



Managing a Case of Ventricular Premature Ectopic Beats: Arrhythmias of Unpredictable Nature: A Case Report

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Abstract: Cardiac arrhythmias occur very commonly in the peri-operative period. In otherwise normal patients undergoing routine non-cardiac procedures this finding is of great concern. The anaesthesia management of a case is discussed here, in which, the occurrence of arrhythmias was anticipated but not expected.

The preoperative clinical evaluation of a 55-year old 48 kg female patient posted for abdominal hysterectomy for uterine Fibroid revealed anaemia (Hb-6gm%), left axis deviation with single ventricular ectopic on ECG and mild cardiomegaly on X-ray chest. Before surgery her anaemia was corrected and expert opinion on ECG and X-ray was reported normal. She was subjected to spinal anaesthesia with 3ml 0.5% Bupivacaine heavy and lumbar epidural catheter was inserted. Intraoperatively patient developed ventricular premature ectopic beats (VPCs) progressing to bigeminy. She was treated with inj. Xylocard 50mg IV bolus followed by 100mg/hour drip and inj. Amiodarone 200mg bolus in 10ml of normal saline over 10 min .followed by 750mg in IV drip at 4drops /min. after a second bolus of 150mg. Postoperatively she was put on Tab. Amiodarone 100mg TDS; tapered to 100mg OD for one month.

This case illustrates multivariate nature of ventricular arrhythmias and the usefulness of pre, intra and postoperative evaluation. The importance of timely



management is stressed and various mechanisms likely to precipitate peri-operative arrhythmias are discussed.

Key words: Arrhythmias, Spinal Anaesthesia, Ventricular Premature Ectopic Beats. (VPCs)

One third of all post-operative complications and more than half the deaths are due to cardiac complications. Several studies have shown that peri-operative heart rate variability is a powerful predictor for postoperative morbidity and long-term mortality. It is therefore important to evaluate a patient critically, make judicious use of diagnostic tools and therapies for the benefit of the patient.

Case Report: A 55year old 48kg para one L1 with uterine fibroid was posted for abdominal hysterectomy. She had complaints of dysmenorrhoea since two years. Her menstrual history was 5/30 regular. No previous history of hospitalization for medical or surgical reason.

Patient was a moderately built middle aged lady. General examination result : pulse-90/min.regular,BP-118/62mmHg,there was pallor, slight pedal oedema, occasional third heart sound was heard in aortic area, RS was clear. Her Hb was 6gm%, platelet count-1.4lac/mm³, prothrombin time 16/15, blood sugar level-105mg%, blood urea level-25mg%, serum creatinine-0.8mg%. ECG showed left axis deviation and a single ventricular ectopic, chest X-ray showed mild cardiomegaly. She had received one blood transfusion two days ago. Therefore she was advised fresh Hb levels; and was referred to medicine to get opinion on X-ray, ECG and CVS findings. In the second pre-anaesthesia check-up after four days, medicine opinion was noted wherein patient was given fitness with Inj Vitcofol, iron preparations in tablet form. They had advised repeat ECG which showed a single ventricular ectopic in lead II and III each. CVS findings were reported to be normal. Gynaecologists had given her three blood transfusions. Fresh Hb level was 9.2gm%.

Patient was subjected to spinal anaesthesia with 3cc of Bupivacaine 0.5% heavy and lumbar epidural catheter inserted. Pulse, BP, respiration, SpO₂, ECG, and level of consciousness were monitored. Anaesthesia up to T7 spinal level was confirmed when surgery started. Patient was sedated with injection Midazolam 0.5mg IV.

Ventricular ectopic beats started appearing on ECG, initially 5-6/min. for about a minute and rapidly progressed to more than 20 VPCs/min. 100% O₂ was started on mask via Bain's circuit, BP was 120/76mm Hg. Injection xylocard 50mg IV bolus was given slowly and Inj. xylocard drip at 100mg/hour was started. VPCs disappeared for 15 minutes, ECG was a normal record with occasional intermittent VPCs, BP was 110/70mm Hg. Patient was receiving 100% O₂ on mask and was absolutely comfortable without sweating, nausea, pain at site. When spinal level receded to T10 she had generalized vague abdominal discomfort so 3ml inj. lidocaine 2% without adrenaline was given through epidural catheter and sensory

level T8 was achieved. Inj. Xylocard was stopped and Inj. Amiodarone 200mg IV bolus in 10ml of normal saline was given over a period of 10min. ABG showed pH = 7.32, pCO₂ 40mm Hg, pO₂ 381mmHg, S Na⁺ 124mEq/lit., K⁺ 3.9mEq/lit., Bicarbonate 20.6mmol/lit., B.E. -5.1mmol/lit., Sat.100%. There was no change in ECG, patient was progressing to Ventricular bigeminy (fig.1). DC cardioversion was kept ready and inj. Amiodarone 750mg in IV at 4 drops/min. was started after giving 150 mg bolus. Patient was monitored on OT table for one hour and was shifted to medical ICU. She developed 2 ectopics during transit. ECG in ICU as shown in the figure (fig.2).

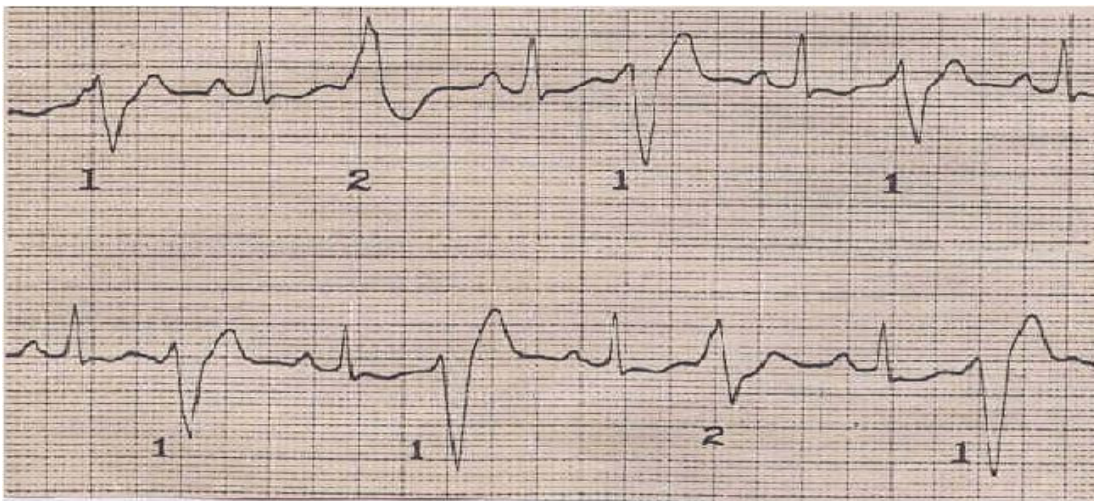


Fig 1 : Ventricular Bigeminy - (VPC from Foci 1 and 2)

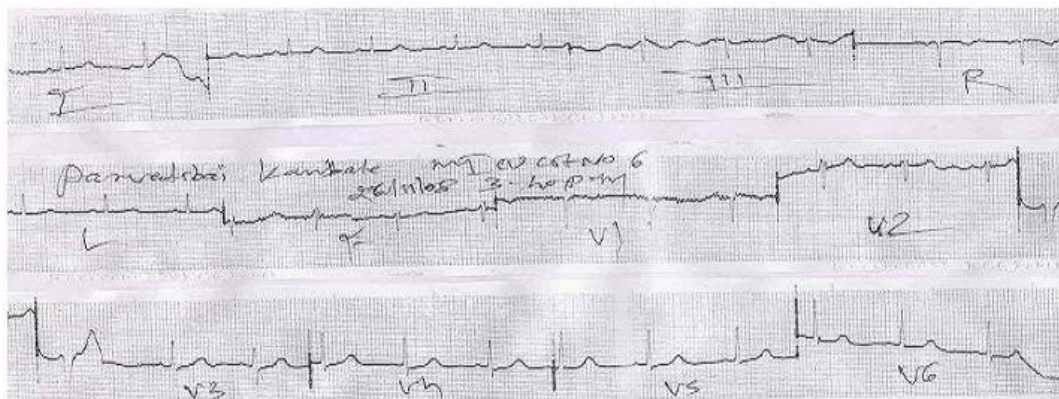


Fig 2 : Post operative ECG

Next day morning, pulse, BP, ECG, ABG findings were normal; patient was shifted to ward, 2D ECHO was advised and patient put on tablet amiodarone 100mg TDS. 2D ECHO was normal with trivial MR, trivial TR, and no regional wall motion abnormality. She was advised to taper tab. amiodarone to 100mg OD for one month. Stress test and review was



asked for after two months. Patient was discharged safely on the seventh day of surgery and daily ECG record showed no ectopics.

Discussion: American Heart Association guidelines focus on evaluation of patients undergoing non-cardiac surgery to reduce the risk of perioperative morbidity and mortality. In asymptomatic patients more than 50 years of age, extensive assessment of history and physical examination is stressed. Our patient was anaemic and anaemia imposes a stress on CVS that may exacerbate myocardial ischemia and heart failure. Haematocrit <28% is also associated with increased incidence of perioperative ischemia. Her ECG showed a single ventricular ectopic and left axis deviation which was not considered to be abnormal. For the cardiomegaly on X-ray chest, pedal oedema and occasional third heart sound, physician's opinion was asked. Factors which predispose this patient to QT-prolongation and a higher risk of resulting arrhythmias are her older age, female sex, myocardial dysfunction due to ventricular dilatation and impaired contractile function. Electrolyte abnormality like hypokalemia, hypomagnesemia are known to cause arrhythmia along with some genetic predisposition which control intrinsic myocardial properties or signalling pathways.

M Hinteseer and M Iribeck have stated that even with normal cardiac structure and function, serum potassium within reference limits and in absence any other QT prolonging medication there was a marked increase in QTc interval upon challenge with DL-Sotalol (2mg/kg). A mis-sense mutation in the form of nucleotide change in the gene encoding the alpha-subunit of human repolarising potassium channel was found to reduce the myocardial repolarisation reserve. They have recommended that awareness and understanding the mechanisms of long QT-syndromes help to identify patients at risk and reduced their exposure to risk factors.

Arrhythmias from local anaesthetic over dosage are known to occur. It is an uncommon but catastrophic complication of regional anaesthesia. Cardiovascular toxicity under regional is mostly reported due to accidental IV injection of Bupivacaine during attempted epidural or brachial plexus block. Bupivacaine depresses the ventricular contractility, causes vasomotor paralysis, and inhibits medullary vasomotor centre. Bupivacaine is known to inhibit the basal and the epinephrine stimulated cAMP production. This limits the success of resuscitative measures used to treat the toxicity.

In this case the dose of Bupivacaine used was not expected to be a toxic one. This volume is generally used for most routine surgeries. Cardiac arrhythmias, especially supra ventricular tachycardia are a well recognized complication after non-cardiac thoracic operations. A relatively sympathotonic status caused by injury to cardiac parasympathetic nerves due to surgical manipulation increases the incidence of arrhythmia.



Post operative sympathetic blockade caused by thoracic epidural decreases the occurrence of tachydysrhythmia has also been reported. Sympathetic blockade is usually 2-6 segments higher than the sensory level. Therefore complete blockade of cardiac accelerator fibres (T1 to T4) is expected if the level of block ascends to T6 level. This, along with paradoxical Bezold-Jarisch reflex, which stimulates mechanoreceptors of left ventricle cause bradycardia. Our patient showed absolutely normal ECG intermittently and maintained BP throughout. Her spinal level at no time reached T6 throughout surgery. The possibility of intravascular injection of Bupivacaine used for spinal is ruled out as flow of CSF was demonstrated clear and the onset of arrhythmias was not immediate. Intravascular placement of epidural catheter is also less likely and drug used epidurally was only 3ml Lignocaine, without adrenaline.

Compromised autonomic nervous system can be a cause. There is a continuous alteration in and interaction between parasympathetic and sympathetic nervous system. Baroreceptors, vasomotor centre, respiratory centre, arterial B.P., respiratory movements constantly modulate autonomic nervous system (ANS) leading to beat to beat fluctuations via SA node. Thus perioperative heart rate variability largely affects morbidity and mortality.

The arrhythmia that this patient showed was of polymorphous ventricular tachycardia type. The axis of ventricular complexes changed its polarity, back and forth. Few peaks pointed upwards and then downwards. The goal of our therapy was to lengthen myocardial refractoriness so that during retrograde conduction, the myocardium is already refractory at the site where impulse returns. Therefore Xylocard and Amiodarone were chosen. All anti-arrhythmics are potentially arrhythmogenic. It is therefore widely accepted that only symptomatic and life threatening arrhythmias should be treated. To prevent sudden death, asymptomatic VPCs or runs of polymorphic ventricular tachycardia require treatment with monitored plasma levels. Interactions with other drug, diseased states should be looked for with caution. Exercise testing on a new anti arrhythmic agent before discharge may give evidence of its safety, has also been reported. With long term treatment given to our patient it is necessary to monitor its long lasting after-effects. The reported side effects are pronounced and therefore fatal recurrence of arrhythmia or sudden death due to bradycardia is feared.

Conclusion: The incidence of perioperative arrhythmias is 90% with patients undergoing cardiac surgery and 70.2% in other surgeries. The emphasis is on the acute management. The presence of structural heart disease and electrolyte imbalance provide a substrate for abnormal automaticity. Hypoglycaemia is reported to give rise to ventricular tachycardia by manifesting as increased sympathetic discharge due to neuroglycopenia. The treatment therefore includes corrective intervention for preventing the imbalance.



Anticipation and prevention of complications along with their early diagnosis and treatment are the most important factors in dealing with regional anaesthesia risks.

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