

# Anaesthetic Management of A Case of Severe Tricuspid Regurgitation Posted for Open Cholecystectomy

Priyanka Bansal<sup>1</sup>, Shilpa Popli<sup>2</sup>

## ABSTRACT

**Introduction:** Isolated tricuspid regurgitation (TR) is very rare and is associated with many conditions, frequently is seen in association with drug abuse, endocarditis or chest trauma.

**Case report:** More commonly TR is associated with other cardiac abnormalities such as end stage aortic or mitral valve disease most often Mitral stenosis, elevated pulmonary artery pressure leads to right ventricular (RV) strain and eventually RV failure with TR. RV can compensate for volume overload. On the other hand pressure overload is not well tolerated by RV.

**Conclusion:** Most symptoms associated with TR are directly related to increased RV afterload.

**Keywords:** Tricuspid regurgitation (TR), Right Ventricle, Pulmonary Vascular Resistance, Volume Overload, Pressure Overload

## INTRODUCTION

Isolated tricuspid regurgitation (TR) most frequently is seen in association with drug abuse, endocarditis or chest trauma. More commonly TR is associated with other cardiac abnormalities such as end stage aortic or mitral valve disease most often Mitral stenosis, elevated pulmonary artery pressure leads to right ventricular (RV) strain and eventually RV failure with TR.<sup>1</sup> Carcinoid syndrome may produce isolated TR. The primary congenital cause of TR is Ebstein's anomaly. We came across a rare case of isolated severe TR. To the best of our knowledge and extensive review of literature, case reporting of isolated case of TR is very rare in literature.<sup>2</sup>

## CASE REPORT

### Preoperative assessment

Our patient was a 50 years female, Sushma, posted for open cholecystectomy. Patient had no history of hypertension, diabetes, bronchial asthma, thyroid disease, tuberculosis. No history of any chest pain, breathlessness, dyspnea on exertion, syncope, orthopnea or paroxysmal nocturnal dyspnea. No history of any trauma or drug allergy. Pt had previous history of six uncomplicated pregnancies and tubectomy 11 years back under local anaesthesia which was uneventful. On examination- BP-116/26 mm hg, PR – 82 per min, irregularly irregular pulse, chest – bilateral air entry present, CVS- pansystolic murmur heard over left sternal border which increases with inspiration. ECG, figure 1, showed right bundle branch block and chest X ray, figure 3, showed significant cardiomegaly. 2D echo, figure 2, was advised which showed dilated RA and RV with moderate to severe TR. Septal wall abnormality was present. Pulmonary artery pressure was 22 mm Hg. No vegetations were seen. Ejection fraction was 50%. Other basic investigations were – HB 14 gm/dl, platelet count -2 lacs, blood sugar 104, blood urea -22, s. Na-138, s. K – 4.0.

### Preoperative period

High risk informed consent taken. Premedication with tab lorazepam 1 mg at 6 am was given along with 150 mg of Tab. Rantac with a sip of water. Fasting status was confirmed before proceeding to OT. 18G cannula was inserted on left arm.

### Intraoperative

**Monitoring-** All routine monitors attached (ECG, NIBP, SpO<sub>2</sub>, ET/CO<sub>2</sub>). Arterial line secured on left arm (brachial artery) for beat to beat BP monitoring. Central line (subclavian) inserted on right side and CVP monitoring done with target to keep CVP 8-12 mm hg. All emergency drugs kept ready. Intraoperative goals made in terms of rate 80 to 100 bpm as tachycardia decreases regurgitation and sinus rhythm to be maintained. Systemic vascular resistance maintained by maintaining BP within 20% of preoperative values and avoiding hypotension. PVR kept low with target of etco<sub>2</sub> between 22 to 25 mm hg and hyperventilation as hypocapnia decreases PVR. (TV was kept 500 ml with RR of 18 -20 per min).

**Induction-** Patient given inj. glycol 0.2 mg followed by inj. fentanyl 120 µg for adequate analgesia. Induction done with inj. etomidate (0.3 mg per kg) 1.8 mg followed by inj. vecuronium 5 mg. After 3 mins proseal LMA was inserted to minimize cardiac changes following induction. Sudden fall in BP was seen after induction- 70/52 mm hg. Inotropic support was started- inj. dobutamine at 5 µg/ kg/ min and titrated according to BP levels of 130-140 / 80- 90 mm hg to maintain circulation.

**Maintenance –** with oxygen and sevoflurane targeted to MAC of 1.3. Nitrous oxide was omitted as it increases pulmonary arterial pressure. Surgery completed. TAP (transverse abdominis plane) block was given with 0.125% bupivacaine (20 ml) for post operative analgesia. After return of spontaneous respiration inj. neostigmine (2.5 mg) with inj. glycopyrrolate (0.5 mg) was given and LMA was removed. Pt awake with spontaneous respiration and intact reflexes.

## DISCUSSION

The increasing no. of patients with valvular heart disease undergoing non cardiac surgery justifies guidelines for proper preoperative evaluation, Intaaortic Ballon Pump or Percutaneous intervention or if required valve replacement or repair.<sup>3,4</sup> The purpose of preoperative evaluation is to lower perioperative morbidity and mortality with minimal expense from preoperative testing and to concentrate on economic investment on high risk

<sup>1</sup>Assistant Professor, <sup>2</sup>Senior Resident, PGIMS, Rohtak, India

**Corresponding author:** Dr Priyanka Bansal, Assistant Professor, PGIMS, Rohtak, India

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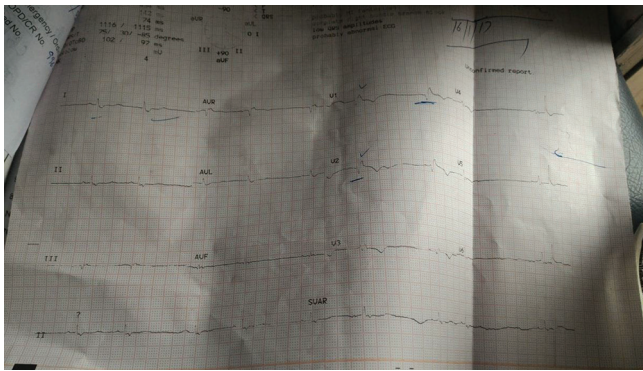


Figure-1: ECG

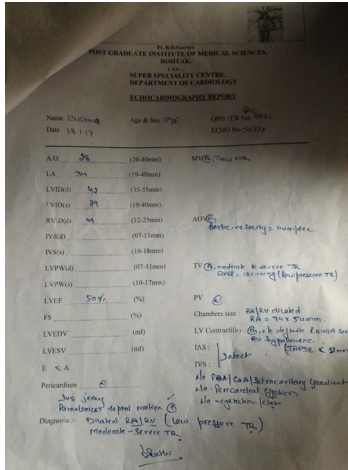


Figure-2: ECHO

patients where specialised tests might modify perioperative management and improve long term benefits.<sup>5,6</sup> Our patient was normal on evaluation and history taking. ECG showed RBBB and chest x-ray showed cardiomegaly. ECG abnormalities fall under minor predictors of risk assessment and cholecystectomy falls under intermediate procedure risk. But since X-ray showed significant cardiomegaly, ECHO was advised which showed ejection fraction 50%, and severe TR, RA and RV dilated with no signs of pulmonary hypertension.<sup>7</sup> We did not go for further invasive or non invasive testing as patient was completely asymptomatic and quite active at work. Isolated TR is well tolerated because RV can compensate for volume overload.<sup>8,9</sup> On the other hand pressure overload is not well tolerated by RV. Most symptoms associated with TR are directly related to increased RV afterload. Therefore when TR is associated with pulmonary vascular hypertension, the impedance to RV ejection produces significant clinical deterioration from decreased cardiac output. Most patients with TR have associated atrial fibrillation due to distension of RA. Patients with valvular heart disease coming for surgery present many challenges to the anaesthesiologist. The 5 variables in dealing with valvular heart disease are important. They are preload, afterload, myocardial contractility, heart rate, rhythm. We proceed with antibiotic prophylaxis to prevent infective endocarditis. AHA recommends ampicillin, gentamicin, or vancomycin. We chose for ampicillin 2g IV half hour before surgery (test dose given in ward). Our aim during surgery was to maintain high or normal heart rate so as to decrease regurgitation. As RV failure is the primary cause of clinical deterioration in patients with TR.<sup>10</sup> Because



Figure-3: X-ray chest

Figure-4: History taking and investigations

the RV is designed geometrically to accommodate volume but not pressure loads, it may require inotropic support especially in setting of positive pressure ventilation or elevated pulmonary vascular resistance. Any suppression of contractility with myocardial depressants may induce severe RV failure. Therefore inotropes were kept ready before induction (dobutamine as it simultaneously decreases pulmonary vascular resistance. Also pulmonary vascular resistance was kept low by hyperventilation with aim to keep  $\text{etCO}_2$  between 22-25 mm Hg as hypocapnia decreases pulmonary vascular resistance. High airway pressures and nitrous oxide that increase pulmonary arterial pressure were avoided.<sup>11</sup> Variations in systemic afterload has little effect on tricuspid regurgitation.

We had certain limitations in our management. We did not go for invasive preoperative tests. The reason being that patient was totally asymptomatic and surgery was of moderate risk. Central venous monitoring was not considered by us.

## CONCLUSION

Isolated tricuspid regurgitation (TR) is very rare. Proper management of pulmonary and systemic pressures is very

important intraoperatively. Investigations vary from case to case.

## REFERENCES

1. Jackson JM, Thomas SJ. Valvular heart disease. In: Kaplan JA, Reich DL, Konstadt SN, eds. *Cardiac anesthesia*, 4th ed. Philadelphia: WB Saunders. 1999:727–85.
2. Jackson JM, Thomas SJ, Lowenstein E. Anesthetic management of patients with valvular heart disease. *Semin Anesth*. 1982;1:239-52.
3. Gorlin R, McMillan IKR, Medd WE, et al. Dynamics of the circulation in aortic valvular disease. *Am J Med*. 1955;18:855-70.
4. Goertz AW, Lindner KH, Schutz W et al. Influence of phenylephrine bolus administration on left ventricular filling dynamics in patients with coronary artery disease and patients with valvular aortic stenosis. *Anesthesiology*. 1994;81:49–58.
5. Yang SS, Bentivoglia LG, Maranhao V, et al. Assessment of valvular regurgitation. In: Yang SS, Bentivoglia LG, Maranhao V, et al., eds. *From cardiac catheterization data to hemodynamic parameters*, 2nd ed. Philadelphia: FA Davis. 1988:152–165.
6. Butterworth JF, Legault C, Royster RL, et al. Factors that predict the use of positive inotropic drug support after cardiac valve surgery. *Anesth Analg*. 1998;86:461-67.
7. Mangano DT. Perioperative cardiac morbidity. *Anesthesiology*. 1990;72:153-84.
8. Eagle KA, Berger PB, Calkins H, et al, eds. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery—executive summary. A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. *Anesth Analg*. 2002;94:1052-64.
9. Forrest JB, Rehder K, Cahalan MK et al. Multicenter study of general anesthesia. III. Predictors of severe perioperative adverse outcomes. *Anesthesiology*. 1992;76:3-15.
10. Goldman L, Calder DL, Nussbaum SR et al. Multifactorial index of cardiac risk in noncardiac surgical procedures. *N Engl J Med*. 1977;297:845-50.
11. Lee TH, Marcantonio RE, Mangione CM et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation*. 1999;100:1043-49.

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