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Persistent hypoxemia in a patient with left ventricular assist device: is it time to think beyond patterns?

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A 47-year-old man was diagnosed with idiopathic dilated cardiomyopathy in 2012. In 2016 he had an implantable cardioverter defibrillator(ICD). In 2017, the progression of heart failure led to a left ventricular assist device(LVAD) HeartWare® as bridge to transplant. Post-operative course was complex, with a five-month stay. After two months at home, he was readmitted for ventricular tachycardia(VT). At the admission LVAD flows were stable around 4L/minute at 2500rpm. A previous transthoracic echocardiogram(TTE) had showed the aortic valve closed at every beat with mild aortic regurgitation(AR). He required several shocks. Remarkably, blood gases showed a progressive hypoxemia making high flow oxygen support necessary. A TTE showed a severe AR in a closed valve and right ventricular impairment. It was likely the consequence of the aortic valve closed for the previous months. Therefore it is key to find the balance between the afterload generated by pump and the inotropism, in order to allow the valve to open. This is the purpose of Lavare cycle. In order to diminish the insufficiency, the speed was decreased to 2400 rpm, but significant AR persisted. The multidisciplinary team decided to perform a transcatheter aortic valve implantation(TAVI). Hypoxemia was not sorted. Another TTE showed that TAVI was well seated, no paravalvular leak. The main differential diagnosis came up. We summarized other causes leading to hypoxemia in three: 1. Reduction of LVAD flows during VT. 2. An underlying lung condition causing intrapulmonary shunt. 3. An unadvised intracardiac shunt (transesophageal echocardiogram's(TOE) sensitivity is between 85-90%). Of note, exacerbations of hypoxemia were related with arrhythmia bursts, with no significant impact on LVAD flow though, remaining above 3.5L/minute. Pulmonary tests ruled out any ventilation/perfusion mismatch. Finally, the decision was to repeat the TOE during one of the episodes of VT. Surprisingly, it showed the presence of a patent foramen oval(PFO), along with a massive right to left interatrial shunt. Previous TTE/TOE were reviewed and this shunt was unadvised during the operation and in sinus rythm. A plausible explanation could be related to the atrioventricular dissociation during the VT, leading to an increase in right atrium pressure, making the shunt more visible. If left atrial pressure exceeds the right atrial, there can be a gradient closing the PFO by approaching the septum primum to the the fossa ovalis. A PFO occluder device was percutaneously positioned and the hypoxemia was finally resolved. This case shows the uniqueness of hypoxemia episodes happening in a patient with LVAD during VT due to a PFO shunt. Due to refractory right ventricular failure, sepsis and lately LVAD thrombosis the patient was not suitable for heart transplant and died soon after. In conclusion, hypoxemia in a LVAD patient needs to be fully investigated, ruling out the presence of any either cardiac or pulmonary shunt.