

# Isolated Severe Ischemic Tricuspid Regurgitation: Successful Surgical Repair

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**Tricuspid regurgitation (TR) is a common association of left-sided valvular lesions that is occasionally seen with ischemic heart disease. However, isolated ischemic TR requiring surgical intervention is extremely rare, with very few reported cases. The case is presented of a 59-year-old woman who, after**

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## Case report

A 59-year-old woman presented initially with increasing nausea and anorexia, followed by tiredness and the development of ascites. She underwent extensive investigations at the referring hospital, including computed tomography scanning of the thorax and upper abdomen, and thoracoscopy to exclude an intraabdominal or intrathoracic malignancy.

Transthoracic and transesophageal echocardiography (TEE) later confirmed gross functional TR with a moderately impaired, dilated right ventricle (right ventricular end-diastolic dimension 60 mm). The patient had a history of recent-onset atrial fibrillation, but no documented myocardial infarction. Cardiac catheterization demonstrated moderate right ventricular function, a grossly enlarged right atrium with pulmonary artery pressure of 40 mmHg, and a right ventricular end-diastolic pressure of 22 mmHg. Coronary angiography showed significant disease in a dominant right coronary artery, with an 80% stenosis at the crux extending into the posterior descending

**extensive non-cardiac investigations, was eventually diagnosed with severe functional TR due to chronic myocardial ischemia. The patient underwent successful surgical treatment.**

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artery (PDA) that was unsuitable for percutaneous intervention.

On initial assessment, the patient appeared cachectic and in gross right heart failure, with ascites and renal failure. She was optimized medically prior to cardiac surgery with extensive diuresis, drainage of the ascites, and peritoneal dialysis.

At surgery, the right heart was grossly dilated and intraoperative TEE confirmed severe TR due to severe annular dilatation. The tricuspid valve leaflets with their chordal attachments were structurally normal. The patient underwent tricuspid valve repair using a 28 mm Edwards MC<sup>3</sup> annuloplasty ring (Edwards Lifesciences LLC Irvine, CA, USA) and a single coronary artery bypass graft to the PDA vessel with a segment of reversed autologous long saphenous vein. Modified ultrafiltration was instituted while the patient was on cardiopulmonary bypass.

The patient required dobutamine (5-10 µg/kg/min) during the early postoperative period, but was extubated 8 h later. Her subsequent recovery was uneventful and she was transferred to the referring hospital on the postoperative day 5 for convalescence. At follow up examination six months later, she was in NYHA functional class I. The patient has since undergone transthoracic echocardiography at follow up; this demonstrated a well-seated annuloplasty ring and minimal TR. In addition, the function of the right ventricle was improved and its dimensions reduced.

## Discussion

Tricuspid regurgitation is a common association of left-sided valve lesions, and is also seen in association

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with congenital abnormalities such as Epstein's anomaly with a structurally abnormal valve. Acute TR may occur as a result of blunt chest trauma (2), after pacemaker insertion (3) or acute myocardial infarction (AMI) (4), or as a result of biopsy-induced regurgitation following cardiac transplantation (5).

Significant TR as a complication of AMI, although exceedingly rare, is mainly structural due to anatomic disruption of the tricuspid valve apparatus; nonetheless, it may be catastrophic.

Functional TR associated with annular dilatation does not usually attain hemodynamic significance, as such chronic regurgitation rarely causes clinical deterioration refractory to diuretic or vasodilator therapy. Few reports have been made of isolated ischemic chronic functional TR requiring surgical treatment (1,6).

Dyssynergy of the ventricular wall adjacent to the papillary muscle (in the absence of papillary muscle infarction) produces valvular regurgitation. In addition, progressive chamber enlargement and right ventricular volume overload produces annular dilatation, leading ultimately to a clinical state of debilitating right heart failure refractory to intensive medical treatment.

Although the present patient had no evidence of previous right ventricular infarction, it is believed that her TR was due to chronic ischemia as she had a moderately impaired dilated right ventricle with no acquired pathology, and a morphologically normal tricuspid valve with dilated annulus with a concomitant significant stenosis at the origin of the PDA vessel. The coronary lesion was not amenable to percutaneous intervention, which precluded surveillance of residual

TR following angioplasty. Furthermore, though ischemic in origin, once TR is advanced and severe due to progressive right atrial enlargement and annular dilatation, it is unlikely to resolve without surgical repair. The continued clinical and echocardiographic improvement provides further credence to an hypothesis of ischemia being the cause of TR.

The present case represents a rare syndrome complex that requires correct diagnosis and subsequent surgical intervention.

#### References

1. Vatterott PJ, Nishimura RA, Gersh BJ, et al. Severe isolated tricuspid insufficiency in coronary artery disease. *Int J Cardiol* 1987;14:295-301
2. Maisano F, Lorusso R, Sandrelli L, et al. Valve repair for traumatic tricuspid regurgitation. *Eur J Cardiothorac Surg* 1996;10:867-873
3. Fishenfeld J, Lamy Y. Laceration of the tricuspid valve by a pacemaker wire. *Chest* 1972;61:697-698
4. Silverman BD, Carabajal NR, Chorchos MA, Taranto AI. Tricuspid regurgitation and acute myocardial infarction. *Arch Intern Med* 1982;142:1394-1395
5. Crumbley AJ, III, Van Bakel AB. Tricuspid valve repair for biopsy-induced regurgitation after cardiac transplantation. *Ann Thorac Surg* 1994;58:1156-1160
6. Szyniszewski AM, Carson PE, Sakwa M, et al. Valve replacement for tricuspid regurgitation appearing late after healing of left ventricular posterior wall and right ventricular acute myocardial infarction. *Am J Cardiol* 1994;73:616-617