not seen within the aortic lumen.

Computerized Tomography, especially with arterial contrast enhancement (CTA) is the investigation of choice, able not only to diagnose and classify the dissection but also evaluate for distal complications. It has reported sensitivity and specificity of nearly 100%^{3,5}.

Non-contrast CT may demonstrate only subtle findings; however, high density mural haematoma is often visible. Displacement of atherosclerotic calcification into the lumen is also a frequently identified.

Post contrast CT (CTA preferably) gives excellent detail. Findings may include^{1-3,5}: intimal flap, double lumen and dilatation of the aorta. The diagnosis in the index case was made using contrast enhanced computerized tomography.

An essential part of the assessment of aortic dissection is identifying the true lumen, as placement of an endoluminal stent graft in the false lumen can have dire consequences. Often distinguishing between the two is obvious, but in some instances, no clear continuation of one lumen with normal artery can be identified. In such instances, a number of features are helpful³.

A true lumen is often compressed by the false lumen, outer wall calcifications (helpful in acute dissections) while a **false lumen** is often larger in size due to higher false luminal pressures

Chronic dissection flaps are often thicker and straighter than those seen in acute dissections³.

Trans-esophageal echocardiography (TOE) has very high sensitivity and specificity for assessment of acute aortic dissection, but due to limited access and invasive nature, it has largely been replaced by CTA or MRA in some⁵. TOE was not done for the index case due to non-availability in our center.

MRA has been reserved for follow-up examinations; rapid non-contrast imaging techniques (e.g. true FISP) may see MRI having a larger role to play in the acute diagnosis, particularly in patients with impaired renal function⁴. It has similar sensitivity and specificity to CTA and TOE⁵ but suffers from limited availability and the difficulties inherent in performing MRI on acutely unwell patients.

Conventional digital subtraction angiography has historically been the gold standard investigation⁹. CTA has now replaced it as the first line investigation, not only due to it being non-invasive but also on account of better delineation of the poorly opacifying false lumen, intramural haematoma and end-organ ischaemia^{4,6,8}.

Angiography although risky is still required for endoluminal repair. The risks of angiography include general risks of angiography plus risk of catheterizing the false lumen and causing aortic rupture.

On CT a number of entities can mimic a dissection⁹ these include; pseudo-dissection due to motion artifact and



Figure 1: Immediate pre-operative period.



Figure 3: Anterior posterior X-ray of affected femur



Figure 2: Lateral radiograph of the right knee.



Figure 4: K-nail being removed

The preoperative radiograph confirms the distal migration into the right knee (Figure 2). There is fracture union, features suggestive of intramedullary chronic osteomyelitis. She had been taking antibiotics due to wound infection following the previous failed implant removal. There were no signs and symptoms suggestive of septicemia.

She had extraction of the K-nail through the knee. Intra-operatively, a septic membrane was removed from the intra-articular surface of the knee and the cartilage of the medial femoral condyle was friable around the extrusion site. There was discharge of purulent effluent during removal which was sent for culture. The nail was 10mm

by 360mm and cloverleaf - shaped. The bone marrow was curetted through the extraction window and irrigated with highly diluted hydrogen peroxide, then antibiotics saline was used for final irrigation and the knee closed with a drain placed extra-articular.

Post-operatively the knee passive flexion improved to >110°. Drain was removed after 72hours. Physiotherapy was scheduled for 2 weeks post-operatively; non weight bearing was prescribed 4 – 6 weeks. Post – operative radiograph revealed area of lucency at the healed fracture site, removed nail track, osteopenic bone and unremoved cerclage wire at fracture site above (Figure 6).

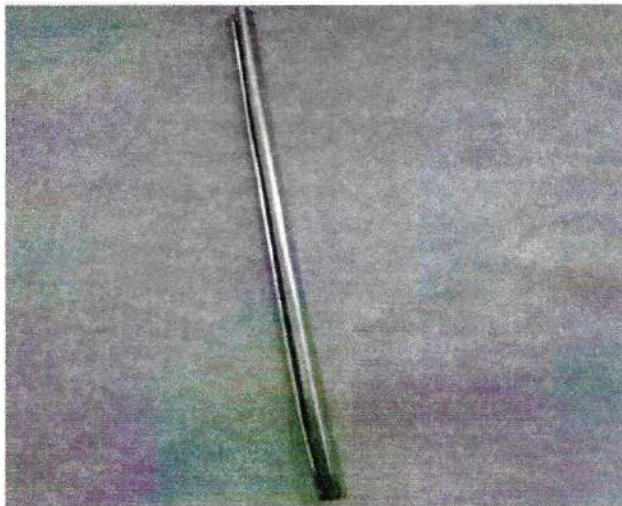


Figure 5: The extracted K-nail



Figure 6: Post-operative radiograph

DISCUSSION

Distal migration of K-nail is a well-documented complication that was common in the era when it was the Gold standard for femoral fracture fixation but that has changed, due to the advent of superior designed intramedullary interlocking nail system. In the index case distal migration was detected 5 years post-insertion but involvement of the knee became symptomatic 2 years later. The reason for migration in this case may be due to faulty implant size selection but the reason for the knee involvement may be iatrogenic. During the failed removal an inexperienced surgeon may have pushed the nail downwards which later penetrated the knee. The attempted removal was not done in our establishment so the exact operative detail can not be given. The presentation by our patient 7 years post-insertion was not different from that reported by other authors whose patients presented between 8 months to 5 years post-insertion.^{4,5}

The radiographic features of a thin sclerotic margin resembling a 'tram line' surrounding the implant and the intra-operative finding

of synovial reaction, turbid fluid akin to purulent effluent on extraction was similar to that reported in literature.⁵

Most of the cases reported by De Belder⁵ had proximal half fractures involving the isthmus, only one had lower most femoral fracture. According to literature distal migration is more common with proximal femoral shaft fractures, same as the index case. The literature reviewed does not indicate which fracture configuration is prone to migration. All his patients had a primary complaint of knee arthropathy except one who was asymptomatic, yet he and other researchers never considered it a possible differential diagnosis in knee pathology.⁵⁻⁹ K-nail or implant induced arthropathy should be borne in mind as a differential diagnosis of knee pathology in adults especially if there is a history of femoral fracture fixation. Young and upcoming surgeons should be reminded that K-nail although obsolete is still being used for management of femoral fractures in resource countries and that the complication described here can occur.

The turbid effluent and synovial tissue were sent for culture which was negative with no organism isolated. This was similar to that reported by De Belder⁵ in his case report of seven patients. Though he did not give reasons but in our case, patient has been on repeated short course antibiotics for a long period.

CONCLUSION

Distally migrated K-nail can be a cause of knee arthropathy. Medical students and young surgeons should consider it as a differential diagnosis of knee pathology.

REFERENCE

1. Ihegihu CC, Ihegihu EY. Spontaneous extrusion of a distally migrated Küntscher intramedullary nail from the left femur through a sinus over the knee joint. *Niger J of Surg Sci* 2014 Jul-Dec; 24(2); 53-5
2. Fischer MS, Fischer S. The inspiring story of Gerhard Küntscher (1900-1972). *J Bone Joint Surg [Am]* 1974;56:208-9
3. Bajwa AS, Schnaid E, Sweet ME. Current indications for open Kuntzsch nailing of femoral shaft fractures. *East and Central African Journal of Surgery*; 5(2); 11-13
4. O'Reilly PH. A complication of intramedullary nailing. *Injury: British Journal of Accident Surgery* 1975 Feb; 6(3); 261-2
5. De Belder KR. Distal migration of the femoral intramedullary nail. *The Journal of Bone and Joint Surgery* 1968 May; 50B(2); 324-332
6. Winqvist RA, Hansen ST Jr. Comminuted fractures of the femoral shaft treated by intramedullary nailing. *Orthop Clin North Am* 1980 Jul; 11(3); 633-48
7. Rehman UH, Anjum PM, Kaimkhani GM, Memon M, Qureshi MA. Interlocking versus Kuntzsch nail in the management of femoral shaft fractures. *Pakistani Journal of Surgery* 2007; 23(4); 265-9
8. Asuquo JE, Orimolade EA, Salawu L, Oladiran AB. Hemophilic arthropathy; A case report. *The Nigerian Postgraduate Medical Journal* 2014 June; 21(2); 203-6
9. Christensen ON. Kuntzsch intramedullary reaming and nail fixation for nonunion of fractures of femur and tibia. *The Journal of Bone and Joint Surgery* 1973 May; 55B (2); 312-9