A Deadly Tear To Fear: An Interesting Case Presentation *

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ABSTRACT

Objectives: 1) To present a case of a patient with aortic dissection. 2) To show how the case arrived to its plausible diagnosis. 3) To discuss other illnesses discovered in the case.

Case Summary: This is a case of a 54-year old, female, Filipino, Catholic, who presented with severe chest pain, substernal in location, with pain intensity of 8/10 associated with diaphoresis and dyspnea leading to fainting spells. Initial impression was cardiogenic shock secondary to Non-ST elevated myocardial infarction. On physical examination, the patient was drowsy and in cardio-respiratory distress. She had symmetrical chest expansion and no retractions were noted. Clear breath sounds were noted in all lung fields. She had an adynamic precordium with normal rate and regular rhythm, however with distant heart sounds. There was no murmur, heave or thrill appreciated. Vital signs at the emergency room showed a blood pressure of 110/80 which eventually became 80/50 mmHg, respiratory rate of 22 cycles per minute, heart rate of 80-100 beats per minute and was afebrile. Patient was scheduled for a stat coronary angiography, however on further reassessment, repeat ECG showed resolution of the inferolateral wall ischemia but this could not explain her fluctuating blood pressure. When the patient underwent the scheduled bedside 2D echo, a moderate cardiac tamponade was discovered with a 4.5 cm aortic dissection. With these findings, patient underwent aortic repair, graft insertion with evacuation of hematoma. She was discharged stable and with no recurrence of chest pain.

Key Words: Aortic Dissection, Cardiac Tamponade, Transthoracic Echocardiogram (TTE), Transesophageal Echocardiogram (TEE)

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INTRODUCTION

In just a blink of an eye, everything can change.

One minute you are enjoying your life, enjoying your youth, enjoying the company of your loved ones and the next minute, you could be on the brink of life and death— you could be fearing for your life.

Things will never be the same again.

Life is very unpredictable. Even if we want to take control of it, there are certain things that are just out of our hands. Just as in the case of this fifty-four year old female, life takes an unexpected turn in just a blink of an eye, as she is riddled with a devastating condition — a condition that would change not just her life, but her family's as well.

CLINICAL HISTORY

General Data:
This is a case of B.L., a 54-year old, female, married, Filipino, Catholic, residing in Manila, who was admitted last March 27, 2016.

Chief Complaint:
"Chest pain"

History of Present Illness:
Few minutes prior to admission, while accompanying her husband who was being seen at the emergency room for dizziness; the patient experienced sudden onset of severe chest pain, substernal in location, with pain intensity of 8/10 associated with diaphoresis and dyspnea leading to fainting spells. She was subsequently admitted due to persistence of symptoms.

Past Medical History
The patient is a known hypertensive for 3 years, with usual blood pressure of 120-140 systolic but she was non-compliant with her maintenance medications of Losartan 50mg once a day. She has no history of asthma or any childhood diseases. She was previously hospitalized due to intestinal amoebiasis but wasn't able to comply with the take home medications. She has no known allergy to any food or medication.

Personal and Social History
The patient is a chronic smoker of 5 pack years (up to present) and she is also an occasional alcoholic beverage drinker. She denies illicit drug use.

Family History
The patient has a family history of hypertension in the maternal side of her family. There is no family history of diabetes mellitus, bronchial asthma, heart, and kidney disease.

Physical Examination
Patient was drowsy and in cardio-respiratory distress, with an initial blood pressure of 110/80, respiratory rate of 22 cycles per minute, heart rate of 80 beats per minute and lips, tongue and buccal mucosa. There was no nasoaural discharge, no tonsillopharyngeal congestion or cervical lymphadenopathies. She had symmetrical chest expansion and no retractions were noted. Clear breath sounds were noted in all lung fields. She had an adynamic precordium with normal rate and regular rhythm, however with distant heart sounds. There was no murmur, heave or thrill appreciated. She had a flat abdomen with normoactive bowel sounds and with no tenderness or organomegaly in all quadrants. She had grossly normal extremities with full and equal pulses. No neurologic deficits were noted at that time.

Initial Impression
The initial Impression at the ER level was cardiogenic shock secondary to Non-ST elevated myocardial infarction.

Course of Admission
At the ER, the patient's ECG revealed an inferolateral wail ischemia (Fig. 1) and her BP was noted to be 80/50; fast drip of 200 cc of PNSS was done and despite the fluid resuscitation, the patient remained hypotensive. Dopamine drip was started at 18 gtts/min and patient was then advised admission. Troponin I and CKMB were extracted which revealed a slight elevation of Troponin I (Table 1), and an x-ray of the chest was done. She was given 4 tablets of aspirin, sublingual Isordil, and IV morphine to relieve the chest pain.

The patient was then admitted and was referred to cardiology service for co-management. Due to drowsiness, sudden onset of headache and unresponsiveness, a plain cranial CT scan was done to rule out a CVD infarct but was noted to be normal. Isoket drip was started at 5cc/hr. The impression at that time was cardiogenic shock secondary to Non-ST elevated Myocardial Infarction. The patient was then prepared for an emergency coronary angiogram and was hooked to triple inotropes to increase her blood pressure. She was taken to the cathlab and coronary angiogram was done which revealed an 80% occlusion of the proximal

left anterior descending artery; thus an intra-aortic balloon was inserted to prevent ischemia or vasospasm. She was taken to the Coronarv Care Unit for observation, with NGT inserted and maintained in adequate oxygenation.

Course in the Ward

On the first hospital day the patient was awake but was irritable. She was noted to have rhonchi bilateral and her urine output was noted to be decreased at 25cc/hr. A urinalysis was done revealing complicated UTI {Table 2) and her creatinine level was increasing by determinations (Table 3). The added impression at that time was a beginning urosepsis vs cardio-renal syndrome.

Her BP was stable at 110/60 and the cardiology service began downtitrating her ionotropes. However, the patient was still oliguric hence she was scheduled for a bedside KUB ultrasound and at the same time, referral to nephrology service was done for the management of urosepsis and increasing creatinine. All the while, the patient was referred for fluctuating BP from 80/50 to 250/120, heart rate was 100s. She was also diaphoretic and pale but there were no murmurs or muffled heart sounds. The ECG was repeated showing resolution of the inferolateral wall ischemia (Fig. 2) but this could not explain her fluctuating blood pressure. It became a challenge then to us as to dig deeper on what may have caused this.

When the patient underwent the scheduled bedside 2D echo, a moderate cardiac tamponade was discovered with a 4.5 cm aortic dissection. The family was told that the patient should be referred to Surgery service for an emergency pericardiostomy. During that time, the risks and benefits of the procedure were well explained to the family and the husband eventually consented. The big question on everyone's minds was did the emergency coronary angiogram with stenting of the LAD caused the cardiac tamponade or was there a more serious underlying cause. The consensus was that if the pericardial fluid drained was bloody, the consideration was iatrogenic or traumatic insertion of the coronary angiogram catheter and the surgeon would have to open the thoracic cavity to repair the damage.

The patient underwent an emergency subxiphoid pericardiostomy and drained 200 cc of serosanguinous pericardial fluid. Since there was blood in the drained fluid, there was then still a dilemma to open and repair the damage or not. Nonetheless, the surgeon opted for a more conservative approach since the fluid was not as bloody as expected and attached a pericardiostomy tube for drainage.

Her BP became stable after draining the pericardial fluid and she was off from the triple ionotropes. What now then be the next move to know the cause of the tamponade?

On the 2nd hospital day, the patient underwent bedside Transesophageal Echocardiogram (TEE). The findings showed an extensive thoracic dissection from the sinu-tubular junction to the ascending aorta with moderate pericardial fluid with thrombosed circumferential intimal flap. It seemed as though she was having the aortic dissection at the emergency room which was why she was hypotensive but the thrombosed intimal flap prevented more blood from seeping out of the aorta hence saving her life. She tolerated the procedure but was noted to have BP fluctuations from 80/40 to 250/100. Again, Inotropes were then given and titrated to stabilize the BP. At this time, CBC results showed a decreasing hemoglobin and hematocrit hence 1 unit of pack RBC was transfused. The plan then, was to push through with the chest CT aortogram with contrast, then she would undergo hemodialysis to prevent further injury to the kidneys (which showed increasing creatinine levels) due to the contrast.

On the 3rd hospital day, she was taken to the Intensive Care Unit and chest CT aortogram was done which showed extensive aortic dissection just a day that passed. This prompted the surgery service to schedule the patient for an aortic dissection repair. A multidisciplinary family conference was held and was attended by all the specialties concerned: TCVS, family medicine, anesthesiology, cardiology service, interventional cardiologist, and nephrology service. It was explained to the family that there was an intimal break of the ascending aorta with spillage causing intramural hematoma and had been going on progressively. The plan was to do aortic repair using aortic graft before the tear would reach the femorals. The procedure was a very complicated one, and the risk of the patient dying on the operating table or after the operation was very high. The operative mortality was 60% with 40% recovery or survival. The mortality rate if there was no intervention was 95-100%. The urgency of the operation and the risks were thoroughly explained and the family decided to push through with the operation. Then, the anti-platelets were put on hold and the necessary blood products were procured. Bedside chest x-ray was done showing pulmonary congestion, yet her oxygenation status was adequate.

On the 4th hospital day, the patient underwent aortic repair, graft insertion with evacuation of hematoma (Figures 3-5). She underwent transfusion with 7 units of packed RBC, 12 units of platelet concentrate, 8 units of fresh frozen plasma, and 4 units of cryoprecipitate. The patient tolerated the procedure well with stable vital signs. Although she was intubated, she was awake and had spontaneous movement.

On the 5-7th hospital day, the patient was extubated and started on incentive spirometry. She was able to tolerate sitting down on the bedside chair. Since her BP was stable, she was transferred to regular room. It was this time that hemodialysis was done and her creatinine levels eventually decreased (Table 4). Her hemoglobin levels also increased.

On the 8th hospital day, patient was seen comfortable with no episodes of chest pain and dyspnea. Vital signs were stable but with febrile episodes. She was noted with hypokalemia which was corrected with oral K+ supplementation. Dopamine and Nicardipine were then discontinued and she was also referred to the Clinical Nutrition Management Service for nutritional evaluation. Removal of dialysis catheter was done. On chest auscultation, she was noted with rales on both lung bases thus, repeat CBC was done which showed increased WBC: 21.44, neutrophils: 74 and lymphocytes: 8.

On the 9th hospital day, she had watery bowel movement, > 10x with food particles. She was started on Hidrasec 1 tab TID and bioflora BID. She was noted with crackles on left base. Piptazo was started initially from 2.25g IV to 4.5grams IV q8. Present IV fluid was decreased to 40 cc/hour and amlodipine to 5mg/tab BID. No fever recurrence was noted then.

On the 10th hospital day, patient had no febrile episodes and loose bowel movement but with one episode of hematochezia. She was still noted to have occasional bibasal crackles with decreased breath sounds, left base but no chest pain and no dyspnea were noted. Patient was started with Rebamipide (Mucosta) 1 tab TID and Pantoprazole was shifted to 40mg IV q12. Oxygen via nasal cannula was discontinued. She was able to tolerate ambulation around the bed without any postural dizziness or cardiopulmonary symptoms, thus was allowed to transfer to a regular room.

On the 11th hospital day, while in a regular room, there were no chest pain and dyspnea; however, she had another episode of hematochezia. Rales with diminished breath sounds left base was found upon auscultation and chest x-ray and left lateral decubitus were requested to evaluate for a possible left pleura! effusion. She was also started on Metronidazole (Dazomet) 1 tablet TID due to a previous amoebiasis infection.

On the 12th hospital day, ultrasound of the left hemithorax was done, instead of the chest x-ray which revealed an interval increase in the amount of free pleura! fluid collection occupying the left mid to lower hemithorax, with an estimated volume of about 298 cc from a previous of 20cc. Passive atelectasis of the adjacent lung was also appreciated. With approximately 200-300 cc of pleural fluid, TCVS service recommended conservative management as patient had no clinical symptoms wherein thoracentesis was warranted.

On the 14th-15th hospital day, patient had no recurrence of hematochezia and febrile episodes. Patient was supposedly for discharge but had to be deferred due to anemia. Latest CBC result showed decreased hemoglobin and hematocrit and increased platelet. She was transfused with 1 unit PRBC properly typed and cross-matched. Hematology referral was also suggested by other service due to presence of thrombocytosis.

On the 16th hospital day, repeat CBC showed hemoglobin of 109, hematocrit 33, white blood cell count of 16.85 and platelet count 148. Possible consideration for the increased platelet was reactive thrombocytosis due to inflammation.

On the 17th hospital day, patient was seen awake, comfortable with no subjective complaints. No recurrence of hematochezia, chest pain and dyspnea. She was then advised for discharged with home medications by ail services.

CASE DISCUSSION

Aortic Dissection is a syndrome caused by an intimal and medial tear in the aorta with propagation of a flase lumen within the aortic media (2). The intimal tear may be a primary event or secondary to hemorrhage within the media. The dissection may occur anywhere along the aorta and extend proximally or distally into other arteries (1,3,4). The wall of the aorta consists of 3 layers: the intima, media, and adventitia. The intima is the innermost layer; the media, the middle layer; the adventitia, the outer layer (7)(fig.6).

Up-to-date data on the epidemiology of aortic dissection are limited. However, in the Oxford Vascular study, the incidence of aortic dissection is estimated at six per hundred thousand persons per year. Many patients with aortic dissection die before presentation to a hospital or prior to diagnosis. There is a missed diagnosis in 38% of patients on initial evaluation. There is a high mortality rate wherein 25% of patients during the 1st 24 hours, 70% in the 1st week, 80% at 2 weeks for proximal dissections and 10% in distal dissections (1,3,4). Hence, early diagnosis is crucial and essential. Our patient was, in a sense, "lucky" to be inside a hospital when the onset of her symptoms occurred.

Aortic dissection can be divided into two types, which are the acute and chronic types, depending on the duration of symptoms. Acute aortic dissection is present when the diagnosis is made within 2 weeks after the initial onset of symptoms, and chronic aortic dissection is present when the initial symptoms are of more than 2 weeks in duration. About one third of patients with aortic dissection fall into the chronic category. The most common site of initiation of aortic dissection is the ascending aorta (50%)(4).

Anatomically, aortic dissection has been classified by two schemes. The DeBakey classification consists of the following three types: I, both the ascending and the descending aorta are involved; II, only the ascending aorta is involved; and III, only the descending aorta is involved (Fig. 7). The other classification is The Stanford, which consists of the following types: Type A, involving the ascending aorta regardless of the entry site location; and Type B, involving the aorta distal to the origin of the left subclavian artery (4)(Fig. 7). For our patient, there was a noted extensive thoracic aortic dissection with thrombosed circumferential intimal flap from the sinotubular junction to the descending thoracic aorta.

This incidence is higher in men than in women and increases with age (4,5). In the International Registry of Acute Aortic Dissection (IRAD) registry, the mean age was 63 years; 65% were men. The prognosis is poorer in women, as a result of atypical presentation and delayed diagnosis. Peak incidence occurs at age 50 to 65 or, for patients with congenital connective tissue disorders (eg, Marfan syndrome, Ehlers Danlos syndrome), at age 20 to 40. The most common risk factor associated with aortic dissection is hypertension, observed in 65-75% of individuals, mostly poorly controlled (5).

Pain is the most common presenting symptom of aortic dissection. The pain of an aortic dissection is midiine and is experienced in the front and back of the trunk, depending on the location of the dissection (4). As seen in our patient, the onset of pain is typically catastrophic, and it reaches a maximum level suddenly. The pain could be sharp, ripping, tearing, or knife-like in nature, but the abruptness is the most specific characteristic of the pain. The pain of aortic dissection does not commonly radiate into the neck, shoulder, or arm, as is typical of the pain of an acute coronary syndrome. Chest pain is more common in Type A dissections while back or abdominal pain is more common in Type B dissections however, clinical presentations may overlap (1,2,4).

When examining the cardiovascular system one must search for the following: 1) signs relating to hemopericardium: pulsus paradoxus, faint or absent heart sounds, distended neck veins, shock, 2) signs relating to aortic root dilatation: wide pulse pressure, diastolic murmur over the aortic area, 3) compression of the true aortic lumen: systolic murmur over any part of the aorta, 4) pulse deficits: a difference of 20mmHg or more in blood pressure between arms, a weaker central or peripheral pulse compared to the contralateral side or palpable thrills or audible bruits over pulses (1,4).

Abdominal examination is essential to exclude other potential causes of the patient's symptoms such as pancreatitis, perforated hollow viscus and ruptured abdominal aortic aneurysm.

The diagnosis of aortic dissection begins with clinical suspicion, which is the most crucial step. The two important steps in the evaluation of patients with suspected aortic dissection are 1) to confirm the presence of dissection and 2) to differentiate between proximal and distal dissections. The diagnosis should be confirmed rapidly and accurately, preferably with an easily available noninvasive modality (4).

Retrograde aortography (Fig. 8) was the gold standard for assessing patients in the 1970s and 1980s, but it has been replaced by cross sectional imaging such as Computed Tomography (CT) scan, Magnetic Resonance Imagine (MRI), Transthoracic Echocardiogram (TTE), Transesophageal Echocardiogram (TEE), which performs better and has a better safety profile (1,2,9).

Although chest radiography and electrocardiography are often ordered in the emergency care setting, these tests cannot establish or exclude the diagnosis of dissecting aortic aneurysm (2,4,9).

Chest radiography lacks the specificity for a diagnosis of aortic dissection, though the classic radiographic sign that is suggestive of aortic dissection is the widening of the mediastinal shadow (Fig. 9), which has been reported in up to 50% of cases of aortic dissection. In our patient, no mediastinal widening was noted (Fig. 10). The mediastinum bulges to the right with dissection of the ascending aorta, and to the left with dissection of the descending thoracic aorta (1,2,3,4,9).

The other chest radiographic signs reported in patients with aortic dissection are altered configuration of the aorta, a localized hump on the aortic arch, a widening of the distal aortic knob past the origin of the left subclavian artery, aortic wall thickness indicated by the width of the aortic shadow beyond intimal calcification, displacement of the calcification in the aortic knob, a double aortic shadow, disparity in the sizes of the ascending and descending aortas, and the presence of a pleural effusion, most commonly on the left. These radiographic signs are suggestive of, but not diagnostic of, aortic dissection (4,9).

Also, ECG changes are nonspecific (chiefly, nonspecific ST-segment/T-wave changes) (4). It is also not helpful in distinguishing aortic dissection from acute coronary syndrome as it is only completely normal in 30% of patients with dissection. In the IRAD series 3% of all aortic dissection patients had ST elevation myocardial infarction pattern; 15% acute ischemic changes and 41% had non-specific ST segment and T wave changes (3,4,9).

If the history suggests aortic dissection, a diagnosis of acute coronary syndrome should not be made even if the ECG has obvious STEMI or NSTEMI patterns as antiplatelet treatment, heparin or thrombolysis may worsen the prognosis of the patient. It is always better to withhold these treatments when aortic dissection is considered and immediately arrange for urgent aortic imaging to clarify the diagnosis (3,4,8,9).

Imaging tests such as the CT scan, MRI, TTE and/or TEE are done once the patient is hemodynamically stable.

The key finding on contrast-enhanced images is the intimal flap separating two lumens (Fig. 11 and 12). Multidetector computed tomography angiography is recommended by the European Society of Cardiology as the first line of investigation for patients with suspected acute dissection (1,9).

The MRI can detect aortic dissection accurately, can delineate the extent of the dissection, can demonstrate the site of the entry tear, can identify the arch vessels that are involved, and can assess the renal artery involvement (Fig. 13). Both the sensitivity and the specificity of MRI are in the range of 95 to 100% (4,9).

The TEE is widely available, is safe in experienced hands, and can be performed quickly and easily at the bedside. These advantages make TEE ideal for use in most patients with aortic dissections, including relatively unstable patients. The sensitivity of TEE has been reported to be as high as 98%, and the specificity ranges from 63 to 96%. Moreover, the TEE is able to identify the entry site of dissection; the presence of thrombus in a false lumen; abnormal flow characteristics; the involvement of coronary and arch vessels; the presence, extent, and hemodynamic significance of pericardial effusion; and the presence and severity of aortic valve regurgitation. The most important diagnostic finding of aortic dissection that can be seen on TEEs is the presence of an undulating intimal flap within the aortic lumen that differentiates a false lumen from a true lumen (1,3,4,9) (Fig. 14).

Surgery is the treatment of choice. Acute Type A aortic dissection has a mortality of 50% within the first 48 hours if not operated (9).

The International Registry of Acute Aortic Dissection has suggested that, if left untreated, proximal (Stanford type A or DeBakey type I or II) dissection carries a one-week mortality of 50-91% owing to complications such as aortic rupture, stroke, visceral ischema, cardiac tamponade, and circulatory failure (4,5,9).

European Society of Cardiology recommends regular cross sectional imaging of the aorta, preferably with magnetic resonance angiography, at one, three, and 12 months after discharge and every six to 12 months thereafter, depending on aortic size.

Various experts also advocate the combined use of echocardiography with axial imaging for routine surveillance. All patients should receive lifelong antihypertensive treatment, including p blockers, with a target blood pressure of 120/80 mm Hg (1,4,5).

In the article published by the International Registry of Acute Aortic Dissections, establishing clear life style goals for patients with thoracic aortic disease is important in improving long-term health and reducing the risk of both fatal and nonfatal complications.

Having regular aerobic exercise, maintaining a low fat and low salt diet, and achieving ideal body weight are all connected to effectively controlling the blood pressure, cholesterol and associated aortic stress (10).

Cessation of smoking and avoidance of tobacco use is also critical. Tobacco use is linked not only with the development of thoracic aortic disease but is highly linked to aortic rupture. Avoidance of cocaine or other stimulating drugs such as methamphetamine is equally important as sudden surges in blood pressure and pulse attributed to such agents has been described as a trigger for aortic catastrophes (5,10).

Having a regular routine of aerobic exercise has multiple benefits in helping patients achieve ideal blood pressure, heart rate, and body weight. A lot of our patients enjoy sports such as basketball, tennis, biking etc., and would want to continue in such activities if possible (5,10).

Another important thing to stress is the importance of adherence to their medications. Such medications include beta blockers and other antihypertensive agents. Patients who suddenly discontinue their medications because they run out of stock or forget their medications at home when travelling, may find themselves in a hypertensive crisis with a potentially catastrophic result (5,10).

In terms of work, patients with thoracic aortic disease generally can function normally in most types of occupations provided that the job does not involve any heavy physical activity or manual labor. Finally, it is important that patients with thoracic aortic disease recognize that aortic disease is usually a lifelong condition that puts them at future risk for acute aortic syndromes. Even those who have received advanced surgical or endovascular therapy must understand that their aortic disease has not been "cured" by the interventions. Educating them about what to do in the event of the sudden onset of chest, back or abdominal pain, or the sudden development of an ischemic complication (i.e., neurologic or limb) and the critical nature of getting to an emergency department promptly, is of the utmost importance. It is similarly important that those who live with or care for such patients understand what action needs to be taken should concerning symptoms arise (5,10).

As primary care physicians, it is part of our duty to establish these goals and instill these important points to our patients.

CONCLUSION

It is not often that we encounter cases of aortic dissection in our practice. However, as primary care physicians, our clinical eye, clinical judgment and skills will be put to the test in the proper identification and management of such a case when the need arises.

Residents in training in Family Medicine, like me, and other primary care physicians are called on to be equipped with the knowledge on how to deal with common as well as uncommon cases.

As with many other diseases, proper history taking is the most essential part of the assessment of a patient as it will be the patient's symptoms that will alert the physician to the possibility of aortic dissection. However, aside from obtaining adequate history, the physical examination of these patients needs to be thorough but also must be done quickly. Proper referrals must be made o ensure that the necessary steps are taken. One mistake or misstep can spell the difference between life or death for a patient, thus "a deadly tear" of the aorta is a "fear" to anyone's life - to remain silent in deep sleep forever.

APPENDIX

FIGURES

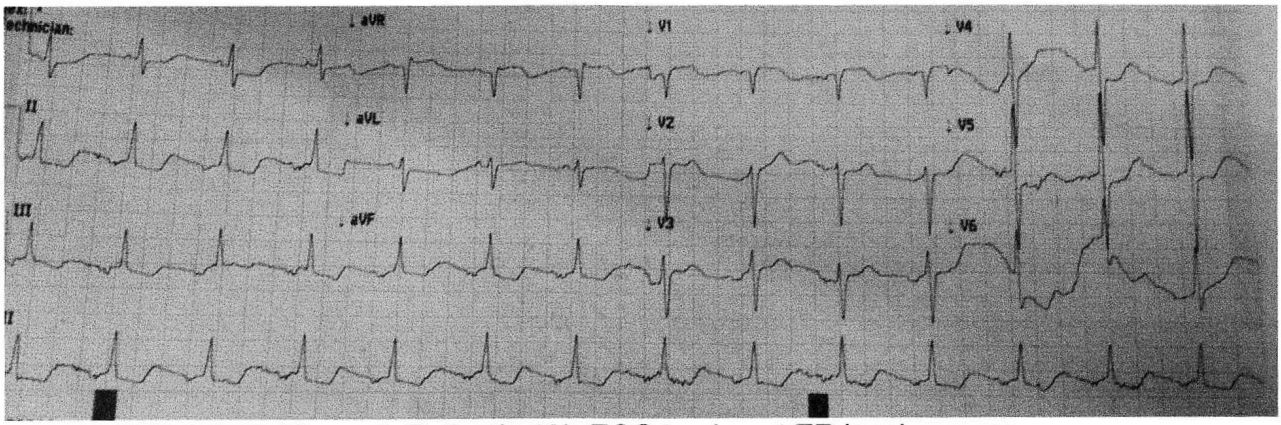


Figure 1. Patient's 12L-ECG tracing at ER level

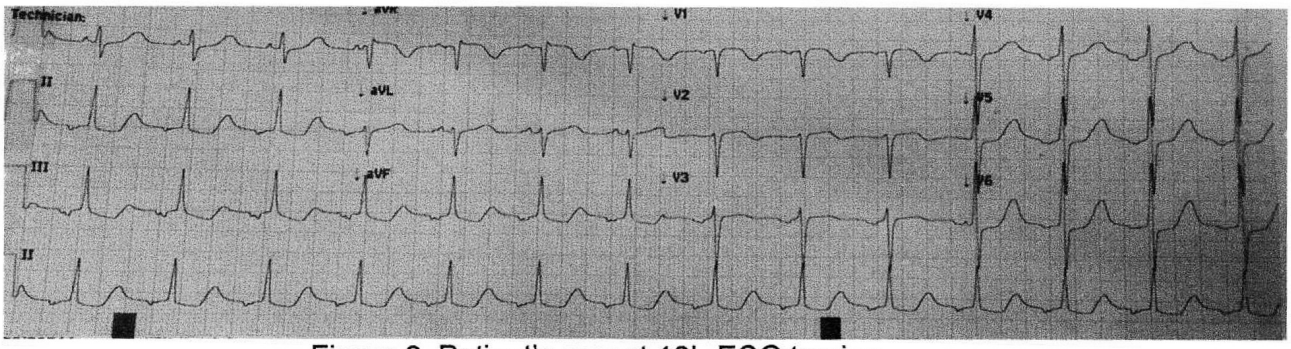


Figure 2. Patient's repeat 12L-ECG tracing

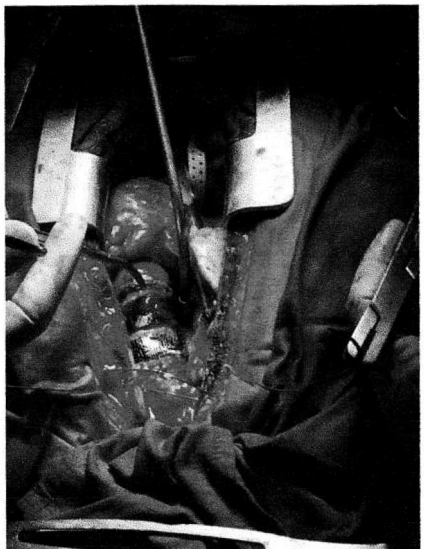


Figure 3. Aortic repair, graft insertion with evacuation of hematoma

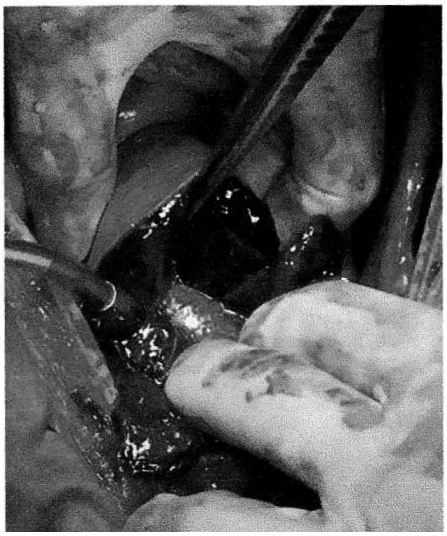


Figure 4. Aortic repair, graft insertion with evacuation of hematoma

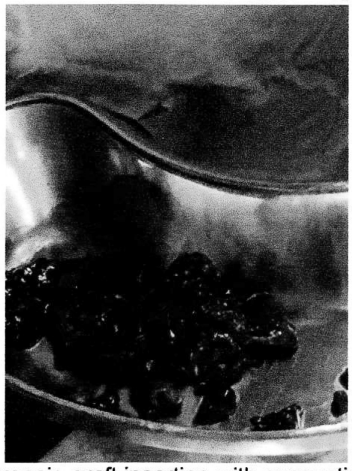


Figure 5. Aortic repair, graft insertion with evacuation of hematoma

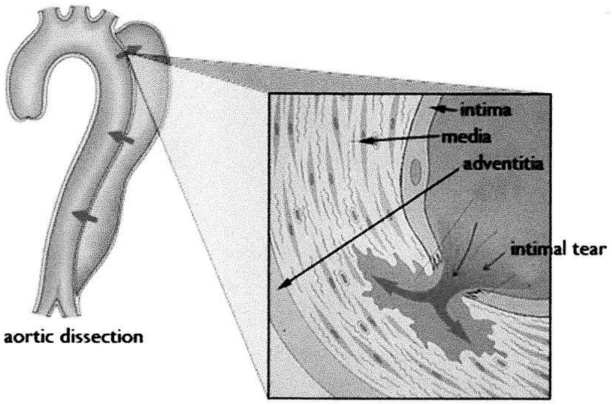
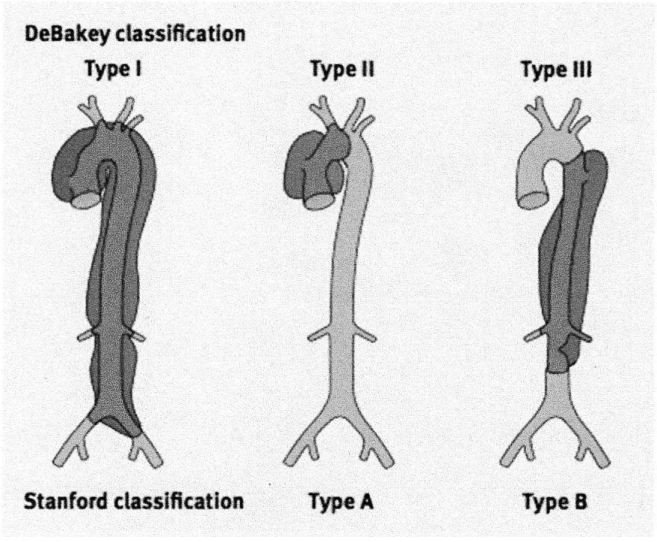
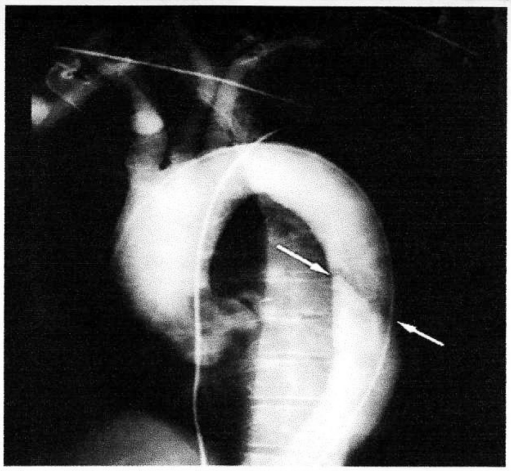


Figure 6. Layers of the aorta and intimal tear.
Source: Layers of the Aorta. From ***The Marfan Syndrome***, by Reed E. Pyeritz, M.D., Ph.D. and Cheryll Gasner, M.N., C./F.N.P. Fifth Edition, July 1999, Revised September 2001.





Descending aortic dissection This aortogram demonstrates dissection of the descending thoracic aorta, arising immediately distal to the origin of the left subclavian artery. An oblique lucency is noted within the lumen of the aorta, which is a diagnostic feature (arrows). Courtesy of Jonathan Kruskal, MD.

Figure 8

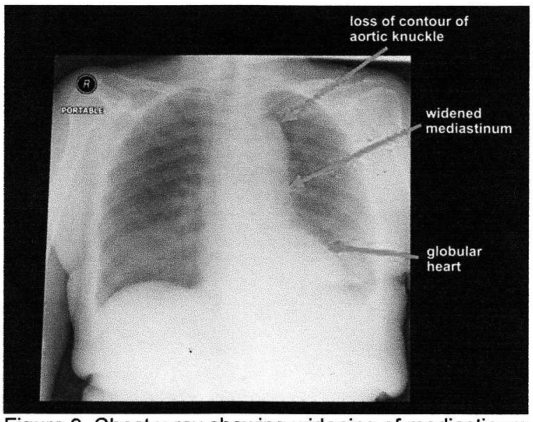


Figure 9. Chest x-ray showing widening of mediastinum

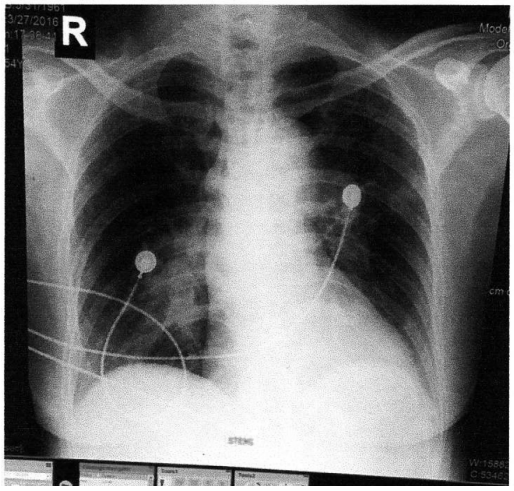
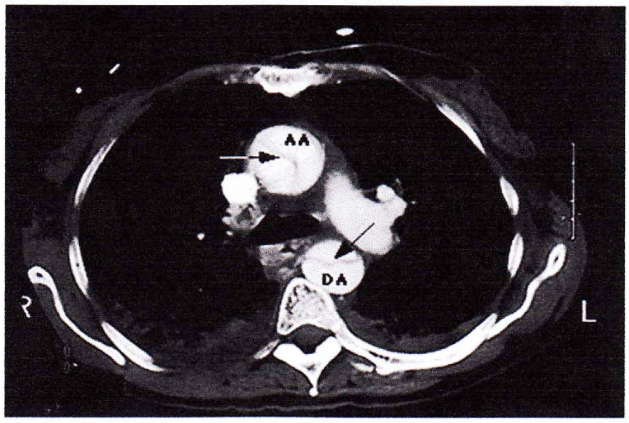


Figure 10. Ches x-ray of patient at ER level



Spiral CT of thoracic aortic dissection
Transverse plane through ascending (AA) and descending (DA) thoracic aorta showing the intimal flap (arrows) and both lumens of a type A aortic dissection. One cannot distinguish between the true and false lumens based on this view alone. Courtesy of Vassilios Raptopoulos, MD.

Figure 11

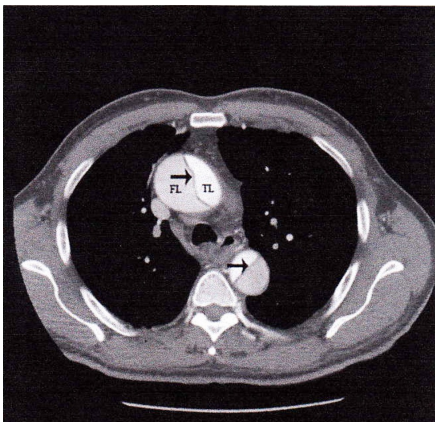
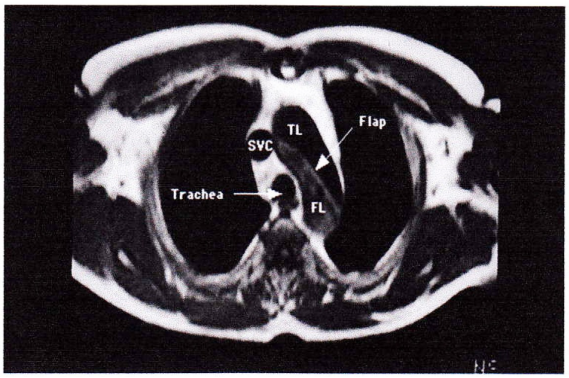
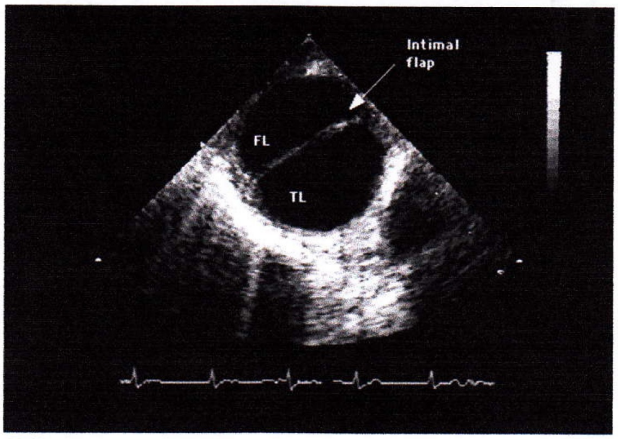


Figure 12. The arrows demonstrate the intimal flaps in both the ascending aorta (anterior) and the descending aorta (posterior) . TL is the true lumen as this has contrast within it whilst the darker flase lumen (FL) does not.



Aortic dissection on MRI Spin-echo magnetic resonance imaging scan of an aortic dissection in the transverse plane at the level of the aortic arch. The true lumen (TL), false lumen (FL) and intimal flac can be easily identified. The tranchea and superior vena cava (SVC) are also seen. Courtesy of Warren Manning, MD



TEE of aortic dissection Transesophageal echocardiography (0° crystal orientation) of type B dissection of the descending thoracic aorta. A thin intimal flap is clearly noted. FL and TL refer the false and true lumen, respectively. Courtesy of P Vignon, MD and Roberto Lang, MD.

Figure 14

TABLES

	Normal Values	
Trop I	0-0.034	**0.035**
CKMB	0-4.55	**2.71**

Table 1

URINALYSIS	
pH	6.0
Glucose	Negative
Protein	1.0
Blood	**200**
WBC	**153.6**
RBC	**4047**
Epithelial cells	41
Bacteria	92.1

Table 2

	Normal Values	**March 27**	**March 28**
BUN	2.5-6.4	5.5	12.4
CREA	46-92	116	155

Table 3

	March 29	**March 30**	**March 31**
BUN	9.6	9.8	10.2
CREA	233	89	103

Table 4

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