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Extensive Aortic Aneurysm in an 83-year-old Woman with History of Chronic Obstructive Pulmonary Disease: a case report

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ABSTRACT

Thoracoabdominal aortic aneurysm (TAAA) is rarely found and commonly asymptomatic. We reported an 83-year-old woman who was admitted to emergency room suffering from shortness of breath and intermittent epigastric pain. The patient was known to have a history of chronic obstructive pulmonary disease (COPD). Chest radiograph, CT scan of the chest, and echocardiograph showed TAAA with intraluminal thrombus along the TAAA. Patient was given symptomatic treatment and medical therapy for AA, including lipid profile optimization, blood pressure and heart contractility control, which aimed to reduce the shear stress and rupture risk of AA. This case report aimed to explain about the COPD as a risk factor of aortic aneurysm (AA) development.

Key words: aortic aneurysm–thoracoabdominal aortic aneurysm–chronic obstructive pulmonary disease

1 INTRODUCTION

Aortic aneurysm (AA) is a condition where the diameter of aorta is dilated for more than 50% of the normal size. In AA, dilation will happen gradually, and it is usually associated with increasing age and atherosclerosis. [1, 2] AA is divided based on the position of the aneurysm. Aneurysm which is located in thoracic aorta is called as thoracic aortic aneurysm (TAA). Aneurysm which is located in the abdominal aorta is called abdominal aortic aneurysm (AAA) and TAA that expands to the abdominal aorta is called thoracoabdominal aortic aneurysm (TAAA) or extensive AA. [2, 3] TAAA is a complex aortic disease. The incidence of TAAA is low, with only 10 new cases diagnosed per 100,000 population-year. [3]

Women are diagnosed with TAAA in older age compared to men and have a higher risk of rupture. [4] AA is often asymptomatic and detected accidentally when doing imaging examinations, such as ultrasound and CT scan, therefore the mortality rate of AA is high. [5] Patient with chronic obstructive pulmonary disease (COPD) in the age above 75 years has a high prevalence of AA, severe lung destruction, and calcification of aortic wall which are risk factors for AA and also has 3.6 times greater risk for rupture. [6–8] We reported a rare case of TAAA and explained about COPD as a risk factor of AA development.

2 CASE REPORT

An 83-year-old woman came to the emergency room with complaint of weakness. She had no appetite for 2 weeks before being admitted to the hospital. The patient complained of shortness of breath and coughing since 3 days before the admission. The patient also complained of intermittent epigastric pain, nausea and vomiting since the last 1 month. Complaints of slashing pain in the chest and abdomen area were denied. The patient was known to have a history of

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COPD for the past 1 year, but complaints of shortness of breath often appear in the past month. History of hypertension, diabetes mellitus, and kidney disease were denied.

Vital signs showed blood pressure 100/70 mmHg, tachycardia (90 bpm), tachypnea (26 times/minute), afebrile, oxygen saturation 96% with 4 lpm O2 via nasal canula. Physical examination showed barrel chest shape with widening of the rib cage. There was an prolonged expiration accompanied by rales and wheezing throughout the chest. There was an abdominal distension and tenderness throughout the abdomen, especially in the epigastric region, normal bowel sound, and no bruit sound. Complete blood count, blood sugar, lipid profile, and kidney function test results were within the normal limit. Electrocardiography (ECG) result supported COPD Figure 1 .

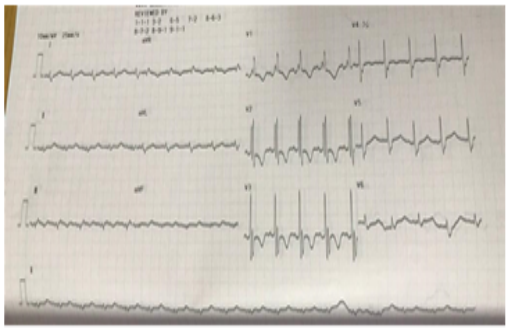

Figure 1. ECG showing sinus rhythm with complete right bundle branch block (RBBB) and low voltage

Chest radiograph showed COPD accompanied by prominent aortic notch and widening of the left posterior mediastinum suspicious of AA with differential diagnosis of mediastinal mass Figure 2 . Echocardiography showed aortic regurgitation, mitral regurgitation, dilation of sinus of Valsalva, ST junction and proximal ascending aorta Figure 3 .

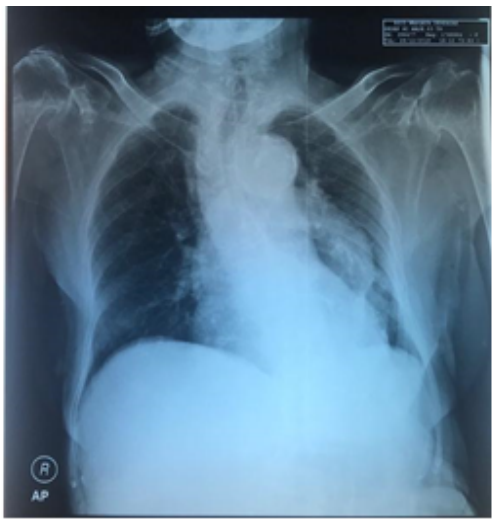

Figure 2. Chest x-ray showing prominent aortic notch, hyperaerated lung, widening of the left posterior mediastinum

Thoracic CT scan showed AA of ascending aorta (4.4 cm in diameter), aortic arch, descending aorta (4.5 cm in diameter) to the thoracoabdominal aorta. Intraluminal thrombus appeared along the TAA. Aortic atherosclerosis appeared with descending aortic wall irregularities as high as the ninth thoracic vertebraeFigure 4 .

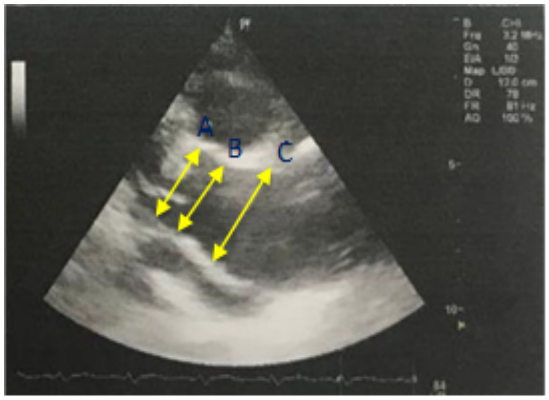

Figure 3. Echocardiograph showing (A) dilatation of sinus of valsava, (B) dilatation of ST junction, (C) dilatation of proximal ascending aorta

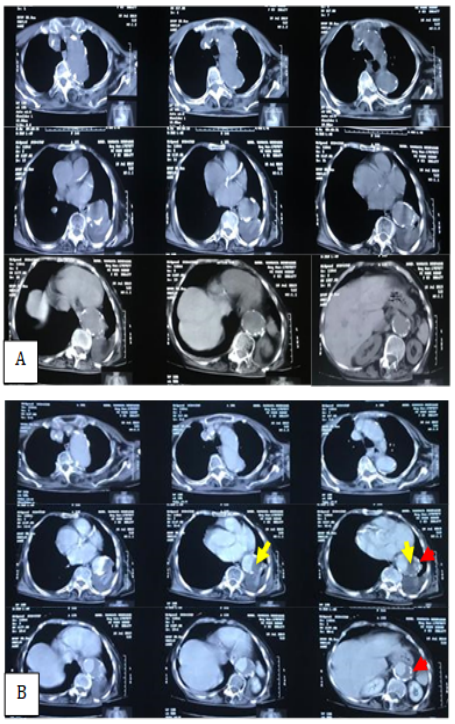

Figure 4. (A) Thoracic CT Scan: pre-contrast. (B) Thoracic CT Scan: post-contrast. Yellow arrow: radioluscent pattern showing intraluminal thrombus. Red arrow: showing irregularities of aneurysm wall.

The patient received symptomatic therapy such as nebulization with salbutamol every 8 hours, intravenous methylprednisolone 62.5 mg twice daily, intravenous pantoprazole 40 mg once daily, and oral paracetamol 1,000 mg thrice daily. Patient was advised to be referred for surgical intervention to treat the TAA, however the family refused due to the potential risks during and after surgery. Therefore, the patient was given medical therapy for AA, such as oral bisoprolol 2.5 mg once daily and oral simvastatin 20 mg once daily.

3 DISCUSSION

In this case, an 83-year-old female patient had a history of COPD. The complaint of shortness of breath was felt more often in the last one month and accompanied by intermittent epigastric pain. The vital signs were within the normal limit. Physical examination of the chest showed a barrel chest shape, elongated expiration accompanied by rales and wheezing throughout the lung. Physical examination of the abdomen showed tenderness throughout the abdomen, especially in the epigastric region and no bruit was obtained. ECG examination is consistent with COPD. We gave initial therapy for COPD and dyspepsia to the patient However, the chest radiograph examination revealed an image of COPD accompanied by prominent aortic notch and widening of the left posterior mediastinum. Therefore, this patient was suspected of having AA with a differential diagnosis of the left posterior mediastinal mass. We further evaluated the CT scan of the chest and found ascending AA (4.4 cm in diameter), aortic arch, and descending aorta (4.5 cm in diameter) to the thoracoabdominal aorta accompanied by an intraluminal thrombus along the TAAA. Aortic atherosclerosis appeared with descending aortic wall irregularities as high as the ninth thoracic vertebrae. The echocardiography result also showed aortic dilation. AA is often diagnosed accidentally when performing imaging tests, such as ultrasound and CT scan, because it is often asymptomatic. Screening studies performed in the general population showed that the prevalence of AAA is 4.9%. [9]

Aneurysm is a deterioration and damage of the aortic wall architecture due to gradual loss of elastin and smooth muscle cells as results of aging process and their progression is influenced by several risk factors. [1, 2, 10] Compared to men, TAA is more commonly diagnosed at older age in women, with mean of age is 75.9 in women vs 62.8 years in men (p=0.01). [3] Meanwhile, AAA is more common in men. In this case, we found a female patient at the age of 83 years old when TAAA was first identified, and the TAAA was originated from TAA which extended to the abdominal aorta.

In this case, the patient complained of shortness of breath which occurred more often in the last one month. It was because patient who has aneurysm tends to experience more airway obstruction. [8] Complaints of intermittent epigastric pain accompanied by nausea and vomiting with stable vital signs can be caused by compression in the area due to an enlarged aorta. However, attention must be given to the abdominal pain because it might be a sign of aortic dissection, especially if abdominal pain is accompanied by other supporting signs, such as decreased consciousness and hypotension. Although complaints of slashing pain in the chest and abdomen were denied, the presence of aortic dissection in this case can not be ruled out. Based on the result of thoracic CT scan, there was ascending aortic wall irregularities as high as ninth thoracic vertebrae, so that the presence of aortic dissection can not be removed due to the limitation of CT scan facilities where we only have CT scan with a single slice of 10 mm thick slices and can not be reformed into coronal and sagital planes.

The mechanism of AA is generally explained as follows. The medial layer of the aortic wall consists mainly of structural proteins, such as collagen and elastin, which contribute to the aortic capacitance and elasticity. Degradation of structural proteins or the presence of defect in their composition causes degeneration of the medial layer and weakening of the aortic wall. Subsequent dilation occurs due to hemodynamic force in the arterial wall and intrinsic change in the composition of the artery wall itself. According to Laplace's law, the wall tension is proportional to the pressure multiplied by the radius of the arterial canal. Then, when the diameter of the aorta increases, the aortic wall tension will also increase, so that it becomes a vicious circle. The condition will be worsen by hypertension. [4] The patient discussed in this case had no history of hypertension.

Risk factors for AA include old age, smoking, hypertension, aortic calcification, hyperlipidemia, and COPD. In this case, the risk factors which were considered to cause AA were old age and COPD. Old age is a risk factor because individuals over 60 years old will progressively deposit calcium in the major arteries which will cause vascular calcification that led to the decreased elasticity of the aorta and other arteries, which can interfere with cardiovascular hemodynamics. [7, 11] Smoking and COPD are closely related, but both are also factors that influence the formation of atherosclerosis which will later be associated with the occurrence of aneurysm. [10] In COPD, there is an inflammation condition that causes an increase of C-reactive protein (CRP) and other systemic inflammatory markers. Increased production of proinflammatory cytokines and tissue factors by monocytes causes increased absorption of LDL by the macrophages, and directly induces expression of adhesion molecules in human endothelial cells. In addition, CRP can be directly deposited to artery walls during atherogenesis which then interacts with other inflammatory mediators to form foam cells, which are the basis of atherosclerosis plaque formation. [12–14] Atherosclerosis predilection is more common in the abdominal aorta than in the thoracic aorta. This is because the smooth muscle cells in the thoracic aorta have a better replicative ability to repair themselves at injury compared to those in the abdominal aorta. [10]

Patients with COPD have a 2.6 times higher risk of AAA. [15, 16] The prevalence of AAA with aortic diameter $\geq$30 cm in COPD patients is 3.7%. COPD patients over 75 years old have a high prevalence of AA, severe lung damage, and calcification of the aortic wall which are the risk factors for AA. [8] Systemic inflammation and hypoxic conditions in COPD contribute to abnormal vasa vasorum circulation to AA. [7] The underlying pathophysiology of AAA and COPD is a persistent pro-inflamatory response associated with proteolysis which causes damage to the arterial wall structure [17–19]

The diagnosis of AA can be made based on imaging test, such as ultrasound, CT scan, or MRI. The most commonly used TAA classification is the Crawford Classification, which has been modified by Safi et al. In this classification, TAA is divided into 5 categories based on the

location of AA. [20] In this case, the patient cannot be classified by using this classification due to the limitation of our facilities. We do not have a CT scan result of the sagittal and coronal plane of the thorax

When aneurysms are formed, they are naturally gradually increased in size and rupture risk. The most serious complication of AA is rupture. AAA with a diameter of 4.0-5.5 cm has an estimated 1-year rupture rate of 1%. [21] When the TAAA diameter exceeds 6 cm, the rupture rate is 14% per year. [22] If TAAA is left untreated, 80% will experience a rupture. [4] TAAA and TAA patients who are non-dissecting with a history of COPD had a rupture risk that increased up to 3.6-folds (p=0.04). [6] Factors that were independently and significantly associated with rupture risk are female gender (OR 4.5, 95% CI 1.98-1.02), smoking (OR 2.1, 95% CI 0.95-4.67), and hypertension (OR 1.04, 95% CI 1.02-1.07). [23] On CT scan of the chest, the patient had an intraluminal thrombus along the thoracoabdominal aorta. This also aggravates the condition of AA. Study investigating the effects of intraluminal thrombus with AA progression showed that increased intraluminal thrombus porosity can cause plasma proteins to diffuse into the aortic wall, thereby affecting AA growth. [24] Because the patient discussed in this case is a woman with COPD history and a large diameter of AA accompanied by extensive intraluminal thrombus, she has high risk of progression and rupture. Therefore, further evaluation and appropriate treatment are required.

The definitive therapy in the management of TAAA is open surgery. In this case, the patient had an aneurysm with diameter around 4.4-4.5 cm. Patients who suffer from TAA with diameter of 4.0-4.9 cm, have an annual risk of rupture of 2%. [25] Although surgical intervention is the only treatment strategy that has been proven to reduce the rupture risk, which is effective and long lasting, this procedure has a mortality rate of >5% in several countries. [26] Reported result of 30-day survival of TAAA intervention is 92-95%. Reported acute kidney failure was 2.1-9.0% and spinal cord ischemia was 1.3-15.5%. [27–29] The mortality rate from descending aortic and TAAA surgery is 8%. [22] The family of the patient refused to be referred for surgery intervention due to the potential risk of complication which might occur during and after surgery.

The main goal of medical therapy in AA is to reduce shear stress by lowering blood pressure and heart contractility. In patients who are not candidates for surgery or refuse surgical treatment, blood pressure control, smoking cessation, and lipid profile optimization should be carried out. Systolic blood pressure (SBP) target is between 100 and 120 mmHg. [30] Prophylaxis using beta blockers may be useful in reducing the level of aortic dilation by reducing the strength of myocardial contractions. Patients who received an ACE inhibitor were less likely to have a rupture of aneurysm compared to those who did not get an ACE inhibitor. [24] We did not give ACE inhibitor for this patient due to her blood pressure always in the lowest threshold of SBP target. The meta-analysis showed a significant decrease in AAA growth in patients who received statin therapy compared to those who did not. [31] Statin can also prevent the expansion of aneurysm. [32, 33] This patient received symptomatic therapy, including intravenous methylprednisolone 62.5 mg twice daily, salbutamol inhalation every 8 hours, oral paracetamol 1 gram thrice daily. Because the patient refused to be referred for surgical intervention, we gave medical therapy for AA including bisoprolol 2.5 mg once daily, simvastatin 40 mg once daily, pantoprazole 40 mg once daily. Our goal was to reduce shear stress and rupture risk. After the main complaint of this patient was improved, the patient was discharged and provided with symptomatic drugs, AA medical therapy, and was advised for routine check.

4 CONCLUSION

Patient with COPD can potentially have various comorbidities, one of which is AA. AA is a very dangerous disease, because it is often asymptomatic. Whereas, based on its natural course, AA will continue to dilate so the risk of rupture increases. Today, the most effective treatment for AA is surgical intervention. However, there are several medical therapies that provide benefits, especially in preventing the occurrence of AA rupture. If the patient is known to have AA, it is recommended to conduct routine surveillance checks to see the development and rupture of AA. Hence, the appropriate interventions can be carried out and can improve the prognosis.

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REFERENCES

[1] Coselli JS, Bozinovski J, LeMaire SA. Open Surgical Repair of 2286 Thoracoabdominal Aortic Aneurysms. The Annals of Thoracic Surgery. 2007;83(2):S862–S864. Available from: https://dx.doi.org/10.1016/j.athoracsur.2006.10.088.

[2] Mancia G, Fagard R, Narkiewicz K, Redón J, Zanchetti A, Böhm M. Authors/Task Force. 2013 ESH/ESC guidelines for the management of arterial hypertension: The Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). Eur Heart J. 2013;34:2159–219.

[3] Jovin IS, Duggal M, Ebisu K, Paek H, Oprea AD, Tranquilli M, et al. Comparison of the Effect on Long-Term Outcomes in Patients With Thoracic Aortic Aneurysms of Taking Versus Not Taking a Statin Drug. The American Journal of Cardiology. 2012;109(7):1050–1054. Available from: https://dx.doi.org/10.1016/j.amjcard.2011.11.038.

[4] Brown LC, Powell JT. Risk Factors for Aneurysm Rupture in Patients Kept Under Ultrasound Surveillance. Annals of Surgery. 1999;230(3):289–289. Available from: https://dx.doi.org/10.1097/00000658-199909000-00002.

[5] Thompson RW, Geraghty PJ, Lee JK. Abdominal Aortic Aneurysms: Basic Mechanisms and Clinical Implications. Current Problems in Surgery. 2002;39(2):110–230. Available from: https://dx.doi.org/10.1067/msg.2002.121421.
[6] Elefteriades JA; 2002.
[7] Zwaka TP, Hombach V, Torzewski J. C-Reactive Protein–Mediated Low Density Lipoprotein Uptake by Macrophages. Circulation. 2001;103(9):1194–1197. Available from: https://dx.doi.org/10.1161/01.cir.103.9.1194.
[8] Wong JE, Zajac P, Aneurysms A. Clinical Guidelines for Primary Care Physicians. Osteopathic Family Physician. 2018;10(1).
[9] Mortality results for randomised controlled trial of early elective surgery or ultrasonographic surveillance for small abdominal aortic aneurysms. The UK Small Aneurysm Trial Participants. Lancet. 1998;352:1649–55.
[10] Frederick JR, Woo YJ; 2012.
[11] Lindsay ME, Dietz HC. Lessons on the pathogenesis of aneurysm from heritable conditions. Nature. 2011;473(7347):308–316. Available from: https://dx.doi.org/10.1038/nature10145.
[12] Ashton HA, Buxton MJ, Day NE, Kim LG, Marteau TM, Scott RA, et al. Multicentre Aneurysm Screening Study Group The Multicentre Aneurysm Screening Study (MASS) into the effect of abdominal aortic aneurysm screening on mortality in men: a randomised controlled trial. Lancet. 2002;360(9345):1531–1540.
[13] Wang SW, Huang YB, Huang JW, Chiu CC, Lai WT, Chen CY. Epidemiology, clinical features, and prescribing patterns of aortic aneurysm in Asian population from. Medicine. 2005;94(41).
[14] Abdul-Hussien H, Hanemaaijer R, Kleemann R, Verhaaren BFJ, van Bockel JH, Lindeman JHN. The pathophysiology of abdominal aortic aneurysm growth: Corresponding and discordant inflammatory and proteolytic processes in abdominal aortic and popliteal artery aneurysms. Journal of Vascular Surgery. 2010;51(6):1479–1487. Available from: https://dx.doi.org/10.1016/j.jvs.2010.01.057.
[15] Andersen CB, Lindholt JS, Urbonavicius S, Halekoh U, Jensen PS, Stubbe J, et al.. Abdominal Aortic Aneurysms Growth Is Associated With High Concentrations of Plasma Proteins in the Intraluminal Thrombus and Diseased Arterial Tissue. Ovid Technologies (Wolters Kluwer Health); 2018. Available from: https://dx.doi.org/10.1161/atvbaha.117.310126.
[16] Sin DD, Man SFP. Why Are Patients With Chronic Obstructive Pulmonary Disease at Increased Risk of Cardiovascular Diseases? Circulation. 2003;107(11):1514–1519. Available from: https://dx.doi.org/10.1161/01.cir.0000056767.69054.b3.
[17] Chung KF, Adcock IM. Multifaceted mechanisms in COPD: inflammation, immunity, and tissue repair and destruction. European Respiratory Journal. 2008;31(6):1334–1356. Available from: https://dx.doi.org/10.1183/09031936.00018908.
[18] Clouse WD, John W Hallett J, Schaff HV, Gayari MM, Illstrup DM, III LJM. Improved Prognosis of Thoracic Aortic Aneurysms. JAMA. 1998;280(22):1926–1926. Available from: https://dx.doi.org/10.1001/jama.280.22.1926.
[19] Ando K, Kaneko N, Doi T, Aoshima M, Takahashi K; 2014.
[20] Baty F, Putora PM, Isenring B, Blum T, Brutsche M. Comorbidities and Burden of COPD: A Population Based Case-Control Study. PLoS ONE. 2013;8(5):e63285–e63285. Available from: https://dx.doi.org/10.1371/journal.pone.0063285.
[21] Svensson LG, Crawford ES, Hess KR. Experience with 1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg. 1993;17:357–68.
[22] Juvonen T, Ergin MA, Galla JD, Lansman SL, Nguyen KH, McCullough JN, et al. Prospective Study of the Natural History of Thoracic Aortic Aneurysms. The Annals of Thoracic Surgery. 1997;63(6):1533–1545. Available from: https://dx.doi.org/10.1016/s0003-4975(97)00414-1.
[23] Multicentre aneurysm screening study (MASS): cost effectiveness analysis of screening for abdominal aortic aneurysms based on four year results from randomised controlled trial. BMJ. 2002;325(7373):1135–1142.
[24] Danyi P, Elefteriades JA, Jovin IS. Medical Therapy of Thoracic Aortic Aneurysms. Ovid Technologies (Wolters Kluwer Health); 2011. Available from: https://dx.doi.org/10.1161/circulationaha.110.006486.
[25] Powell J, Greenhalgh RM. Cellular, enzymatic, and genetic factors in the pathogenesis of abdominal aortic aneurysms. Elsevier BV; 1989. Available from: https://dx.doi.org/10.1016/0741-5214(89)90049-9.
[26] Meijer CA, Kokje VBC, van Tongeren RBM, Hamming JF, van Bockel JH, Möller GM, et al. An Association between Chronic Obstructive Pulmonary Disease and Abdominal Aortic Aneurysm beyond Smoking. European Journal of Vascular and Endovascular Surgery. 2012;44(2):153–157. Available from: https://dx.doi.org/10.1016/j.ejvs.2012.05.016.
[27] Stein LH, Berger J, Tranquilli M, Elefteraides JA. Effect of Statin Drugs on Thoracic Aortic Aneurysms. The American Journal of Cardiology. 2013;112(8):1240–1245. Available from: https://dx.doi.org/10.1016/j.amjcard.2013.05.081.
[28] Lin TC, Shih CC. Review of treatment for thoracoabdominal aortic aneurysm, and the modern experience of multibranched endograft in Taiwan. Acta Cardiologica Sinica. 2017;33(1):1–1.
[29] Allison MA, Criqui MH, Wright CM. Patterns and Risk Factors for Systemic Calcified Atherosclerosis. Arteriosclerosis, Thrombosis, and Vascular Biology. 2004;24(2):331–336. Available from: https://dx.doi.org/10.1161/01.atv.0000110786.02097.0c.
[30] Safi HJ, Miller CC. Spinal cord protection in descending thoracic and thoracoabdominal aortic repair. Elsevier BV; 1999. Available from: https://dx.doi.org/10.1016/s0003-4975(99)00397-5.
[31] Flessenkaemper I, Loddenkemper R, Roll S, Enke-Melzer K, Wurps H, Bauer T. Screening of COPD patients for abdominal aortic aneurysm. International Journal of Chronic Obstructive Pulmonary Disease. 2015;10:1085–1085. Available from: https://dx.doi.org/10.2147/copd.s81439.
[32] Pasceri V, Willerson JT, Yeh ETH. Direct Proinflammatory Effect of C-Reactive Protein on Human Endothelial Cells. Circulation. 2000;102(18):2165–2168. Available from: https://dx.doi.org/10.1161/01.cir.102.18.2165.
[33] Estrera AL, Miller CC, Chen EP, Meada R, Torres RH, Porat EE, et al. Descending Thoracic Aortic Aneurysm Repair: 12-Year Experience Using Distal Aortic Perfusion and Cerebrospinal Fluid Drainage. The Annals of Thoracic Surgery. 2005;80(4):1290–1296. Available from: https://dx.doi.org/10.1016/j.athoracsur.2005.02.021.

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