Original Article

Aortic dissection in Siriraj patients: Computed tomography findings

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Abstract

Background CTA has replaced angiography in both diagnosis and evaluation of aortic dissection. Most findings are associated with true and false lumens which account for the most important information in both diagnosis and management.

Objective: To describe computed tomographic (CT) findings including types based on Stanford classification, true and false lumens, acute and chronic aortic dissections, relation to origins of aortic branches, complications and other related findings.

Methods: Computed tomographic angiography (CTA) scans of one hundred and twenty patients with aortic dissection during 2007 to 2016 were retrospectively reviewed. The findings indicating types, true and false lumens, acute and chronic, origination of aortic branches, complication and other related findings are categorized.

Result: Most true lumens were smaller, having outer wall calcification. Most false lumens were larger, showing beak sign, cobweb sign, and intraluminal thrombi. However, the larger lumens could be true lumens as well as the smaller lumen could be a false lumen and outer wall calcification could be seen in a false lumen. The larger true lumens and the smaller false lumens with outer wall calcifications were more often found in chronic aortic dissection than acute aortic dissection. Both acute and chronic aortic dissections were more Stanford type B than type A. Complications included rupture, hemopericardium, hemothorax, hemomediastinum and distal organ infarction, which were more frequent in acute dissection. Intrathoracic complications were more commonly caused by type A acute dissection. Renal infarction was the most common complication in type B acute aortic dissection.

Conclusion: Most CT findings of aortic dissection in this study were typical. Atypical findings were also found in both acute and chronic aortic dissections. Outer wall calcifications of false lumens in acute aortic dissection were found in 2 cases.

Keywords: Aortic dissection, Stanford classification, Computed Tomography angiography, Siriraj patients

Introduction

Dissection is the result of a spontaneous longitudinal separation of the aortic intima and adventitia caused by circulating blood gaining access to and splitting the media of the aortic wall. The intimal tear allows blood to enter the media from the vessel lumen. The blood-filled space within the medial layer becomes the false lumen (Figure 1). This results in two lumens- a true lumen and a false lumen- with the false lumen having pressures greater than or equal to those in the true lumen [1, 2]. It is important for the radiologist to determine the luminal origins of branch vessels in patients who may undergo surgical repair or percutaneous treatment with endovascular grafts

In most CT examinations, the identity of the true lumen may be determined by its continuity with an un-dissected portion of aorta but in some patients, this continuity is difficult to appreciate or impossible to establish continuity. However, there are many other imaging findings which help to base the distinction between true and false lumens.

There are many prior studies about these findings in CT angiography (CTA) [2-6]. They had summarized the findings which associated true or false lumen: true lumen associate with sign of smaller lumen and outer wall calcification and false lumen associate with larger lumen, beak sign, cobweb sign and intraluminal thrombus (Figure 2).

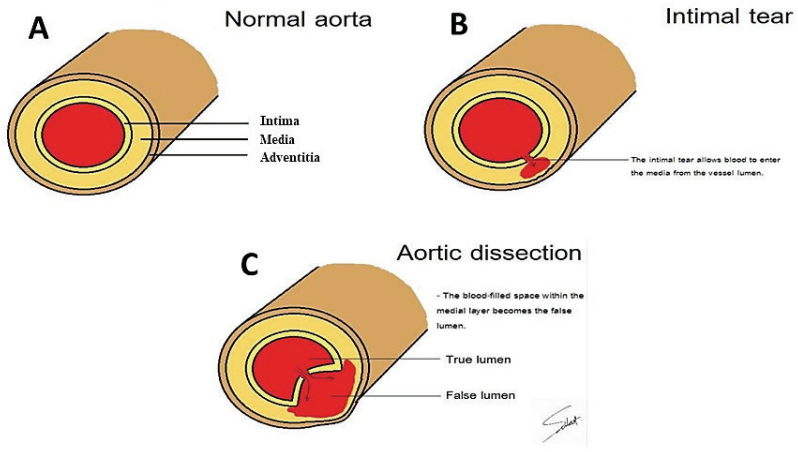

Figure 2. *The mechanism of aortic dissection :*
A : Normal aortic lumen in cross section,
B : Initial intimal tear allows blood to enter the media from vessel lumen,
C : Blood-filled space within the media become false lumen.

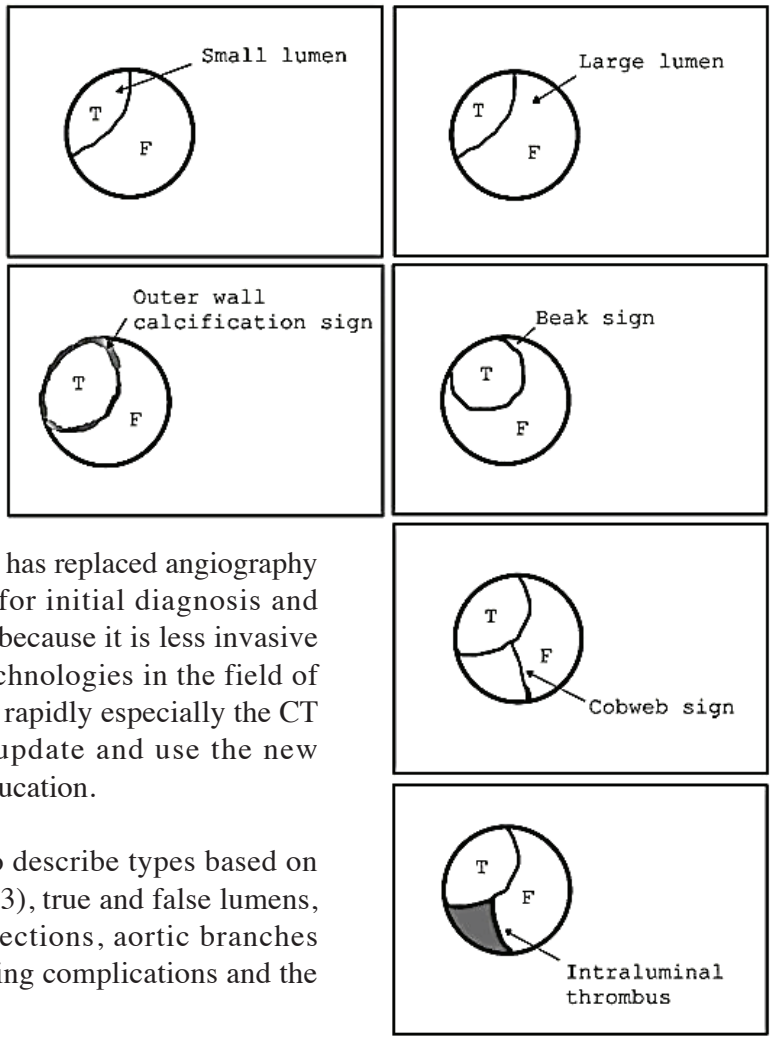

Figure 2. *The pictures reveal signs of true and false lumens in axial plane of aorta.*
: Left column reveals sign of true lumen; Small lumen and outer wall calcification.
: Right column shows sign of true lumen; large lumen, beak sign, cobweb sign and intraluminal thrombus.

Currently, MDCT angiography has replaced angiography and use as the gold standard for initial diagnosis and evaluation of aortic dissection because it is less invasive and is widely used.[7] The technologies in the field of radiology have been developed rapidly especially the CT scanner. It is interesting to update and use the new technologies for continuous education.

The purpose of this study is to describe types based on Stanford classification (Figure 3), true and false lumens, acute and chronic aortic dissections, aortic branches origination, findings of dissecting complications and the other findings.

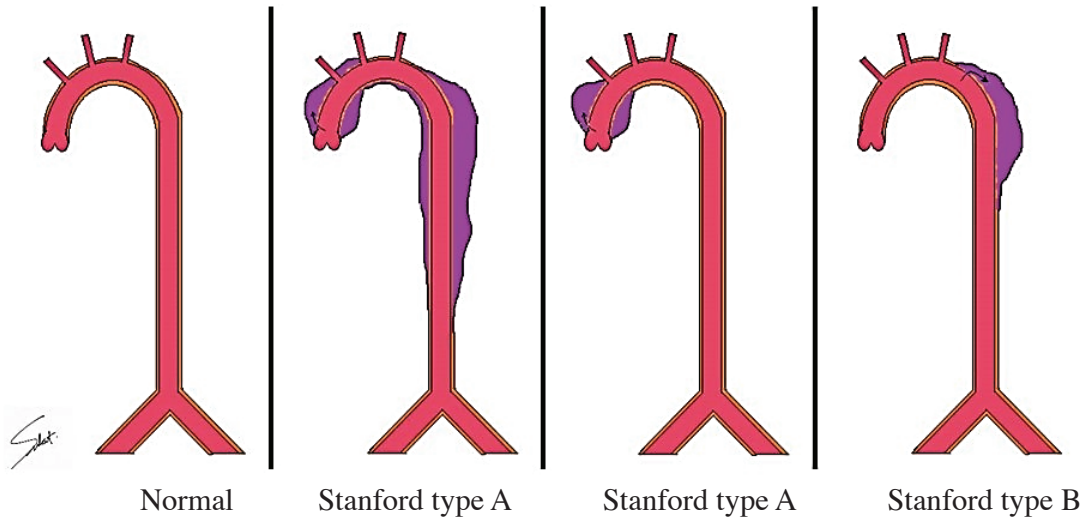

Figure 3. *Aortic dissection: Stanford classification*

Materials and methods

Study population

Acute and chronic aortic dissection patients in Siriraj hospital since January 2007 to January 2016 who underwent CT angiography of thoracic and abdominal aorta were reviewed and categorized.

If more than one CT examination were available for a particular patient, the earliest acute scan or the latest chronic scan would be used for the purposes of this study. One patient was excluded because there was no history of onset to categorize acute or chronic aortic dissection.

Imaging

All one hundred and nineteen patients underwent MDCT angiography imaging at our institution using a dedicated aortic protocol with a 1.25-mm-slice thickness, 120 kV and 300 m A. Intravenous administration of 120 mL of iodinated contrast material was done with a rate of 5 mL/sec. Patients were scanned from the aortic arch to the aortic bifurcation (superior to inferior). Imaging analysis was performed on a PACS system.

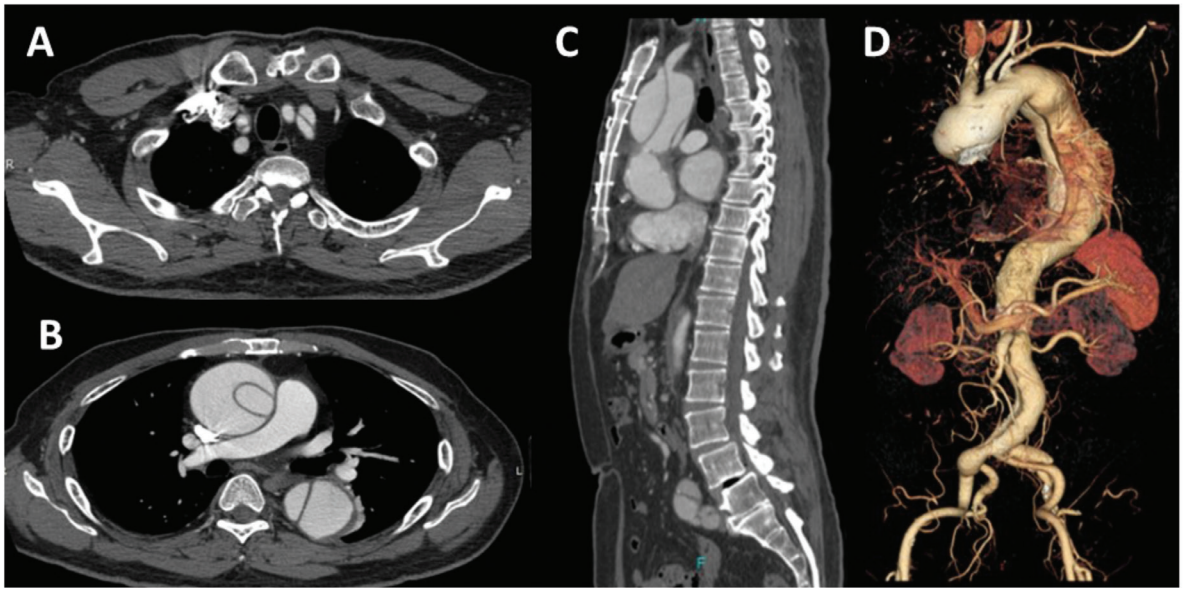

Figure 4. *Aortic dissection Stanford classification type A.*
A and B : axial plane images
C : sagittal plane image
D : volume rendering image

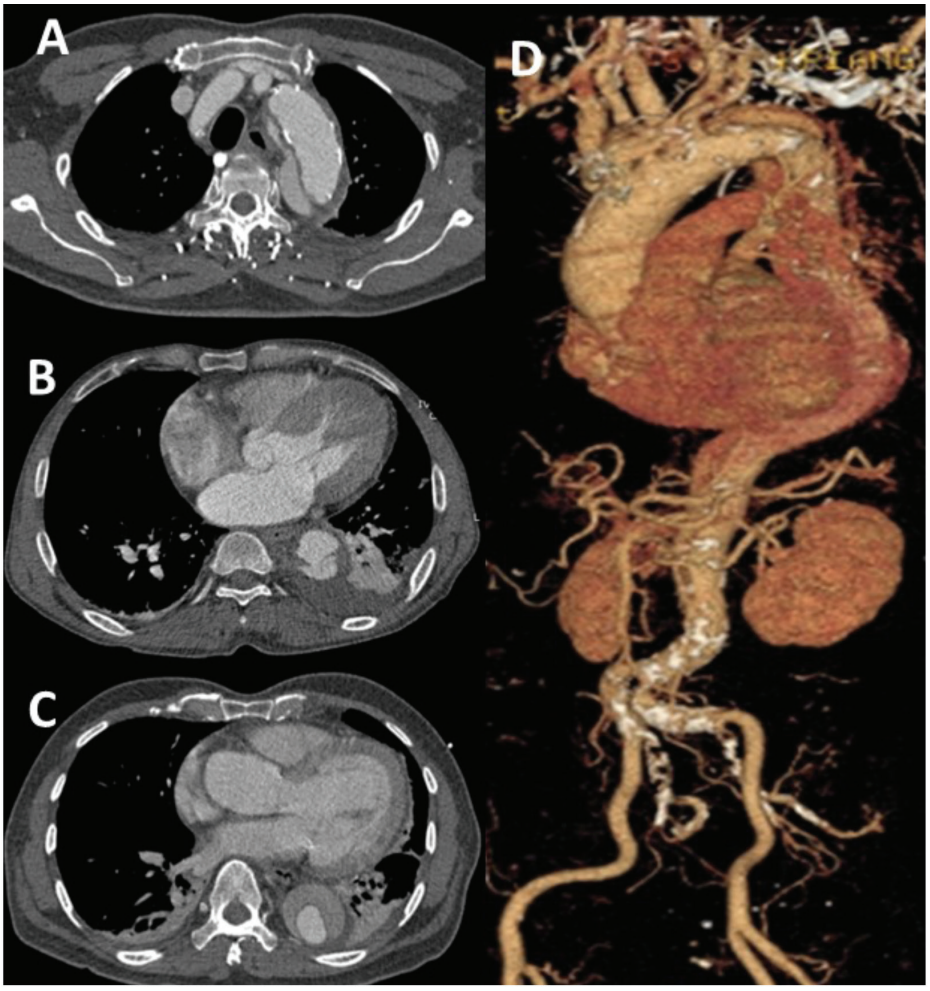

Figure 5. *Aortic dissection Stanford classification type B. A, B and C : axial plane images.*
D : volume rendering image

All images were retrospectively reviewed by three observers (two expert cardiovascular radiologist and training radiology resident) who were unawared of the acute versus chronic nature of each patient's disease. Both acute and chronic dissections were classified based on Stanford classification as type A which affected ascending aorta (Figure 4) and arch and type B which affected beyond left subclavian artery (Figure 5).

Each scan was evaluated for the presence of the following imaging features on one or more images in each lumen of the aorta: the beak sign, outer wall calcification, intraluminal thrombus and cobweb sign. The beak sign was defined as the presence of an acute angle between the dissection flap and the outer wall; the space formed by the acute angle could be filled with high-attenuation material (contrast-enhanced blood) or low-attenuation material (hematoma). Cobweb sign was defined as thin, linear radiolucent filling defects in the lumen that were attached to the wall at one end; the other end could be attached to the flap or the wall or have no attachment.

To determine which lumen was true and which was false, we followed the two lumens in the descending aorta proximally and distally on the CT scans. The lumen in the dissected portion of aorta that was continuous with the lumen of a non-dissected portion of aorta was deemed to be the true lumen. If a lumen ended in a blind sac, it was deemed to be the false lumen. These standards were used to define the lumens in all patients. On the postoperative study, the lumen within the remaining native dissected portion of aorta that was continuous with the aortic interposition graft was deemed to represent the true lumen.

In this study the origination of artery branches of aorta-brachiocephalic artery, left common carotid artery, left subclavian artery, celiac trunk, superior mesenteric artery, and bilateral renal arteries-which were originated from true or false limen were also recorded. Moreover complication of aortic dissection such as rupture, hemothorax, hemopericardium, mediastinum hematoma and distal organ infarction were also revealed in our study.

Statistic

Descriptive statistics were calculated. Statistical analyses were performed by using software (SPSS for Window).

Results

One hundred and nineteen patients were included in the study. There were 38 type A dissections and 81 type B dissections, 66 of acute dissections and 53 of chronic dissections. The mean age patients with acute and chronic dissection were 53 and 58 years, respectively. These all data were shown in Table 1.

Underlying diseases of patients who had acute or chronic aortic dissections were denoted in Table 2. Systemic hypertension was the most common underlying disease of acute and chronic aortic dissection cases.

The incidental finding of chronic aortic dissection type A was found in 1 patient who had squamous cell cancer of right parotic gland.

CT findings of true and false lumens in acute and chronic cases are delineated in Table 3 and Figure 6. All true lumens showed outer wall calcifications (Figure 7). Most true lumens were smaller than false lumens (Figure 8). Findings of false lumens included beak sign, cobweb sign, larger lumen and intraluminal thrombus sign, However, larger lumens could be seen in true lumens (Figure 9) as well smaller lumens and outer wall calcifications could be seen in false lumens. One patient (2%) with acute aortic dissection type B showed a focal intraluminal thrombus in the true lumen (Figure 10). The beak sign and cobwebs were not seen in the true lumen (Figure 11 and 12).

Table1. *The patients were included in this study.*

	Acute		Chronic		Total
	A	B	A	B	
Patients(number)	25(21%)	41(34%)	13(11%)	40(34%)	119

Table2. *The table shows underlying disease of both acute and chronic aortic dissection patients. The incidental finding of acute aortic dissection type B was found in 2 patients who were known case of rectal cancer and idiopathic thrombocytopenia, respectively*

Underlying diseases	Acute		Chronic	
	A (n= 25 scans)	B (n= 41 scans)	A (n= 13 scans)	B (n= 40 scans)
Hypertension	5(20%)	17(41%)	7(53%)	23(57.5%)
Diabetic mellitus	0(0%)	4(10%)	3(23%)	3(7.5)
Dyslipidemia	3(12%)	6(14%)	0(0%)	6(15%)
Chronic kidney disease	2(8%)	0(0%)	0(0%)	1(2.5%)
Coronary arterial disease	0(0%)	3(7%)	0(0%)	5(12.5%)
Atrial fibrillation	1(4%)	3(7%)	0(0%)	3(7.5)
Marfan syndrome	2(8%)	2(4%)	0(0%)	1(2.5%)
Aortic aneurysm(ascending aorta)	1(4%)	0(0%)	0(0%)	0(0%)
Systemic lupus erythematosus	2(8%)	0(0%)	0(0%)	0(0%)
Larsen syndrome	0(0%)	1(2%)	0(0%)	0(0%)
Transient ischemic attack	0(0%)	1(2%)	0(0%)	0(0%)
Trauma	0(0%)	1(2%)	0(0%)	0(0%)
Iatrogenic (TACE)	0(0%)	2(4%)	0(0%)	0(0%)
Incidental	0(0%)	2(4%)[1]	1(7%)[2]	1(2.5%)

Table3. *CT findings for the true and false lumens in acute and chronic cases*

Sign		Acute		Chronic	
		A (n= 25 scans)	B (n= 41 scans)	A (n= 13 scans)	B (n= 40 scans)
True lumen	Smaller lumen	25(100%)	39(95%)	12(92%)	33(82.5%)
	Larger lumen	0(0%)	3(7%)	1(7%)	7(17.5%)
	Beak sign	0(0%)	0(0%)	0(0%)	0(0%)
	Cobweb sign	0(0%)	0(0%)	0(0%)	0(0%)
	Intraluminal thrombus	0(0%)	1(2%)	0(0%)	0(0%)
	Outer wall calcification	16(64%)	31(75%)	9(69%)	32(80%)
False lumen	Smaller lumen	0(0%)	3(7%)	1(7%)	7(17.5%)
	Larger lumen	25(100%)	38(92%)	13(100%)	33(82.5%)
	Beak sign	22(88%)	32(78%)	11(84%)	19(47.5%)
	Cobweb sign	2(8%)	8(19%)	7(53%)	7(17.5%)
	Intraluminal thrombus	7(28%)	27(65%)	13(100%)	36(90%)
	Outer wall calcification	0(0%)	2(5%)	0(0%)	6(15%)

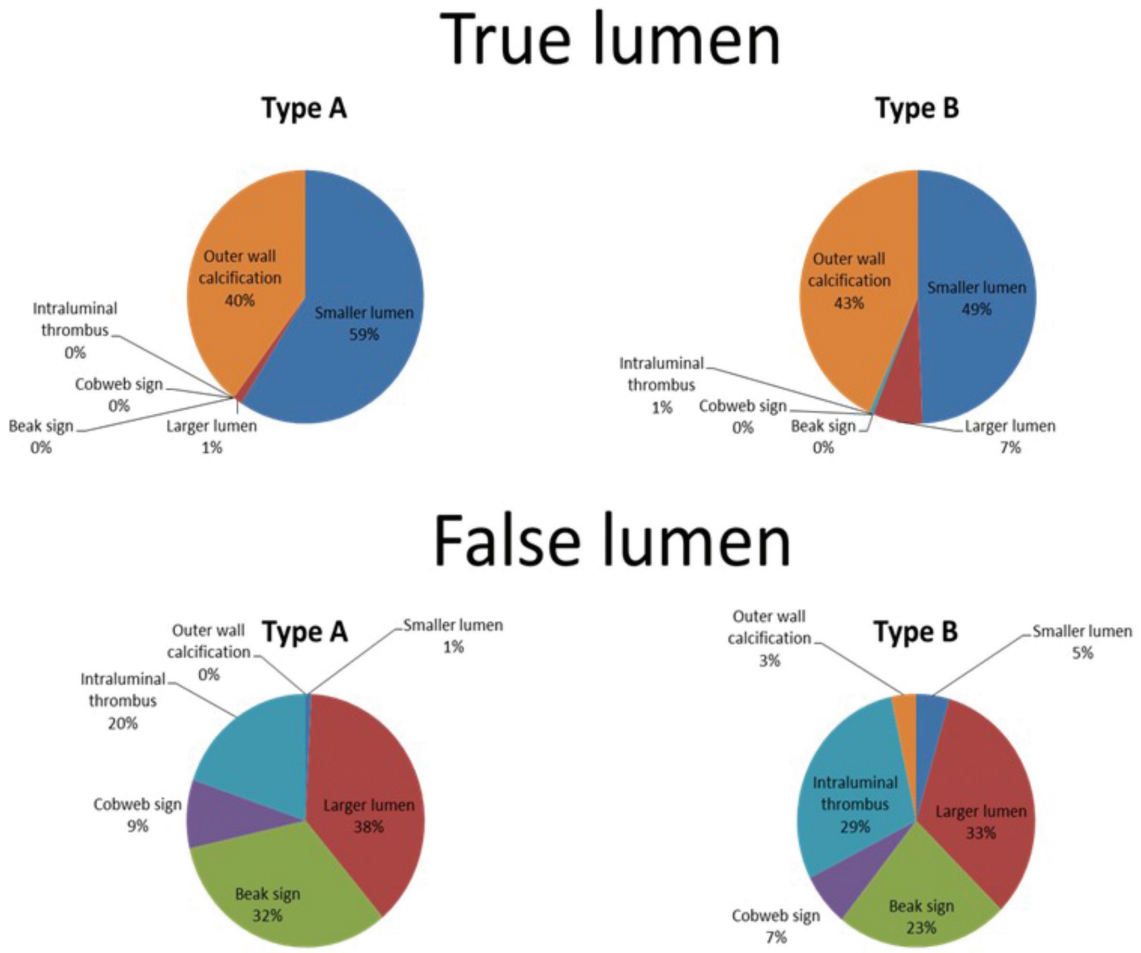

Figure 6. *Findings of true lumen and false lumen in aortic dissection Stanford classification type A and B.*

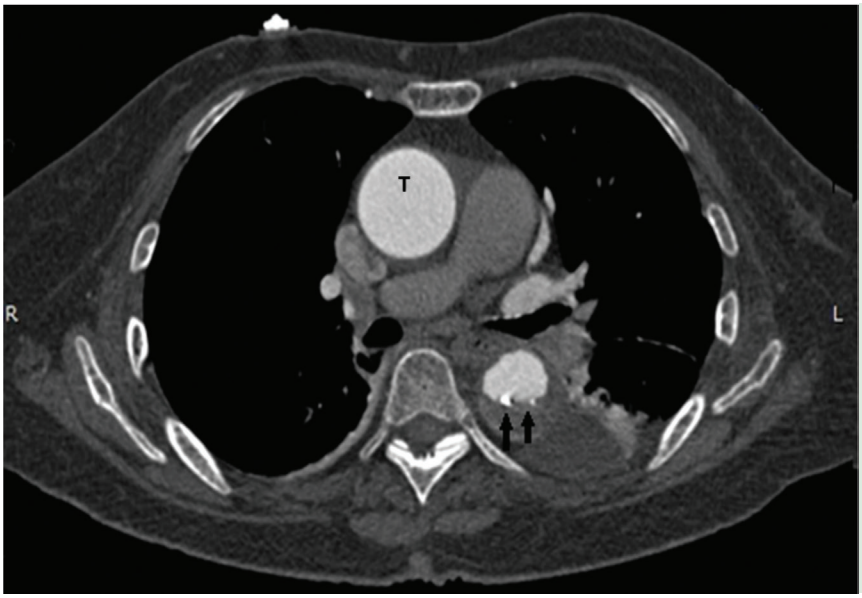

Figure 7. *A 75-year-old man with history of hypertension presented with chronic aortic dissection type B. This picture demonstrates contrast early filling in true lumen (T) and delay in false lumen. There are calcifications at outer wall of true lumen (black arrows). The other findings are left pleural effusion and bilateral atelectasis.*

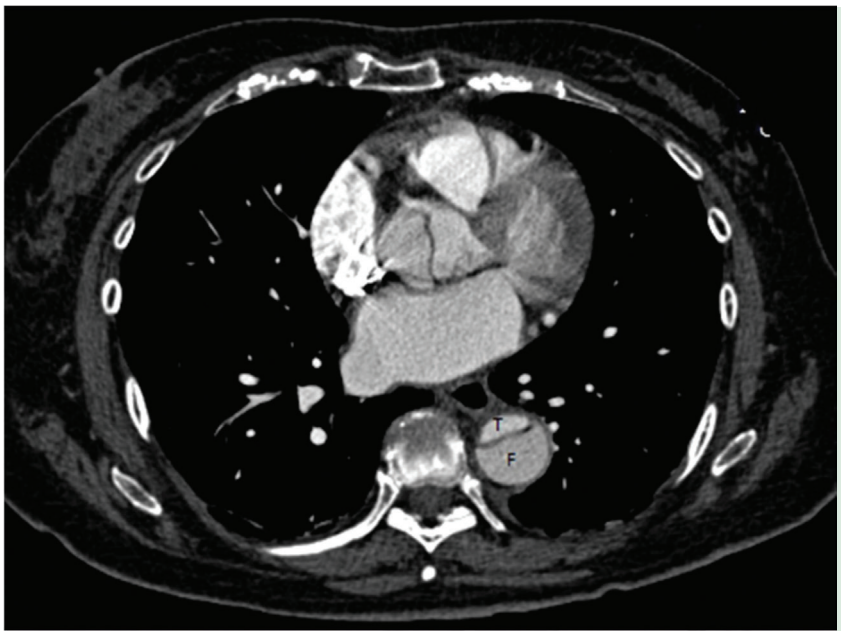

Figure 8. *A 65-year-old woman with history of hypertension, presented with acute aortic dissection type B. The true lumen (T) is smaller than false lumen (F).*

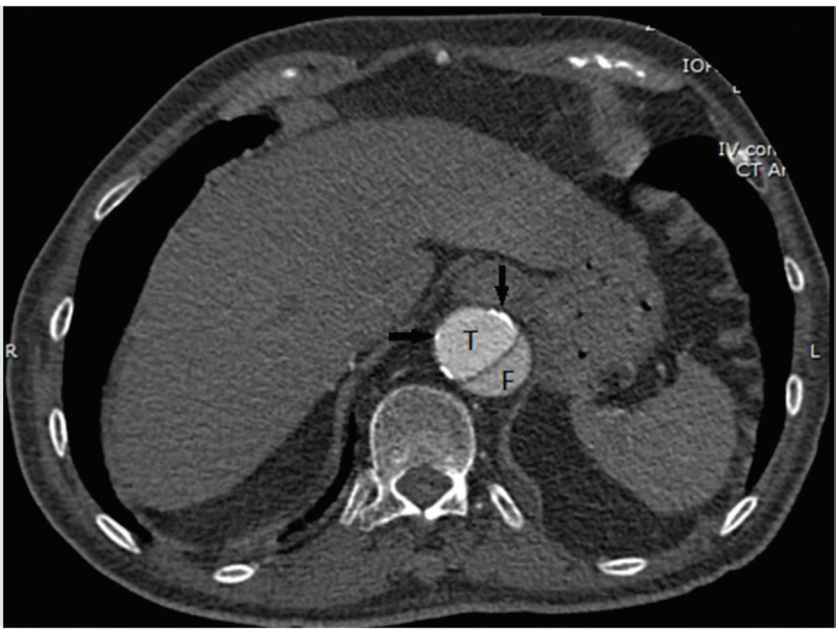

Figure 9. *A 78-year-old man with history of T2DM and hypertension, presented with chronic aortic dissection type B. This picture reveals contrast early fill in true lumen (T) but delay in false lumen (F). The true lumen (T) shows calcified outer wall (black arrows and larger lumen than false one.*

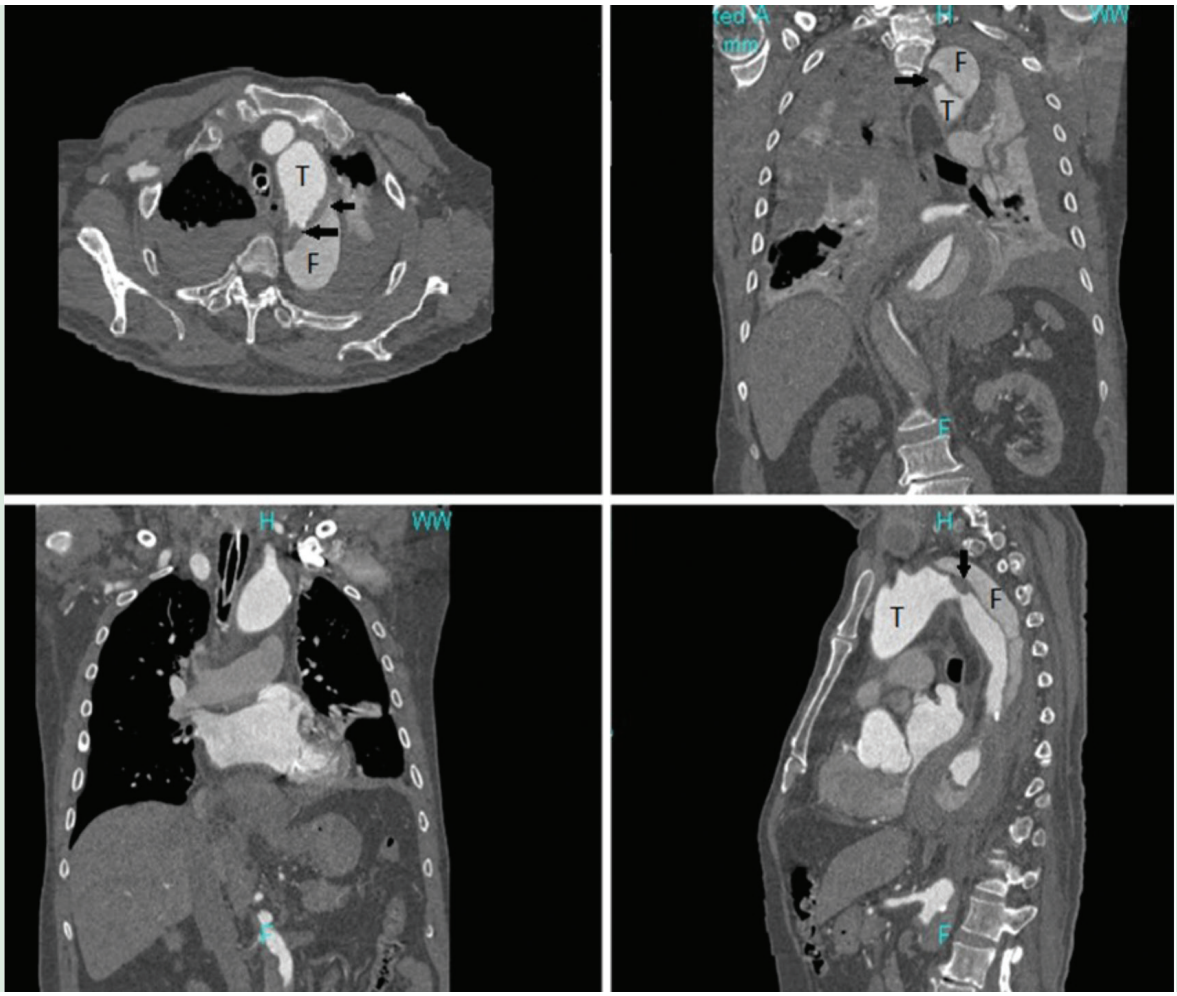

Figure 10. *A 70 year-old man with history of atrial fibrillation and non-ST elevation myocardial infarction, presented with acute type B aortic dissection. MPR pictures of CTA thoracic aorta. The true lumen (T) is continued from ascending aorta and aortic arch and more contrast fill than the false lumen (F). These pictures show the focal intraluminal thrombus in the true lumen (black arrows) just distal to aortic arch level which is not typical of true lumen.*

Figure 11. *A 64-year-old woman without any underlying disease presented with acute onset of chest pain. The diagnosis was acute aortic dissection type A. The CTA abdominal aorta demonstrates acute angle between the dissection flap and the outer wall (black arrows) that is beak sign. (T=true lumen, F=false lumen).*

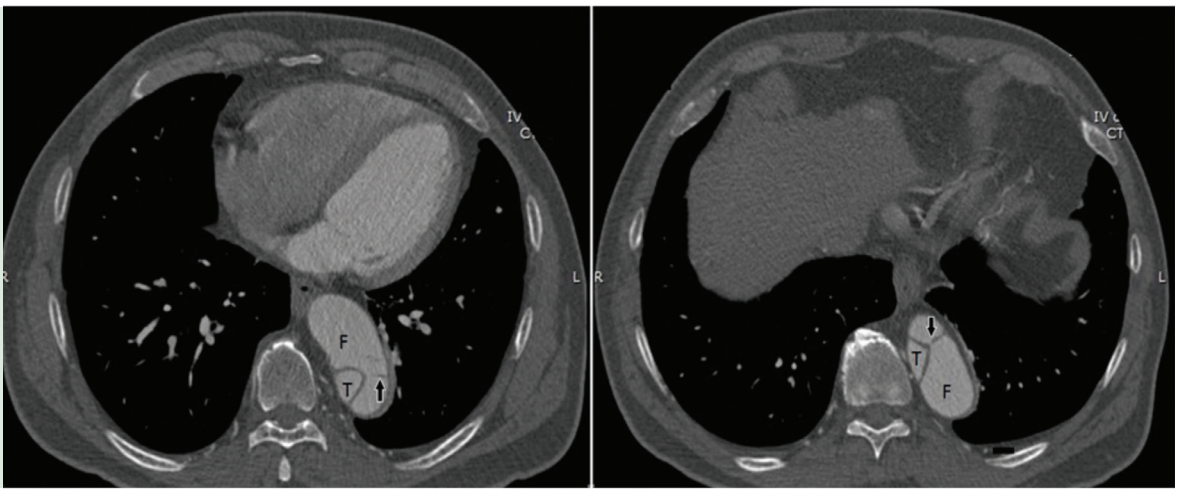

Figure 12. *A 60-year-old man with history of squamous cell carcinoma at right parotid gland, presented with incidental finding of aortic dissection. Both pictures are follow-up films after conservative treatment. These finding shows thin linear filling defect (black arrows) crossing in false lumen (F) which is Cobweb sign*

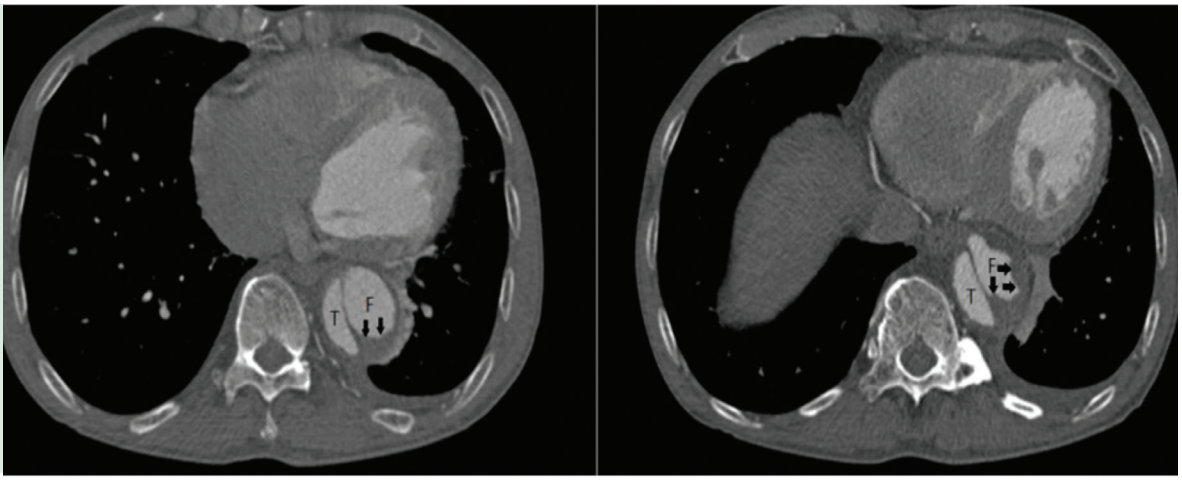

Figure 13. *A 75-year-old man with no underlying disease, presented with acute aortic dissection type B. These pictures demonstrate eccentric filling defect (black arrows) in the false lumen (F) which's compatible with intraluminal thrombus. The other finding is minimal left pleural effusion.*

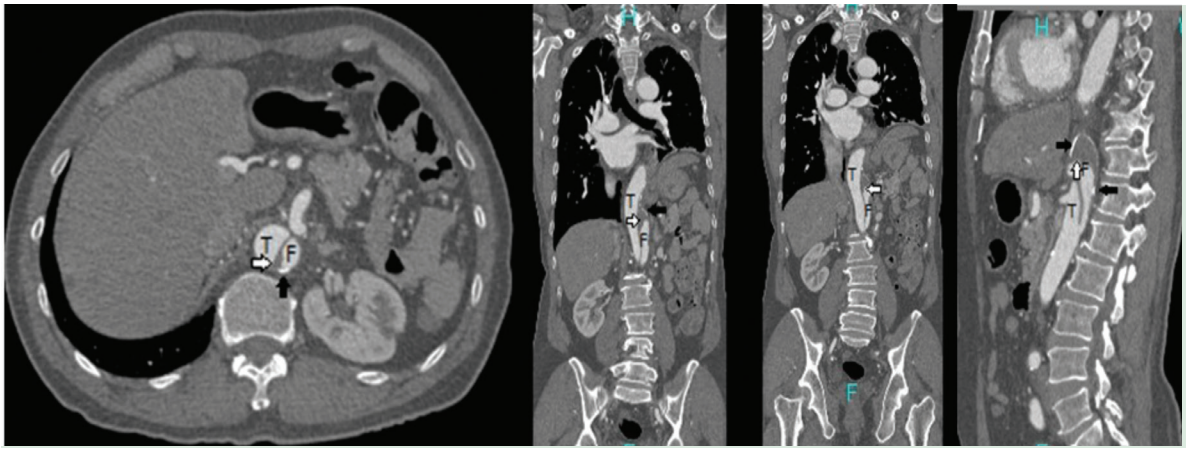

Figure 14. *A 69 year-old man with history of hepatocellular carcinoma, presented with immediate iatrogenic acute type B aortic dissection post trans-arterial chemoembolization. MPR pictures of CTA thoracic aorta show focal intraluminal thrombus (white arrows) in the false lumen (F), calcification at outer wall (black arrows) of false lumen and smaller size of false lumen than true one (T). These findings are not typical of false lumen.*

It is interesting to note that the larger true lumens were seen in chronic aortic dissection more than acute aortic dissection were type B more than type A. The smaller false lumens with outer wall calcifications were noticed in chronic aortic dissection more than acute aortic dissection which also were type B more than type A(Figure 13 and 14). Origins of aortic branches varied in both acute and chronic aortic dissections especially ones which originated from descending aorta (Table 4). The complications of aortic dissection are death, rupture dissection, hemopericardium, hemothorax, hemomediastinum and distal organ infarction (renal infarction, bowel infarction, limb infarction or stroke) [6]. The other findings that our study found were pericardial effusion, pleural effusion and heart failure.The findings as describe above are delineated in figures15-22 and table 5.

Table 4. *This table shows the origination of major aortic branches which from true and false lumen in acute and chronic aortic dissection patient.*

Branch	Acute				Chronic			
	A (n= 25scans)		B (n= 41 scans)		A (n= 13 scans)		B (n= 40 scans)	
	TRUE	FALSE	TRUE	FALSE	TRUE	FALSE	TRUE	FALSE
Brachiocephalic a.	18(72%)	2(8%)	1(2%)	0(0%)	9(69%)	1(7%)	2(5%)	0(0%)
Left common carotid a.	20(80%)	1(4%)	1(2%)	0(0%)	11(84%)	0(0%)	2(5%)	0(0%)
Left subclavian a.	21(84%)	0(0%)	1(2%)	0(0%)	11(84%)	0(0%)	2(5%)	0(0%)
Celiac trunk	14(56%)	10(40%)	34(83%)	9(22%)	12(92%)	1(7%)	30(75%)	9(22.5%)
Superior mesenteric a.	20(80%)	1(4%)	36(88%)	3(7%)	13(100%)	0(0%)	33(82.5%)	5(12.5%)
Left renal a.	12(48%)	8(32%)	29(70%)	12(29%)	5(38%)	7(54%)	28(70%)	10(25%)
Right renal a.	22(88%)	2(8%)	31(75%)	9(22%)	13(100%)	0(0%)	35(87.5%)	4(10%)

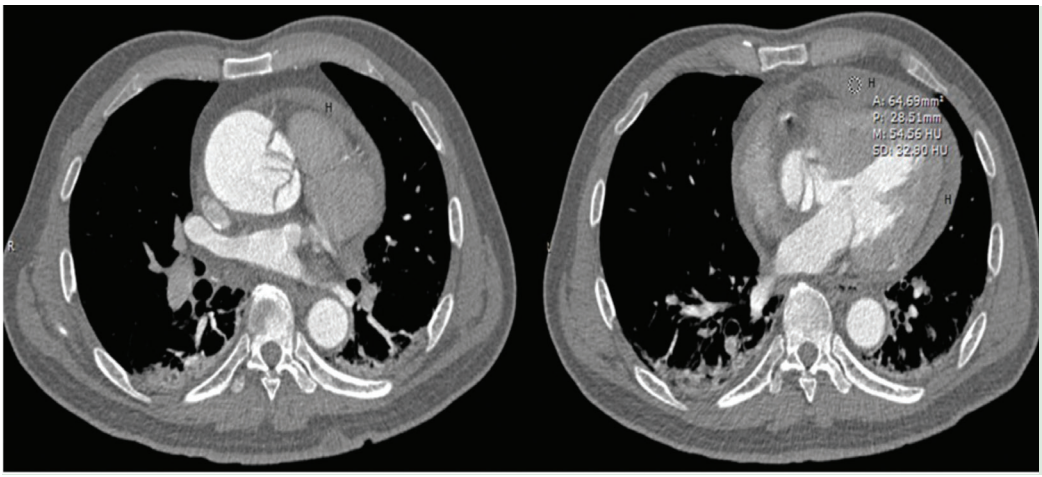

Figure 15. *A 56-year-old man with no underlying disease, presented with acute aortic dissection type A. These pictures show high density pericardial fluid (HU about 54), which suggest hemopericardium (H).*

Table 5. *Aortic dissection complications and other findings in this study.*

Complications and the other findings	Acute		Chronic	
	A(n= 25 scans)	B(n= 41 scans)	A(n= 13 scans)	B(n= 40 scans)
Death	7(28%)	3(7%)	0(0%)	0(0%)
Rupture aortic dissection	2(8%)	0(0%)	0(0%)	0(0%)
Hemopericardium	8(32%)	1(2%)	0(0%)	0(0%)
Hemothorax	2(8%)	3(7%)	0(0%)	0(0%)
Mediastinal hematoma	2(8%)	2(5%)	0(0%)	0(0%)
Renal infarction	1(4%)	6(14%)	1(7%)	2(5%)
Bowel ischemia	1(4%)	0(0%)	0(0%)	0(0%)
Pericardial effusion	2(8%)	0(0%)	0(0%)	0(0%)
Pleural effusion	8(32%)	18(44%)	0(0%)	3(7.5%)
Acites	2(8%)	1(2%)	1(7%)	0(0%)
Heart failure	0(0%)	1(2%)	0(0%)	1(2.5%)

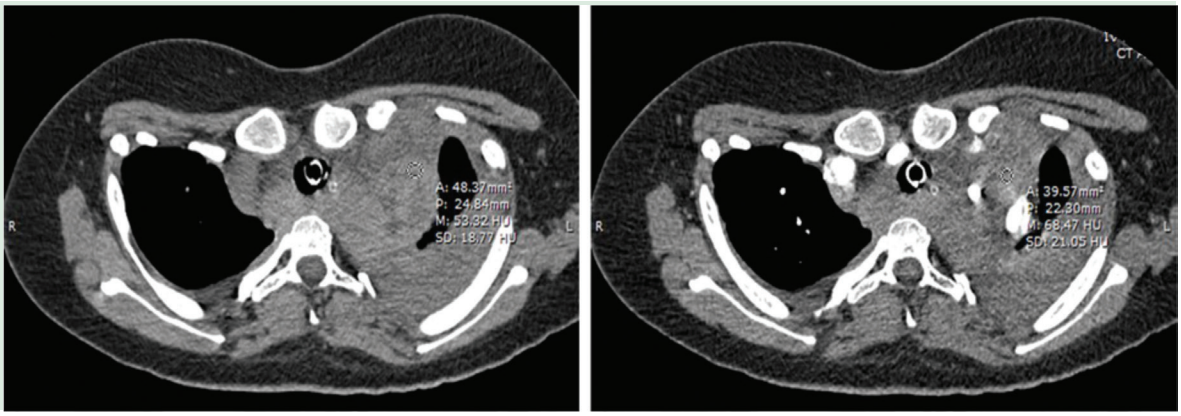

Figure 16. *A 19-year-old woman, known case of Larsen syndrome, presented with acute aortic dissection type B. These pictures show high density lesion (HU about 68) at the mediastinum, which suggest hemomediastinum.*

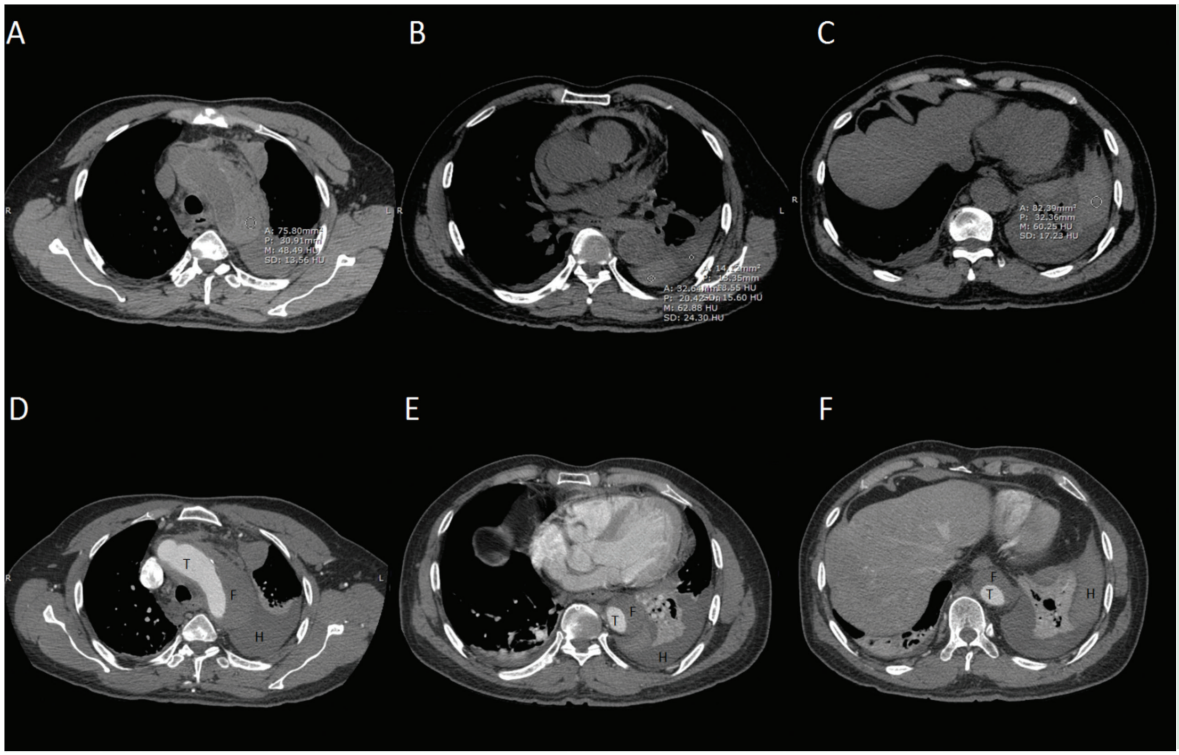

Figure 17. *A 63-year-old man with history of hypertension, presented with acute chest pain. These pictures show acute aortic dissection. There is high density intramural thrombus (HU about 48) and high density left pleural fluid (HU about 60). This case was diagnosis of acute aortic dissection type A with of rupture dissection and left hemothorax.*

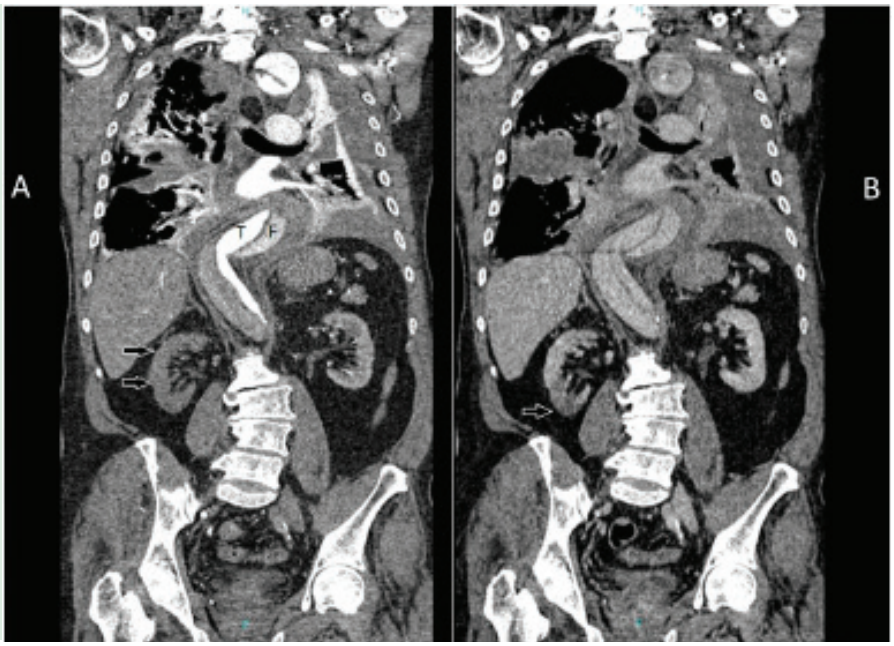

Figure 18. *A 79-year-old woman with HT and DM, presented with acute aortic dissection type B. In the arterial phase of coronal plane of whole aorta (A) shows relative delay enhancement of right kidney as compare to the left. In delay phase of coronal plane of whole aorta (B) demonstrates contrast enhancing defect at lower pole of right kidney (black arrow) which suggestive of right lower pole kidney infarction. (T=true lumen, F=false lumen).*

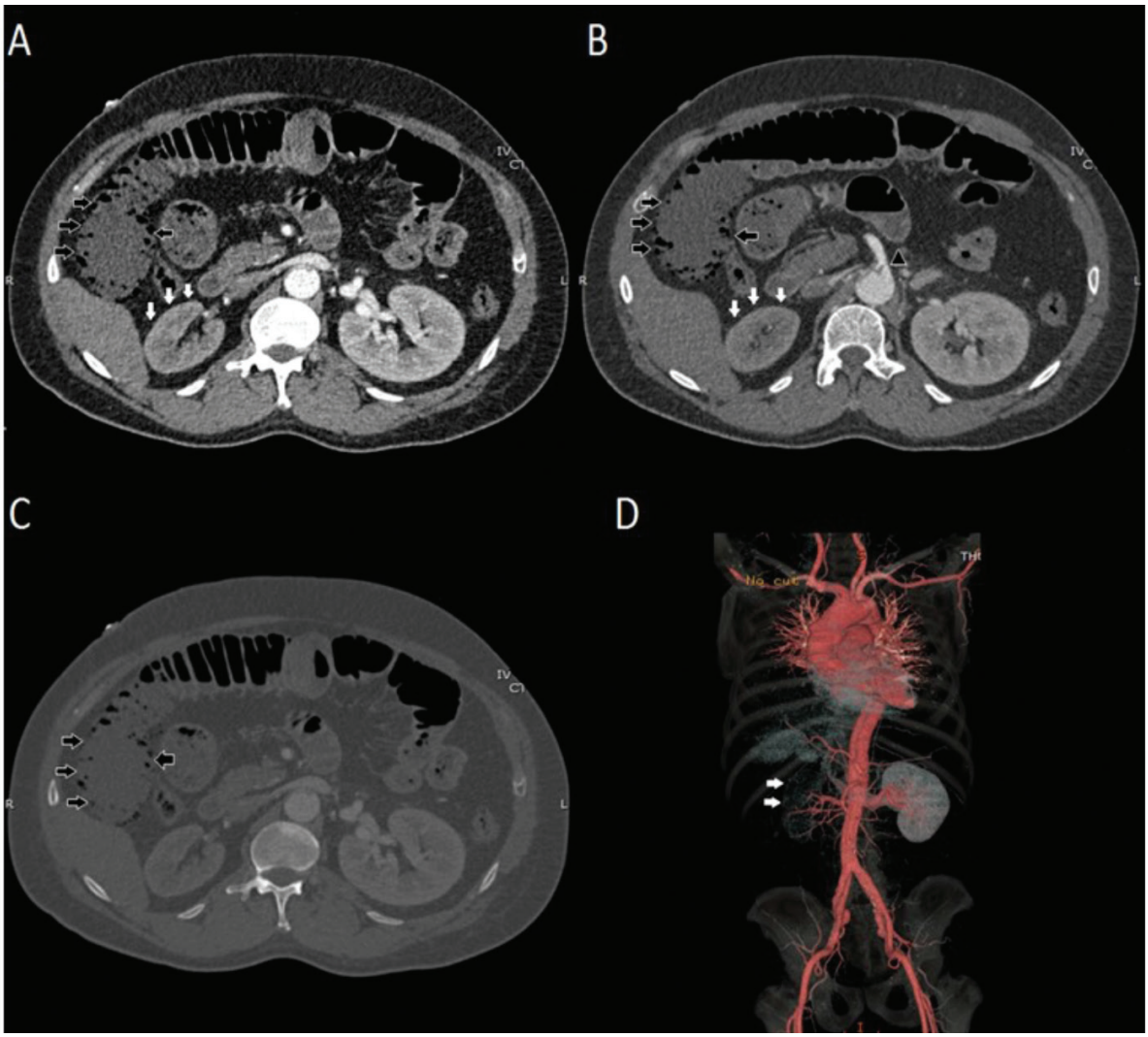

Figure 19. *A 45-year-old man, no underlying disease, presented with acute aortic dissection type A. A : arterial phase. B : portovenous phase, C : bone window. D : volume rendering of CTA whole aorta. Right renal artery, superior mesenteric artery and celiac trunk origins are compromised (arrow head). There is short segment small bowel infarction which shows pneumatosis intestinalis (black arrows). There is also renal infarction (white arrows) which show relatively poor enhancement than the left one.*

Most complications were depicted in acute aortic dissection. There were ten patients who died of aortic dissection. Death was found only in acute group mostly type A. This study showed that intra-thoracic complications of aortic dissection-ruptured aortic dissection, hemopericardium, hemothorax and mediastinal hematoma – were more frequently seen in acute than chronic aortic dissection and type A was found more than type B in acute aortic dissection (Figure 15,16 and 17).

One case had underlying disease of extremely rare genetic disease which is often classified as a type of osteochondrodysplasia. This case had previous ascending aortic aneurysm then presented with acute chest pain. The CTA revealed acute aortic dissection type B with mediastinum hematoma and left pleural effusion (Figure 16 and 20).

Renal infarctions were most frequently seen in type B acute aortic dissection (Figure 18). There was a one type A acute aortic dissection that compromised celiac trunk, superior mesenteric artery and right renal artery resulting in short segment of small bowel infarction and right renal infarction (Figure 19).

The other findings included pleural effusion, pericardial effusion, ascites, and heart failure. The most common among these was pleural effusion. Pericardial effusions were found in two cases which were type A acute aortic dissection.

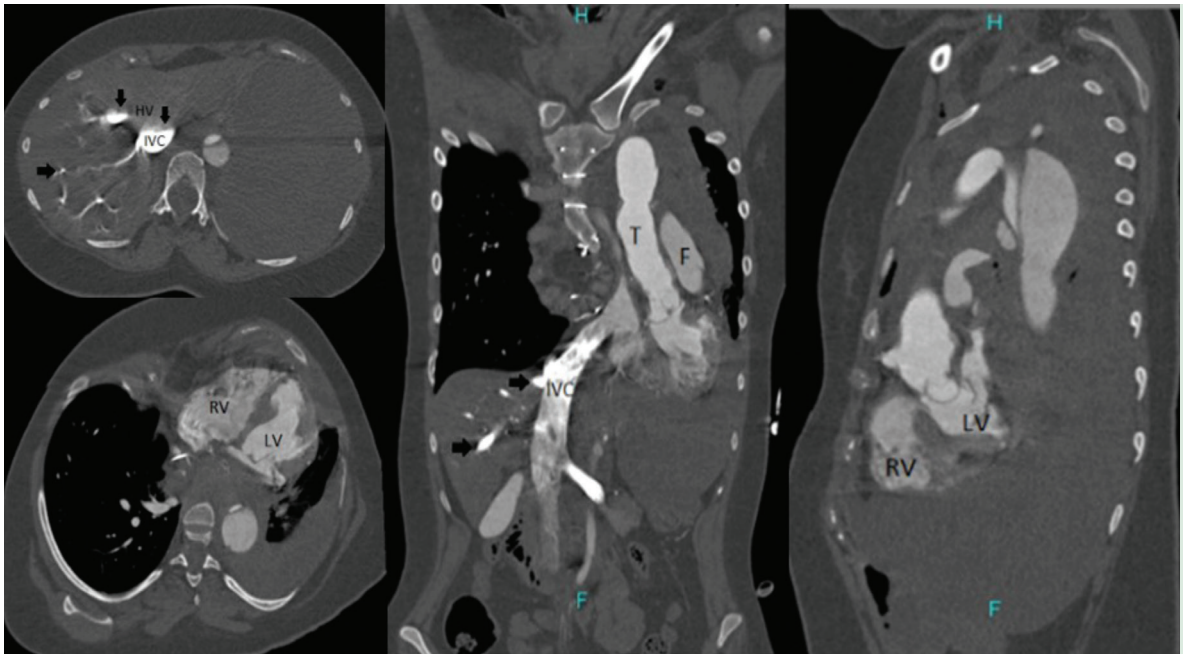

Figure 20. *A 19-year-old woman with Larsen syndrome, presented with acute aortic dissection type B. There is contrast reflux to all hepatic veins (HV) in arterial phase and right ventricular (RV) dilatation which suspect of right side heart failure. (IVC=inferior vena cava, LV=left ventricle, T=true lumen, F=false lumen)*

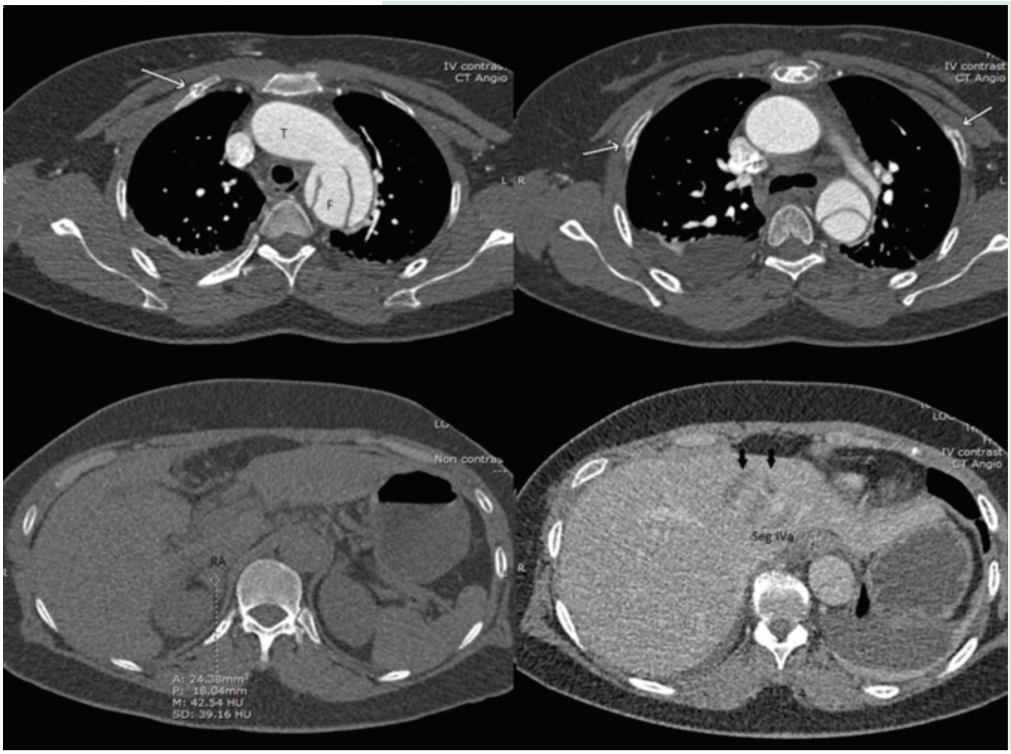

Figure 21. *A 55-year-old woman with no underlying disease, presented with truck injury. There is acute aortic dissection type B. There are multiple associated traumatic injury; multiple bilateral ribs fracture (white arrows), right adrenal gland hematoma (RA), and liver laceration at hepatic segment 4a (black arrows).*

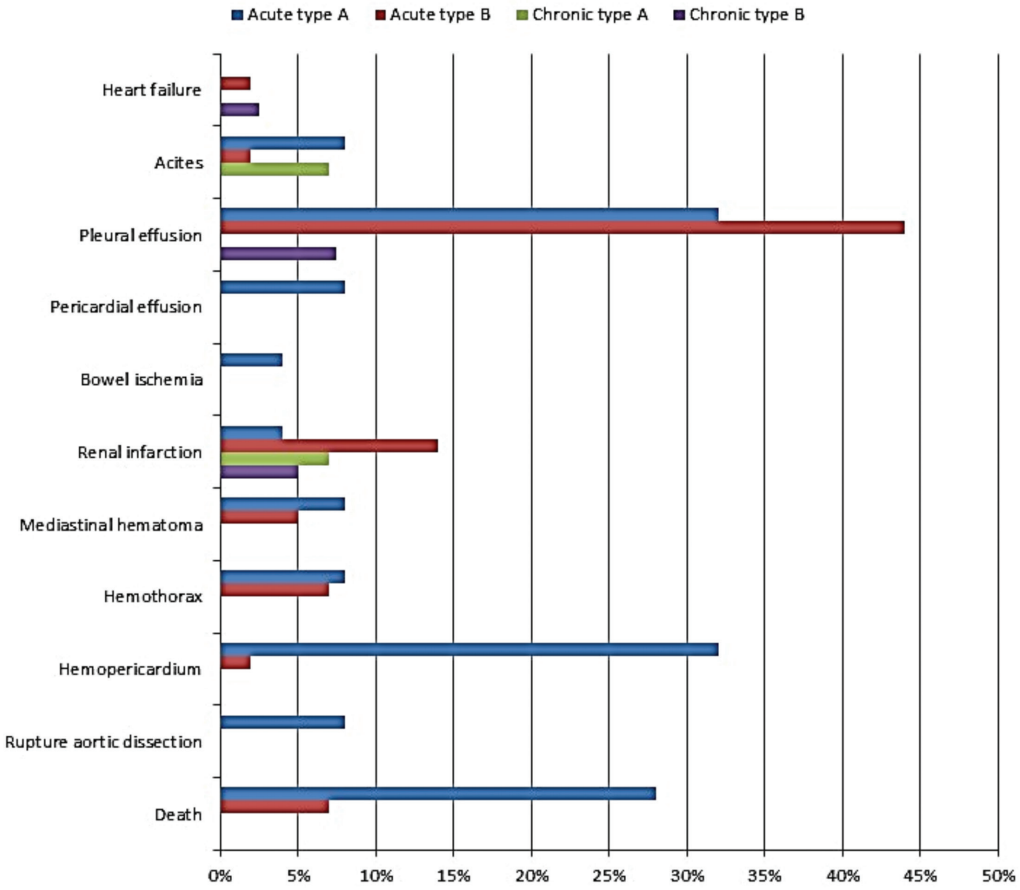

Figure 22. *Aortic dissection complications and other findings in our study cases*

Discussion

Aortic dissection is the result of aortic intima tear and separation of the aortic intima and adventitia which is caused by circulating blood access into and splitting the media of the aortic wall. The blood-filled space within the medial layer becomes the false lumen. True lumens are always determined by continuity with an un-dissected portion of aorta. There are many risk factors or associated diseased of aortic dissection. Like previous studies, the most common associated disease of aortic dissection in our study was hypertension

The aortic dissection can be classified into acute or chronic types by using history of onset. If onset is less than 2 weeks, it is classified as acute aortic dissection, while chronic type which has onset more than 2 weeks.(8-10) The aortic dissection is also classified by using the location of disease in Stanford classification to identify in type A and B. Type A involves the ascending aorta and type B involves descending aorta distal to left subclavian artery. The chronic aortic dissection was defined as persisting dissection more than 2 weeks after the acute event, as mentioned by Mark A LePage et. al. [1]

There are many important arterial branches from aorta. In this condition, aortic branches can be originated from true or false lumen. The dissection may also compromise aortic branches that impairs blood flow to distal organs.

In some severe cases, the dissecting blood can disrupt the aortic wall causing rupture. Extravasating blood can spill into pericardium, mediastinum or pleural cavity.

As described above, the aortic dissection, especially acute, is the serious emergency cardiovascular disease with high mortality rate. Diagnosis of this condition is very crucial. The initial benefit investigation is computed tomography angiography (CTA) of whole aorta, which is the gold standard now.

The CTA findings of aortic dissection about true lumen, false lumen and complications of aortic dissection have been described in previous studies. The suggestive findings of true lumen are smaller in size and outer wall calcifications. The suggestive findings of false lumen are larger in size, beak sign, cobweb sign and intraluminal thrombi.

Similarly, our study showed that majority of true lumens findings were smaller size and outer wall calcifications and the majority of false lumen findings were beak sign, cobweb sign, larger size and intraluminal thrombi. The study from Mark A LePage et al. described that outer wall calcification was ever present in the false lumens on scans of acute dissections [1] but in our study showed two cases of acute aortic dissection with calcified outer walls of false lumens.

One had hepatocellular carcinoma with immediate iatrogenic aortic dissection after trans-arterial chemo-embolization and the other one had hypertension who presented with type B acute aortic dissection. Our study also demonstrated that typical findings of both true and false lumens. The larger lumens especially in chronic type) and focal intraluminal thrombus (found one case in acute type) can be seen in true lumens. The smaller lumens and outer wall calcifications can be seen in the false lumens especially in chronic type aortic dissection. However, like other studies, the beak and cobweb signs were not seen in the true lumens.

Identification of the luminal origins of aortic branches in patients who may undergo surgical repair or percutaneous treatment with endovascular grafts is important to avoid some complications caused by occlusion of origin arterial branches such as renal or bowel infarction. The frequency of major aortic branches originating from true or false lumens. In our study, the complication findings were commonly found in acute aortic dissections.

The complications of aortic dissection were death, ruptured dissection, dissection, hemopericardium, hemothorax, hemomediastinum and distal organ infarction (renal infarction, bowel infarction, limb infarction or stroke) [6]. The other findings that our study found were pericardial effusion, pleural effusion and heart failure [6]

The most severe complication was death mostly found in acute Stanford type A. Nobody died in in chronic group. Intra-thoracic complications were depicted in depicted in acute dissection more than chronic dissection and type A more than type B. Pleural effusion was the most common other findings. Pericardial effusions were only seen in type A acute aortic dissection. Renal infarction was the most common intra-abdominal complication and was exclusively seen in acute type B aortic dissection.

CTA is the most beneficial study for evaluation of all type of aortic dissection**.** It gives the information for diagnosis, classification, plan of treatment and follow up.

In summary, typical findings helped identify true and false lumens, origination of major aortic branches, and complications.

This study also demonstrated atypical findings of both true and false lumens that give more information to classify the aortic dissection. The frequency of major aortic branch origins varied that could not be used to predict the type.

The complication findings help the radiologist to concern in each dissecting type. Further studies with newer radiology technology may show different or new information.

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