

Treatment for acute type B intramural hematoma and patent ductus arteriosus in an adult using a covered stent

A 69-year-old woman was admitted to our emergency department of the first hospital of Jilin University for persistent chest and back pain since 3 days ago. Her family history was negative for any cardiovascular disease, and she had a 20-year history of cigarette smoking. Her initial vital signs were documented as follows: blood pressure 126/62 mm Hg, heart rate 80 beats/min, respiratory rate 18 breaths/min and temperature 36.4 °C. Continuous murmurs were auscultated at the second intercostal space along the left sternal border. Laboratory investigations including routine blood test, coagulation studies and basic metabolic panels revealed no abnormalities. Electrocardiography revealed normal findings. Emergent contrast-enhanced computed tomography angiography (CTA) revealed type B intramural hematoma secondary to atherosclerotic ulcer of proximal descending aorta and coexistence of patent ductus arteriosus (PDA) (Fig. 1a-1d), indicating blood flow from left to right due to contrast jetting in the main pulmonary. A catheter-based approach was planned following multidisciplinary team decision. Under general anesthesia, the patient's right femoral artery was

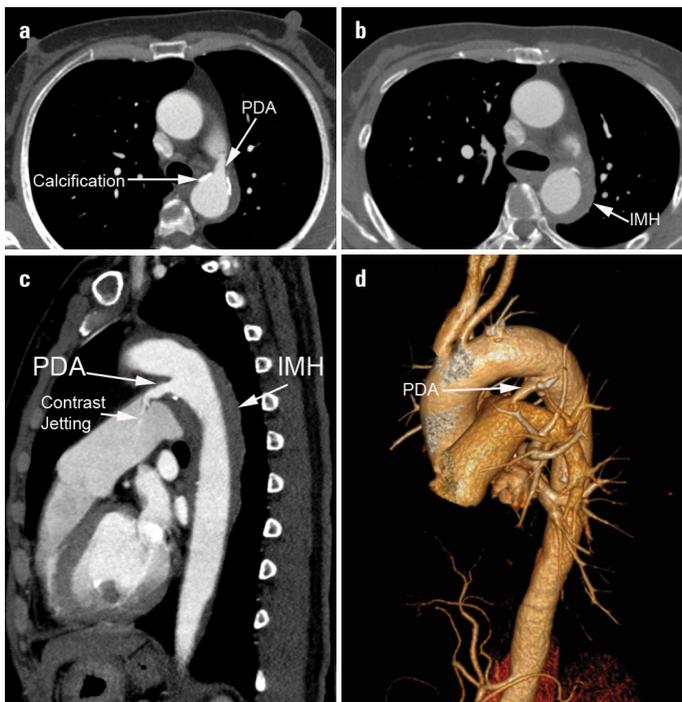


Figure 1. (a, b) Axial CTA images showed significant calcification of thoracic descending aorta surrounding PDA. (c) Sagittal reconstruction image showed contrast jetting in the main pulmonary indicating left to right shunt across the PDA. (d) Volume rendering image showed PDA

dissected and controlled. An endovascular stent graft (Relay: 28-M132200282385S, Bolton) was introduced distal to the left subclavian artery, covering the atherosclerotic ulcer and PDA. Angiography after this procedure revealed well-implanted graft stent, and PDA was not detected. The postoperative course was uneventful. Follow-up CTA at 3 months revealed satisfactory outcomes with complete closure of PDA and existence of aortic wall thickening (Fig. 2a, 2b).

Aortic intramural hematoma (IMH) is a life-threatening disease that usually presents with aortic pain and is characterized by sudden chest pain that may radiate to the back or neck (1). Few cases of IMH cases are asymptomatic and are incidentally diagnosed. A majority (90%) of IMH cases are caused by spontaneous hemorrhage of vasa vasorum in the media of aortic wall, and approximately 5% of patients with IMH are associated with atherosclerotic plaque erosion through the intima into the media (2). Other etiologies of IMH may include trauma and catheter-based aortic procedure. Reportedly, the absence of communication between the lumen and aortic wall during imaging or surgery is the hallmark of spontaneous IMH, but with the wide use of high-resolution CT, some cases of IMH have been detected with an intimal defect, such as ulcerative protrusion. Complications of IMH include expansion or extension of IMH, rupture, conversion to aortic dissection, branch vessel compromise, and aneurysm or pseudoaneurysm formation. An aortic ulcerative protrusion was identified at the proximal thoracic descending aorta, with circumferential mural thickening of descending aorta in the patient in our study. More interestingly, PDA was simultaneously detected by CTA in our patient. Usually, the arterial duct closes within 24–48 hours after birth (3). Most PDAs are mainly diagnosed in children, accounting for 6%–11% of all congenital heart diseases, and are rarely detected in adults. PDAs have diverse lengths, calibers, and shapes, all of which impact the degree of left-to-right shunt and corresponding hemodynamic consequences (4).

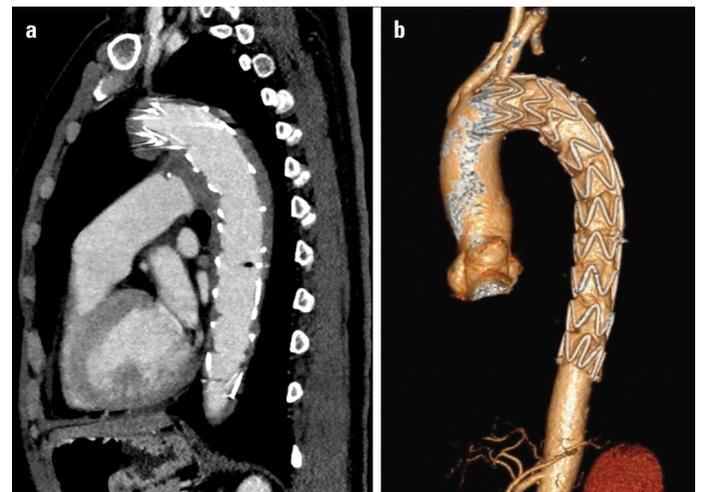


Figure 2. (a, b) CTA showed the absence of PDA, aortic wall thickening and well-implanted graft stent

A small PDA allows only a small left to right shunt, and is often clinically silent. Conversely, several complications including pulmonary hypertension, heart failure, aneurysm, atrial fibrillation, infectious endocarditis, pulmonary artery and aortic dissection, may be associated with a large PDA. The clinical manifestations of PDA vary greatly according to the size of ductus, the pressure across the ductus, patient's age, and the presence or absence of pulmonary hypertension. In the patient in our study, there was a relatively long tube of PDA and focal tiny lumen on pulmonary artery side, which offers substantial resistance to blood flow. Such PDA allows a small left to right shunt and restricts the transmission of aortic pressure to pulmonary artery. Therefore, this patient tolerated PDA without obvious symptoms for several years. Due to the existence of left to right shunt, significant thoracic descending aortic atherosclerosis had occurred surrounding PDA.

In conclusion, to our knowledge this is the first report of PDA coexistent with type B IMH secondary to atherosclerotic ulcer and ulcerative protrusion in an adult patient. Multiple aortic pathologies can be simultaneously delineated by CTA, which helps clinicians determine therapeutic strategy. However, appropriate management for this complex disease entity has not been fully documented yet. Endovascular repair of thoracic aortic disease is widely used mainly because of its invasiveness. In our patient, endovascular treatment demonstrated a less invasive option with promising results, which was verified by postoperative follow-up CTA.

Conflicts of interest: The authors declare that they have no conflict of interest.

Informed consent: Informed consent was obtained from the patient in the study.

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An unusual cause of pulmonary hypertension; partial anomalous pulmonary venous return to coronary sinus detected on transesophageal echocardiography

An asymptomatic male patient aged 25 years was admitted to the cardiology polyclinic with two transesophageal echocardiography (TEE) reports from separate hospitals indicating no abnormalities. Transthoracic echocardiography revealed mild tricuspid valve regurgitation and mild right chamber dilation. Mean pulmonary arterial pressure measured on pulmonary valve was 37 mm Hg. Coronary sinus (CS) was dilated with an abnormal flow (Fig. 1a, 1b). TEE was performed to detect the etiology of pulmonary hypertension (PHT); it showed that the left upper pulmonary vein was separated from the left atrium by a septum (Video 1) and the

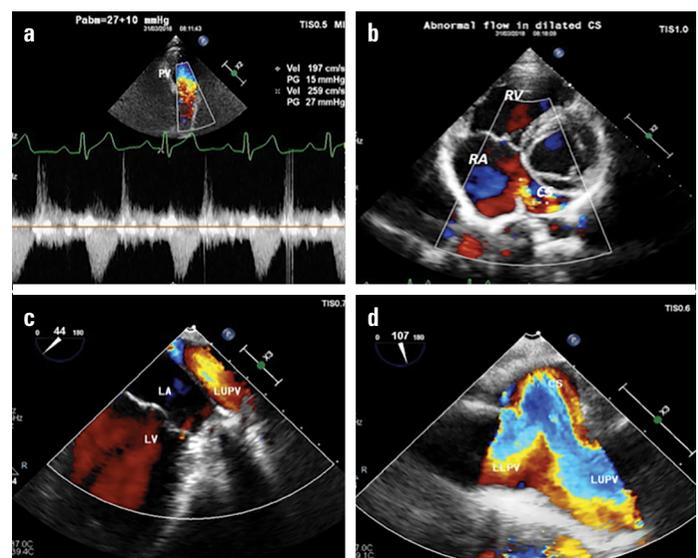


Figure 1. (a) Mean pulmonary arterial pressure measured on pulmonary valve was 27+10: 37 mm Hg. (b) Coronary sinus (CS) was dilated with an abnormal flow. (c) Transesophageal echocardiography showed that the left superior pulmonary vein was separated from the left atrium by a septum and was draining into CS. (d) The left upper and lower pulmonary veins were both draining into the CS. PABm - mean pulmonary arterial pressure, PV - pulmonary valve, RA - right atrium, RV - right ventricle, LA - left atrium, LV - left ventricle, LUPV - left upper pulmonary vein, LLPV - left lower pulmonary vein