

Case Report

Aortic dissection: case series

Bhavana Venkata Nagabhushana Rao*

Department of Medicine, Queens NRI Hospital, Visakhapatnam, India

Received: 01 February 2016

Accepted: 01 March 2016

***Correspondence:**

Dr. Bhavana Venkata Nagabhushana Rao,

E-mail: bhavanavnrao@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Aortic dissection may not be attended by a physician in his lifetime, but he should possess all the clinical acumen to deal with as it is a catastrophic disease. Early and accurate diagnosis will save a life. Here we present three cases we faced in sequence over a period of two months. A case of extensive dissection arch to thoracic aorta, its display in detail. Second case eliciting ambiguity between coronary ischemia and aortic dissection. Management difficulties of such clinician situation are discussed. Third case, the fracture of a renal artery stent leading to severe hypertension, abdominal pain, and aortic dissection. Such case was not described in the literature to our knowledge.

Keywords: Aortic dissection, Hypertension, Renal artery stenosis, Abdominal pain

INTRODUCTION

Aortic dissection is a rare disease whose early detection is of paramount importance for a safer outcome. This is a disease of the elderly, affecting 6th to 8th decades; men are affected a little more and earlier than women. Incidence in the general population being 2.6 to 3.5 per 100,000 person years.¹ High blood pressure is the most important risk factor, 72% of patients has hypertension other factors being atherosclerosis, pre-existing aortic aneurysm, bicuspid aortic valve, inflammatory and degenerative diseases of the aorta, coronary artery bypass surgery, cardiac catheterization, aortic valve replacement, chest injury and acute deceleration motor vehicle injuries.² Crack cocaine is a major cause in the Afro-American population. Half of the dissections in females below 40 ages are in gestation. Degeneration of collagen and elastin in aortic intima is the primary predisposing factor. Blood passes through a tear in the intima in to aortic media, separating the media from the intima and creating a false lumen. Many classifications exist for aortic dissection, mainly depending upon whether ascending aorta is involved or not, if involved being type 1 if not type 2 as in Stanford classifications.

CASE REPORT

First Case

Sixty years old female was admitted to casualty with complaints of weakness, backache, epigastric pain and pain of both axilla of one hour duration. She was a hypertensive on telmisartan 40 mg a day. She was alright until 5 days ago, she had sudden epigastric pain radiating to back and left axilla. She was profusely sweating and cold calmy at that time. She was taken a hospital nearby and an electrocardiogram (ECG) was taken which did not reveal any abnormality. She was administered pantoprazole and IV fluids intravenously. She felt fine afterwards and went home. Next day ultrasonography of upper abdomen was done which did not reveal any abnormality. But breathlessness, weakness, and restlessness persisted. At the time of admission to our hospital, she was pale, restless and hypertensive. Her Hb 10 mg/dl, leukocytes 15,000 and platelets were 50,000. ECG was normal and chest X ray revealed left lower zone consolidation suspicion of pneumonia. Upper GI endoscopy, an echocardiogram and cardiac marker were normal. She was put on intravenous piperacillin/tazobactam and azithromycin. Metoprolol was added to

control the blood pressure. She felt better during the day and at midnight she developed restlessness and breathlessness and found to be hypoxic. She improved with noninvasive ventilation. ECG revealed no abnormality except for sinus tachycardia. Chest X ray showed mediastinal widening and bilateral pleural effusions, massive on left side (Figure 1). In the perspective of this clinical picture with mediastinal widening on x-ray aortic dissection was suspected and CT angiogram was done, which demonstrated a massive aortic dissection arch to descending aorta (Figure 2 and 3). Cardiovascular surgeon was consulted, as the hospital did-not have the acumen to deal with such case she could not be taken to the surgical procedure. Meanwhile patient developed intractable hypotension and she couldn't be revived.



Figure 1: Chest X ray demonstrating cardiomegaly, mediastinal widening, bilateral pleural effusions more on left.

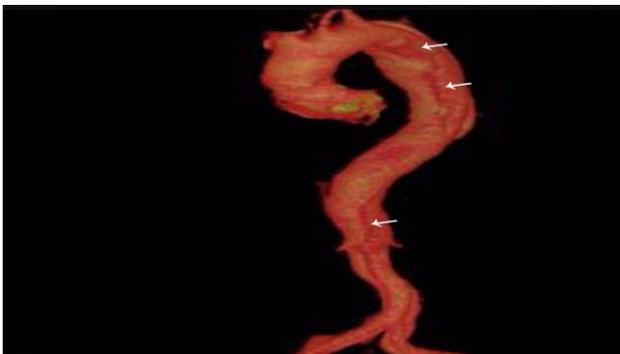


Figure 2: CT angiogram depicting aortic dissection from arch to descending aorta

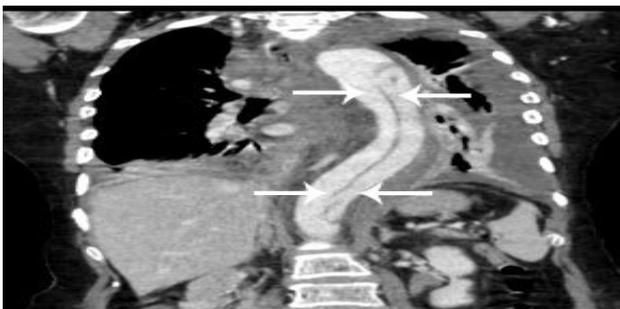


Figure 3: CT angiogram depicting aortic dissection from arch to descending aorta.

Case Two

Sixty one year old a female patient was brought to my clinic with symptoms of extreme weakness. She was known to have hypertension. She was given prednisone 60 mg a day for an eye problem two weeks ago, since then she discontinued her antihypertensive medicines. When she got into my clinic, she was unwilling to sit in the chair and preferred to lie down on the couch. She was feeling dizzy, had no chest pain, shortness of breath or sweating. Her systolic blood pressure was 60 mmHg. She was rushed to the emergency department and intravenous fluids were infused rapidly. Her blood pressure returned to normal and the dizziness subsided. Chest X ray was taken which was normal and cardiac markers were within normal limits. ECG revealed T inversion V₁ to V₅ chest leads with ST depression (Figure 4). In the scenario of abnormal ECG in elderly patient with hypotension and dizziness acute coronary syndrome was suspected, she was commenced on anti-platelet drugs and low molecular heparin. Following morning we did an echocardiography (Echo), which showed dissection of ascending aorta with mild aortic regurgitation. Transesophageal echo and CT angiogram confirmed the diagnosis (Figure 5). Anti-platelet drugs and anticoagulant were stopped at once. She was given metoprolol and ramipril. Cardiovascular surgeon was consulted. He opinioned that surgery was not warranted as dissection was small and no progressive. She was followed up with electrocardiogram every month for 6 months and every third month next six months.

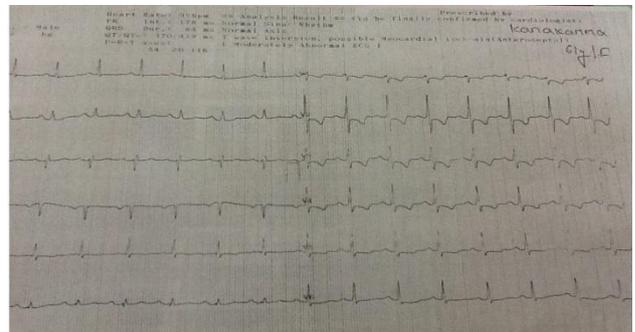


Figure 4: ECG showing anterior T inversion suggestive of ischemia.



Figure 5: CT chest demonstrating dissection of ascending aorta.

Case Three

Twenty seven year old male was admitted to our hospital with complaints of upper abdominal pain, reeling sensation, headache and general weakness. He was known to have bilateral renal artery stenosis and left renal artery was stented one year ago. He was seropositive for HCV. At the time of admission blood pressure was 190/120 mmHg, peripheral pulses were normally felt and he was having abdominal pain. ECG, chest X ray, other laboratory investigations were normal except for serum creatinine 2 mg. His blood pressure was controlled with nitroglycerin infusion, in addition to arkamin 100 micrograms three times a day prazosin extended release 5 mg twice a day, metoprolol extended release 50 mg twice daily and torsemide 20 mg twice daily. We did CT renal angiography to assess the renal circulation in the view of the acute rise in blood pressure in a patient with renal artery stent in situ. CT angio revealed a fracture of the stent and stenosis of the renal artery distal to the stent (Figure 6). Incidentally a focal dissection of the abdominal aorta at the level of gastroesophageal junction was noted (Figure 7). An acute rise of blood pressure might have caused the dissection abdominal aorta being the cause of his abdominal pain. The patient improved once the renal artery stent was replaced. He was discharged home, blood pressure and renal functions were normal at review.



Figure 6: CT angiogram showing fractured stent of left renal artery.



Figure 7: CT angiogram demonstrating abdominal aortic dissection at the level of gastro esophageal junction.

DISCUSSION

Aortic dissection a catastrophic disease, early and accurate diagnosis and immediate treatment are essential for survival. Even with all the advances in medicine, 40% patients die immediately, 30% patients admitted to hospital first thought to have another disease and only 15% of all the victims are diagnosed antemortem. Severe chest pain anteriorly or posteriorly as severe at the inception as it ever becomes is a common mode of presentation. The pain may radiate anywhere in the chest or abdomen. It can be associated with syncope, dizziness, cerebrovascular accident, myocardial infarction, heart failure, aortic regurgitation or hypotension.³ Our first patient had epigastric pain radiating back, the second one had dizziness with hypotension and third had abdominal pain. Location pain will depend upon the level of dissection. High blood pressure is the commonest cause of dissection, all our patients were hypertensives. In first patient dissection progressed and developed mediastinal widening, giving clues to the diagnosis. Clinically 96 percentages of acute aortic dissection could be identified based upon combination three clinical features abrupt onset of the abdomen or chest pain, mediastinal or aortic widening on chest X ray, a variation in the pulse of extremities or carotids and/or deference of blood pressure more than 20 mm Hg in between the arms.⁴

D dimer, if it's normal is a useful screening test to identify who do not have an aortic dissection and thus costly investigations like angiogram, MRI can be avoided. CT angiogram is a gold standard test for acute aortic dissection and available in most cardiac centers. The first case was a candidate for surgical intervention, it could not be provided as our vicinity doesn't have that expertise even if we had, it could have been a difficult endeavour as she developed intractable hypotension. ECG is useful to exclude acute MI but it may not be very useful if dissection leads to coronary ischaemia.⁵ Our second case had dizziness, hypotension and abnormal ECG suggestive of acute coronary syndrome necessitating anti platelet drugs and anticoagulants. It could have led us to a dangerous situation as the patient had a dissection, if we could not have picked up by echocardiogram. Hence it is essential to keep aortic dissection in differential diagnosis in the presence of severe chest pain even ECG is suggestive of ischaemia. Transoesophageal echocardiogram is a very specific diagnostic tool for ascending and thoracic aortic dissections. Second patient developed mild aortic regurgitation and one third of ascending aortic dissections precipitate aortic regurgitation, the regurgitant murmur better heard right of sternum. Third patient had hypertension crisis due to fracture of renal artery stent a known infrequent complication.⁶ But such patient developing an aortic dissection was not reported in the literature to our search. Abdominal aortic dissections could present with abdominal pain as in this patient. Acute dissections involving the ascending aorta are considered surgical emergencies. Surgical options include

excision of the intimal tear, obliteration of entry into the false lumen proximally or reconstruction of the aorta with interposition of synthetic vascular graft. When regurgitation complicates dissection re-suspension of aortic leaflets may restore competency or may have to opt for aortic valve replacement. Surgeons now a days use tissue glue to oppose the dissected aortic layers or after resection to place the dacron prosthesis. Mortality and morbidity have reduced except for the occasional complication of late necrosis. Endovascular techniques are in use included balloon fenestration or to stent the branch arteries whose flow is compromised. Instead of surgical repairs intraluminal stent grafts can be placed in distal dissections by percutaneous transfemoral approach. If hemopericardium complicates dissection patient should be taken to the theatre to repair of the aorta and intraoperative drainage of hemopericardium. If haemodynamically compromised aspiration of enough fluid just to rise the blood pressure will be of much benefit.

Medical therapy is the treatment of choice for distal uncomplicated dissections, stable isolated arch dissections and stable dissections of more than two weeks. Medical therapy attempts at reduction of blood pressure to 100 - 120 mmHg or to the lowest level that can be tolerated without compromising the renal and cerebral circulations and pulse rate of 60-65 per minute. Intravenous beta blockers either labetalol or metoprolol is the drug of choice. If systolic blood pressure still remains high nitroprusside can be used, but the prior beta blockade is warranted. If beta blockers are contraindicated either verapamil or diltiazem can be tried. Nifedipine to be avoided as it may increase the heart rate. Refractory hypertension may result from hyperreninemia due to the renal circulation being compromised by dissecting flap, in such situations intravenous enalapril is drug of choice. If a patient develops significant hypotension rapid volume expansion to be attempted. If vasopressors absolutely required noradrenaline is preferred over dopamine as dopamine may increase left ventricular force of contraction in high doses. But dopamine in lower dosage can be utilized to improve renal perfusion.

CONCLUSION

Aortic dissection a rare catastrophic disease and high index of clinical suspicion is essential to make an early fruitful diagnosis. Triad of chest pain, abnormal chest X ray, pulse and pressure differences in the limbs prompt us to suspect dissection. Abnormal ECG suggestive of ischaemia doesn't rule out a dissection. A sudden surge of blood pressure may precipitate a dissection even in young people.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

REFERENCES

1. Mészáros I, Mórocz J, Szlávi J, Schmidt J, Tornóci L, Nagy L, et al. Epidemiology and clinicopathology of aortic dissection. *Chest*. 2000;117(5):1271-8.
2. Larson EW, Edwards WD. Risk factors for aortic dissection: a necropsy study of 161 cases. *Am J Cardiol*. 1984;53(6):849-55.
3. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA*. 2000;283(7):897-903.
4. von Kodolitsch Y, Schwartz AG, Nienaber CA. Clinical prediction of acute aortic dissection. *Arch Intern Med*. 2000;160(19):2977-82.
5. Bossone E, Mehta RH, Trimarchi S, Cooper JV, Smith DE et al. Coronary artery involvement in patients with acute type A aortic dissection: Clinical characteristics and in-hospital outcomes. *J Am Coll Card*. 2003;41(6S1):235.
6. Stone PA, Campbell JE, Aburahma AF, Hamdan M, Broce M, Nanjundappa A, Bates MC. Ten-year experience with renal artery in-stent stenosis. *J Vasc Surg*. 2011;53(4):1026-31.

Cite this article as: Rao BVN. Aortic dissection: a case series. *Int J Res Med Sci* 2016;4:1268-71.