

CLINICAL CASES: SEE THAT CASE AT LEAST ONCE

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An unrecognized mechanism of functional tricuspid regurgitation revealed by transthoracic three-dimensional echocardiography

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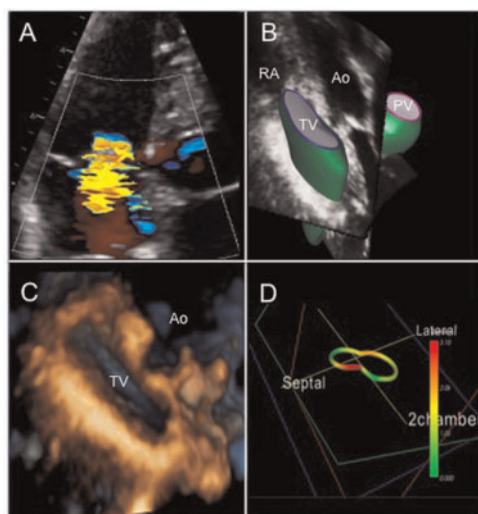
Introduction. Functional tricuspid regurgitation (FTR) commonly occurs secondary to tricuspid annulus (TA) dilation, pulmonary hypertension or right ventricular (RV) dysfunction. We report a case of FTR associated with a severe distortion of TA shape due to dilated ascending aorta (AAo), revealed by transthoracic three-dimensional echocardiography (3DE).

Description of Case Report and Imaging Techniques. A 81-year-old female with arterial hypertension was admitted for exertional dyspnea. A transthoracic echocardiogram showed left ventricular hypertrophy with normal size and function, and a significant dilation of AAo (maximum 51 mm at tubular level) associated with tricuspid aortic valve. Notably, a moderate-severe tricuspid regurgitation (TR) of unclear cause was found (Panel A). A 3DE study was performed, showing normal RV volumes and function (Panel B), a small RA and a severe distortion of TA shape due to extrinsic compression by the dilated AAo (Panel C). Using custom software, TV annulus quantitative analysis revealed a highly asymmetric TA (47x13 mm) with marked ellipticity (Panel D). TA maximal diameter was oriented along the anterolateral-posteromedial direction, and it was more than two fold larger than the diameter displayed in the RV-focused 4-chamber view (19 mm). The TA size in terms of 3D area was actually small (5.6 cm²). The patient was discharged with medical therapy and was scheduled for 6-month follow-up.

Questions, problems or possible differential diagnosis. In our case, conventional two-dimensional (2D) Doppler echocardiography identified a dilated AAo and a significant moderate-severe TR, but was unable to identify the underlying mechanism, as there was no leaflet abnormality, no sign of pulmonary hypertension, no right heart chamber dilation or RV dysfunction, and the measured TA diameter was normal. 3DE was key to rule out the most common mechanisms of TR, and identify a peculiar pathogenesis of valve dysfunction, i.e. extrinsic compression by the dilated AAo.

Answers and discussion. Despite their close anatomic position, the functional interaction between TV and AAo has not been yet reported as a possible mechanism of FTR. This is the first report on the fact that FTR can develop not only as a consequence of TA dilation and rounded shape, but also of severe TA deformation due to extrinsic compression by dilated AAo.

Conclusions and implications for clinical practice. In patients with dilated AAo and FTR of unclear mechanism, a 3DE study should be performed to rule out an extrinsic compression of the TA. The measurement of TA from the conventional 4-chamber RV-focused view is likely to underestimate the maximal TA dimension due to the marked TA ellipticity in such cases. The incidence of coexisting significant TR in the setting of dilated AAo is currently unknown, as well as whether FTR is supposed to resolve after successful surgical treatment of AAo dilation. Future studies are needed to clarify the diagnostic, therapeutic and prognostic implications of our findings.



Abstract 1174 Figure.

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Left ventricular pseudoaneurysm after radiofrequency ablation of premature ventricular contractions

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Left ventricular (LV) pseudoaneurysm is a rare and dangerous echocardiographic finding, usually caused by myocardial infarction. In this case we describe elderly woman who survived after LV free wall rupture. She got under the care of physician in a subacute period with LV pseudoaneurysm.

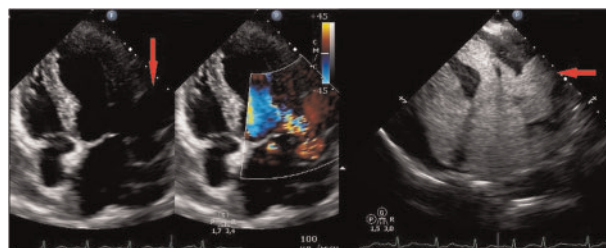
79-year-old woman was admitted to our hospital with acute severe chest pain irradiated to the neck, jaw and back, accompanied by sweating, diarrhoea, hypotension and palpitation. Transient loss of vision and right side numbness appeared in the intensive care unit. 2.5 months earlier the radiofrequency ablation (RA) of PVC was performed and the patient was discharged without complications. Two weeks after the ablation she underwent treatment for transient right hemiparesis and dysarthria. She also complained of chest pain during that period, but echocardiography was normal except for minimal pericardial effusion (PE) near LV. At the admission ECG was normal except for sinus tachycardia. SpO₂ was 98%. Chest X-ray showed slight heart enlargement. Serum troponin I level was slightly elevated. At the transthoracic echocardiography here was moderate PE localized near right ventricle and anterolateral LV wall. Anterolateral PE was less echogenic and was separated by septa. Moderate right atrium diastolic collapse was presented, but there were no signs of cardiac tamponade. Aorta and heart valves were normal. No evidence of pulmonary hypertension was found. Basal anterolateral free wall defect was suspected during color Doppler examination according to low velocity to-and-fro flow between LV cavity and pericardial effusion. Echocardiography with left-sided contrast (sulphur hexafluoride microbubbles) revealed communication between LV cavity and anterolateral pericardial space near left heart chambers through the free wall rupture (red narrow). There were no microbubbles in the right side effusion. Contrast enhanced CT scan confirmed rupture and pseudoaneurysm formed of pericardium adhered to anterolateral free wall. No coronary artery disease was found. There was no contrast accumulation in right-sided PE and its density was typical for lysed blood.

Patient was transferred to a specialized hospital. Due to high operative risk and technical difficulties urgent procedure was declined. Unfortunately she died of multiple organ failure soon after elective surgery.

Acute chest pain accompanied by neurological signs after left heart interventional procedures demands exclusion of aortic dissection. Pericardial effusion is typical for aortic root dissection, but also may be a result of heart perforation during procedure. In this case previous history of RA and contrast-enhanced echocardiography helped to diagnose a dangerous complication – LV free wall rupture.

Typically, LV free wall rupture leads to sudden death from immediate cardiac tamponade. Perhaps pericardial adhesion formed as a consequence of ablation limited the spread of blood, and large pseudoaneurysm was formed.

Thus in case of pericardial effusion after RA free wall rupture should be excluded. Contrast enhanced echocardiography or enhanced CT scan are useful in such cases. Contrast propagation into pericardial effusion is a sign of rupture.



Abstract 1175 Figure.

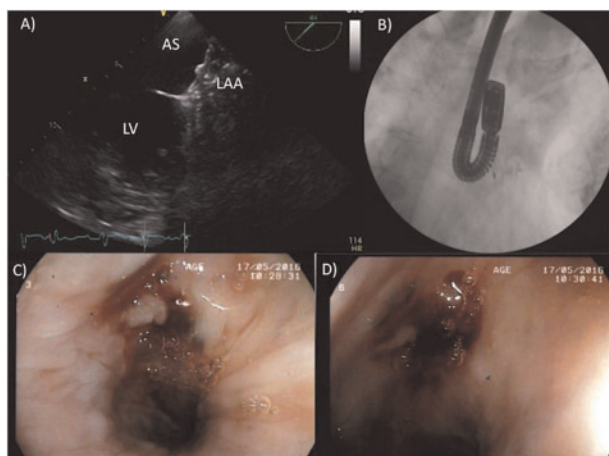
1176

Successful management of buckling of echocardiographic transesophageal probe

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The safety of transesophageal echocardiography (TEE) has been extensively documented and the TEE overall complication rate ranges from 0.18% to 2.8%; the major complications are extremely rare. We report a case of a 75-year-old patient with a ulcerative colitis and persistent atrial fibrillation, who underwent transcatheter appendage closure. Five day safter the procedure a transitory ischemic attack occurred. TEE was promptly performed in order to exclude the device malapposition. The patient undergoing procedure with standard premedication (lidocaine spray, scopolamine butylbromide 20 mg, and midazolam 2 mg). Before the exam, the operator checked the proper probe function, and confirmed that the probe is in the unlocked position. The probe was inserted to the back of the pharynx without difficulties and was slowly advanced down the esophagus. At 30 cm from buccal rhyme no images of cardiac chambers were available. The operator then placed the probe at 50 cm from buccal rhyme and midesophageal images were surprisingly depicted (Panel A). The images were blurred and the manipulation of the probe became difficult. Even after a small incremental addition of midazolam and scopolamine butylbromide the probe was fixed and could be pulled back only up to 30 cm from buccal rhyme. The patient was immediately referred to endoscopy laboratory and fluoroscopic evaluation revealed severe buckling of the probe in the middle third of the esophagus (Panel B). Then, under angiographic guidance the probe was carefully advanced into stomach in which buckled probe was unfolded and then pulled back to esophagus. The patient was always conscious, haemodynamically stable, with no respiratory distress. After the probe extraction, the gastroscopy was performed in order to assess the state of esophageal mucosa and revealed an ulcer with adherent clot and a small laceration (Panel C and D). A complete buckling of the TEE probe which was fixed in the esophagus needs a prompt gastroenterology intervention before forcing more and the fluoroscopy is mandatory to make the diagnosis.



Abstract 1176 Figure.

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An extremely rare tumor of cardiovascular system

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Introduction: Pulmonary artery sarcoma (PAS) is an extremely rare and highly malignant tumor. Less than 300 cases have been reported worldwide, and in those patients without surgical treatment the median survival is about 1.5 months.

Case report: A 50-year-old male patient (pt) presented with a one-month history of progressive breathlessness, fatigue, and dry cough. The pt searched twice the emergency department and was empirically medicated for respiratory infection, without clinical improvement. As the chest radiography showed cardiomegaly, a transthoracic echocardiography (TTE) was performed on an outpatient basis. It showed a dilated right ventricle, with hypokinesia of the mid-apical segments, mild tricuspid regurgitation, high pulmonary artery systolic pressure (PASP), 68mmHg, mild dilation of the main pulmonary artery (PA) and right and left pulmonary arteries (RPA and LPA, respectively), and a large mass with apparent origin in main pulmonary artery (PA) with extension to RPA and LPA and into the right ventricular outflow tract (RVOT), suggestive of tumor versus thrombus. He was hospitalized for further study. Systemic anticoagulation was started with unfractionated heparin. Computed tomography (CT) pulmonary angiography revealed a filling defect in the main PA, RPA, and LPA, with hypodense content adhering to the vessels' wall and with extension to RVOT. Positron emission tomography (PET)-CT revealed an abnormal [18F]-2-fluoro-deoxy-D-glucose uptake within the wall of

the main PA, RPA, LPA and right ventricle (maximum standardized uptake value [SUVmax] 23). Given the findings suggestive of malignancy, the pt underwent exploratory median sternotomy. Extemporaneous biopsy confirmed the diagnosis of pleomorphic sarcoma of the pulmonary artery (PAS). There were no cleavage planes towards adjacent tissues, and so tumor was considered unresectable. Pt was started on neoadjuvant chemotherapy with liposomal doxorubicin monthly. Currently, the pt is in the third month of follow-up, in the 2nd cycle of chemotherapy, with periodic clinical evaluations.

Conclusion: PAS is an extremely rare tumor of the cardiovascular system, with non-specific clinical manifestations. Surgical treatment remains the cornerstone of management. The prognosis is very poor in those without possible surgical resection, with a median survival reported of 1.5 months. A high index of suspicion for early diagnosis is the key to successful treatment and prolonged survival.

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Pneumopericardium: a rare complication of esophageal cancer

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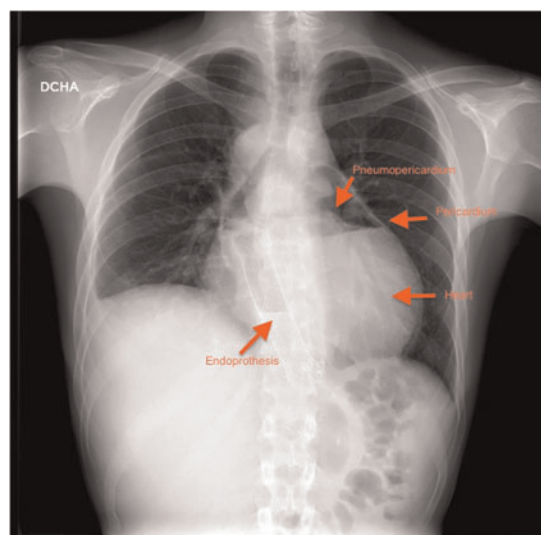
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Introduction: Pneumopericardium is a rare entity, usually as the result of an abnormal communication between a neighbour structure (lungs, oesophagus) and the pericardium. We report a case of esophagopericardial fistula (EPF) that occurred in a patient carrying an esophageal endoprosthesis.

Case Report: a 28-year-old male assisted to the emergency department with chest pain for three days. He was previously diagnosed of an esophageal carcinoma treated palliatively with an esophageal endoprosthesis. At admission, he referred typical pericardial chest pain accompanied with fever. Physical examination revealed tachycardia (110 bpm), diminished heart sounds, a friction rub and hypotension (90/60 mmHg). Electrocardiogram was normal, and the chest x-ray demonstrated a radiolucent area along the left heart border. Later he underwent a computed tomography that confirmed the diagnosis of pneumopericardium, suggesting the presence of an EPF caused by cancer invasion (Fig 1). An echocardiogram did not show any sign of heart dysfunction. Initially, a new endoprosthesis was placed in order to solve the fistula, but he continued a bad evolution in the following 48 hours and finally died.

Discussion: Pneumopericardium is a serious entity, divided according to its origin into traumatic and non-traumatic. The first one includes aorto-coronary bypass surgery, iatrogenic procedures (gastroscopy, thoracocentesis, etc); the second one includes benign and malignant disease, as it is our case. Our patient developed an EPF secondary to an esophageal perforation. Although benign disease is the most common cause of EPF (accounting 76% of all causes), our patient presented with a malignant neoplasia. Clinical features include dyspnea, acute chest pain, fever and, depending on the amount of air there can be hemodynamic compromise with tachycardia, hypotension or oliguria. In our patient, a complication related to his neoplasia was suspected, mainly mediastinitis, finding at the end this EPF. Chest x-ray is the most important test, and fifty percent of the patients can have pneumopericardium in the roentgenogram. The electrocardiogram is usually normal, and echocardiogram can show the "air gap sign", a dark echo that shadows the image during the cardiac cycle, although this sign can also be seen in pneumomediastinum. EPF portends a bad prognosis and urgent treatment consists on reparation of the fistula. In our patient, a new endoprosthesis was placed, but he deteriorated clinically and died.

Conclusions: Pneumopericardium due to EPF is a rare but life threatening condition. Early diagnosis is mandatory for successful management.



Abstract 1178 Figure.

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Left atrial dissection after myocardial infarction

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Introduction:

60 years-old man, with previous history of poliomyelitis in infancy with deformity of lower limbs, alcoholism, hepatitis C infection, smoking, type two diabetes mellitus, left nephrectomy due to pyonephrosis and without previous history of cardiac disease, presented at the emergency department with new-onset chest pain and dyspnoea. The patient was hemodynamically stable, with a loud systolic murmur, without thrill, at cardiac auscultation. Electrocardiogram showed ST segment elevation of 0.1 mV in inferior leads. Blood analyses revealed Troponin I 16.4 ng/mL (cut-off for acute coronary syndrome: 0.12 ng/mL). The main diagnostic hypothesis was ST segment elevation myocardial infarction (STEMI). Coronariography was performed and revealed common trunk plus three vessels disease. Angioplasty was not performed.

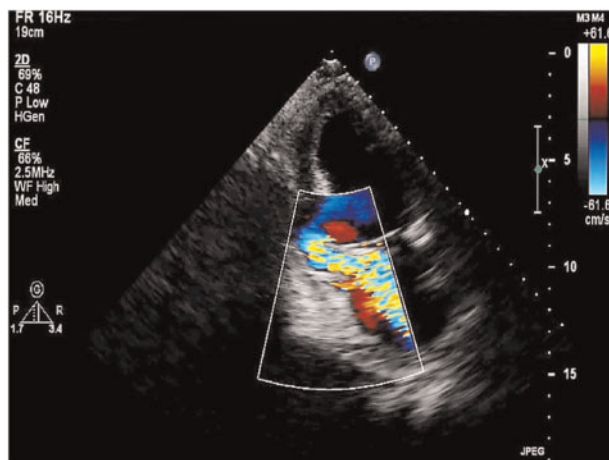
Description of the problem: Transthoracic echocardiogram revealed an aneurysm of the basal segment of left-ventricle's inferior wall with apparent communication to the left atria with major para-mitral regurgitation and left atria dissection (Figure). Transesophageal echocardiogram and latter cardiac magnetic resonance imaging were performed and confirmed those findings. Patient developed acute heart failure with generalized subcutaneous oedema, bilateral pleural effusion and severe symptomatic hyponatremia (minimum sodium 107mmol/mL).

Discussion: Left atrial dissection was assumed as a mechanical complication of myocardial infarction. Thus, cardiac surgery to correct the major para-mitral regurgitation was crucial to revert the acute heart failure.

Stabilization of the patient and the optimal timing for surgery were the main problems: early surgery would comprise a high mortality risk due to patient instability, whereas delayed surgery would allow for easier left atrial dissection repair in scarred tissues but with increased risk of death due to progression of acute heart failure or left atrial rupture while waiting for surgery.

Decision: It was decided not to perform emergent cardiac reparative surgery due to the high risk of the procedure in a patient with decompensated acute heart failure. Clinical stabilization of the patient was successfully achieved with high-dose diuretic therapy (furosemide intravenous perfusion 2 mg/kg/day). Cardiac surgery was performed after clinical stabilization with reparation of left atrial dissection, closure of the para-mitral communication between left ventricle and left atria with bovine pericardium and coronary artery bypass graft with left internal mammary artery to the left anterior descending artery. Patient recovered successfully after surgery with clinical resolution of heart failure.

Conclusion: This case report illustrates a rare mechanical complication after myocardial infarction, with left atrial dissection and major para-mitral regurgitation, in which clinical stabilization was challenging but possible, with positive results with delayed reparative cardiac surgery.



Abstract 1179 Figure.

1180

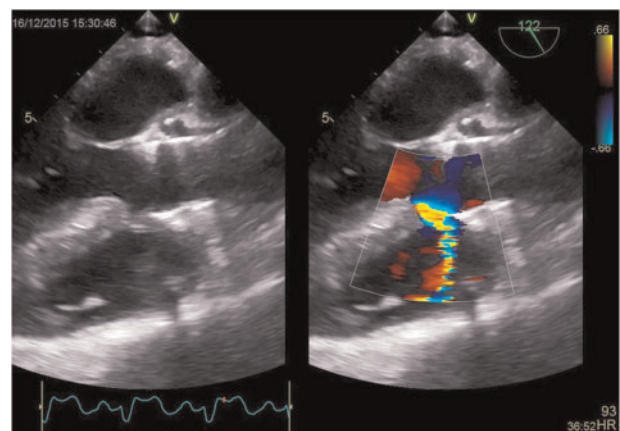
Late appearance of a ventricular septal defect after transcatheter aortic valve replacement: a rare complication

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A 82-year-old woman with a history of previous pulmonary embolism and rheumatoid arthritis treated with corticosteroids with recent onset of effort angina and syncope was diagnosed with isolated severe aortic stenosis. The Heart Team deemed the

patient at high surgical risk and proposed a percutaneous approach to the aortic stenosis. The patient underwent a transcatheter aortic valve replacement (TAVR) with a 29 mm CoreValve Evolute R prosthesis via transfemoral approach. A post-dilation was needed because of a severe periprosthetic leak, with a residual acceptable echocardiographic and angiographic evaluation. The patient was discharged in good clinical conditions. She presented at a follow-up visit three weeks later complaining the onset and progressive worsening of fatigue, shortness of breath and leg swelling. A harsh, holosystolic heart murmur was appreciated. A transthoracic echocardiogram revealed the presence of a perimembranous subaortic ventricular septal defect (VSD) determining a moderate degree high velocity left-to-right shunt (diameter 7 mm), with a Qp/Qs ratio of 1.8:1. A severe pulmonary hypertension was detected, with normal right ventricular size and function. A normal function of the aortic bioprosthesis with a moderate periprosthetic leak were observed. We hypothesized that the VSD had been caused by a subacute erosion of the perimembranous interventricular septum from the frame of the bioprosthesis stent, probably favoured by balloon post-dilation. The Heart Team decided for an urgent VSD repair with a transcatheter closure device. A percutaneous VSD closure was performed with an Amplatzer Muscular VSD occluder, with a residual mild shunt and no interference with the aortic bioprosthetic leaflets. During the procedure a third-degree atrioventricular block developed, hence a pacemaker was implanted at once. The patient was transferred to the Cardiac Intensive Care Unit. She developed a low cardiac output syndrome primarily due to a worsening of right ventricular function, and died 8 days later of multiorgan failure.



Abstract 1180 Figure.

1181

Doppler flow velocities pattern in a thrombophilic patient with an lvad thrombosis

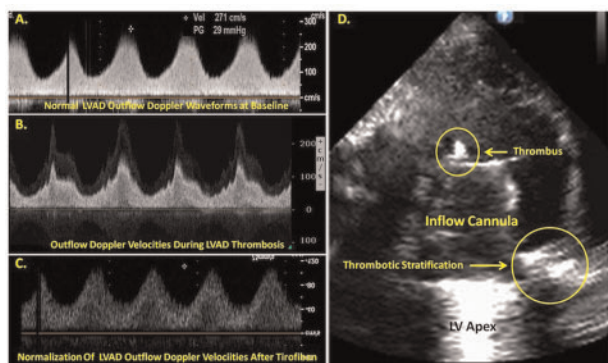
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Introduction: Thrombosis leading to LVAD failure is a medical emergency for which there is a lack of consensus on the most practical imaging protocol to detect this potentially fatal condition.

Case Report: A 66 years old patients was evaluated for an LVAD Destination therapy. His past medical history was significant for a severe ischemic cardiomyopathy with a severe LV impaired function (EF 25%) and a mild/moderate RV dysfunction (RV strain -14%). He was also known for a thrombophilic state due to a MTHFR mutation and a genetic resistance to Clopidogrel. Because of a previous triple bypass, a anterior-lateral thoracotomy surgical approach was chosen for the placement of the LVAD outflow cannula in the descending aorta. The post-operative transesophageal Echo was normal with the exception of a significant spontaneous echo-contrast despite the regular opening of the aortic valve. The flow velocities at the level of the Inflow/Outflow cannula were normal. After few months he developed a low cardiac output state with increase hemolysis index and a "low-flow" alarm suggestive for a LVAD pump thrombosis. Transesophageal Echo showed a significant alteration of the Doppler flow waveform of the outflow cannula with a normal systolic wave and reduced diastolic velocities with "a damped aspect" (IMAGE A, B) due to a poor LV unloading in the presence of a LVAD pump thrombosis, successfully treated with a prolonged Tirofiban infusions and consequent normalization of the doppler (IMAGE C). Following TEE examination revealed a small thrombus at the top level of the inflow cannula and a thrombotic stratification of the LV apex next to the Inflow cannula (IMAGE D) successfully managed with both anti-platelets and anticoagulation therapy.

Conclusion/Implication to Clinical Practice: we here report a new and easily recognizable Doppler velocities pattern pathognomonic for Continuous-flow LVAD thrombosis in the clinical context of a LV apical thrombus.



Abstract 1181 Figure.

1182

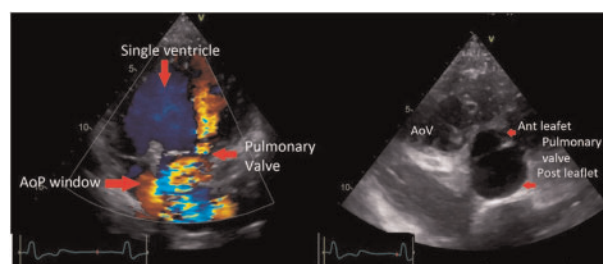
An unusual cause of aortic diastolic reflux in a failed single ventricle palliation

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Introduction: Even in present times we find cases where palliation and correction for single ventricle patients are performed either too late or inappropriately. Cases where survival of a single ventricle infant is possible into childhood are extremely rare and if the Fontan procedure is not performed in due time, the case becomes a real challenge for cardiologists and surgeons, with ill defined management. **Case description:** We present the case of a 15 year old girl who came to our clinic after having a long history of palliation and postponed repairs. She was diagnosed at birth with complex congenital heart disease, but without palliation until the age of three, when a first pulmonary banding was performed, but not followed by a Glenn or Fontan. Four years later an intraluminal banding was inserted and, in an attempt to alleviate the pulmonary hypertension (PHT), a surgical aorto-pulmonary window was created. **Patient evaluation:** The echocardiography showed the structural aspect of double inlet right ventricle with malposition of the great arteries and an associated atrial septal defect. Ventricular function was preserved, with mild-to-moderate atrioventricular valves regurgitation. There was free shunting through the aorto-pulmonary window, with very

significant diastolic reflux, corresponding to an equivalent severe aortic regurgitation. Of note was the presence of a bicuspid pulmonary valve (Figure right panel), a rare finding, with moderate regurgitation. The blood flow from the aorto-pulmonary window is directed towards the pulmonary valve, joining the regurgitant jet (Figure left panel). The invasive hemodynamic study revealed the presence of precapillary PHT with a PVR index of 5.5 WUI and a mean pulmonary pressure of 41 mmHg, a flow ratio of 1.9 and a gradient of 60 mmHg at the level of the banding, all prohibitive for a single ventricle route. Other causes of aortic diastolic reflux had to be excluded, such as aortic valve regurgitation due to a bicuspid valve or endocarditis. The valve was tricuspid and there were no clinical or imaging indications of endocarditis. The main complication of the diastolic reflux is coronary steal, but the patient does not show signs of cardiac ischemia. **Discussions:** This case brings into discussion a rare case of double inlet right ventricle surviving past infancy, where surgical treatment was performed late and incompletely, resulting in a failed single ventricle route. The context in which the decision to surgically create an aorto-pulmonary window is not clear, as recent data dismissed this procedure as not suitable, due to very significant coronary steal through diastolic reflux. To add to the complex hemodynamics, the patient associates an atrial septal defect and also a bicuspid pulmonary valve with significant regurgitation, adding to the deleterious effect of the aorto-pulmonary window. Currently, the patient is being evaluated in order to determine the safety of closing the vascular communication, in the context of severe PHT. Given that a cavo-pulmonary shunt is prohibitive, pulmonary vasodilator therapy was started with bosentan and sildenafil. The exercise capacity is reduced, but without signs of cardiac failure. **Conclusions:** This case highlights how otherwise incidental findings, like a bicuspid pulmonary valve, can become significant in complex scenarios. Surgical procedures performed in single ventricle patients after the optimal age are to be carefully analyzed, as the hemodynamic effects of combined abnormalities can lead to worse outcomes than conservatory treatment.



Abstract 1182 Figure.