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Anaesthetic Management of a Case of Multiple Ventricular Premature Contraction Posted for Spine Surgery: A Case Report

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Abstract: ***Ventricular premature contractions (VPCs) are ectopic beats arising from the ventricular myocardium below the AV node. VPCs which are occasional and presents with no symptoms are very common in daily clinical practice. However, VPCs which are multiple (>5) or are associated with cardiac anomalies can progress to life threatening arrhythmias. Inhalational agents, electrolyte and metabolic abnormalities under anaesthesia can precipitate multiple VPCs. Here, we present a case of a 29 years old male patient with occasional VPCs posted for implant removal of lumbar spine in our institution. After 30 minutes of starting surgery under general anaesthesia, patient developed multiple VPCs with hemodynamic instability which was managed successfully by prompt treatment with injection loxicard and injection metoprolol iv. and surgery went uneventful. This case report highlights various factors which could precipitate VPCs under anaesthesia and concludes that judicious and prompt use of anti - arrhythmic agents can prevent an asymptomatic VPCs transforming into fatal arrhythmias.***

Keywords: Ventricular premature contraction, arrhythmias, Holter, ECHO, Hypoxia, hypokalemia

1. Introduction

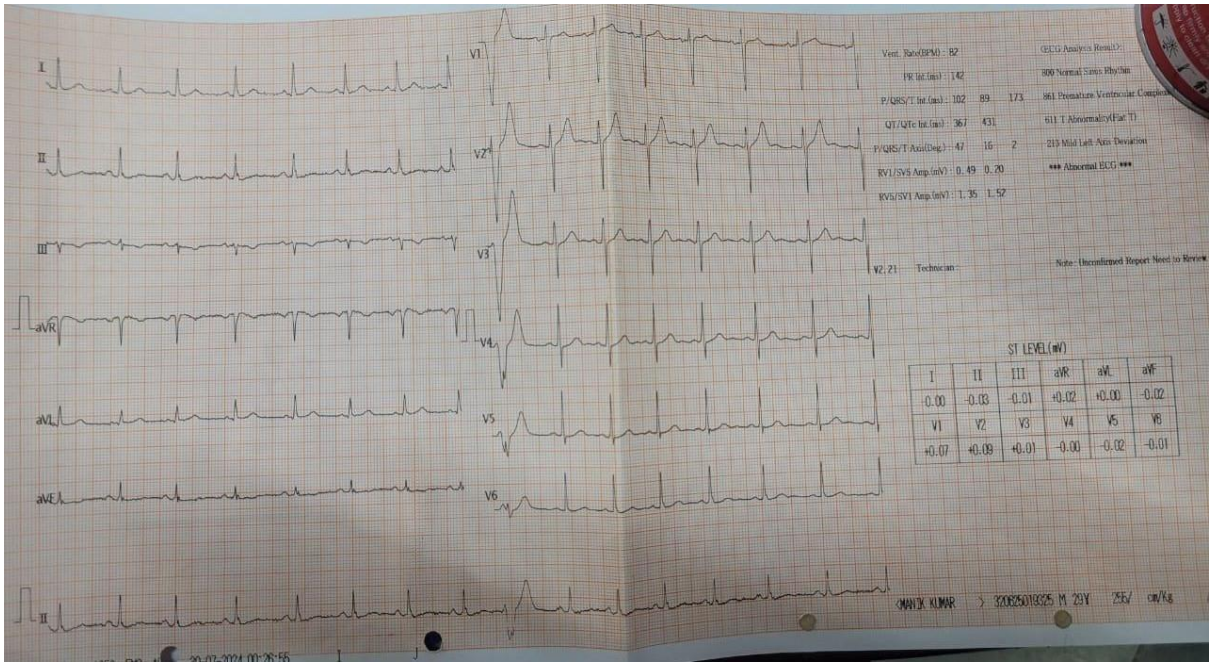
Ventricular premature contractions (VPCs) are ectopic beats arising from ventricular myocardium below the atrioventricular node (AV). It gives rise to wide bizarre QRS complex. Non cardiac causes include stimulants like caffeine, metabolic and electrolyte abnormalities. In the absence of heart disease, VPCs are associated with little or no increase in developing into a dangerous arrhythmia. [1] Multiple VPCs can cause upsetting symptoms in patients and can lead to life - threatening arrhythmias. [2] This case report can provide the reader, an approach to manage a case of multiple ventricular premature contraction complicating a spine surgery.

2. Case Report

A 29 - year - old male patient was posted for implant removal of lumbar spine L5. The patient had burst fracture of the L5 spine 5 years back for which he was operated and screw fixation was done in emergency which went uneventful.

In pre anesthetic checkup vitals were, pulse rate - 100/min, BP - 130/80mm hg, RR - 20/min, SPO2 - 100% on room air. Chest - B/L clear with no added sound, METS >4, MP grade was 1. He had no associated co - morbidities.

All the investigations were within normal limit, except ECG which showed sinus tachycardia and ectopics as shown in lead V1 to V6. (fig - 1)



Patient was referred for cardiology opinion. Holter, Echo and cardiac MRI was advised by cardiologist. Echo was normal

with left ventricular ejection fraction 60%, MRI was normal and Holter showed frequent VPCs including couplets and quadrigeminy.

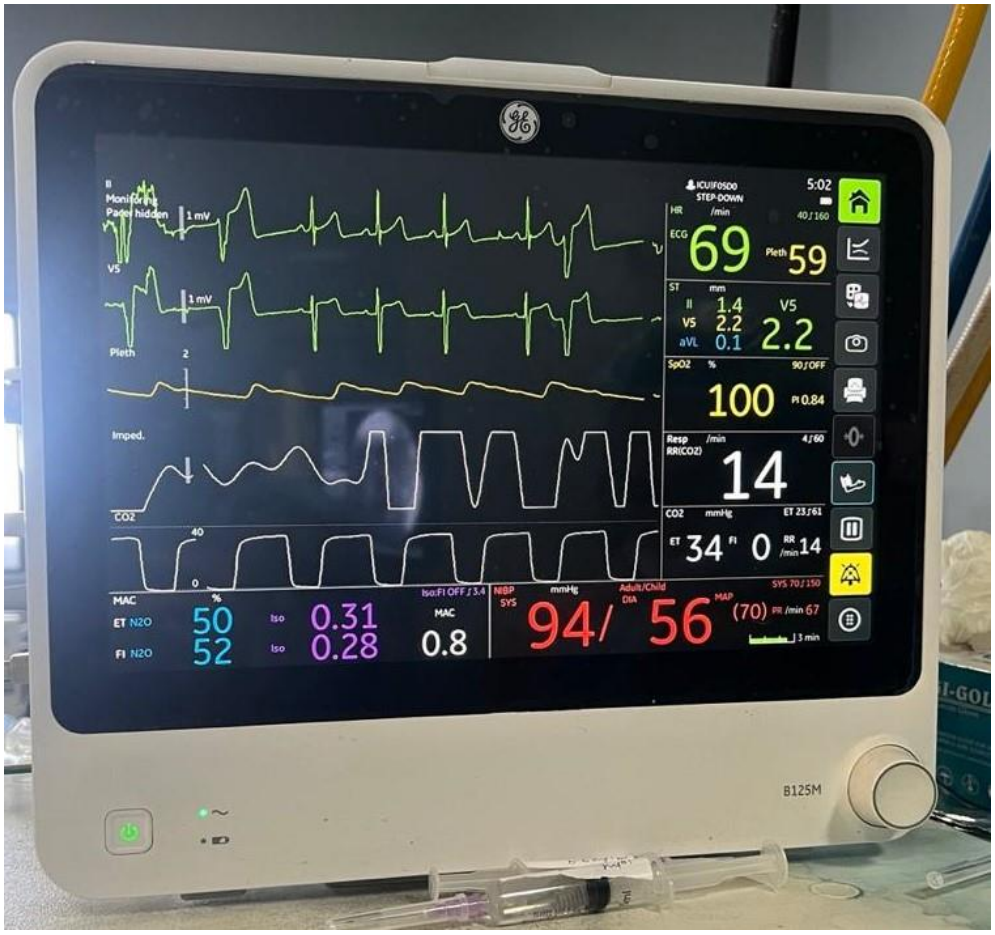
Patient was advised tab bisoprolol 2.5 mg OD after breakfast. Surgery was postponed for one week. After one week ECG was done and it showed 1 - 2 VPCS/minute but patient did not have any clinical symptoms. The risk of delaying surgery for the complete optimization of the heart condition was weighed against the risk of anaesthesia and it was decided to take up the case after explaining the high cardiac risk.

After obtaining informed and written high - risk consent, patient was shifted to OT and all routine ASA standard monitors were attached and iv access was secured. Baseline hemodynamic parameters were as follows: HR - 78/min, BP - 128/76 mmhg, and respiratory rate - 20/min, SPO2 - 100%. The 3 lead ECG monitor displayed sinus rhythm with 2 - 3 VPCs/minute. Inj glycopyrrolate 0.2 mg im was given in the preoperative room 20 minutes before taking up the case. Patient was premedicated with inj midazolam 1.5 mg iv and fentanyl in a dose of 2mcg /kg iv. After 3 mins of preoxygenation patient was induced with inj propofol 2mg/kg iv. Muscle relaxation was done with inj vecuronium 8 mg iv followed by tracheal intubation with wire reinforced endotracheal tube size 8. Anesthesia was maintained with isoflurane, N2O and oxygen along with intermittent boluses of vecuronium and fentanyl.

Patient was handed over to the surgeon after proper positioning and padding in prone position and surgery started.

We encountered 1 - 2 VPCs/min with stable hemodynamic parameters initially. No treatment was given. After 30 minutes of starting surgery in prone position, number of VPCs were more frequent (Observed>10/min) for which injection lignocaine 1.5mg/kg iv was given and infusion started at 2 mg/hr. We continued with surgery, suddenly patients started developing more frequent VPCs worsening and amounting to slow ventricular tachycardia / idionodal rhythm along with hypotension and bradycardia. (Fig - 2)

We stopped Isoflurane. After the telephonic consultation with cardiologist inj metoprolol 5 mg was given IV. After giving injection metoprolol 5 mg iv, no of VPCs reduced to 2 - 3 per minute. (fig - 3)



With the normalization of ECG rhythm, blood pressure of patient also improved. Anaesthesia was maintained by propofol infusion. Surgery was continued for total 3 hrs and the patient was extubated after giving reversal in deep plane of anesthesia. Patient was transferred to ICU for observation and was discharged on post operative day five.

3. Discussion

VPCs arise from abnormal site in the ventricular myocardium and give rise to wide bizarre QRS complex. The VPCs are common in daily clinical practices and these patients are largely asymptomatic. Premature ventricular complexes are more common with increasing age and in conditions like coronary artery insufficiency, myocardial infarction, valvular heart disease. Non cardiac causes include caffeine, cocaine, alcohol, acidosis, hypoxemia and electrolyte abnormalities. [3] New - onset VPC should be considered life - threatening as it may progress to serious ventricular arrhythmias like tachycardia and ventricular fibrillation. [4] VPCs are more likely to lead to ventricular fibrillation if they are multiple (10 or more ectopic impulses/min), multifocal or bigeminal; occur near the vulnerable period of the preceding ventricular repolarization (R - on - T phenomenon); or appear in short - long - short coupling sequences, [4, 5] or the presence of impulse salvos (i. e, 3 - 5 consecutive impulses).

It is important to find out whether underlying structural heart disease is present and left ventricular function is impaired. The routine investigations should include resting 12 lead ECG, Echocardiography, ambulatory Holter recordings and exercise tolerance test.

Echocardiography is important as both ventricular function and the presence or absence of structural heart disease are important consideration in assessing the need for further interventions and treatment [2]. Structural heart disease and poor left ventricular function are the key factors in determining the need for treatment and assessing the prognosis. [6, 7]

VPCs during anaesthesia can be caused by multiple factors such as electrolyte and acid base disorders, hypokalemia, hypoxia, hypercarbia, hypothermia and light plane of anaesthesia.

The management of VPCs start with treating the underlying causes. Asymptomatic and healthy patients with occasional VPCs do not need treatment. However, multiple VPCs (>5 beats/min), or VPCs with haemodynamic disturbance require prompt treatment. Beta blockers are the safest initial choice [1]. I. V lignocaine in a dose of 1.5 mg/kg followed by 1 - 4 mg /min can also be used for its membrane stabilizing effect that can prevent a sustained VPCs from culminating into a ventricular tachycardia. Esmolol, propranolol, procainamide, quinidine, disopyramide, atropine, verapamil, and overdrive pacing are other therapeutic modalities. [3, 4]

Anaesthetic agents, surgical stress and light plane of anaesthesia can precipitate VPCs even in a normal patient. In this case also, we attribute anaesthetic agent as the cause of multiple VPCs in a patient who already had occasional VPCs with no known cause. There is various literature which suggests the association of volatile anaesthetic agents and abnormal cardiac rhythm in patients under anaesthesia. [8, 9,

10] Volatile anaesthetics has potential to cause bradycardia, AV conduction abnormality and prolongation of QTc interval. Reversible AV dissociation has been reported in humans anaesthetised with N_2O and volatile anaesthetics. [11] In this patient cardioversion was not required as prompt recognition and pharmacological treatment of the case restored sinus rhythm. We continued surgery and it was finished uneventful, however it is prudent that such cases with ECG and Holter showing VPCs should be optimised preoperatively and decision to proceed with elective surgery should be based on multidisciplinary inputs from cardiologist, anaesthetists and surgeons.

Anaesthetic risk is largely related to the severity of underlying ventricular dysfunction. However, we must treat all the underlying causes which precipitates VPCs under anaesthesia and must not forget the role of inhalational anaesthetics in the causation of abnormal cardiac rhythm. However, further studies are needed to reaffirm this hypothesis.

4. Conclusion

Ventricular premature contractions are very common during anaesthesia. There are various factors responsible for causing VPCs under anaesthesia. The anaesthesiologist should rule out all the possible causes of arrhythmia during intraoperative period before starting specific intervention. Patients with preoperative VPCs should be thoroughly investigated and optimized to rule out any cardiac pathology. Managing such cases requires multidisciplinary inputs. A vigilant intraoperative monitoring and prompt treatment avoids an impending cardiac mortality in such cases.

Declaration

Conflict of interest - nil

Funding - nil

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