

In early experience with C-SES, incomplete neointimal coverage and insufficient expansion of the stent struts were reported by investigators (8). Specifically, compared with paclitaxel-eluting stents, SESs tend to be associated with more rapid neoatherosclerotic changes perhaps because of a difference in the polymer coating on the stent strut surface. SESs have been shown to promote the formation of lipid-rich yellow neointima, which is associated with unstable plaques that have a higher potential of rupture and thrombotic sequelae (9).

Importantly, the discontinuation of dual antiplatelet therapy in itself has not been shown to be a risk factor for VLST (10). However, our case questions this statement because VLST occurred >7 years after DES implantation and 3 months after discontinuation of clopidogrel therapy in our case.

Conclusion

In conclusion, should we recommend lifelong dual antiplatelet therapy in the absence of any contraindication for first-generation DESs (especially C-SESs)?

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Video 1. Coronary angiography revealing thrombotic occlusion in the left coronary artery.

Video 2. A drug-eluting stent (CYPHER) was deployed on the left coronary artery.

Video 3. Very late stent thrombosis causing subtotal occlusion distal to the drug-eluting stent in the left coronary artery.

Video 4. Percutaneous coronary intervention was performed for stent thrombosis on the left coronary artery

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Successful treatment of a renal arteriovenous fistula with pulmonary hypertension occurring 38 years after nephrectomy

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Introduction

Iatrogenic arteriovenous fistula (AVF) is a rare cause of pulmonary hypertension (PH) and high-output heart failure (HOHF). We report a case with PH and 15-years heart failure history secondary to an iatrogenic renal AVF due to nephrectomy performed 38 years ago who underwent closure.

Case Report

A 61-year-old female patient has applied to an external healthcare center with a complaint of dyspnea. Upon detection of PH by transthoracic echocardiography (TTE), the patient was referred to our clinic for further evaluation of PH. The patient with progressive dyspnea for 15 years has had World Health Organization (WHO) class III symptoms, orthopnea, and leg edema

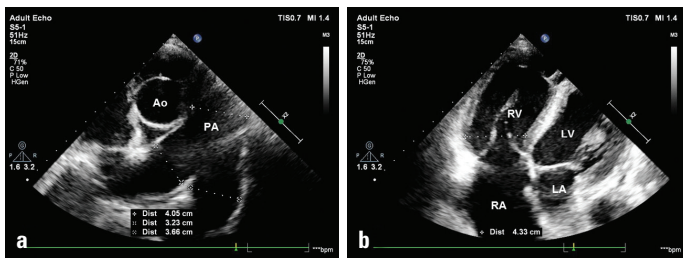


Figure 1. (a) Parasternal short axis view of dilated pulmonary arteries. (b) Apical four chamber view. Ao - aorta, LA - left atrium, LV - left ventricle, PA - pulmonary artery, RA - right atrium, RV - right ventricle

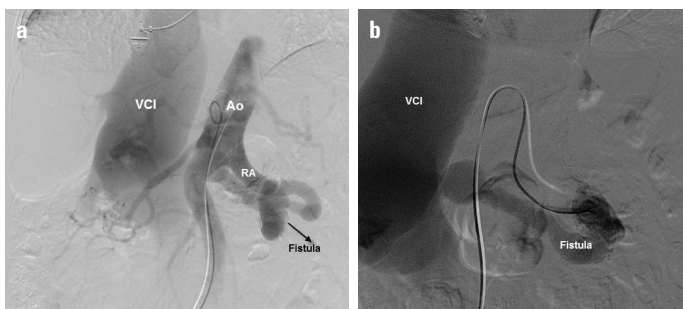


Figure 2. (a) Abdominal aortography showing a fistula between the renal artery and vein. (b) Selective imaging of the fistula. Ao - aorta, RA - renal artery, VCI - vena cava inferior

for approximately 1 year. The patient has been admitted to many centers for many years with these complaints and has been diagnosed with heart failure and hypertension. Her past medical history included hysterectomy and oophorectomy, 12 years ago; cholecystectomy, 20 years ago; and left nephrectomy because of infection and nephrolithiasis, 38 years ago.

Physical examination revealed that the patient was in no distress at rest. Her blood pressure level was 110/70 mm Hg and heart rate was 90 beats per minute and regular. TTE showed normal left ventricular (LV) systolic function, LV hypertrophy, mild mitral regurgitation, and moderate tricuspid regurgitation (TR). Estimated systolic pulmonary artery pressure (sPAP) of the patient was measured as 80 mm Hg from TR velocity. Severe dilatation was observed at pulmonary arteries (main pulmonary artery, 49 mm and branches, 35 mm) (Fig. 1a). There were also left atrial (50 mm) and mild right chambers enlargements (Fig. 1b). An intracardiac shunt could not be detected. The estimated pulmonary vascular resistance (PVR) was measured at approximately 2.5 Wood units (WU). Estimated CO level was approximately 8.3 L/min. Transesophageal echocardiography confirmed the absence of any intracardiac shunts. After echocardiographic evaluation, more detailed physical examination was performed and a continuous bruit was heard in the upper abdomen, more intense in the left hypochondrium region.

Computed tomography (CT) angiography showed increased pulmonary vascularity and dilated pulmonary trunk and right ventricle, but no signs of pulmonary embolism. Pulmonary venous connections were normal. A high-resolution CT showed normal lung parenchyma. Pulmonary function tests and measurement of

blood gases were normal. With right-heart catheterization (RHC), oxygen saturations were found to be high in the right chamber and the highest at the level of IVC (84.4%). RHC confirmed PH with a mean PAP of 40 mm Hg. Pulmonary capillary wedge pressure was 20 mm Hg and PVR was 2 WU. In addition, CO (by Fick method) and Qp/Qs were measured as 9 L/min and 2.91, respectively. Because of the presence of upper abdominal bruit, high CO, and shunt findings, abdominal aortography was performed following RHC. A fistula was found between the left renal artery and vein (Fig. 2, Video 1). It was thought that this renal AVF was probably linked to vascular injury originated from the nephrectomy performed 38 years ago. It was decided to close the fistula because of high CO findings. The fistula was embolized with two Amplatzer Vascular Plug II (St. Jude Medical, Inc., St. Paul, MN, USA) devices by an interventional radiology expert. The procedure was successfully achieved (Video 2). Fifteen days after embolization, the patient improved symptomatically with WHO class II symptoms. Echocardiography demonstrated normal estimated CO level (6 L/min); lower estimated sPAP (45 mm Hg); mild TR; and reduced right ventricular, left atrial size (40 mm), and PA diameter (42 mm). NT-proBNP levels were 2340 and 722 pg/mL at 15 and 30 days after embolization, respectively. The patient's follow-up will continue.

Discussion

We described a case with HOHF and PH due to renal AVF probably occurring several years after nephrectomy. According to our information, the present case is the first in the medical literature. Renal AVF secondary to vascular injury after nephrectomy is an uncommon anomaly. Cases of HOHF, without PH, related to post-nephrectomy renal AVF are rarely reported (1, 2).

Ohm's law suggests that PH is secondary to increased pulmonary resistance or increased pulmonary flow (3). High CO induces shear stress in the pulmonary circulation, which can lead to pulmonary vascular remodeling and an increase in PVR. Paradoxically, low systemic vascular resistance may be accompanied by pulmonary vasoconstriction caused by other unmetabolized vasoactive substances (4). The most common causes of high CO and increased pulmonary flow include anemia, renal disease, cirrhosis, hyperthyroidism, AVF, and intracardiac shunts (5). The aim of this case report is to make PH physicians aware that AVF needs to be considered as a determinant of PH in patients with a high CO state. When other causes of a high CO state are excluded, AVF as a determinant of PH should be considered. In addition, a correct diagnostic approach could not be prepared until the abdominal bruit was discovered in the present case. The fact the examiners in PH referral centers initially did not detect the existence of the hyperdynamic circulation and bruits in these patients and consider it as a potential etiology.

Concern exists regarding perioperative management of hemodynamic changes during closure of fistula as the sudden

increase in LV afterload can lead to acute cardiac decompensation. Contrary to a case developing biventricular failure after closure of a long-standing large AVF (6), there are publications showing rapid reversibility of heart failure with closure of AVF (7). In our case, despite a long medical history, closure of AVF did not lead to periprocedural decompensation. Positive changes were observed even after short follow-up of 15 days.

Conclusion

Iatrogenic AVF occurring after nephrectomy, although rare, can lead to HOHF and PH over years. Repair of AVF may be well-tolerated in the periprocedural period, and a long-standing history should not preclude repair.

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Video 1. Selective imaging of the fistula from renal artery to vena cava inferior.

Video 2. Successful threatment of the fistula with two closure devices.

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