Coronary Artery Involvement in Type A Aortic Dissection: Fate of the Coronaries

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September 4, 2022

Abstract

Background: Type A aortic dissection (TAAD) involves a tear in the intimal layer of the thoracic aorta proximal to the left subclavian artery, and hence, carries a high risk of mortality and morbidity and requires urgent intervention. This dissection can extend into the main coronary arteries. Coronary artery involvement in TAAD can either be due to retrograde extension of the dissection flap into the coronaries or compression and/or blockage of these vessels by the dissection flap, possibly causing myocardial ischaemia. Due to the emergent nature of TAAD, coronary involvement is often missed during diagnosis, thereby delaying the required intervention. Aims: The main scope of this review is to summarise the literature on the incidence, mechanism, diagnosis, and treatment of coronary artery involvement in TAAD. Methods: A comprehensive literature search was performed using multiple electronic databases, including PubMed, Ovid, Scopus and Embase, to identify and extract relevant studies. Results: Incidence of coronary artery involvement in TAAD was seldom reported in the literature, however, some studies have described patients diagnosed either preoperatively, intraoperatively following aortic clamping, or even during autopsy. Among the few studies that reported on this matter, the treatment choice for coronary involvement in TAAD was varied, with the majority revascularizing the coronary arteries using coronary artery bypass grafting or direct local repair of the vessels. It is well-established that coronary artery involvement in TAAD adds to the already high mortality and morbidity associated with this disease. Lastly, the right main coronary artery was often more implicated than the left. Conclusion: This review reiterates the significance of an accurate diagnosis and timely and effective interventions to improve prognosis. Finally, further large cohort studies and longer trials are needed to reach a definitive consensus on the best approach for coronary involvement in TAAD.

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COI and funding: None

Ethical approval: No ethical approval required for this review.

Authors' contributions: Authors FK, MJ, MT and SZCPT were involved in literature review design, literature search, and manuscript writing. Authors IMW, IM, BV and MB were involved in manuscript revision.

Data availability: The data that support the findings of this study are publicly available in electronic databases such as PubMed, Ovid, Scopus and Embase.

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Conclusion : This review reiterates the significance of an accurate diagnosis and timely and effective interventions to improve prognosis. Finally, further large cohort studies and longer trials are needed to reach a definitive consensus on the best approach for coronary involvement in TAAD.

Keywords: Aortic Dissection, Type A Aortic Dissection (TAAD), Aortic Surgery, Coronary Artery, CAD, CABG.

Introduction

Aortic dissection (AD) is a life-threatening medical condition that annually affects 3-4 people per 100,000 [1][2]. The underlying pathology is defined by a tear in the aortic intima producing medial layer separation, creating a space known as the false lumen (FL) into which bloods flows. If this entry tear originates proximal to the left subclavian artery (LSA) (Zone 0-2), the event is classified as Type A AD (TAAD); otherwise, the dissection is Type B (TBAD) with the originating entry tear located distal to the LSA and without arch involvement [1][3]. AD can be further classified based on timeframe into acute (<15 days since symptom onset), subacute (15-90 days since symptom onset), and chronic (>90 days since symptom onset) [1][3]. Acute TAAD has the highest mortality, which can reach 50% within the first 48 hours without prompt intervention [4].

Treatment for acute TAAD involves open surgical repair/replacement of the affected segment, with or without total arch replacement (TAR) using frozen elephant trunk (FET), if the aortic arch is involved [5]. The extent of dissection flap in TAAD can sometimes involve the main coronaries, either by causing coronary artery dissection or by blocking the coronary ostia, causing myocardial ischaemia. Given the acute setting of TAAD, coronary involvement is often missed [6].

This review will highlight the mechanisms behind TAAD with concomitant coronary involvement as well as the diagnostic methods and corrective approaches. A comprehensive literature search was conducted using multiple electronic databases including PubMed, Ovid, Scopus and Embase to identify and extract all the evidence in the literature on coronary artery involvement in TAAD.

Diagnosing Type A Aortic Dissection with Concomitant Coronary Malperfusion

Despite great advances in the field of aortovascular surgery, TAAD with concomitant coronary artery involvement still poses a significant diagnostic challenge. With its rare incidence, and variable clinical presentation, coronary involvement is often difficult to diagnose and may go undetected for a significant amount of time [6]. Due to the paucity of literature on this topic, acute coronary involvement has been loosely associated with coronary dissection, malperfusion, or occlusion and resulting ischaemia. Unfortunately, many research papers have not differentiated between these specific anatomic scenarios.

It is important to note that coronary involvement in TAAD does not strictly lead to myocardial ischaemia and infarction. This is evidenced in a study by Chen et al. [7] where 6 out of 20 patients (30%) presented with aortic dissection and acute coronary involvement without evidence of myocardial ischaemia. This has also been supported by other studies in the literature, where patients who presented with the dissection extending retrogradely to the coronary ostia were assessed for myocardial ischaemia, and remarkably between 21.4% and 25% showed no such evidence [6-8]. The most common mechanism of myocardial ischaemia as a result of acute aortic dissection is the retrograde extension of the dissection into the coronary arteries [8]. In addition to this, coronary malperfusion can result from compression of the coronary arteries by the dissection flap or a haematoma [6].

The Neri classification was repeatedly cited throughout the literature to highlight the different mechanisms behind coronary malperfusion [9]. As illustrated in **Figure 1** , Neri et al. [9] described 3 main types of coronary lesions that result from aortic dissection. Type A is considered coronary ostium dissection and is limited to the ostium, while type B features a coronary false channel. On the other hand, type C coronary artery dissection involves detachment of the coronary circumference and associated intussusception. As described by the authors, ostial dissection may not always result in coronary malperfusion, unless a local flap occludes blood flow, essentially working as a 'trapdoor'. When a dehiscence in the coronary wall forms, a

coronary false channel can be created, allowing for a retrograde extension of the dissection into the coronary artery. The distal extent of this coronary artery dissection can vary amongst patients. When the coronary artery is separated from the aortic root, as seen in type C, occlusion of the blood flow can subsequently result in coronary ischaemia and myocardial damage [9].

Table 1 summarises the patient demographics and comorbidities of those that were diagnosed with TAAD and concomitant coronary involvement which we identified upon searching the literature. Additionally, it depicts the mode of diagnosis, which coronary arteries were involved, and the cause of coronary malperfusion, if reported. Reported incidence of coronary malperfusion in association with acute aortic dissection has been variable, ranging from 6.1% to 14.1% in the literature [6,7]. As shown in the table, there was a mixed gender dominance in this group of patients. However, paradoxically, patients who developed coronary malperfusion were younger (p=0.001) and were also at a lower risk of intramural haematomas (p=0.001), as demonstrated by Chen et al. [7]. As expected, a large number of patients had a history of coronary artery disease, hypertension, chronic obstructive pulmonary disease, diabetes and were known smokers. Some patients also had Marfan's syndrome, and this was found to be significantly associated with coronary involvement (p=0.008), in addition to hypertension (p=0.003). Moreover, patients who developed coronary involvement were associated with an increased aortic regurgitation rate (p=0.001), which often warranted valve replacement [7].

The mode of diagnosis of concomitant coronary malperfusion varied greatly, as evident in **Table 1** . Patients were diagnosed either preoperatively, or following aortic clamping amidst surgery, or even during autopsy. Prior to surgery, some patients had presented with new ST-segment elevation on ECG, in addition to abnormalities in the motion of the left ventricle (echocardiogram) and very high levels of serum creatine kinase. A recent systematic review and meta-analysis suggested that ECG changes indicating ischaemia were not helpful in diagnosing patients with aortic dissection [10]. Although ECG changes tend to be more consistent with coronary involvement, overall, less than around 50% of aortic dissections are discernible on ECG [11]. Some studies have also evaluated the benefit of using coronary angiography to diagnose coronary involvement prior to surgery, however, such an approach showed no effect on coronary artery bypass grafting (CABG) rates and did not improve the prognosis for these patients. Additionally, and interestingly, it was even noted that performing this investigation can cause critical delays in treatment [12].

As also seen in **Table 1** , the right coronary artery was more commonly involved in aortic dissection, hence, myocardial ischaemia is expected to impact the inferior, rather than the anteroseptal walls, more commonly [13]. These findings are concurrent with a study involving 236 aortic dissection patients, whom the right coronary artery was more commonly affected [14]. Although higher rates of myocardial ischaemia have been reported related to the right coronary artery, a significant link has been established between left coronary involvement and pre-operative cardiopulmonary arrest (p=0.004) [15]. Furthermore, the mechanisms of coronary malperfusion described earlier in this paper are all identified in the Table, with the main cause being coronary dissection due to retrograde extension of the aortic dissection, as expected.

It is also important to note that various patients presented with haemodynamic instability, often requiring cardiopulmonary resuscitation (CPR) and other interventions such as percutaneous cardiopulmonary support (PCPS) preoperatively [6]. The role of such interventions in producing a successful outcome in cases of coronary involvement must be evaluated in future studies. Moreover, many patients also presented with hemopericardium, cardiac tamponade, intramural hematoma, pleural effusion, limb ischaemia, and other complications that may have had an impact on treatment options and prognosis [7].

The Surgical Approach

When a patient presents acutely with aortic dissection symptoms, the appropriate investigations and medical management are initiated. Once the diagnosis is confirmed with imaging (echocardiogram, CT, MRI), more invasive surgical treatment is usually undertaken [16,17]. This section will describe the surgical approach for managing TAAD with concomitant coronary involvement, in addition to other treatment modalities reported in the literature.

In TAAD, surgical aortic repair is the definitive management of choice in most circumstances. The aim of this procedure is to resect the intimal tear and, depending on the location and extent of the tear, replace the aortic root, ascending aorta and/or the arch (which can be partial or total) [16]. Other factors that dictate the type of surgical intervention are branch vessel impairment, coronary artery involvement, and aortic valve involvement [17].

TAAD Repair

The majority of studies reported performing an open aortic repair through a median sternotomy, with cardiopulmonary bypass, as illustrated in Table 2. If the tear is in the ascending aorta, this portion is resected and replaced with a synthetic graft. The distal anastomosis is often done open, under deep hypothermic circulatory arrest. Then, perfusion is resumed, followed by establishment of the proximal anastomosis [6]., Eren and others described using the Bentall technique for these tears in selected cases with root enlargement or dissection [8]. If the tear is in the aortic arch, partial or complete replacement of that segment can be done [6].

Surgical management of coronary malperfusion is dependent on when coronary involvement is identified. In the study by Kawahito et al. [6], myocardial protective measures were established early during the surgery. Systemic cooling was done instantly after CPB was started. When ventricular fibrillation was identified, the ascending aorta was clamped and incised. If the coronary ostia were involved or if annuloaortic ectasia was present, the aortic root was replaced using the composite valve-graft conduit technique. For myocardial protection, cardioplegia can be delivered through the coronary sinus in a retrograde manner or antegradely through a non-dissected ostium. During systemic cooling and rewarming, myocardial protection was administered frequently, every 20 minutes. If coronary involvement is identified earlier, the cardioplegic solution may be passed through constructed CABG grafts and the coronary ostium, before aortic repair is started [6]. However, some studies, like the one by Eren et al. [8], used continuous retrograde cold blood cardioplegia uninterruptedly through the coronary sinus. [8]. If coronary involvement was evident only after aortic declamping (through impaired myocardial performance), saphenous vein grafting can be performed during the rewarming phase.

The surgical approach is a theme of controversy in the literature and current practice, even the timing of intervention has also been challenged. A delay in surgical management is thought to allow time for inflammation to abate and is thought to yield benefit in peripheral malperfusion. Despite this, in patients presenting with TAAD with coronary involvement, delaying surgical management can result in permanent myocardial ischaemia and necrosis [7].

Coronary Revascularisation

Figures 2, 3 and 4 illustrate the reconstruction technique for Type A, B and C coronary dissections as well as the underlying pathology behind each, respectively.

Regarding the choice of coronary management, Kawahito et al., and others described their preference of CABG over local coronary repair for revascularisation out of fear of operating on friable arteries and the delicacy and complexity associated with local coronary ostial repair [6]. Neri et al. [9], on the other hand, explained the various benefits of local repair for coronary revascularisation and their encouraging results with this technique. They noted the reduced risk of competitive flow from CABG grafts and discouragement of re-dissection of the coronary arteries because antegrade flow has been established. Additionally, as described in type A coronary dissection, where the coronary ostium is involved, reconstruction can be done without revascularising (by CABG) vessels that have unknown measure of impairment [9]. Despite this, it is important to keep in mind that local repair cannot take place without utilising the damaged and fragile dissected coronary arteries, and this may not be feasible in all cases [13]. A 2014 study by Xiao et al. [18] also described total arch replacement for TAAD in 33 patients, with concomitant CABG to treat 2 RCA involvements.

What is the optimal approach?

Neri et al. [9] proposed their classification system in order to facilitate the understanding of the extent of

the coronary artery dissection and its most appropriate management. For type A coronary dissections, a continuous suture to affix the interrupted arterial layers is sufficient, allowing for the ostium to be continuous with the aortic wall. If this is not possible, the authors describe excising the ostial button and reattaching this using gelatin– resorcin-formalin glue and a running suture line to connect the ostial button to the graft. Meanwhile, in type B coronary dissections, a cardiac patch, in the form of a saphenous vein or autologous pericardium, is used with a running suture to repair the dissected layers. Type C coronary dissection management involves the vessel being transected and an end-to-end, bevelled saphenous vein reconstruction being performed [9,13].

Lajevardi et al. [19] described a case of full intimo-intimal intussusception producing left coronary occlusion in a 73-year-old patient with concomitant TAAD. The circumferential tear found at aortotomy was located 3 cm from the sinotubular junction in the ascending aorta, causing left coronary artery occlusion and aortic regurgitation. This case was successfully treated with replacement of the affected ascending aorta with aortic valve resuspension [19].

Despite the Neri classification being quoted repeatedly in the literature, a 2013 study suggested a more novel denomination of coronary malperfusion for surgical management guidance [7]. Changes suggested by Chen et al. [7] include adding a fourth category, Type D, which highlights dissection into the sinuses of Valsalva or through the aortic annulus into the left ventricular outflow tract, resulting in commissural detachment and aortic regurgitation.

It can be suggested that surgical management decisions should be based on three factors. Firstly, one must consider the presence of annuloaortic ectasia, often present in patients with connective tissue disorders like Marfan's and those with a bicuspid aortic valve. In this case, a modified Bentall or valve-sparing procedure is recommended. Secondly, the presence or absence of a damaged coronary ostium, which often must be resected. And finally, the absence of an ostial tear as other recommendations based on specific coronary dissection anatomy were described by Chen et al. [7]. Nevertheless, CABG is required to revascularize the coronary arteries.

Despite guidelines by Neri and Chen, specific management of coronary involvement in TAAD remains controversial, with studies recommending using CABG under all circumstances and others suggesting using solely local repair in type A coronary dissection [13]. Pêgo-Fernandes et al. [20] reported repairing 11 dissections with Dacron grafts. However, for coronary involvement repair, the authors used saphenous vein bypass grafts in 63.6% (7/11) of patients. The remaining patients had right coronary dissection, which was re-implanted using saphenous vein grafts or an Impra^{TM} vascular graft (Becton-Dickinson, NJ, USA). For some of these cases, a Cabrol-like technique was used, employing a smaller Dacron graft to connect the aortic graft to the coronary ostia. This provides 'tension-free' method for attachment. [20].

The Percutaneous Approach

Lentini et al. [13] documented managing the dissected coronary arteries by preliminary stent implantation and passive perfusion balloon catheters. The authors emphasized that these interventions, although yielding some benefit, do not treat the underlying aortic dissection and will act only as a temporary solution to restore coronary blood flow until definitive surgical repair can take place [13]. In 2013, Imoto et al. [15] reported 75 Type A dissection patients, out of whom 7 were treated with pre-operative coronary stents, and 23 with bypass grafting. If the dissection occurred in the left main trunk, a stent was inserted during catheterisation followed by open surgical repair of the aorta. However, when the right coronary artery was involved, a stent was only placed prior to surgical repair if the dissection was diagnosed during catheterization. Balloon inflation was done early when the stents were inserted to reduce the ischaemic damage. Fourteen patients were found to have entry to ruptures sites to the root of the aorta and underwent aortic root replacement as a result. Twelve of these patients also had a direct anastomosis performed to the damaged coronary artery involved, using a synthetic graft. In addition, gelatin-resorcin-formaldehyde or fibrin glue were used to repair any local damage to the arteries, or if the ostia was also involved. Out of all techniques used to repair the coronary arteries, including stenting, biological glue, aortic root reconstruction, and even CABG, there was

no significance found in the incidence of operative mortality (p=0.192). However, incidence of low cardiac output syndrome was significantly lower where coronary stents were utilised (p=0.042) [15]. These findings by Imoto and colleagues [15] are summarised in**Table 3** .

Studies that evaluate various surgical strategies for coronary involvement and associated outcomes are very limited in the literature. Further appraisal of our current coronary repair techniques is vital. In a systematic review of 31 studies, only 1 reported the concomitant use of percutaneous coronary intervention (PCI) in addition to TAAD repair [21]. Despite this, PCI can yield significant benefit to patients who are deemed unsuitable for surgical repair, such as those with advanced age, significant comorbidities, and those with neurological deficits.

Zimpfer et al. [22] successfully treated a TAAD patient with endovascular stent-graft placement. A graft was passed through the right coronary artery and extended from the sinotubular junction to the brachiocephalic trunk. A temporary pacemaker was also used to reduce cardiac output to ensure safe and controlled placement of the stent-graft. The authors reported complete success with full exclusion of the dissection and no evidence of malperfusion in the coronary or supraaortic vessels. In addition, no associated complications were reported during the early follow-up period (30 days) [22].

Outcomes

It is well-established in the literature that the presence of coronary involvement in aortic dissection increases mortality (8.2% vs. 3.3%, p<0.019) [7]. This review found that the main cause of mortality intraoperatively was the inability to wean patients off CPB, for instance due to critical left or right ventricular malperfusion and consequent mechanical failure. Imoto et al. attributed this to pressure differences that may occur after repair between the true and false lumen at the aortic root. Pooling of blood into the false lumen may prevent a drop in the lumen pressure during diastole, which may subsequently compress the true lumen [15].

The higher mortality rate in patients with coronary involvement may also be attributed to delays in diagnosis and often even refusal of surgical treatment in the scenario of association coronary dissection treatment. Hesitation to operate may be critical, given that mortality increases by 1% with each hourly delay [23]. **Table 4** summarises the complications associated with coronary involvement concomitant with TAAD.

Conclusion

Coronary involvement in TAAD increases morbidity and mortality if undetected or untreated; thus, it is important to identify this condition early in the diagnosis of TAAD and to manage it accordingly. However, further large cohort studies and longer trials are needed to reach a definitive consensus on the best approach for coronary involvement in TAAD.

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Table 1. Patient demographics, co-morbidities, mode of diagnosis, coronary arteries involved and the cause

of coronary malperfusion in the studies included.

Study	Year	Study population (n)	Mean age (years)	Gender (Male: Female)	Comorbidities/Associated Disease	Comorbidities/Associated Disease	Comorbidities/Associated Disease	Comorbidities/Associated Disease	Comorbidities/Associated Disease	Method/Associated diagnosis (for coronary involvement)	Coronary arteries involved (Right, Left or Both)	C c n a f s (i c
					Hypertension	Diabetes	Smoking	Previous CAD	Other			

Kawahito 2003 et al.	12	60.8 ± 8.3	4:8	66.67% (8/12)		0%	Marfan's (8.33%, 1/12)	9 patients developed MI prior to surgery (ST elevation on ECG, abnormal LV wall motion on ECHO and high CK levels. 3 patients developed dissection after aortic declamping (seen in intraoperative TEE, postoperative coronary angiogram, or at autopsy.	Right-8 Left-2 Both-2	C 2 C n d 7 C n d 3 M f c c T A T E T C

Eren et al.	2007	14	56.7 ± 8.4 years	11:3	100% (14/14)		21.43% (3/14)	Renal insufficiency (14.29%, 2/14) COPD (35.71%, 5/14) Marfan's (14.29%, 2/14).	11 patients had ST elevation on ECG, abnormal LV wall motion on ECHO and very CK levels. 3 patients were found to have dissection intraoperatively using TEE	Right-8 Left-2 Both-4	I – T A T P T C

Chen et al.	2013	20	51.8 ± 11.8	14:6	60% (12/20)	15% (3/20)	25% (5/20)	5% (1/20)	COPD (5%, 1/20) Marfan's (10%, 2/20)	14 patients had myocardial ischaemia preoperatively. 6 patients were diagnosed during surgery.	Right- 15 Left- 5	
Neri et al.	2001	24	65.5 years (41-78 years)	14:10	100% (24/24)			0%	COPD (12%, 3/24) Renal insufficiency (4%, 1/24)	ECG, echocardiogram, serum CK levels +/- coronary angiography.	Right- 11 Left- 4 Both- 9	

Imoto et al.	2013	75	60.8 ± 12.2 years	35:40	67% (50/75)		0%	Renal Failure (3%, 2/75) Marfans (3%, 2/75) Cerebrovascular disease (5%, 4/75) Valvular heart disease (4%, 3/75) History of type B aortic dissection (5%, 4/75)	Intraoperative examination, coronary angiography or TEE. ECG/Echo also used to diagnose myocardial ischaemia/infarction	Right-26 Left-19 Both- 3	C I
Pego-Fernandes et al.	1999	11	48 years (37-62 years)	6:5	81.8% (9/11)			Marfan's (18.2%, 2/11)	ECG, Cineangiograms, Intraoperative examination	Right-9 Both-2	I a c
Lajevardi et al.	2012	1	73	Female	100% (1/1)			Alzheimer's	ECG, Coronary angiography, Intraoperative TEE	Left	C (i i s t a c f t i t a i a

CAD: Coronary Artery Dissection, MI: Myocardial Infarction, ECG: Electrocardiogram, Echo: Echocardiogram, CK: Serum Kinase, TEE: Transesophageal Echocardiogram, LV: Left Ventricle, COPD: Chronic Obstructive Pulmonary Disease

Table 2. Summary of studies Included and the Procedures Reported to Repair the TAAD and Coronary Involvement.

Authors	Year	Surgery for TAAD	Extent of aorta replacement (root, ascending, arch)	Surgery for coronary arteries	Postoperative interventions (PCI, CABG, Others)
Kawahito et al.	2003	Open aortic repair	Ascending	CABG with saphenous veins	Not reported
Eren et al.	2007	Open aortic repair	Root, ascending, arch	CABG with saphenous veins	Not reported
Chen at al.	2013	Open aortic repair	Ascending, the proximal transverse arch or hemiarch	CABG	Not reported
Neri et al.	2001	Open aortic repair	Ascending aorta and hemiarch	CABG Local repair Patch repair Short graft	Not reported
Imoto et al.	2013	Open aortic repair	Ascending, total arch, hemiarch and stent-graft closure of the descending aortic	Coronary artery stent (prior to aortic repair) CABG Local repair Synthetic graft (if root repair took place)	Not reported
Pego-Fernandes et al.	1999	Open aortic repair	Ascending	CABG or Gore-Tex graft (to the coronary ostia or right coronary artery) Reimplantation of dissected coronary	Not reported
Lajevardi et al.	2012	Open aortic repair	Ascending	aortic valve resuspension (due to intussusception)	Not reported

CABG: Coronary Artery Bypass Graft

Table 3. Coronary repair procedure and the associated mortality and low cardiac output syndrome rates in patients adopted from Imoto et al. [15].

Procedure	Mortality intraoperatively n (%)	Low Cardiac Output Syndrome n (%)
Stenting	1/7 (14.3)	1/7 (14.3)
Aortic Root Replacement	1/5 (20.0)	3/5 (60.0
Coronary Artery Bypass Graft	11/23 (52.4)	13/23 (61.9)
Biological Glue	3/13 (23.1)	3/13 (23.1)

Table 4. Summary of studies included and the associated morbidity and mortality rates.

Authors	Year	Morbidity					Mortality		
		Cerebral infarction/bleeding	Heart Failure	Renal Failure	Re-intervention	Other	Intraoperative	Early (within 30 days)	La (af yea
Kawahito et al.	2003	16.67% (2/12)	8.33% (1/12)	8.33% (1/12)	8.33% (1/12)	Respiratory failure (8.33%, 1/12), Leg ischaemia (8.33%, 1/12)	25% (3/12)	8.33% (1/12)	8.3 (1/
Eren et al.	2007	-	-	9.09%(1/11)	-	Respiratory Failure (9.09%, 1/11)	14.29% (2/14)	7.14% (1/14)	-
Chen at al.	2013						5% (1/20)	15% (3/20)	
Neri et al.	2001			25% (6/24)	8.33% (2/24)		12.5% (3/24)	8.33% (2/24)	3/
Imoto et al.	2013						In-hospital mortality: 24% (18/75)	In-hospital mortality: 24% (18/75)	
Pego-Fernandes et al.	1999					-	18.2% (2/11)	9.1% (1/11)	18 (2/
Lajevardi et al.	2012	Not reported	Not reported	Not reported	Not reported	Not reported	0%	Not reported	

Figure 1. The Neri Classification illustrating Type A, B and C coronary dissection. See description in text. Reproduced from Neri et al. [9] (Elsevier open access). Copyright permission obtained.

Figure 2. Illustration of underlying pathology behind Type A coronary dissection as well as its reconstruction technique. See description in text. Reproduced from Neri et al. [9] (Elsevier open access). Copyright permission obtained.

Figure 3. Illustration of underlying pathology behind Type B coronary dissection as well as its reconstruction technique. See description in text. Reproduced from Neri et al. [9] (Elsevier open access). Copyright permission obtained.

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