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Cardiovascular Surgery and Interventions

Case Report Open Access

Ten-year follow-up of coronary revascularization in a pediatric case with homozygous familial hypercholesterolemia

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ABSTRACT

Homozygous familial hypercholesterolemia can cause coronary artery stenosis during childhood. Herein, we report a six-year-old boy who underwent coronary artery bypass grafting and liver transplantation at the same clinic four months later.

Keywords: Coronary artery bypass grafting, Familial hypercholesterolemia; liver transplantation; pediatric coronary bypass.

Homozygous familial hypercholesterolemia (HFH) is a rare, but a serious genetic disorder with a mutation in the low-density lipoprotein (LDL) receptor.[1] It is characterized by severely elevated serum LDL levels and cutaneous, tendinous xanthomas, and an increased risk of premature coronary artery disease (CAD), which may necessitate coronary artery bypass grafting (CABG) in children.

Herein, we report a six-year-old boy who underwent CABG and liver transplantation at the same clinic four months later.

CASE REPORT

A six-year-old boy was admitted to our clinic with a history of chest pain in May 2006. He was diagnosed with HFH six months ago, and was scheduled for liver transplantation. He received medical therapy including atorvastatin 40 mg daily and ezetimibe 10 mg daily. With the treatment, total cholesterol was 768 mg/dL, LDL cholesterol 679 mg/dL, high-density lipoprotein (HDL) cholesterol 24 mg/dL, and triglycerides 85 mg/dL in the blood tests. Other parameters were within normal ranges. In the preoperative period, stable angina was detected and coronary angiography was performed. There was severe stenosis at the proximal left main coronary artery (LMCA) (90%) and multiple mild stenotic lesions at the other coronary arteries (Figure 1). Thallium myocardial scintigraphy was performed and myocardial ischemia was seen only at the left anterior location. Echocardiography showed normal ejection fraction with 65% and aortic valve and aortic wall thickening without gradient. Carotid arteries were examined with Doppler ultrasound, and there were no evidence of stenosis, despite small atheromatous plaque formations.

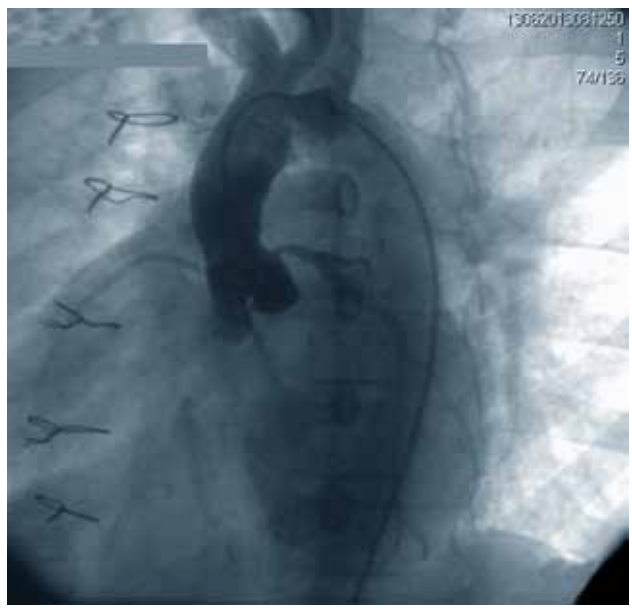

Figure 1. Preoperative aortography.

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Myocardial revascularization was indicated. The patient was scheduled for CABG. A written informed consent was obtained from his parents. After general anesthesia and via a midline incision, the chest was opened and the left internal mammary artery (LIMA) was harvested. Using on-pump technique, we bypassed the LAD with LIMA. Due to the small size of the thorax and the presence of ischemia only at the anterior part of the myocardium, a bypass to circumflex artery was not performed. Surgery was completed without any complication.

The postoperative course in the intensive care unit was uneventfully and he was discharged home on Day 5. He was prescribed acetylsalicylic acid 80 mg daily, atorvastatin 40 mg daily, and ezetimibe 10 mg daily with diet modification. The cardiac status was checked for the transplantation surgery one month after CABG. We performed a stress test and found no ischemic changes. At three months of follow-up, total cholesterol was 868 mg/dL, HDL cholesterol 29 mg/dL, LDL cholesterol 810 mg/dL, and triglycerides 121 mg/dL. His chest pain resolved.

The patient was, then, referred to the liver transplantation unit three months after the operation. The liver transplantation was done at four months with living donor transplantation technique.

Ten days after transplantation surgery, total cholesterol was 215 mg/dL, HDL cholesterol 20 mg/dL, LDL cholesterol 152 mg/dL, and triglycerides 214 mg/dL. In the first year of follow-up, xanthomas regressed.

After the transplantation, his serum cholesterol level was 150 mg/dL, and he did not suffer from any complaint related to cardiac ischemia.

Based on his outpatient follow-up results, the patient was stable. Treadmill tests were performed with 12 month intervals to evaluate the ischemia of the myocardium; however, there was no evidence of ischemia. In June 2013, we performed coronary angiography to control the graft (Figure 2). The LIMA-to-LAD anastomosis was stable without any evidence of a new cardiac disease. In addition, there were intimal thickening at the abdominal aorta and its main branches which did not cause any clinical complaint (Figure 3).

DISCUSSION

Homozygous familial hypercholesterolemia is an autosomal dominant disorder which is associated with accelerated atherosclerosis and the development of multiple tendon xanthomas.[1] The primary challenge is LDL receptor disorder in the liver that is why liver transplantation is indicated, if medical therapy fails to lower the blood lipid levels. Patients with HFH carries out a high risk to develop CAD usually in the

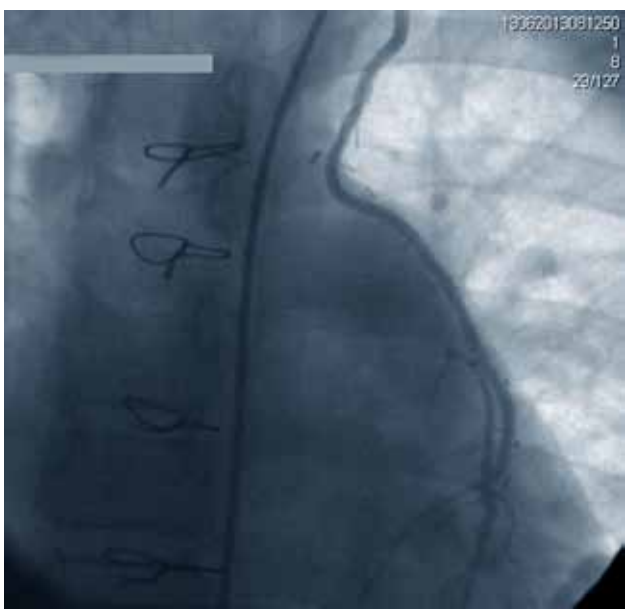

Figure 2. Postoperative control angiography.

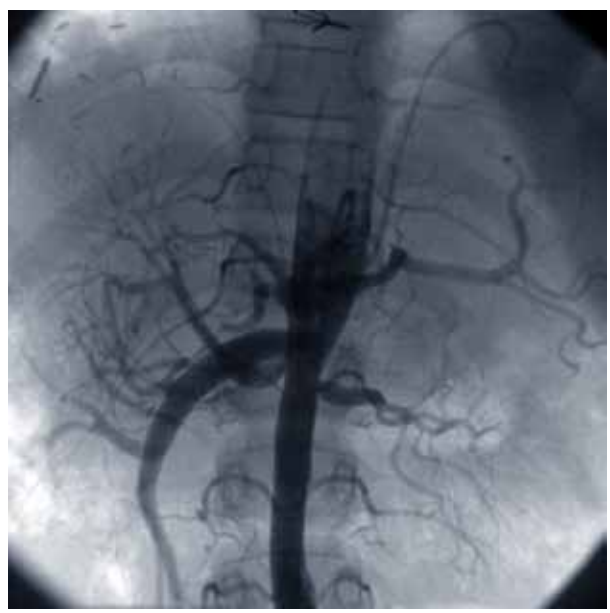

Figure 3. Abdominal aortography.

second or third decades of life; however, very early onset of CAD in childhood has been also reported in the literature.[1] The youngest child reported for CABG with familial hypercholesterolemia was a seven-year-old boy[2] However, our case is nominated to be the youngest patient for CABG surgery in the literature.

Recently, the use of arterial conduits for coronary problems in infants and children has been widely adopted with excellent long-term patency and growth in children.[3,4] Variations in the adaptation to growth of children have been shown between the arterial and venous grafts with thoracic artery growing in a proportion to somatic growth, while saphenous vein grafts tend to course in a more linear way without any increase in the length or diameter.[5-7] In our case, the choice of LIMA depended on its growth potential. However, the small size of the thorax, LIMA and coronary arteries makes it difficult to position the thorax, to harvest LIMA, and to anastomose it to a small coronary artery. For multiple coronary artery anastomoses, the right internal mammary artery can be also used either as T-graft or as separately.

Very high blood LDL levels should be controlled in patients with HFH via drugs, plasmapheresis, or liver transplantation.[9] In patients refractory to medical treatments, transplantation is the most effective and most durable way to control LDL levels and its complications,[10] as the problem is disorder of LDL receptors at the liver. A new liver with a normal working LDL receptor allows the patient to manipulate the blood LDL level.

The first description of the living-related liver transplantation procedure procured from a living donor (the mother) and transplanted in a child dates back to 1988.[8,9] Living-related liver transplants soon came to account for a substantial number of pediatric cases performed in many centers throughout the world, and the only possibility for liver transplants in countries, where cadaveric organ procurement was not allowed until a few years ago.

In our case, the absence of the lateral wall ischemia of the myocardium gave us the courage not to do bypass to the circumflex artery, since we did not prefer using neither a venous graft nor a right internal mammary artery graft at such a young case for the stretching potential of these grafts as the child grows up. On the other hand, there were soft atheromatous plaques at the aorta which may cause distally embolism. We cannulated aorta, as it would be difficulty to do bypass surgery with off-pump techniques in a child; however, we avoided an additional manipulation with proximal anastomosis which we did not consider that the patient needed it, due to the lack of the lateral wall ischemia.

In conclusion, coronary revascularization in childhood is a rare procedure. The patients with homozygous familial hypercholesterolemia must be followed closely for the development of coronary atherosclerosis particularly in patients scheduled for liver transplantation. The surgical results for cardiac revascularization are usually satisfactory in ischemic patients. However, long-term survival of the patients depends on strictly controlling lipid levels, due to the ongoing nature of the disease. Our case is the youngest patient with homozygous familial hypercholesterolemia undergoing coronary artery bypass grafting procedure with the longest follow-up.

Declaration of conflicting interests

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A left atrial thrombus forming an antibioma: a rare entity

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ABSTRACT

An antibioma is defined as a hard edematous swelling containing sterile pus following treatment of an abscess with long-term antibiotherapy. Abscess of the heart, as any abscess elsewhere in the body, develops in two principal ways: *(i)* by dissemination from a distant infectious focus *(ii)* by direct extension of an infectious process located in the heart itself. Even the fresh blood clot in the heart may become infected. Herein, we report the first case of the left atrial thrombus forming an antibioma following the Lutembacher's repair.

Keywords: Antibioma; Lutembacher's syndrome; mitral valve replacement; thrombus left atrial appendage.

Antibioma, which is a rare anomaly of the heart, is defined as a hard edematous swelling containing sterile pus following treatment of an abscess with long-term antibiotherapy. In the literature, a left atrial (LA) antibioma has never been reported. Herein, we present an unusual case of the LA thrombus forming an antibioma following the Lutembacher's repair.

CASE REPORT

A 28-year-old female was admitted with complaints of dyspnea on exertion. She was in the New York Heart Association Class II (NYHA II) for four years. Through a detailed examination, she was diagnosed with Lutembacher's syndrome. A two-dimensional echocardiography revealed ostium secundum atrial septal defect and severe mitral stenosis. There was no history of pulmonary tuberculosis or thrombus in the left atrium or appendage. Ostium secundum atrial septal defect was closed with an autologous-untreated pericardial patch and mitral valve was replaced with a 29-tilting disc mechanical prosthetic heart valve. Surgery was uneventful. On postoperative Day 2, the patient developed high-grade fever (38 °C). Blood culture showed a heavy growth of *Streptococcus* which was treated according to *Streptococcus*-sensitive antibiotics. She was extubated in the fifth postoperative day. The patient was on an anticoagulant with international normalized ratio (INR) set at 2.5 to 3.0. In the ninth postoperative day, transthoracic echocardiography showed a large LA thrombus (Figure 1), despite oral anticoagulants and target INR. She was, then, put on heparin; however, repeated echocardiography did not show any improvement. The patient developed tachypnea and chest X-ray and computed tomography (CT) scan showed patchy confluent areas of consolidation in both the lungs, more dense in the lower lobes. Thoracic CT also showed mosaic perfusion along with the areas of ground-glass changes in both the lungs with an organized thrombus in the posterior part of the LA (Figure 2) measuring 4x3 cm in size without a thrombus in the LA appendage. The patient was scheduled for LA clot removal. Intraoperatively, there was a large, 4x3x3 cm, well-defined globular mass with very thin glistening covering of the left atrium just beneath the right inferior pulmonary vein away from the prosthetic valve and the patch (Figure 3). During manipulation, the mass ruptured and turbid brown-colored fluid came out. The wall of the mass was excised and pus was drained. The pus was sent for culture, which did not grow any organism initially. The LA was cleaned with normal saline and vancomycin. Following the redo surgery, the patient required tracheostomy due to prolonged intubation and she continued to have spikes of fever with blood culture growing *Candida albicans*. On Day 118 days of the intensive care unit (ICU) stay, the patient developed multi-organ failure secondary to

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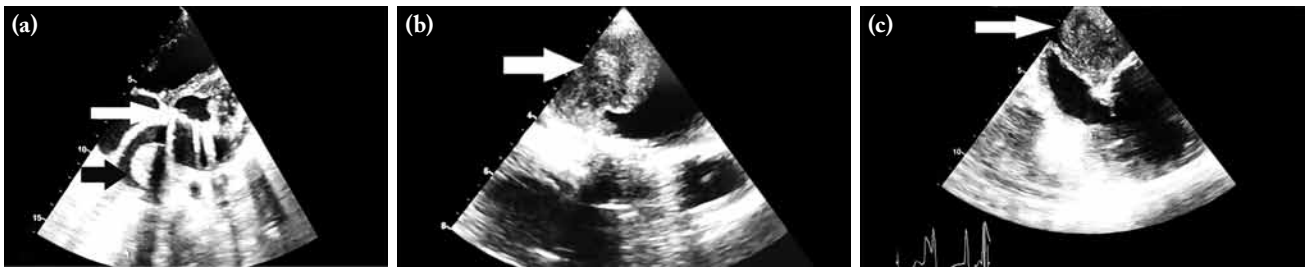

Figure 1. Echocardiography. **(a)** Transthoracic echocardiography showing the prosthetic valve (white arrow) and the left atrial thrombus (black arrow). **(b)** Transesophageal echocardiography showing the left atrial thrombus (white arrow). **(c)** Transesophageal echocardiography showing the left atrial thrombus (white arrow).

fungal septicemia. Histopathologica report suggested an infected thrombus and fluid culture was reported as the growth of *Stenotrophomonas maltophilia*.

DISCUSSION

Antibioma of the heart is a rare occurrence which has not yet been documented in the literature. It is defined as a hard edematous swelling containing sterile pus following the treatment of an abscess with long-term antibiotherapy.[3] Abscess of the heart, as any abscess elsewhere in the body, develops due to *(i)* the dissemination from a distant infectious focus, *(ii)* the direct extension of an infectious process located in the heart itself,[1] or *(iii)* fresh blood clot in the heart becoming infected, as in our case.[1-3] Prosthetic valve

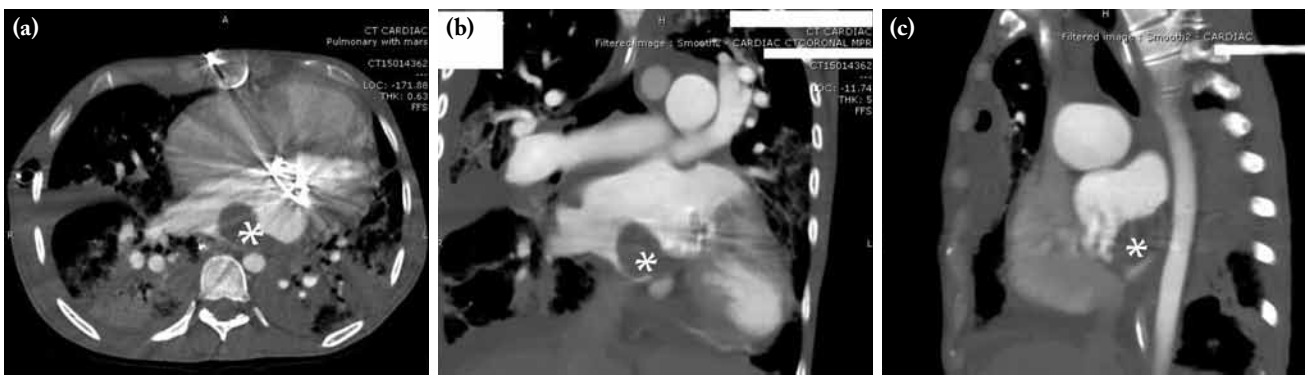

Figure 2. Computed tomography scans. **(a)** A coronal section showing prosthetic valve and the left atrial thrombus (*). **(b)** A sagittal section showing the left atrial thrombus (*). **(c)** A lateral view showing the left atrial thrombus (*).

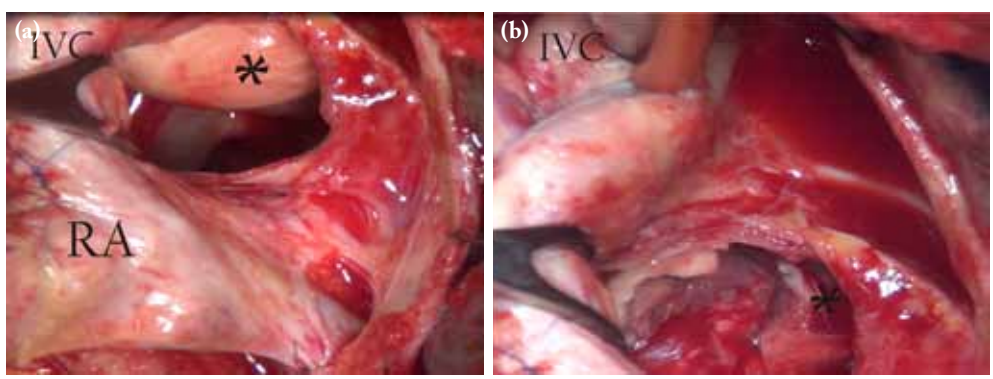

Figure 3. Intraoperative images. **(a)** An intraoperative image showing an opened left atrium and antibioma (*). **(b)** An intraoperative image showing the debrided area of the left atrial antibioma (*).
IVC: Inferior vena cava; RA: Right atrium.

endocarditis can lead to abscess formation; however, it is usually limited to the annulus and may infect the newly formed clot in the vicinity.[2] In our case, we initially suspected of prosthetic valve endocarditis; however, prosthetic valve appeared normal and a new thrombus formed at the posterior wall of left atrium, near the right inferior pulmonary vein. The main cause probably would be *(i)* damage to the endothelium and raw exposed muscle with persistent atrial fibrillation would have become the source for the clot[3] or *(ii)* the atrial septal defect closure with a pericardial patch would have left suture or raw pericardial tissue at the lower most end of the patch, which might have formed the clot which became infected over a period of time due to prolonged ICU stay and hospital-acquired infection, or *(iii)* possibility of very low cannulation of inferior vena cava and deep reinforcement of the suture would form the pocket where the blood became stagnated and formed a thrombus at the lower end of the interatrial septum, in particular. This was probably the cause in our case. In such cases, diagnosis can be obtained by echocardiography and antibioma can be suspected, if the clot is in unusual position, as in our case, and does not respond to intravenous heparin. Computed tomography offers the exact location and the content of the mass. Surgical evacuation thorough normal saline wash with antibiotics is the primary treatment of choice. However, the postoperative course remains stormy and outcome may be dismal.

In conclusion, antibioma of the left atrium is a very rare occurrence with no published literature to date. Therefore, we believe that early recognition and aggressive management may yield satisfactory outcomes.

Declaration of conflicting interests

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Cardiovascular Surgery and Interventions

Case Report Open Access

Idiopathic pulmonary artery aneurysm: is it really idiopathic?

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ABSTRACT

Pulmonary artery aneurysms are rare cardiac abnormalities. Although various factors have been proposed, their etiology and pathophysiology are not well-understood. Herein, we report our surgical approach for a symptomatic pulmonary artery aneurysm in an 11-year-old girl, in whom we detected an intraluminal structure during surgery.

Keywords: Idiopathic; pulmonary artery aneurysm; turbulent flow.

A pulmonary artery (PA) aneurysm is a rare lesion with an approximate incidence of 1/14,000 in autopsies.[1] In the literature, various etiologic causes have been previously reported, of which idiopathic PA aneurysm being the most common.[1] The main complaints of a PA aneurysm include shortness of breath, cough, and chest pain.[2] There is no guideline for surgical intervention in PA aneurysm and management still remains controversial. In general, early surgical intervention is recommended for symptomatic cases and large PA aneurysm sizes to avoid possible fatal ruptures.[2] Herein, we report a rare case of a PA aneurysm and its successful management with surgical intervention. Besides, we propose a new hypothesis for a cause of a PA aneurysm in our case.

CASE REPORT

An 11-year-old female patient was referred to our institution with a complaint of hoarseness and shortness of breath (New York Heart Association [NYHA] Class II) which were progressively increasing with the past six months. There was no history of cough, orthopnea, hemoptysis, fever, chest pain, syncope or trauma. Physical examination findings were completely normal, except grade III/IV harsh systolic murmur over the pulmonic area. Electrocardiography showed sinus rhythm. Chest X-ray demonstrated an aneurysmal main PA segment without cardiomegaly and with normal lung fields. Transthoracic echocardiography (TTE) revealed a main PA aneurysm without any other cardiac abnormalities. The patient was also examined for connective tissue disorders, which produced negative results. Cardiac magnetic resonance imaging (MRI) was done to further evaluate the aneurysm and showed a lesion with 3 cm fusiform dilatation of the main PA towards the left PA above the pulmonary valve. Distal main PA and branch PAs were normal in size (Figure 1). During catheterization, maximal diameter was 38 mm (X-score: 4.9) and the main PA pressure was 26 mmHg (Figure 2). Interestingly, the filling of the right PA with contrast agent was delayed and relatively slower than that of left PA. However, it was initially considered an artefact, as there was no accompanying cardiac or vascular abnormality, as assessed by TTE, MRI and angiography.

As the patient was symptomatic with a main PA Z-score of 4.9, we performed elective surgery. A mid-line sternotomy was performed. The main PA was aneurysmatic towards the left PA. Cardiopulmonary bypass under mild hypothermia was initiated. Vertical pulmonary arteriotomy revealed no thrombi or vegetations. The right ventricular outflow tract, pulmonary valve, and branch PAs were normal. However, an intraluminal membranous flap-like structure which partially obstructed the right PA origin was detected. The membrane and aneurysmatic arterial segment were resected. Redundant arterial wall was reconstructed, according to an appropriate Hegar

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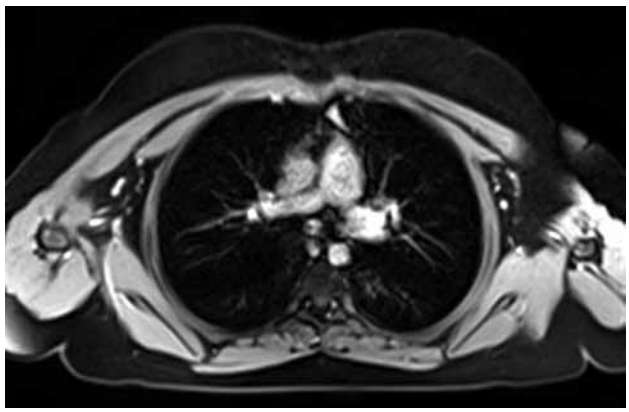

Figure 1. A coronal section of cardiac magnetic resonance imaging showing fusiform dilatation of main pulmonary artery towards left pulmonary artery and normal-size branch pulmonary arteries.

dilator size. Postoperative course was uneventful. Prior to discharge, repeated echocardiography showed no gradient across the pulmonary valve and the patient was, then, was discharged in the fifth postoperative day. Histopathological examination of the arterial wall revealed a lost smooth muscle layer which was replaced by diffuse mural fibrosis. Elastic fibers were disrupted and fragmented. There were no vasculopathic changes in the adventitia (Figure 3).

DISCUSSION

Aneurysms of the main PA are rare clinical entities with an unknown etiology and pathogenesis.[1] Almost half of the cases have accompanying congenital heart disease, the most common being patent ductus arteriosus.[3] Behçet's and Marfan syndromes, injuries, pulmonary hypertension, infections, and idiopathic factors are the other common underlying causes of these aneurysms.[3] The diagnosis should be based on TTE and cardiac catheterization findings to ascertain the extent of the aneurysm and rule out accompanying intracardiac pathologies.[2,3] In addition, MRI is an essential diagnostic tool, as it is noninvasive and optimal for the detection of intimal flaps.[2,3] Furthermore, in case of suspected connective tissue disorders, rheumatologic panel should be tested. Our case suffered from dyspnea and hoarseness. The first was reported as the most common symptom of patients with a PA aneurysm, while the latter is a rare symptom. In a PA aneurysm, hoarseness resulted from stretching of the recurrent laryngeal nerve.[2,3] For diagnostic work-up, several imaging modalities were used, including TTE, catheterization, and MRI; however, none showed an accompanying abnormality. Furthermore, rheumatologic panel was completely normal.

The clinical course of large aneurysms is unpredictable. According to the Laplace's law, wall tension is directly proportional to the radius of the vessel and intraluminal pressure, whereas it is inversely related to the wall thickness.[4] Thus, higher intraluminal pressures with thinning of the vessel wall lead to an increased risk of dilatation and rupture.[4] Also, it may cause massive hemoptysis.[2] In the literature, there is still a debate on the optimal size of the main

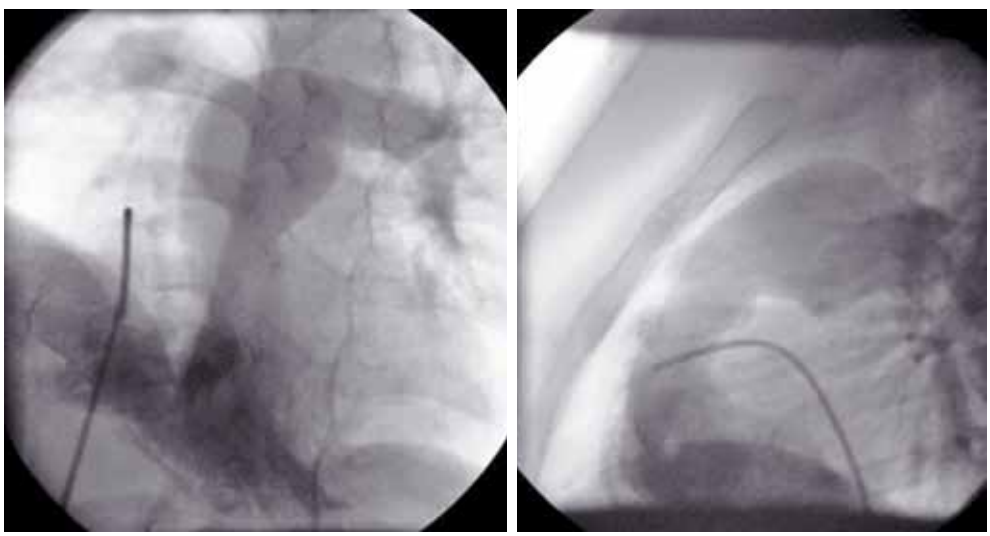

Figure 2. Anteroposterior and left lateral views during cardiac catheterization showing a main pulmonary artery aneurysm.

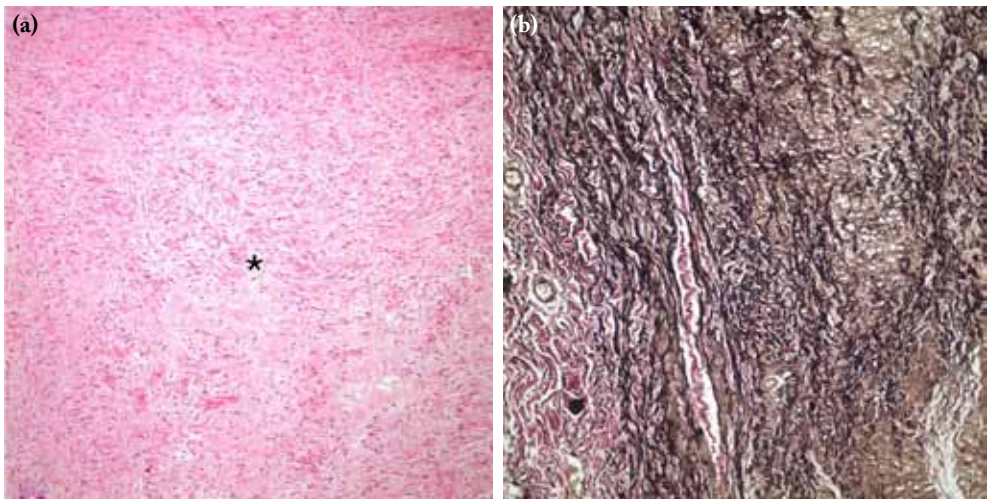

Figure 3. Histopathological examination of the arterial wall. **(a)** Loss of smooth muscle layer (*) (H-E x 10). **(b)** Disrupted and fragmented elastic fibers (black stained) (Verhoeff's stain x 20).

PA aneurysms for surgical indication. On the other hand, it is commonly accepted that symptomatic aneurysms should be surgically treated to prevent the risk of rupture.[2] As there is no clear cut-off level of the aneurysm size for surgery, we preferred using X-score (4.9) as a surgical indication. Therefore, we believe that using X-scores may be crucial in timing of surgery.

Furthermore, various surgical techniques have been described to date, including aneurysm plication, pericardial patch reconstruction, and graft interposition.[3] Review of case series revealed that these techniques were equally effective.[3] In our case, we used arterial resection with reduction arterioplasty to preserve the intact pulmonary valve function and integrity of the branch PAs.

On the other hand, an interesting finding in our case was the existence of an intraluminal membranous flap-like structure which partially obstructed the right PA origin. Slow, late-phase filling of the right PA during preoperative angiography was most likely related to this structure. Unfortunately, preoperative MRI did not show any flap-like structure in PAs. We hypothesize that membrane around the origin of the right PA led to turbulent flow which was injuring the internal wall of left PA. Consistent with this hypothesis, we noticed that the main PA aneurysm was leaning towards the left PA. Previous reports showed that different flow dynamics and disturbed flow conditions might damage the endothelium, thereby, offering the initial step to the degeneration of the arterial wall.[4] Consequently, they may cause aneurysm formation, as turbulence significantly increases the pressure and fluid shear stress over the arterial wall. Histopathological findings also supported our hypothesis. In previously reported cases, normal histology was reported.[1,5] However, we found a complete loss of the muscle layer and replacement fibrosis without accompanying adventitial alterations. Therefore, these alterations may have occurred as a result of turbulent flow caused by the membranous structure.

In conclusion, despite numerous etiologic factors of pulmonary artery aneurysms have been identified, surgeons should keep in mind that some biomechanical causes may be the underlying factors of aneurysms in so-called idiopathic cases.

Declaration of conflicting interests

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Cardiovascular Surgery and Interventions

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A giant benign epicardial lipomatosis of the left ventricle

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ABSTRACT

Fatty masses of the heart are relatively uncommon. Herein, we report an extremely rare case of a giant benign epicardial lipomatosis arising from the left ventricle. A 26-year-old female presented with atypical chest pain and easy fatigability for the past six months. Chest X-ray showed an increased cardiothoracic ratio. Echocardiography revealed a large homogenous mass in the pericardial space near the lateral surface of the left ventricle. Computed tomography and magnetic resonance imaging of the chest showed a large fat-density mass with well-defined margins, arising from the left ventricle. The patient underwent surgical removal of the mass without cardiopulmonary bypass. She recovered well and discharged in the seventh postoperative day.

Keywords: Benign cardiac tumor; cardiac malignancy; epicardial lipomatosis; left ventricle.

Primary cardiac tumors account for 5 to 10% of all neoplasms of the heart and pericardium with an incidence ranging from 0.0001 to 0.05% in autopsies.[1] Lipomas are the second most common benign cardiac tumors.[2] These tumors are often asymptomatic in nature and usually detected incidentally, mostly during autopsies.[1-3] Although a lipoma is a benign tumor, it can lead to compression of cardiac chambers and cause life-threatening complications, when the tumor size increases.[1-6] Herein, we report a rare case of a giant benign epicardial lipomatosis arising from the left ventricle.

CASE REPORT

A 26-year-old female had atypical chest pain and easy fatigability for the past six months. Chest X-ray showed a cardiothoracic ratio of 70%. Echocardiography (ECHO) showed a large homogenous, echogenic mass in the pericardial space located along the lateral surface of the heart, arising from the epicardium and extending posterior, anterolateral surfaces of the left ventricle (LV) including the apex with minimal pericardial effusion (Figure 1a). The biventricular function was good with no regional wall motion abnormalities. Computed tomography (CT) of the chest showed a large fat-density mass, extending from the arch of the aorta superiorly to the dome of the diaphragm inferiorly in the left hemithorax. The mass had a well-defined lateral margin limited by the pericardium peripherally and its medial border was encasing the left atrium, LV and a part of right ventricle (RV). The mass also encased the left anterior descending artery and derived its blood supply from the branches of the left coronary artery (Figure 1b). The tumor was found to be causing a mass effect on the left lower lobe of the lung and its vasculature. Cardiac magnetic resonance imaging (MRI) showed an abnormal soft tissue enhancement in the anterolateral surface of the heart occupying the anterolateral, posterior and inferior surfaces of the LV and anteriorly extending over the RV (Figure 1c). The mass was well-defined and had the same signal intensity as subcutaneous fat, in all the images and occupying almost the whole of the left hemithorax (Figure 1d). The mass was in close contact with the cardiac chambers, but not compressing any chambers or great vessels.

Surgical operation

The patient was taken up for surgery via median sternotomy without cardiopulmonary bypass (CPB). During opening the pericardium, a very large yellowish tumor with a smooth surface was detected covering the

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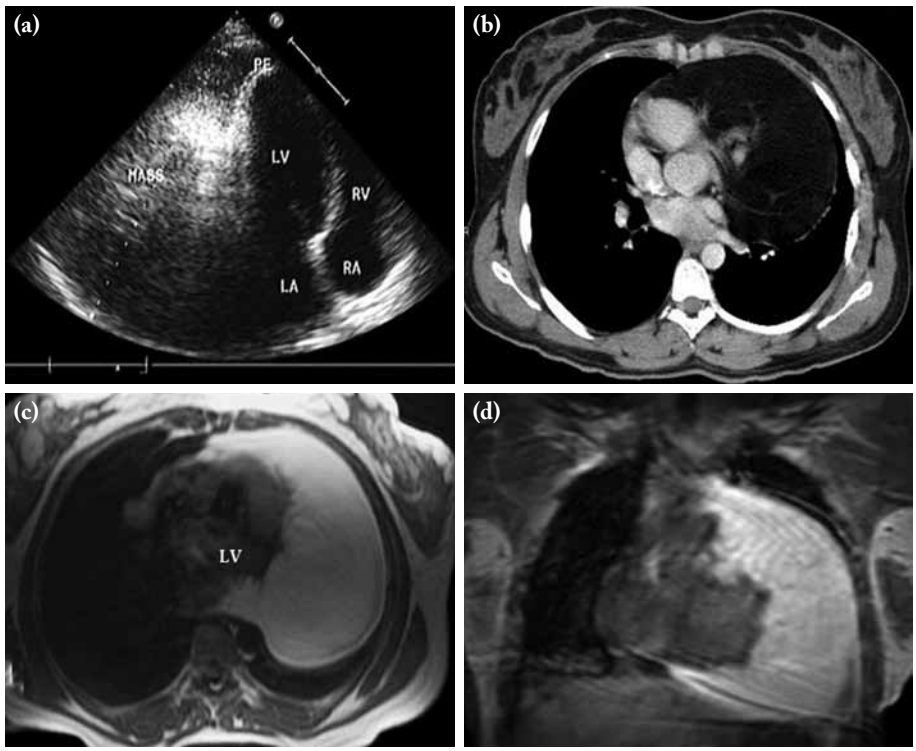

Figure 1. **(a)** Echocardiography showing a homogenous echogenic mass in the pericardial space along the lateral surface of the heart. **(b)** A computed tomography scan showing a large fat density mass with major vessels. **(c)** A magnetic resonance imaging scan showing a large fatty mass arising from the anterolateral and posterior surfaces of the left ventricle. **(d)** A magnetic resonance imaging scan showing the mass occupying almost the whole left hemithorax.

LV: Left ventricle; RV: Right ventricle; RA: Right atrium; LA: Left atrium; PE: Pericardial effusion.

entire heart and bulging into the wound, which was unable to be easily lifted out of the pericardial sac (Figure 2a). It had no definite stalk or pedicle and was firmly adherent to the LV. The tumor was highly vascular and received its blood supply from the left anterior descending artery and lots of feeding vessels arose directly from the epicardium of the LV. Major vascular branches supplying the tumor were ligated and clipped (Figure 2b). While the major portion of the tumor was excised, a thin rim of the tissue was left behind (Figure 2c), due to its firm adherence to the LV myocardium & extreme vascularity. The excised specimen weighed around 850 g. The postoperative course was uneventful and the patient was discharged in the seventh postoperative day. Histopathological examination showed the presence of fibroadipose tissues which revealed mature adipocytes arranged in lobules without cellular atypia, suggestive of lipomatosis (Figure 2d).

DISCUSSION

Benign tumors of the heart were rare. Lipomas are the second most common benign cardiac tumors.[2] Cardiac lipomas represent about 10% of all cardiac tumors and are thought to originate either from the subendocardium, subpericardium or the myocardium.[3] The majority of the cardiac lipomas are subendocardial or epicardial, and only 25% are found in the myocardium.[1-4] Clinical presentation usually depends on the size and location of the tumor. They usually originate from the epicardial fat tissue and grow slowly into the pericardial sac; in case of asymptomatic cases, they often achieve enormous dimensions before producing any symptoms.[1-4] Many tumors are clinically silent, being found only during necropsy or on routine chest X-rays.[1-4] These tumors cause symptoms through compression of the heart. Anginal pain due to coronary arterial compression, atrial arrhythmias,

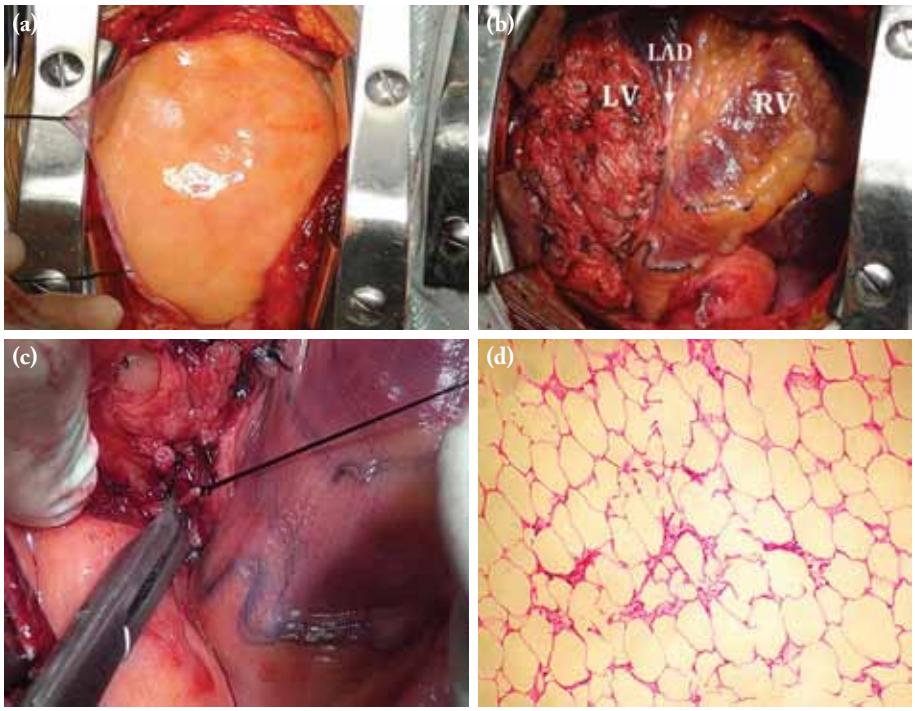

Figure 2. **(a)** An intraoperative view showing the mass just after opening the pericardium. **(b)** Major vessels supplying the tumor which were ligated and clipped. **(c)** Showing the left ventricle after excision of the major portion of the tumor and normal right ventricle. **(d)** Histopathological examination with a paraffin section of lipomatosis showing mature adipocytes (H-E x 400).

LAD: Left anterior descending artery; LV: Left ventricle; RV: Right ventricle.

and effort intolerance has been also reported.[4] The subendocardial tumors with an intracavitary extension may cause symptoms characteristic of their location; they usually produce early symptoms, such as congestive heart failure, supraventricular or ventricular arrhythmias, syncope, or sudden cardiac death.[5] Chest X-ray usually does not differentiate between pericardial effusion and a subepicardial lipoma. In addition, ECHO accurately defines the location and extent of the mass.[1-4] However, the acoustic appearance of an intra-pericardial lipoma is not diagnostic. Subepicardial lipomas usually appear relatively ECHO-lucent and may be erroneously interpreted as the pericardial fluid.[4] Also, CT and MRI can demonstrate lipomatous tumors.[1-4] The latter can distinguish between lipomas and other tumors, such as liposarcomas. It also provides a clear definition of blood vessels to precisely detect the location, size, and extension of the tumor.[1-4] It also demonstrates the relationship of the tumor to the coronary arteries[1] and estimates the intramyocardial extension. These are important factors in determining tumor resectability and help planning the management approach.[4] Although some epicardial tumors may be removed without CPB, most intramural and intracavitary tumors should be excised with the use of CPB, as cardiac manipulation may release part of the tumor, causing an embolism. Following the first successful removal of an intrapericardial lipoma by Maurer in 1952, several sporadic cases have been reported.[1-6] Benign lipomas can be mostly excised completely with low morbidity and mortality and with excellent long-term results.[1] In the majority of cases, intrapericardial lipomas may be removed without CPB. However, CPB may be required in cases of recurrence and when there is an intracardiac extension. The decision to resect such epicardial lipomatosis is going to be debulking, as it is densely adherent to the heart and major coronary arteries. Off pump debulking allows ease of identifying bleeders which is going to be a much more difficult job on the pump, since the heart is arrested and bleeders would not be seen clearly. Intraoperatively, it is important to excise the entire

tumor along with the pedicle to prevent recurrences. The rate of lipoma recurrence after total and subtotal resection is very low.[6] Our case seems to be exceptional, as this was a giant benign lipomatous tumor without any pedicle directly arising from the LV surface. To the best of our knowledge, this is the first case reported in the literature.

In conclusion, most of the patients with intrapericardial lipomas are asymptomatic for a long time due to the slow growth of the tumor. Imaging modalities such as computed tomography and magnetic resonance imaging are the main diagnostic tools. Surgical resection is necessary to prevent tumor compression syndromes of the heart. Finally, the status of malignancy needs to ruled out and confirmed by histological examination.

Declaration of conflicting interests

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Cardiovascular Surgery and Interventions

How to do it? Open Access

Descending thoracic aorta to femoral bypass grafting for total abdominal aortic occlusion

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ABSTRACT

Descending thoracic aorta to femoral bypass graft is an alternative procedure for revascularization of the lower extremities to conventional techniques, when the abdominal aorta is complete obstructed. In general, this technique is performed by surgeons, when abdomen is unable to be opened due to the presence of an abdominal infection, fistulas, or hostile abdomen. In this article, we present our four-year outcomes of descending thoracic aorta to femoral bypass with cross-femoral bypass in four patients. There was no mortality and all grafts were patent. Descending thoracic aorta to femoral bypass grafting has excellent overall long-term results.

Keywords: Abdominal aortic occlusion; aortofemoral bypass; descending thoracic.

The thoracic aorta to femoral bypass grafting is an ideal procedure for a small subgroup of patients, comprising those with an occluded old aorta to femoral bypass graft, those with a lead-pipe calcified infrarenal aorta which is unsuitable as an inflow source, and those with a hostile abdomen (i.e., ileal conduit, ileostomy or colostomy or a previous aortic graft infection). Complete obstruction of the abdominal aorta at the renal artery level is often a surgical challenge in case of a long-standing thrombosis, short suprarenal aortic space, and extensive periarterial inflammatory reaction. Therefore, a descending thoracic aorta to femoral bypass graft is used as an alternative for revascularization. Herein, we present our four-year outcomes of descending thoracic aorta to femoral bypass grafting with cross-femoral bypass in four patients.

SURGICAL TECHNIQUE

Descending thoracic aorta to femoral bypass grafting was performed in four patients who had a juxtarenal complete obstruction of the abdominal aorta in our hospital (Figure 1). All patients were males with a mean age of 65.5 (range, 59-72) years and they had severe atherosclerotic disease and hypercholesterolemia. None of them was, however, diabetic. They had claudication and ischemic pain of the lower extremity. Two of them were ex-smokers with normal pulmonary function test results [Forced expiratory volume in one second (FEV_1) ≥75]. The patients had no renal disease, and biochemical screening results were normal. Coronary angiography revealed coronary atherosclerosis in only one patient. Coronary artery bypass surgery grafting was performed by off-pump technique to this patient before three months.

A written informed consent was obtained from each patient. Selective intubation with double-lumen tubes were performed under fentanyl anesthesia. The patients were positioned to position the hip flat; however, the torso was slightly rotated to the right. Four incisions were made: a left posterolateral thoracotomy, two groin incisions, and a lateral abdominal incision. The descending thoracic aorta was approached through the thoracotomy at the level of the sixth-seventh intercostal space. The left lung was decompressed by selective intubation, and the aorta was cleanly dissected. Common femoral arteries were obtained via two groin incisions. The psoas were reached through the lateral abdominal incisions and with an extra-peritoneal approach. A retroperitoneal tunnel was, then, fashioned for the passage of the

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graft. Once an 8 mm silver, INTERGARD collagen-coated knitted polyester vascular prosthesis graft was carried from this tunnel, the patient underwent systemic heparinization (100 U/kg). The proximal end of the graft was reached the through the aortic hiatus at the diaphragm level. When the blood pressure was reduced by vasodilators, the descending thoracic aorta was controlled with side-clamp, and an end-to-side anastomosis was performed. The distal end of the graft was fashioned with an end-to-side anastomosis to the left common femoral artery. An 8 mm INTERGARD collagen-coated knitted polyester vascular prosthesis graft was, then, performed through the left femoral to the right femoral crossover bypass. The side-aortic clamp was removed and the grafts were visualized with pulsing. Finally, after the bleeding was controlled, the lung was re-inflated, and the chest was closed in a standard fashion over a chest tube.

There was no postoperative mortality. Except one patient, all were extubated in the operation room without a need for respiratory support. However, one patient required respiratory support for six hours. There were no pleural effusion; however, two patients had pulmonary atelectasis for four days. Oral antiaggregant (acetylsalicylic acid) treatment was started in the postoperative third day, following prophylaxis of low-molecular-weight heparin in the postoperative early period. The mean length of intensive care and hospital stays was two days and eight days, respectively. Distal pulses of the lower extremities were sensed at discharge. The graft failure was not seen within the first month of follow-up. Computed tomography (CT) angiography revealed that all grafts were patent at four years (Figure 2).

DISCUSSION

Bypass grafting which originate from the descending thoracic aorta to femoral arteries are well-described, although they are not commonly used in the first-line setting. The descending thoracic aorta to femoral bypass grafting was first described in 1961 in two cases.[1] The thoracic aorta is a good inflow option, as its exposure is relatively straight forward and this segment is usually free of significant atherosclerosis.[2] Most authors have suggested that the use of the descending thoracic aorta with a retroperitoneal tunnel yields more acceptable operative risks and patency rates. Five year patency rates of the procedure has been reported as 86% in the literature.[3]

Axillobifemoral bypass is the most common first choice as an alternative to aortofemoral incomplete

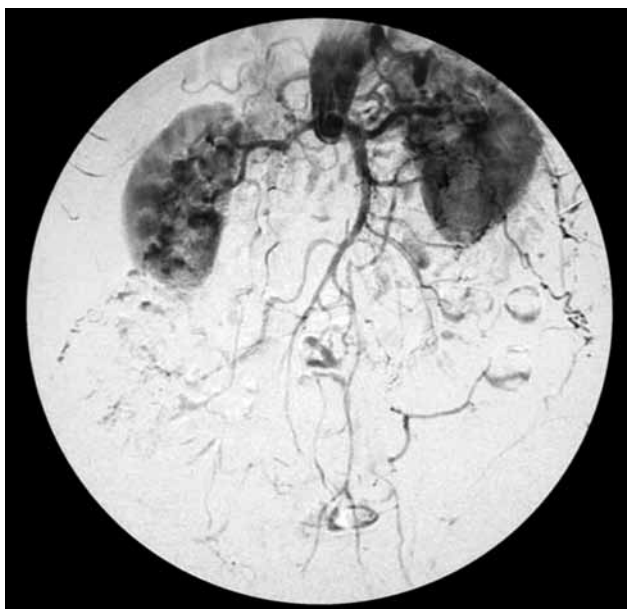

Figure 1. Preoperatively angiography of 60-year-old male patient.

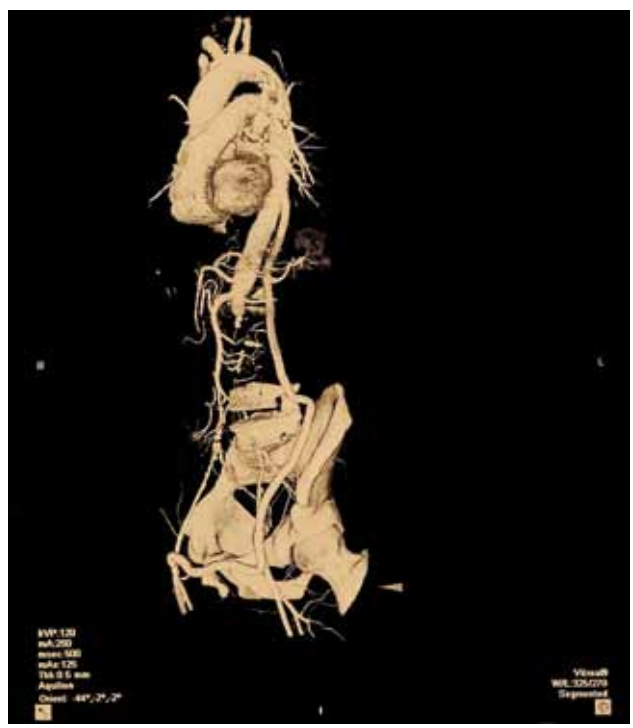

Figure 2. Control computed tomographic angiography of 60-year-old male patient in postoperative first mount.

obstruction of the abdominal aorta at the level of renal arteries.[4] However, the long-term patency is lower, due to the longer graft length with potential compression along the superficial course.[1]

In our technique, we used the descending thoracic aorta, rather than the axillary artery. In most reports, the bifurcated graft is favored to thoracofemoral and femorofemoral crossover grafts.[5,6] We used this graft in only one patient.

Furthermore, thoracic aorta is controlled either with a side-biting clamp or with two completely occluding aortic clamps placed in close proximity to each other. One of the disadvantages of completely occluding clamping is that renal circulation can be impaired, if the time of proximal anastomosis to aorta is hold over, and spinal cord and mesenteric ischemia can develop. Partial aortic control with side-clamping is successful in most cases, although it is not recommended, when the aorta is heavily diseased and calcified. Additionally, it makes the anastomosis more difficult, as the margins of aortotomy become closely opposed.[1] A partial side-clamp was placed in our patients, and it did not complicate the anastomosis.

Baird et al.[6] described ascending aorta to femoral bypass grafting through a median sternotomy. The advantage of this technique is that concomitant coronary revascularization can be performed, when indicated. One of our patients who had coronary artery disease underwent off-pump coronary artery bypass grafting three months before thoracofemoral grafting. Subsequently, we performed revascularization of the lower extremities by descending thoracic aorta to femoral and femorofemoral crossover bypass grafting.

In the present study, all patients were eligible descending thoracic aorta to femoral bypass grafting with excellent overall long-term results. Despite pulmonary difficulties, we believe that this procedure is a satisfactory alternative for the treatment of juxtarenal complete abdominal aortic occlusions, as the thoracic aorta is no more atherosclerotic, and there is high blood flow in this segment to the axillary artery.

In conclusion, endovascular treatment of infrarenal aortic occlusions is feasible in most cases with favorable midterm patency rates. However, due to relatively high procedurally-related complication rates, cautious selection of the patients and taking appropriate preventive measures are necessary to improve the immediate outcomes.[7]

Declaration of conflicting interests

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Cardiovascular Surgery and Interventions

How to do it? Open Access

An alternative peripheral arterial cannulation in minimally invasive and robotic cardiac surgery

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ABSTRACT

Peripheral cannulation is a major step for establishing of cardiopulmonary bypass in minimally invasive cardiac surgery and in certain open chest procedures. Traditionally, a transverse arteriotomy incision or a purse suture over the arterial wall can be used during open cannulation of the femoral artery. Herein, we present an alternative technique for femoral artery cannulation with the Seldinger method, which uses double-pledgeted horizontal sutures on the anterior wall of the femoral artery.

Keywords: Minimally invasive cardiac surgery; peripheral cannulation; robotic cardiac surgery.

Peripheral cannulation is a major step for establishing of cardiopulmonary bypass in minimally invasive cardiac surgery and in certain open chest procedures. Traditionally, a transverse arteriotomy incision or a purse suture over the arterial wall can be used during open cannulation of the femoral artery. Herein, we present an alternative technique for femoral artery cannulation with the Seldinger method, which uses double-pledgeted horizontal sutures on the anterior wall of the femoral artery.

SURGICAL TECHNIQUE

After marking the course of the femoral artery in the groin, an oblique 3 cm incision was made 1 cm above the inguinal crease. The subcutaneous tissue and femoral sheath were opened using a scissor. Dissection was made laterally to the femoral vessels to avoid lymphatic injury and lymphorrhea. Only anterior surfaces of the femoral artery and vein were dissected and exposed (Figure 1). The fascia and surrounding tissue around the vessels were kept intact. Systemic heparinization was made, before the suture placement and cannulation. A double pledgeted U-suture of 5/0 polytetrafluoroethylene was, then, placed at the anterior side of the common femoral artery (Figure 2). Bites were superficially taken through the adventitial layer of the femoral artery. There should be two pledgeted U-sutures on the anterior surface of the femoral artery. The

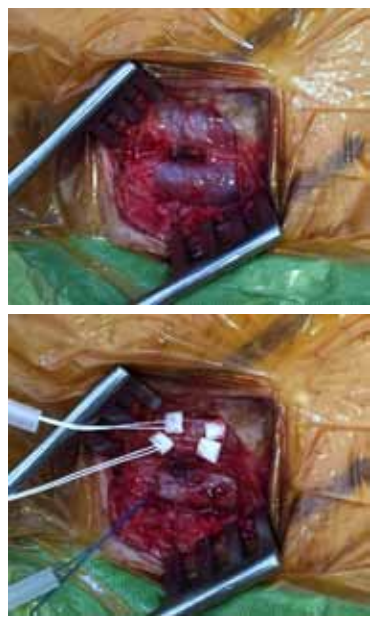

Figure 1. Surgical exposure of common femoral artery and vein (upper image), placement of double-pledgeted polytetrafluoroethylene sutures on the femoral artery and prolene sutures on the common femoral vein (lower image).

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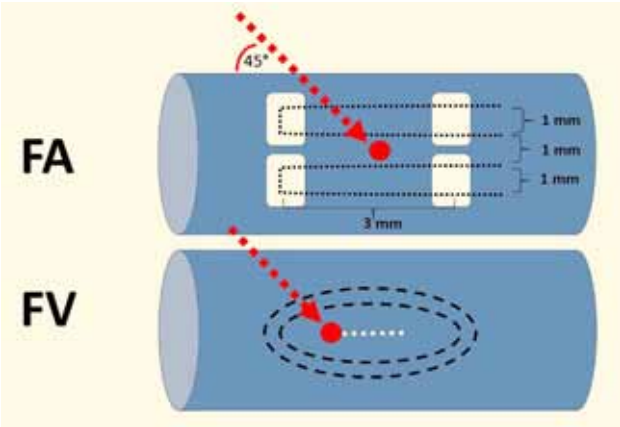

Figure 2. Placement of the sutures. Double-pledgeted sutures are horizontally placed through the adventitial layer of the femoral artery (FA) (top image). Red dot shows the puncture site in the middle of two-layered horizontal sutures. Note the distances of sutures. Double purse suture on the anterior surface of the common femoral vein (FV) (bottom image). Red dot shows the puncture site and dotted white line shows the 3 mm incision horizontal and superior incision before insertion of venous cannula.

Figure 3. Double-pledgeted sutures on the femoral artery and a 3 mm incision over the femoral vein before insertion of the venous cannula.

distance between each suture line should be 1 mm, and the length of each bite should be 3 mm long horizontally. If the anterior wall was calcified, the sutures were placed more laterally or medially in a suitable plaque-free area. Femoral access was, then, performed using the Seldinger technique in the midpoint between the two pledgeted sutures. After the dilatation of the artery with a 15-F or 17-F

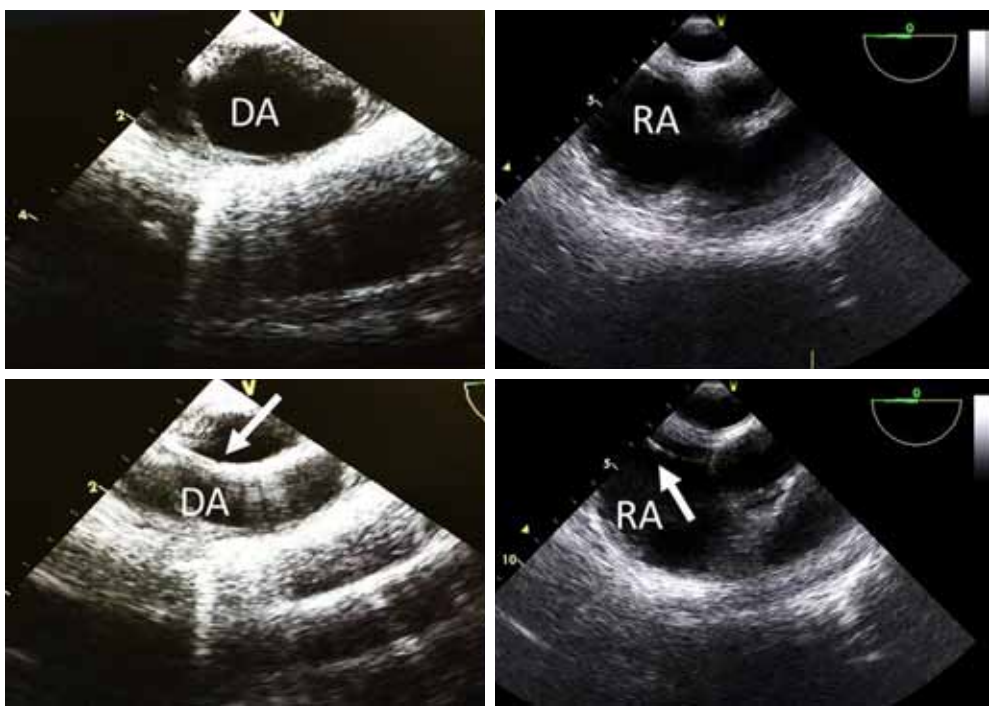

Figure 4. An intraoperative transesophageal echocardiography guidance view during peripheral cannulation. In the left lower view, guidewire (arrow) is shown in the descending aorta (DA). In the right lower view, guidewire is seen in the right atrium (RA).

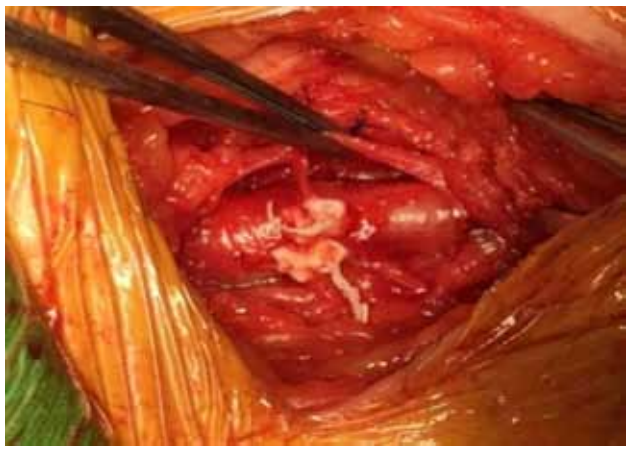

Figure 5. Femoral artery and vein after tying sutures.

dilatator over the 0.035-inch guidewire, the arterial cannula was inserted. An oval shaped, double purse-string suture (5/0 polypropylene) was placed at the anterior side of the common femoral vein. Each suture should be 1 mm away from each other. Using the Seldinger technique, the vein was punctured inferiorly to the oval-shaped purse suture (Figure 3). The puncture site was dilated using a 15-F or 17-F dilatator over the 0.035-inch guidewire. Before the insertion of the venous cannula, the anterior wall of the femoral vein, lying in the oval purse suture, was incised 2 mm superiorly (Figures 2 and 3). The venous cannula was, then, immediately inserted through the vein with gentle maneuvers. All these steps of peripheral cannulation were performed under the guidance of transesophageal echocardiography (Figure 4). At the end of the procedure, the arterial cannula was removed, and the cannulation site was washed out in an antegrade and retrograde fashion. All two pledgeted sutures were, then, ligated (Figure 5). Sutures on the vein were also tied, before the delivery of the protamine following cardiopulmonary bypass.

A postoperative computed tomography angiography image revealed a natural course of the femoral artery with a gentle curve (Figure 6). The

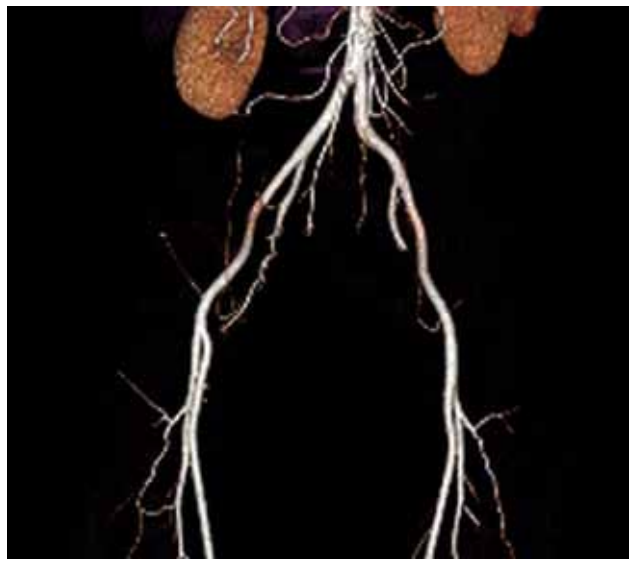

Figure 6. A computed tomographic image following peripheral cannulation of the femoral vessels.

diameters of the proximal and distal segment of the common femoral artery in the cannulation site were similar without a stenosis or occlusion.

In conclusion, in patients undergoing minimally invasive cardiac surgery, particularly with mini-incisions such as port-access operations or robotic approach, peripheral cannulation is of paramount importance for establishing cardiopulmonary bypass. Our experience shows that this technique is simple, safe, and feasible for peripheral cannulation. Over the past five years, this technique has been routinely used in more than 300 minimally invasive and robotic procedures, as well as redo surgeries in our hospital. No procedure-related morbidity including leg ischemia perioperatively, or a vascular morbidity such stenosis, thrombosis, dissection or occlusion has been reported during follow-up to date.

Declaration of conflicting interests

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