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Suspected case of amisulpride induced peri-operative ventricular bigeminy with bradycardia: A case report

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ABSTRACT
A patient who had perioperative ventricular bigeminy with bradycardia while undergoing total abdominal hysterectomy is reported. Diagnosis of bigeminy was confirmed by ECG. The cause of such an arrhythmia is thought to be due to amisulpride. The patient was successfully managed symptomatically. The importance of proper clinical evaluation and vigilant intraoperative monitoring has been highlighted.

KEYWORDS: Amisulpride; Bradycardia; ventricular bigeminy**;** premature ventricular complex

I. INTRODUCTION

During general anaesthesia, the incidence of cardiac arrhythmias is as high as 60%.[1] Bigeminy is a type of cardiac arrhythmia where normal heartbeat is followed by an ectopic beat resulting in what is commonly known as "coupled beats". The peripheral pulse during such episodes of arrythmia is known as pulsus bigeminus. Clinically bigeminy might be innocuous unless it does not cause haemodynamic instability as it has the potential to convert into ventricular fibrillation or asystole. Correctable factors such as sensitisation of heart by some volatile anaesthetics, hypomagnesemia, hypokalaemia, hypoxia, light plane of anaesthesia and inadequate analgesia have been implicated in the aetiology of arrhythmias.[1,2] Post operative nausea and vomiting (PONV)is one of the commonest complain other than pain ranging to almost 30% of the general population.[3]Female gender and previous history of PONV are one of the risk factors. Amisulpride is a dopamine antagonist which is used in psychosis. Now studies have shown that it is efficacious in PONV prophylaxis and has a better safety profile as compared to other drugs used for PONV.[4–7]In our case, the patient had a history of PONV and hence we decided to administer amisulpride which is now recommended for PONV prophylaxis.[4–7] Patient had no previous exposure to amisulpride. There are at least 3 studies showing patients developed bradycardia after oral amisulpride intake but here we are reporting a case of ventricular bigeminy induced bradycardia attributed to amisulpride.[8–10]

II. CASE REPORT

A 62years old, 85 kg female patient, a nurse by profession, posted for total abdominal hysterectomy with a history of hypertension since 1year on tablet telmisartan 40mg once daily and tablet cilnidipine 10mg once daily and recently diagnosed hypothyroidism on tablet levo-thyroxine 25mcg once daily. Patient was diagnosed with covid-19 in 2020 and received treatment in critical care unit for 7 days. Patient had previously undergone bilateral tubal ligation under total intravenous anaesthesia, appendicectomy under general anaesthesia and dilatation and curettage under total intravenous anaesthesia. Previous surgeries were mainly uneventful with history of post op nausea vomiting (PONV) during tubal ligation and appendicectomy. The patient also gave a history of an episode of throat uneasiness after she tried to gargle tablet aspirin mixed in warm water for throat irritation in 1995 without any medical advice. Patient had mild dyspnoea on exertion which was consistent with her age and built with MET>4. Preoperative physical examination and routine investigations were done and all were within acceptable range. After discussing the anaesthetic management plan with the surgeon and patient, consent was obtained for the same. During the PAC, her main concern was PONV and we had assured her that we would look into the matter and the drug amisulpride was discussed with her. The patient was given 0.5 mg of tablet clonazepam the night before surgery and tablet ranitidine 150mg the night before surgery and the morning of surgery. Antibiotic ceftriaxone 1gm was given intravenously 1hr before surgery. The patient was taken in and all ASA standard monitors were connected. The base line pulse was 70/min and the blood pressure was 150/90 mm Hg. Injection amisulpride 5mg was given as an infusion over 20 minutes. Preoxygenation was done for 3

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mins with 100% oxygenation. Pre medication was done with injection fentanyl 100mcg. Induction was done with injection ketamine 25mg, injection propofol 110mg and injection vecuronium 4mg IV. A cuffed endotracheal tube of size ID 6.5mm was inserted and position was confirmed by chest auscultation and EtCO2. The patient was maintained with O2, N2O, sevoflurane and IPPV. Injection dexamethasone 4mg was given intravenously post induction. There was no significant hemodynamic change during intubation or till 15 minutes of induction after which there was sudden onset of ventricular bigeminy associated with bradycardia and the pulse well down to 32/min. Injection atropine 0.3mg intravenous was given and the pulse bounced back to 60 with disappearance of bigeminy, only to reappear for two more episodes during a 2hour period. The blood pressure remained stable throughout and there were no clinical manifestations to suggest pain. There were no signs of skin manifestation, rise of body temperature, change in EtCO2 or oxygen saturation. The patient was reversed using injection neostigmine 2.5mg and injection glycopylorrate 0.4mg intravenous and extubated after thorough oropharyngeal suctioning and shifted only after achieving necessary clinical parameters. Patient was concious, oriented and mostly pain free when she was shifted to post op room with pulse 72/min, blood pressure of 140/80mmHg and saturation of 97% with oxygen via nasal cannula. 5 mins after shifting, the pulse of the patient became irregular with missed beats. The lead II and V5 of ECG showed vetricularectopics of bigemy pattern with heart rate fluctuating from 30-50/min. Injection atropine 0.3mg intravenous was given and the heart rate improved from to 70s transiently, only to revert back to bigeminy & back to sinus rhythm every 5-10 minutes, spontaneously. A 12 lead ECG was done at this point and the patient was kept under observation for 1 hour before she was shifted to critical care unit and kept under observation [Figure 1]. These episodes lasted for a total period of 10 hours before reverting back to normal. Repeat ECG after 10 hours was done and it was similar to that of the preoperative pattern [Figure 2]. Next day the patient was shifted to ward and holter monitoring was done which was suggestive of a normal sinus rhythm with a ventricular ectopic. The serum electrolytes namely sodium, potassium and calcium all were within normal limits. The pain was well controlled with opioid and then non opioid analgesics.

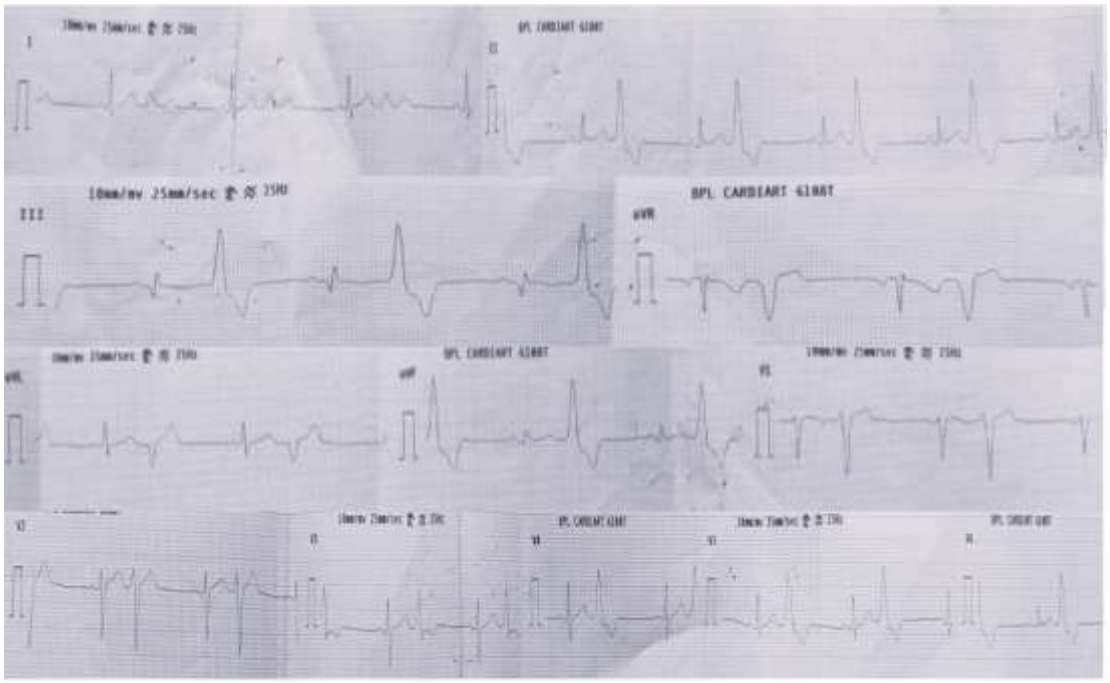

Figure 1: Post-op ECG just after shifting the patient to post op room

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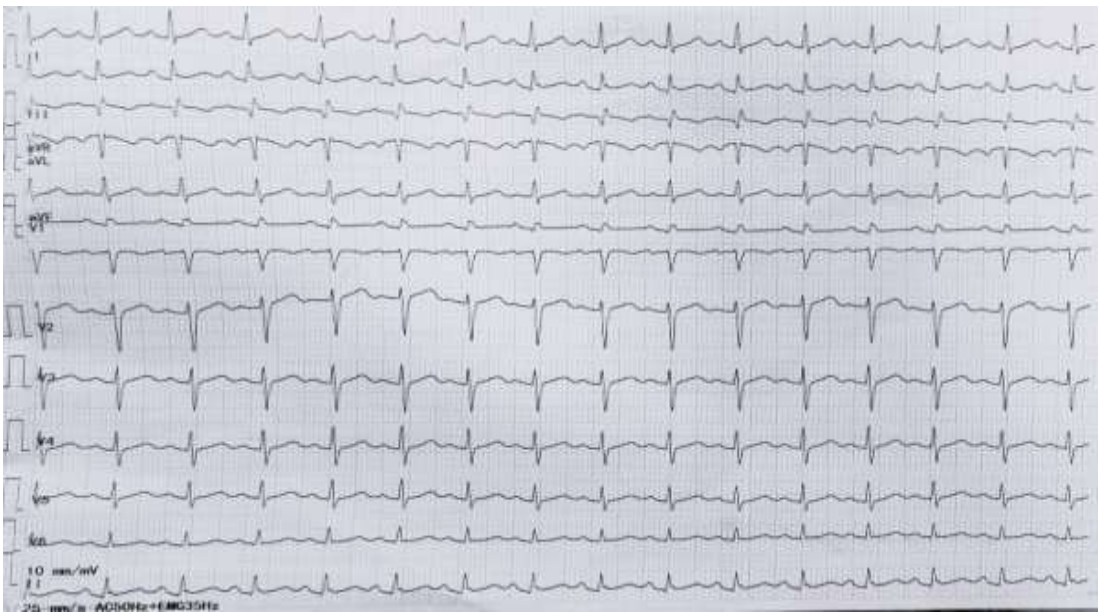

Figure 2: ECG after 10 hours post op in critical care unit

III. DISCUSSION

Amisulpride, a potent dopamine (D_2 and D_3) receptor antagonist is already in use in oral form for psychosis. It is grouped under the category of atypical anti psychotics. It has a good safety profile with a reduced incidence of extrapyramidal, central nervous system, cardiovascular and gastrointestinal side effects. It has an additional benefit of very minimal QT prolongation in the therapeutic range when used for PONV which is a major concern with droperidol.[3–7]There are been multiple studies showing the efficacy of the drug in PONV prophylaxis as well as a rescue antiemetic at various doses like 1mg, 5mg, 10mg and 20mg with good success rates. After an intravenous administration, the peak plasma concentration is achieved at the end of the infusion period and the plasma concentration decreases by 50% within almost 15 minutes.[5] The drug undergoes very minimal metabolism.Following intravenous administration, two thirds is excreted in urine and one third is excreted through faeces. The mean elimination half-life is almost 4-5 hours. Most of the side effects associated with amisulpride is reported mostly in oral use that too in a dose above 600mg alone or in combination with other antipsychotics. Very few case reports on the complications have been published so far that mainly includes QT prolongation, hypertension, hypotension, arrhythmia or bradycardia.[3–7]There have been multiple previous publications describing bradycardia but not at the dose given for PONV prophylaxis. The ventricular premature complexes (PVC) might arise from one or multiple foci with typical ECG findings of wide QRS, absence of preceding P wave, opposite deflection of ST segment and a compensatory pause. Two or three PVCs separated by one normal sinus beat is called bigeminy. Three consecutive PVCs is generally considered ventricular tachycardia and are dangerous if occurring in the middle third of the T wave as it might result into ventricular tachycardia or fibrillation. Palpitations, syncopal attacks are commonly associated with PVCs but our patient had no such complains. Patients with underlying cardiac issues are at a higher risk. Use of defibrillator should be considered in case of life-threatening arrhythmia leading to hemodynamic instability arising from multiple PVCs. Antiarrhythmics should be used only if PVCs progresses to ventricular tachycardia. β-blockers are the most commonly used drugs in ventricular ectopics.[2] In our case, the arrythmia was associated with bradycardia and the VPCs subsided once bradycardia was corrected with atropine.

IV. CONCLUSION

Our case demonstrates the chance of bradycardia and arrhythmias even in lower doses of amisulpride as a side effect. It stresses upon the need to evaluate all perioperatively administered drugs and not only those known to cause side effects.

CONFLICT OF INTEREST

There are no conflicts of interest.

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