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Atrial septal defect closure following mitral transcatheter edge-to-edge repair

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Case report: 87-year-old female patient with history of atrial fibrillation and known severe mitral and torrential tricuspidal regurgitation and a reduced left and right ventricular ejection fraction EF LV 20%, TAPSE 13mm was admitted to the Coronary Unit due to global cardiac decompensation complicated by acute kidney injury with anuria. Following the initiation of inotropic and vasopressor support with dobutamine and norepinephrine, aggressive diuretic therapy was administered. Adequate diuresis was achieved only after the introduction of sequential nephron blockade with acetozolamide, furosemide, chlortalidone, and spironolactone. The patient subsequently achieved hemodynamic stabilization and successfully underwent M-TEER (mitral transcatheter edge-to-edge repair). The procedure resulted in a reduction of mitral regurgitation to mild and tricuspid regurgitation from torrential to severe. During the subsequent hospital course, the patient experienced significant hemodynamic deterioration. Repeat transesophageal echocardiography revealed progression of iatrogenic interatrial septal defect with Qp:Qs 2.6, accompanied by recurrent torrential tricuspid regurgitation and moderate mitral regurgitation. Following initial stabilization with high dose inotropic and vasopressor support, successful percutaneous closure of ASD was performed using an Amplatzer occluder, resulting in a significant reduction in central venous pressure. Hower, the subsequent course was complicated by further clinical deterioration, with severe left and right heart failure and anuria. Stroke volume was markedly reduced to 10ml/m^2. Intensive medical management was reinstituted, including high dose inotropic and vasopressor support combined with repeated intensive diuretic therapy. Over week, the patient clinical, hemodynamic and echocardiographic status gradually improved.

Conclusion: Following M-TEER, reduction of mitral regurgitation from severe to mild likely resulted in an increase in left ventricular afterload. In the setting of severely impaired baseline left ventricular systolic function, the left ventricular may have been unable to adequately accommodate this abrupt change in loading condition, leading to increased left atrial pressure. The resulting pressure gradient across the iatrogenic atrial septal defect facilitated left-to-right shunt as a potential mechanism of left atrial decompression. The increased shunt volume overload contributed to right ventricular volume overload, recurrent torrential regurgitation, and right heart-sided heart failure. This case illustrates the complex interplay between left ventricular afterload, left atrial pressure, iatrogenic interatrial shunting, and right ventricular function following M-TEER. It underscores the importance of individualized hemodynamic assessment when considering iatrogenic ASD closure in patients with advanced biventricular dysfunction, as eliminating a potentially harmful left-to-right shunt may also remove a compensatory route for left atrial decompression. Careful postprocedural monitoring and tailored medical management remain essential while the ventricles adapt to the new hemodynamic condition.[1]

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LITERATURE

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