Sustained Ventricular Arrhythmias in an Asymptomatic Child Posted for Laparoscopic Rectopexy: An Anesthetist's Dilemma?

The Editor,

Sustained ventricular premature complexes (VPCs) are rare in the pediatric population, especially with structurally normal heart. We would like to describe a case where we observed sustained ventricular arrhythmias in an asymptomatic child posted for surgery.

A 14-year-old boy weighing 56 kg (body mass index-24.49) with a history of rectal prolapse was posted for laparoscopic rectopexy under general anesthesia. Preanesthetic evaluation was unremarkable. He was premedicated in the preoperative area with 1 mg of intravenous (IV) midazolam. Anesthetic induction was done with 250 mg of IV thiopentone sodium, 100 μg of IV fentanyl, and 25 mg of IV atracurium. After securing the airway with a 7.0 mm uncuffed endotracheal tube, anesthesia was maintained with oxygen, air, isoflurane, and intermittent positive pressure ventilation. Ventricular ectopics with a heart rate of 70/min were observed on the monitor. There was no response to IV glycopyrrolate 0.2 μg, IV atropine 0.6 mg, or 50 mg of IV xylocard. Instead, it soon progressed to ventricular bigeminy with a blood pressure of 88/60 mmHg. The case was cancelled due to sustained arrhythmias. Postoperatively, the patient was hemodynamically stable with normal sinus rhythm. On further cardiac evaluation, he was found to have resting VPCs which disappeared on exercise. Echocardiography revealed a structurally normal heart. The child was started on oral metoprolol 25 mg twice daily as advised by the cardiologist.

He was posted for the same surgery after a week. Preinduction, electrocardiography (ECG) showed ventricular ectopics with hemodynamic values in the normal range. Two large bore IV cannulas and arterial line were secured. He was induced with IV ketamine 100 mg, 100 μg of IV fentanyl, 0.6 mg IV atropine, and 25 mg IV atracurium. Anesthetic plane was deepened with isoflurane and IV xylocard 60 mg was given before laryngoscopy. Postinduction, the patient developed ventricular bigeminy with a heart rate of 60–80 beats/min and blood pressure of 80–90/50–55 mmHg [Figure 1a].

Some of the common causes for ventricular arrhythmias such as lighter planes of anesthesia, hypoxia, hypercarbia, acidosis, and dyselectronemias were ruled out. There was no response to treatment with IV calcium gluconate 1000 mg and IV xylocard 60 mg. Isoprenaline infusion was started at 0.1 μg/kg/min for its nonselective beta agonistic action to increase the heart rate and also to reduce QT prolongation. There was only transient response to isoprenaline infusion. Hence, surgeons were persuaded to abandon laparoscopic rectopexy and sclerosant was injected. Postextubation, his

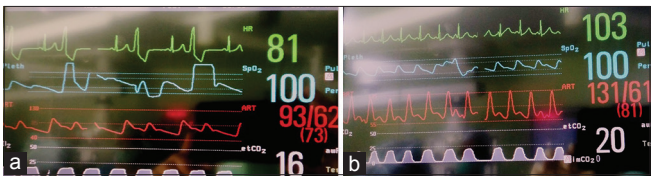

Figure 1: (a) Postinduction, electrocardiography showing ventricular bigeminy figure. (b) Postextubation, electrocardiography patient did not have ectopics

heart rate was 103 with a blood pressure of 131/61 mmHg with no ectopics [Figure 1b]. He was advised to undergo a complete workup with the cardiologist and endocrinologist.

Although most VPCs are benign in pediatrics, they can be due to various causes such as congenital heart disease, cardiomyopathies, prolonged QT syndrome, electrolyte abnormalities, and Brugada syndrome.[1] A reference to cardiologist is necessary when the diagnosis is new or ECG abnormalities are seen. On the first occasion, our patient was incidentally detected to have ventricular bigeminy on induction of anesthesia. Cardiologist opined it to be of benign origin as it disappeared on exercise.[2,3] Hence, he was taken up for surgery the second time, but ventricular bigeminy was still observed with hemodynamic values in lower limit of normal with no response to treatment.

The natural history of VPCs in children with an anatomically normal heart differs depending on the origin of the VPCs. They are classified as VPCs with right bundle branch block (RBBB), VPCs with left bundle branch block, catecholamine-induced ventricular tachycardia, and accelerated idioventricular rhythm (AIVR). VPCs originating from the left ventricle (VPC with RBBB) usually disappear during childhood. In contrast, VPCs originating from the right ventricle and AIVR are reported to be significant and should be regularly followed by a cardiologist.[4,5]

With regard to our patient, he suffered from obesity which may lead to ECG abnormalities and left ventricular hypertrophy. However, postoperative echocardiogram revealed a structurally normal heart. Perioperative adrenergic stimulation causing ventricular ectopic activity was offset with IV midazolam, adequate depth of anesthesia, and analgesia.

It is noteworthy that many anesthetic and antidysrhythmic agents have prodysrhythmic effects and prolonged QT interval. In fact, the prolongation of QT interval can itself precipitate progression of VPCs to dysrhythmias.[6] Atropine should be contemplated in such cases as it can prolong QT interval and may be the cause for worsening of ECG rhythm.[7,8]

In conclusion, we consider the progression of ventricular ectopic to bigeminal rhythm to be an atropine-induced ventricular bigeminy, emphasizing the fact that routine use of antiarrhythmic agents is not indicated in asymptomatic patients. Thus, it is a concern for the anesthesiologist to differentiate children with benign arrhythmias in whom anesthesia can be safely induced and those cases at risk which need to be postponed for further follow-up.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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