

## CASE REPORT

### IMMEDIATE POST-OPERATIVE VENTRICULAR BIGEMINY IN A PATIENT OPERATED FOR OVARIAN CYSTECTOMY: A CASE REPORT

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**ABSTRACT:** A 30 years female who was recently diagnosed as RHD with MR was posted for Rt. Ovarian cystectomy. Immediately after extubation she was having continuous ventricular bigeminy which persisted even after giving 60mg of 2% lignocaine i.v. Later 2mg of metoprolol was given which stopped the ventricular ectopics from occurring. The case report discusses the successful treatment of the ventricular bigeminy and the prevention of any complications from arising in this particular patient.

**KEYWORDS:** Ventricular bigeminy, Ventricular premature complex (VPC), Ventricular ectopic, Rheumatic heart disease, Mitral regurgitation, Metoprolol.

**INTRODUCTION:** Ventricular Bigeminy is a type of ventricular premature complex (VPC) where every normal sinus beat is followed by a ventricular ectopic. The characteristic feature here is wide QRS (0.14s) complexes with abnormal ST-T changes followed by a long pause. Ventricular arrhythmias are among the commonest arrhythmias that occur in patient with or without heart disease. The peripheral pulse associated with such episodes is known as pulsus bigeminus. The most feared complication is its potential to develop into ventricular fibrillation and asystole. Hypokalemia and light plane of anaesthesia are some of the few correctable causes of ventricular bigeminy.<sup>[1]</sup> Early and appropriate management of these causes will prevent it from progressing into cardiac arrests.

**CASE REPORT:** A 30 years old female weighing 50 kg was diagnosed with complex Rt. Ovarian cyst and was admitted in Gynaecology ward of our hospital. She had symptoms of white discharge for 1 year duration which was neither foul smelling nor blood stained. She had no history of diabetes or hypertension. Her pre-operative potassium level was 4.2meq/L, and Haemoglobin was 11.9g/dl. Her Liver function tests, renal function tests and Thyroid function tests were normal. Her chest x-ray PA view and ECG were well within normal limits.

Since the patient reportedly had complaints of dyspnoea and palpitation frequently in the past 5 years she was referred for a cardiologist opinion. Echocardiography results showed that there was mildly dilated LA (4.0cm), with thickened anterior mitral leaflet and posterior mitral leaflet. There was restricted mobility of posterior mitral leaflet. There was moderate to severe mitral regurgitation and the ejection fraction was about 68%. There was no mitral stenosis, aortic stenosis or regurgitation, no pulmonary arterial hypertension, pleural effusion or clot. A diagnosis of Rheumatic Heart Disease with moderate to severe MR was made by the cardiologist

The patient was assessed under ASA III because of her cardiac problems 3 days prior to her surgery and general anaesthesia was planned for the surgery.

On the morning of the surgery day, the patient was given IE prophylaxis (Inj. Ampicillin 2gm iv and Inj. Gentamycin 80 mg iv) and shifted to the operation theatre. The patient was given general

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anaesthesia and monitored intra-operatively using Ecg, pulseoximetry and EtCO<sub>2</sub> monitor. The entire operative period was incident free.

After the operation, she was extubated smoothly. Recovery was normal with eye opening and the patient was responding to oral commands. The vitals of the patient was also normal with heart rate of 64/min and blood pressure around 100/60 mmHg. When she was about to be shifted to post-operative recovery ward, ventricular ectopics started. Initially there was 5 to 6 ventricular ectopic beats/min which gradually progressed to ventricular bigeminy. There were also small periods of normal sinus rhythm without any ventricular ectopics when the heart rate was around 45-50/min. During the ventricular bigeminy the heart rate almost doubled to around 90-100/min. Immediately 60 mg of 2% lignocaine i.v. was given to the patient but proved futile in controlling the ectopic.

After 5 min, 1mg of metoprolol was given and immediately the frequency of the ventricular ectopics came down to 5-6/min. Again 1mg metoprolol i.v. was repeated which completely stopped the ectopics. The patient was completely reverted to sinus rhythm within 10 min. All this time the patient was conscious and was obeying oral commands. The patient was shifted to the ICU and was monitored for 2 days for any signs of ectopics. Luckily there was no incidence of ventricular ectopic or bigeminy in that period. The serum potassium level was found to be around 3.6 meq/L in the immediate post-operative period. She was shifted to post-operative ward on the third day and was discharged on the 7<sup>th</sup> post-operative day.

**DISCUSSION:** There is said to be 60% incidence of cardiac arrhythmias particularly during general anaesthesia.<sup>[2]</sup> There is also increased potential for progression into ventricular fibrillation or intraoperative asystole. The cardiac risk is said to be increased in the perioperative period if there is more than 5 ventricular ectopics per minute.<sup>[3]</sup> Old age, previous cardiac pathology like myocardial infarction, digitalis toxicity, light plane of anaesthesia, hypokalemia and volatile agents are some of the causes of ventricular pre mature contractions.<sup>[1,4]</sup>

In this particular case the main attributing factor is the presence of mitral regurgitation which might have triggered myocardial ischaemia causing ectopic foci to arise from the ventricles leading to ventricular bigeminy. VPC's occurring after T wave precipitates ventricular tachycardia and ventricular fibrillation. This is the most dreaded complication of ventricular bigeminy which may cause sudden death.

Volatile agents increases the conduction time through the Bundle of His and Purkinje system thereby facilitating re-entry phenomenon.<sup>[5]</sup> There is stimulation of alpha 1 adrenergic receptors especially in the heart which results in the sensitization of the sympathetic receptors to the catecholamines and facilitates arrhythmia.<sup>[6]</sup>

Oculo cardiac reflex, a variant of Trigemino-cardiac reflex (TCR) is also reported to cause Ventricular bigeminy.<sup>[7,8]</sup> Surya Kumar et al reported in their case by an increase in the ICP caused due to Valsalva Manoeuvre triggered TCR which due to stimulation of trigeminal nucleus caused the ventricular bigeminy.<sup>[9]</sup>

Any disturbance in the potassium balance causes cardiac arrhythmia.<sup>[10,11]</sup> Prominent U waves along with ST segment and T wave depression is a significant sign of hypokalemia.<sup>[12]</sup> In a hypokalemic patient, hyperventilation leads to respiratory alkalosis precipitating arrhythmias by further reducing potassium.<sup>[4]</sup> Hypokalaemia treatment has lead to the reversal of ventricular bigeminy as observed by Ganny et al in their case report.<sup>[4]</sup> But Wong et al have differed by not committing hypokalaemia as an independent risk factor for cardiac arrhythmias.<sup>[11]</sup>

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Inadequate analgesia causes sympathetic stimulation leading to outpouring of catecholamines especially adrenaline. This causes cardiac arrhythmias due to the overstimulation of sympathetic receptors in the heart.

Congestive cardiac failure, hypoxia, hypercapnia, prolonged Q-T syndrome, atropine administration, transfusion of stored blood and alcohol ingestion are some of the lesser known causes of VPC's.<sup>[13,14]</sup>

For asymptomatic or mild symptomatic VPCs, patient education and reassurance, anxiolytic drugs and avoidance of aggravating factor like stress and caffeine-containing products is the recommended treatment.<sup>[15]</sup> Patients with no cardiac pathology but having high frequency of ventricular ectopics (>1000 in 24 hours) need long-term follow-up, with periodic reassessment of LV function.<sup>[16]</sup> For symptomatic patients with underlying cardiac pathology Beta-blockers and implantable cardiac defibrillators are considered as main therapy and catheter ablation is kept as adjunctive therapy.<sup>[15]</sup>

The definitive treatment is to treat the underlying cause such as correction of hypokalaemia, giving adequate analgesia and replacement to volatile agents having less arrhythmogenic potential as was done by Ganny et al in their patients.<sup>[4]</sup> Synchronised cardio version is needed in patients having ventricular tachycardia. In case of haemodynamic instability overdrive cardiac pacing should be considered. Corrective surgery is required for ventricular arrhythmias due to congenital cardiac disease.<sup>[17]</sup>

In our patient who had underlying cardiac pathology Inj. Lignocaine 60 mg i.v bolus dose which was used initially to treat proved to be futile with no effect. So we have used Inj. metoprolol 2mg i.v. in two divided doses which had immediate response by curtailing the ectopic and eventually abolishing the ventricular bigeminy.

**CONCLUSION:** Even though Ventricular bigeminy are easier to diagnose, vigourous monitoring and early recognition of the causal factors are the most important factor in the treatment. In this patient the earlier diagnosis of cardiac pathology during assessment and intraoperative monitoring for ventricular arrhythmias was important in the management of ventricular bigeminy and also prevented the possibility of the dreaded complication like ventricular fibrillation and cardiac asystole from arising.

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