Case Report

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Fatal and Non-fatal Spontaneous and Iatrogenic Acute Aortic Dissection type A: report of two cases

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Abstract:

Aim: To describe iatrogenic and spontaneous presentations of acute aortic dissection (AAD) type A.

Method: We report two cases of male patients with iatrogenic non-fatal and spontaneous fatal acute aortic dissection type A.

Results: Two male patients are discussed with different aetiology of acute aortic dissection type A. The first patient of 68-years-old, having progressive angina pectoris developed iatrogenic acquired AAD secondary to diagnostic procedure of coronary angiography. Emergent surgical procedure was performed. The second patient of 66-years-old, with known history of aortic dissection type B, presented with acute chest and back pain. The condition was misdiagnosed for acute pulmonary embolism followed by sudden death. Autopsy findings are discussed.

Conclusions: Iatrogenic acute aortic dissection type A is a rare complication of routine coronary angiography which requires emergent surgical intervention. Spontaneous AAD may be erroneously seen for acute pulmonary embolism

Keywords: Spontaneous Acute Aortic Dissection Type A, Iatrogenic Aortic Dissection Type A, Diagnostic Imaging Technique, Emergent Surgical Intervention.

Introduction:

Acute aortic syndrome (AAS) is classified according to Stanford classification into proximal (type A) and distal (type B) types [1]. AAS encompasses a group of life-threatening type A- and B- aortic conditions (aortic dissection (AD), intramural haematoma (IMH) and penetrating atherosclerotic ulcer (PAU)) that are increasingly recognized due to sophistication, rapid and wide spread application of non-invasive imaging modalities. The syndrome requires high index of clinical suspicion, rapid recognition and appropriate treatment (including operative techniques and cannulation methods) to optimize outcome and improve prognosis [2]. In a recent study, acute AD was initially not suspected in almost one-third of the subjects [3]. Diagnosis is established based on clinical features enforced by rapid imaging techniques and supported by serum biomarkers. Multi-slice computed tomography scanning (MSCT), transthoracic (TTE) and/or Trans oesophageal echocardiography (TEE) or magnetic resonance imaging (MRI) are most common-

ly used as the diagnostic imaging technique [2, 4-7]. Currently, the widely used modality for the initial diagnosis is MSCT [8]. Population-based studies suggest that the incidence of acute AD ranges from 2 to 3.5 cases per 100,000 person-years [2, 9]. Type A dissections (AD, IMH and PAU) involve the ascending aorta and in almost all cases require emergent surgical intervention in highly specialized institutes with broad diagnostic and therapeutic facilities [10] and is associated with operative mortality rates up to 35% [11]. Two cases are presented demonstrating iatrogenic non-fatal and spontaneous fatal presentations of the disease.

Case reports:

Case 1

Iatrogenic type A-AD during routine coronary angiography (CAG) of a 68-year-old male who was analysed due to a 6-year chest pain at rest and on exertion. His only risk factor for coronary artery disease was treated arterial hypertension. Exercise tolerance test-

ing demonstrated silent ischemia. He was treated medically and planned for elective CAG. Routinely right Judkins-4 catheter was used with smooth cannulation of the right coronary artery (RCA). The first projection of the RCA in left anterior oblique position revealed severe atherosclerotic changes (Fig. 1A, black arrow). At the second injection of the RCA in right anterior oblique projection, contrast was directly injected into the aortic wall creating an iatrogenic dissection (Fig. 1B-D, white arrow). The left coronary artery (Fig. 1E, black arrow) showed diffuse severe obstructive lesions. The patient had no chest or back pain and he was hemodynamically stable. The diagnosis was confirmed by MSCT revealing

ing iatrogenic acute type A-AD (Fig. 1F, white arrow and arrowheads). The patient was immediately sent for emergent operation. At operation, a large entry was found cranial of the RCA ostium and the false lumen was thrombosed. No pericardial effusion was visible. Bio glue was injected between the layers and supra-coronary ascending aorta replacement/repair with 28 Gel weave aorta tubular graft was performed. Coronary artery bypasses grafting with left internal mammary artery to the left anterior descending artery and venous jump graft to the intermediate, obtuse marginal and descending posterior arteries. The post-operative period was uncomplicated.

Figure 1: Coronary angiogram. The first projection of the RCA in left anterior oblique position revealed severe atherosclerotic changes (Fig. 1A, black arrow). At the second injection of the RCA in right anterior oblique projection, contrast was directly injected into the aortic wall creating an iatrogenic dissection (Fig. 1B-D, white arrow). The left coronary artery (Fig. 1E, black arrow) showed diffuse severe obstructive lesions. The diagnosis was confirmed by MSCT revealing iatrogenic acute type A-AD (Fig. 1F, white arrow and arrowheads).

Case 2

A 66-year-old man with antecedent medical history including cardiac asthma based on hypertensive crisis in 2002, type B aortic dissection in 2002 (Fig. 2A) beginning at the anterior part of the aortic arch propagating to the descending aorta (Fig. 2B), mild renal impairment (Kreatinine 121 μ mol/l and GFR 52) with cyst (72 mm) in the left kidney and contracted right kidney. He was asymptomatic and the blood pressure was well controlled under supervision of his general practitioner. His blood pressure was well managed with a beta blocker, calcium re-entry blocker and statin. He was annually followed-up at the out-patient clinic. On October 17, 2013, at night he was awakened with chest and back pain. He was seen by the paramedics and because of a unchanged appearing ECG, he was reassured and not taken to the hospital. The next morning he was seen by his general practitioner. Due to elevated D-dimer of > 18.000 (normal < 500), suspicion of pulmonary embolism was arisen and he was advised by his general practitioner to consult the pulmonologist. The high sensitivity troponin and NT-proBNP were slightly elevated 21 and 38, respectively. On the

way to the hospital he became unconscious and died.

At autopsy, the heart weighed 569 gram, cardiac tamponed with thrombus and haemorrhagic fluid filled through type an aortic dissection (Fig. 2C and D). Pathology specimen of the ascending aorta with spontaneous acute dissection type-A showing C) the dissection (black arrow) with D) mural haematoma and thrombus formation (white arrow). The entire ascending aorta was involved in the dissection reaching the level of the aortic valve leaflets. The coronary and carotid arteries were free of dissection. At the level of the descending thoracic aorta a splitting was found leading to an old and new dissection lumen. The dissection course was from the proximal aorta to the splitting point. The old type B dissection showed an occluded old lumen with a functional pseudo-lumen reaching the iliac arteries. The right kidney was atrophic (40 grams) and left kidney was hypertrophic (299 grams) with a cyst filled with clear yellow fluid. No pulmonary embolism, tumour or infection was found. Myocardial staining demonstrated no recent infarction.

Figure 2: Antecedent type B aortic dissection in 2002 (Fig. 2A) beginning at the anterior part of the aortic arch propagating to the descending aorta (Fig. 2B), at autopsy, cardiac tamponed with thrombus and haemorrhagic fluid filled through type an aortic dissection (Fig. 2C and D). Pathology specimen of the ascending aorta with spontaneous acute dissection type-A showing C) the dissection (black arrow) with D) mural haematoma and thrombus formation (white arrow).

Discussion:

Aortic dissection (AD), first recognized in 1761^[12] and as early as 1802, Maunoir described the process of AD^[13]. The incidence of AD is estimated by several authors varying from 2.9/10,000/year^[9] to 5-30 per million population/year^[14, 15].

Aetiology and aetiopathogenesis: (spontaneous, acquired iatrogenic and acquired accidental)

Degeneration of the aortic wall, in particular myxoid and mucoid degradation and deterioration of the aortic media layer, has been recognized as an etiologic factor of AD increasing its susceptibility to rupture^[16]. AD occurs usually spontaneous^[17] but acquired caused by iatrogenic trauma during invasive cardiovascular diagnostic procedure^[18], after coronary surgical intervention^[19] and delayed dissection of the donor aorta 22 years following orthotopic heart transplantation^[20] have been reported and accounts for 5% of cases^[21]. In one of our patients (patient 5), acquired iatrogenic acute AD, without chest or back pain, occurred during diagnostic coronary artery catheterization. Rarely, traumatic chest injury may be incriminated for AD^[22, 23]. Acquired accidental aorta injuries due to deceleration trauma or blunt chest injury are often involving the descending aorta (type B)^[24-26] and rarely the ascending aorta^[22]. In contrast, as a result of Marfan syndrome AD was detected in the younger patient group (< 70 years)^[8]. Meijboom et al reported that pregnancy in Marfan syndrome is relatively safe up to an aortic root diameter of 45 mm^[27]. Few cases, with successful repair, have been reported describing acute aortic dissection which may rarely occur especially during the third trimester of pregnancy in Marfan patients. These cases had an aortic root dilatation of 60 and 52 mm, respectively^[28, 29].

Clinical presentation and differential diagnosis:

AD is an infrequent but highly fatal disorder which is associated initially with acute chest pain in 92-96% and without chest pain in 4-8% of patients^[9, 30]. In our first case, the acquired iatrogenic AD was painless. Earlier reports have elucidated that one-third of cases were initially missed^[31] and that AD was clinically suspected only in 15% of patients before death^[9]. Although infrequent, AD should enter into the differential diagnosis of patients either presenting with acute chest pain associated with or without arterial hypertension and/or aortic regurgitation or mimicking ST-segment elevation myocardial infarction^[21, 32]. AD should be differentiated from acute pulmonary embolism and acute coronary syndromes^[32]. Mehta et al., reported that acute type A-AD as a result of hypertension, atherosclerosis and iatrogenic post cardiac surgery, were commonly found in the elderly (>70 years)^[8].

Diagnostic modalities:

Multi-slice computed tomography scanning (MSCT), transthoracic (TTE) and/or Trans oesophageal echocardiography (TEE) or magnetic resonance imaging (MRI) are most commonly used as the diagnostic imaging technique^[2, 4-7]. Recently, the widely used modality for the initial diagnosis is MSCT^[8]. It is emphasized that high index of clinical suspicion and early imaging studies (MSCT and/or TEE) may lead to early recognition and hence proper timely surgical intervention to improve outcome and prognosis. Recently,

using 32-slice MSCT scanning, a false diagnosis of type A-AD in a pregnant woman having a normal aorta has been reported^[33].

Electrocardiography: In the series of Evangelista et al., normal ECG without ST-T segment changes was found in 38% of patients^[30]. ECG demonstrated evidence of acute myocardial infarction in 5% of patients with type A-AD^[21]. On the other hand, ECG with acute coronary syndrome- resembling electrocardiographic profiles were described in approximately 25% of patients which was associated with more complications and in type A-AD was an independent predictor of in-hospital mortality^[6]. In addition, a normal ECG can increase the suspicion of the occurrence of AD. The initial ECG in our patients showed either normal or non-diagnostic findings, such as nonspecific ST segment abnormalities, sinus bradycardia or sinus tachycardia and voltage criteria for left ventricular hypertrophy.

Chest x-ray: It was demonstrated in two large series that the chest x-ray was normal in 10-20% of the cases^[21, 30] and mediastinal widening was observed more frequently in type A than type B-AD (58% vs. 43%)^[30]. A normal chest x-ray is not capable of ruling out the suspicion of AD.

Biomarkers: Recently, from our group, Linssen et al. demonstrated that plasma N-terminal pro-Brain-type natriuretic peptide (NT-proBNP) independently predicted all-cause mortality and cardiovascular events in the general population^[34]. In contrast to C-reactive protein (CRP), BNP is a marker of specific pathophysiological processes, especially in relation to ventricular dysfunction and ischemic burden^[35]. Many supplementary biomarkers (CRP, D-dimer, NT-proBNP) could be helpful in early diagnosis and rapid classification of acute chest pain guiding management and predicting the prognosis^[36]. In a recent retrospective study by Sakamoto et al., they suggested that a cut-off value of 5.0 µg/ml of D-dimer could differentiate between AD and pulmonary embolism from acute myocardial infarction^[37]. They recommended to perform MSCT first in a patient with D-dimer value > 5.0 µg/ml^[37] to rule out AD. C-Reactive Protein values were demonstrated to have positive correlation with aortic diameter (> 48 mm) and age (> 65 years) in predicting in-hospital development of clinical events and mortality^[38]. All available conventional biomarkers are nonspecific and should be used with caution for differentiation between chest pain syndromes (acute aortic syndrome, acute coronary syndrome and pulmonary embolism), in conclusion, there is yet no a reliable biomarker readily available to differentiate between the different acute chest pain syndromes^[39]. Elevated D-dimer may justify urgent contrast enhanced MSCT scanning to differentiate between the different acute aortic, coronary or pulmonary vascular syndromes.

Mortality: Acute type A-AD is highly fatal disorder with an early mortality of 1-2% per hour. The published in-hospital mortality rate of spontaneous AD was 22.1%^[18]. Iatrogenic AD is associated with higher mortality rate than spontaneous AD (35% vs. 24%)^[21, 40, 41]. Among the predictors for early mortality are aortic diameter of > 50 mm and involvement of ascending aorta^[5]. Acute coronary syndrome-like ECG finding could raise the possibility of misdiagnosis and in type A-AD it was, besides syncope at presentation and

advanced age, a strong independent predictor of in-hospital mortality^[6]. Vanhuysse et al., reported higher operative mortality rate of 40% in patients aged > 80 years compared to 18% in patients aged < 80 years^[42]. On the other hand, Kawahito et al., observed no significant differences in the hospital mortality, actuarial survival or event free rates of surgically treated patients, for type A-AD, aged more than 75 or less than 75 years^[43]. Hospital mortality rates varied from 10.5% (<75 years) to 13.0% (> 75 years)^[43] and overall rate of 13.4% in a population with a mean age of 60 years^[44]. Acute type A-AD accounts for 14% of maternal cardiac mortalities in the United Kingdom^[45]. In 2000, in the autopsy series (84 patients with 86 AD) of Meszaros et al., in only 13 patients (13/84=15%), AD was clinically suspected before death. Aortic dissection was proven at autopsy in 80/84 (95%), by surgical procedure in 6/84 (7%) and by ultrasound and MSCT scan in 2/84 (2.4%) of patients who remained alive^[9]. Post-mortem, atherosclerosis of the aorta and ostial stenosis of the coronary arteries were found in (11/80=14%) and (76/80=95%), respectively. Finally, rupture of the aorta causing death was detected in (69/86=80%) of patients^[9]. In a single-centre prospective study, it was found that in-hospital mortality was significantly associated with elevated D-dimer, CRP and aortic diameter^[46].

Conclusions: Iatrogenic acute aortic dissection type A is a rare complication of routine coronary angiography which requires emergent surgical intervention. Spontaneous acute type A-AD may be erroneously seen for acute pulmonary embolism.

Reference:

1. Crawford ES, Svensson LG, Coselli JS, Safi HJ, Hess KR. [Surgical treatment of aneurysm and/or dissection of the ascending aorta, transverse aortic arch, and ascending aorta and transverse aortic arch](#). Factors influencing survival in 717 patients. J Thorac Cardiovasc Surg 1989 Nov;98(5 Pt 1):659-73.
2. Nienaber CA, Powell JT. [Management of acute aortic syndromes](#). Eur Heart J 2011 Aug 2.
3. Jansen Klomp WW, Brandon Bravo Bruinsma GJ, Peelen LM, Nierich AP, Grandjean JG, van 't Hof AW. [Clinical recognition of acute aortic dissections: insights from a large single-centre cohort study](#). Neth Heart J 2017 Mar;25(3):200-6.
4. Kim JK, Park SW, Jeong JO, Lee SC, Gwon HC, Kim K, et al. [Clinical features and prognosis of acute aortic intramural hemorrhage compared with those of acute aortic dissection: a single center experience](#). Jpn Heart J 2001 Jan;42(1):91-100.
5. Evangelista A, Dominguez R, Sebastia C, Salas A, Permyer-Miralda G, Avegliano G, et al. [Prognostic value of clinical and morphologic findings in short-term evolution of aortic intramural haematoma](#). Therapeutic implications. Eur Heart J 2004 Jan;25(1):81-7.
6. Biagini E, Lofiego C, Ferlito M, Fattori R, Rocchi G, Graziosi M, et al. [Frequency, determinants, and clinical relevance of acute coronary syndrome-like electrocardiographic findings in patients with acute aortic syndrome](#). Am J Cardiol 2007 Sep 15;100(6):1013-9.
7. Meredith EL, Masani ND. [Echocardiography in the emergency assessment of acute aortic syndromes](#). Eur J Echocardiogr 2009

Jan;10(1):i31-i39.

8. Mehta RH, O'Gara PT, Bossone E, Nienaber CA, Myrmel T, Cooper JV, et al. Acute type A aortic dissection in the elderly: clinical characteristics, management, and outcomes in the current era. J Am Coll Cardiol 2002 Aug 21;40(4):685-92.
9. Meszaros I, Morocz J, Szilavi J, Schmidt J, Tornoci L, Nagy L, et al. Epidemiology and clinicopathology of aortic dissection. Chest 2000 May;117(5):1271-8.
10. Menon V, Sengupta J, Unzek S. Optimal management of acute aortic dissection. Curr Treat Options Cardiovasc Med 2009 Apr;11(2):146-55.
11. Andersen ND, Ganapathi AM, Hanna JM, Williams JB, Gaca JG, Hughes GC. Outcomes of acute type a dissection repair before and after implementation of a multidisciplinary thoracic aortic surgery program. J Am Coll Cardiol 2014 May 6;63(17):1796-803.
12. Nichols F. Observations concerning the body of his late majesty. Philos Trans R Soc Lond 1761;265-74.
13. Maunoir JP. Memoires physiologiques et pratiques sur l'anéurysme at la ligature des arteres. Geneva, JJ Paschoud 1802.
14. Manfredini R, Boari B, Gallerani M, Salmi R, Bossone E, Distant A, et al. Chronobiology of rupture and dissection of aortic aneurysms. J Vasc Surg 2004 Aug;40(2):382-8.
15. Bickerstaff LK, Pairolero PC, Hollier LH, Melton LJ, Van Peenen HJ, Cherry KJ, et al. Thoracic aortic aneurysms: a population-based study. Surgery 1982 Dec;92(6):1103-8.
16. Loire R, Man J. [Aortic insufficiency and degenerative aortic disease. Anatomic study and etiology]. Ann Cardiol Angeiol (Paris) 1990 Jun;39(6):327-31.
17. Hostiuc S, Dermengiu D, Ceausu M, Capatina CO, Luca L, Hostiuc M. [Sudden death due to dissection of the thoracic aorta associated with dissection and rupture of the pulmonary artery: report of two cases](#). Forensic Sci Int 2014 Mar;236:e9-13.
18. Aalberts JJ, Boonstra PW, van den Berg MP, Waterbolk TW. [In-hospital mortality and three-year survival after repaired acute type A aortic dissection](#). Neth Heart J 2009 Jun;17(6):226-31.
19. Said SAM, Somer ST, Bloemendaal K. Dissecting aneurysm of the ascending aorta: An uncommon complication of CABG. Cardiologie 1, 103-105. 1994.

Ref Type: Journal (Full)

20. Skalsky I, Urban M, Malouskova J, Szarszoi O, Pirk J, Netuka I. [Aortic dissections following heart transplantations](#). J Card Surg 2012 Jan;27(1):125-7.
21. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. [The International Registry of Acute Aortic Dissection \(IRAD\): new insights into an old disease](#). JAMA 2000 Feb 16;283(7):897-903.
22. Said SA, Keeris LM, van IG. A soccer victim: late rupture of the ascending aorta after violent physical trauma. Int J Cardiol 1998 Feb 28;63(3):309-12.
23. Zivkovic V, Nikolic S. [An uncommon accident and unusual](#)

cause of death--a fatal domestic ram attack. J Forensic Sci 2013 Jul;58(4):1065-8.

24. Ansari AH, Ahmed AS, Lal NP. Traumatic aortic injury: a case report. Ulus Travma Acil Cerrahi Derg 2009 Nov;15(6):621-3.

25. Nano G, Mazzaccaro D, Malacrida G, Occhiuto MT, Stegher S, Tealdi DG. Delayed endovascular treatment of descending aorta stent graft collapse in a patient treated for post- traumatic aortic rupture: a case report. J Cardiothorac Surg 2011;6:76.

26. Cademartiri F, Salamousas BV, Luccichenti G, Pedrazzini M. Post-traumatic descending aorta intramural haematoma fortuitously witnessed during a magnetic resonance examination of the spine. Acta Biomed 2004 Dec;75(3):185-7.

27. Meijboom LJ, Vos FE, Timmermans J, Boers GH, Zwinderman AH, Mulder BJ. Pregnancy and aortic root growth in the Marfan syndrome: a prospective study. Eur Heart J 2005 May;26(9):914-20.

28. Nonga BN, Pasquet A, Noirhomme P, El-Khoury G. Successful bovine arch replacement for a type A acute aortic dissection in a pregnant woman with severe haemodynamic compromise. Interact Cardiovasc Thorac Surg 2012 Aug;15(2):309-10.

29. Guo C, Xu D, Wang C. Successful treatment for acute aortic dissection in pregnancy---Bentall procedure concomitant with cesarean section. J Cardiothorac Surg 2011;6:139.

30. Evangelista A, Padilla F, Lopez-Ayerbe J, Calvo F, Lopez-Perez JM, Sanchez V, et al. Spanish Acute Aortic Syndrome Study (RESA). Better diagnosis is not reflected in reduced mortality. Rev Esp Cardiol 2009 Mar;62(3):255-62.

31. Spittell PC, Spittell JA, Jr., Joyce JW, Tajik AJ, Edwards WD, Schaff HV, et al. Clinical features and differential diagnosis of aortic dissection: experience with 236 cases (1980 through 1990). Mayo Clin Proc 1993 Jul;68(7):642-51.

32. Gu YL, Svilaas T, van der Horst IC, Zijlstra F. Conditions mimicking acute ST-segment elevation myocardial infarction in patients referred for primary percutaneous coronary intervention. Neth Heart J 2008 Oct;16(10):325-31.

33. Hamilton MC, Nightingale AK, Masey S, Stuart AG, Angelini G, Hopkins R, et al. A case report of a normal aorta misdiagnosed as type A dissection by modern multidetector computed tomography. Eur Radiol 2010 Aug;20(8):1856-8.

34. Linssen GC, Bakker SJ, Voors AA, Gansevoort RT, Hillege HL, de Jong PE, et al. N-terminal pro-B-type natriuretic peptide is an independent predictor of cardiovascular morbidity and mor-

ality in the general population. Eur Heart J 2010 Jan;31(1):120-7.

35. Leon de la Fuente, Naesgaard PA, Nilsen ST, Woie L, Aarsland T, Gallo P, et al. B-type natriuretic peptide and high sensitive C-reactive protein predict 2-year all cause mortality in chest pain patients: a prospective observational study from Salta, Argentina. BMC Cardiovasc Disord 2011;11:57.

36. Wen D, Zhou XL, Li JJ, Hui RT. Biomarkers in aortic dissection. Clin Chim Acta 2011 Apr 11;412(9-10):688-95.

37. Sakamoto K, Yamamoto Y, Okamatsu H, Okabe M. D-dimer is helpful for differentiating acute aortic dissection and acute pulmonary embolism from acute myocardial infarction. Hellenic J Cardiol 2011 Mar;52(2):123-7.

38. Wen D, Wu HY, Jiang XJ, Zhang HM, Zhou XL, Li JJ, et al. Role of plasma C-reactive protein and white blood cell count in predicting in-hospital clinical events of acute type A aortic dissection. Chin Med J (Engl) 2011 Sep;124(17):2678-82.

39. Eggebrecht H, Naber CK, Bruch C, Kroger K, von BC, Schmermund A, et al. Value of plasma fibrin D-dimers for detection of acute aortic dissection. J Am Coll Cardiol 2004 Aug 18;44(4):804-9.

40. Januzzi JL, Isselbacher EM, Fattori R, Cooper JV, Smith DE, Fang J, et al. Characterizing the young patient with aortic dissection: results from the International Registry of Aortic Dissection (IRAD). J Am Coll Cardiol 2004 Feb 18;43(4):665-9.

41. Tsai TT, Nienaber CA, Eagle KA. Acute aortic syndromes. Circulation 2005 Dec 13;112(24):3802-13.

42. Vanhuyse F, Maureira P, Laurent N, Lekehal M, Grandmougain D, Villemot JP. Surgery for acute type A aortic dissection in octogenarians. J Card Surg 2012 Jan;27(1):65-9.

43. Kawahito K, Adachi H, Yamaguchi A, Ino T. Early and late surgical outcomes of acute type A aortic dissection in patients aged 75 years and older. Ann Thorac Surg 2000 Nov;70(5):1455-9.

44. Halstead JC, Meier M, Etz C, Spielvogel D, Bodian C, Wurm M, et al. The fate of the distal aorta after repair of acute type A aortic dissection. J Thorac Cardiovasc Surg 2007 Jan;133(1):127-35.

45. Johnston C, Schroeder F, Fletcher SN, Bigham C, Wendler R. Type A aortic dissection in pregnancy. Int J Obstet Anesth 2011 Dec 2.

46. Wen D, Du X, Dong JZ, Zhou XL, Ma CS. Value of D-dimer and C reactive protein in predicting inhospital death in acute aortic dissection. Heart 2013 Aug;99(16):1192-7.