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A case of heart transplantation from a donor with atrial septal defect secondum

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Introduction: Number of patients waiting for a heart transplant is increasing but the number of donated heart is too small. But, the heart of a donor with congenital heart disease is rarely used for heart transplantation. Inhere VSD doner's heart was used for transplantation. **Case Report:** A 63-year-old man was presented with advanced heart failure from old myocardial infarction. Right heart catheterization showed high pulmonary arterial pressure with mean PAP of 65mmHg, with pulmonary vascular resistance of 1.4 wood unit. Both atrial filling pressure were also elevated with mean pulmonary capillary wedge pressure of 56, mean RA pressure of 15mmHg. He was admitted after the acceptance of 44-year-old donor heart for transplantation. Repeated transthoracic echocardiography revealed that there was an atrial septal defect secondum in the donor heart(figure 1). Our patient had unexpected offer, we hesitated to get a heart with ASD and RV enlargement. After thorough discussion with heart transplantation team, we decided to accept a heart. We expected that donor heart will tolerate the high PVR of the recipient immediate postoperative period, because donor heart might have been get used to high volume/pressure overload from left to right shunt. Also, the donor was local donor at our hospital, so we could save ischemic time. ASD was treated with patch closure before the anastomosis (Figure 2). High dose inotropic agent was needed for postoperative 3 days as well as CVVHD. After 3 weeks after operation, he was discharged to home at healthy status with tacrolimus/MMF/prednisolone maintenance. He has been well until 3 months now. **Discussion:** Use of a donor heart with congenital heart disease is very limited in transplantation. ASD donor is usually discarded due to the high risk of post-operative RV failure. However, ASD heart may tolerate the recipient's high PVR, because it is well-trained at high PAP condition. Considering there is significant lack of donor heart while many desperate heart transplantation candidates exist, use of marginal donor could be an option to solve this problem.

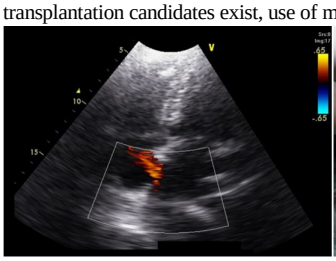

Figure 1
Transthoracic echocardiography revealed atrial septak defect sencondum in the donor heart

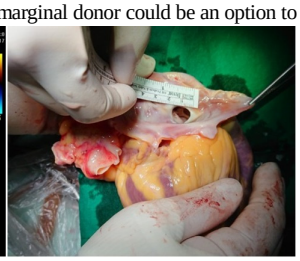

Figure 2
ASD was patch closed before anastomosis

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Aborted Sudden Cardiac Death in a young man with Hypertrophic Cardiomyopathy and WPW syndrome

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Both hypertrophic cardiomyopathy (HCM) and Wolff-Parkinson-White (WPW) syndrome are frequent causes of sudden cardiac death (SCD) in the young. Here, we describe an aborted SCD in a young man who was found to have concomitant HCM and WPW syndrome with clearly documented reentrant tachyarrhythmia. A 19-year-old male suddenly fell down after overnight duty. After several minutes of bystander cardiopulmonary resuscitation, a single AED shock successfully terminated his ventricular fibrillation (VF, Figure 1A), and he recovered fully. His baseline ECG was notable for short PR interval with slurred QRS upstroke, indicative of ventricular pre-excitation (Figure 1B). Echocardiographic examination revealed markedly thickened interventricular septum and systolic anterior motion of mitral valve with increased pressure gradient, consistent with HCM with dynamic obstruction (Figure 2). Also, abnormal vascular response was noted during treadmill test. Isoproterenol provocation test was performed due to his recurrent episodic palpitation, documenting a narrow QRS tachycardia (Figure 2). Subsequent electrophysiologic study identified an accessory pathway (Figure 3) with reproducibly-inducible atrioventricular reentrant tachycardia, which was successfully eliminated by radiofrequency catheter ablation. He remained uneventfully so far with β-blocker. Concomitant HCM and WPW syndrome is a very rare condition. SCD survivors of HCM generally undergo ICD implantation. WPW syndrome itself is also a SCD-prone disease, however, ablation of arrhythmogenic substrate is indicated rather than ICD. Association between tachyarrhythmia and SCD has been well-acknowledged in HCM: rapid repetitive conduction to ventricle causing spontaneous rhythm degeneration into VF. Factors responsible for this phenomenon includes outflow tract obstruction, inherent vulnerability to ischemia and arrhythmia, and abnormal vascular response, which were all presented in our case and might play synergistic roles. In fact, he refused ICD therapy, therefore we eliminated his potential VF trigger and prescribed β-blocker. Nevertheless, close monitoring of symptom recurrence should be ensured for this young man.