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Case Report Open Access

Atropine-Induced Bigeminy by Treating Bradycardia-Dependent Ectopy

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Abstract

We report the case of a 9-year-old healthy boy who underwent day-surgery herniotomy under combined general and regional anesthesia. After inhalational induction, borderline bradycardia with Premature Ventricular Contractions (PVCs) was observed in ECG tracing, which worsened despite the adequate anesthetic management to avoid and treat arrhythmogenic triggers. Subsequent diagnostic tests were performed to rule out an underlying heart disease. Thereby, we conduct a brief review of ventricular ectopy in children with structurally normal hearts, both in the clinical and perioperative setting; and finally linked to our case in order to find out the cause of the abnormal electrical activity.

Keywords: Pediatrics; Anesthesia; Ectopy; Premature ventricular contractions; Bigeminy; Atropine

Introduction

Ventricular ectopy consists in extra impulses originating from an area distal to His–Purkinje system. It´s classified as simple (PVCs) or complex (polimorphic PVCs, bigeminy, couplets, non-sustained tachycardia). PVCs are the most common ventricular arrhythmia in pediatric population, varying the prevalence with age in healthy children: 15-20% of neonates/infants, 10% of children and 20-30% of adolescents. Ventricular ectopy can be related to cardiac causes like heart diseases (structural or functional abnormalities) or cardiac surgery; and non-cardiac causes like electrolyte disorders (hypokaliemia, hypomagnesemia, hypercalcemia), pH alterations (acidosis, alkalosis), drugs (antidepresants, adrenergics, anesthetics) or stress response (surgery, infection). PVCs may worsen the prognosis of children with cardiac pathology while they are generally considered benign in children with a structurally normal heart [1-4].

Case Description

A 9-year-old boy, weighing 60 kg and classified as ASA-I (American Society of Anesthesiologists physical status classification system), underwent day-surgery inguinal hernia repair under combined general and regional anesthesia. Patient was premedicated with oral midazolam 10 mg. After inhalational induction with sevoflurane 8%, isolated PVCs were detected in ECG, with normal respiratory parameters ($SatO_2$ 99% and $EtCO_2$ 35 mmHg), hemodynamic values in lower limit of normal (FC 70 bpm and BP 90/60 mmHg) and without abnormal heart sounds or murmurs. Hence we decided to continue the procedure, decreasing sevoflurane concentration to 2.5% (FiO_2 0.4) for anesthetic maintenance and administering atropine (0.6 mg) to treat borderline sinus bradycardia, both of which–sevoflurane and bradycardia-could induce PVCs. As heart rate increased (FC 90-100 bpm), PVCs became more frequent and finally assumed a bigeminal pattern (Figure 1). Fentanyl (75 μg) was injected prior to supraglottic airway device placement and ilioinguinal/iliohypogastric block subsequently was performed for analgesia (levobupivacaine 0.25% 12 ml). Ventricular bigeminy persisted throughout the 40 min procedure, with no changes in monitored parameters and no complications during anesthesia emergence. In post-anesthesia care unit, a 12-lead ECG (Figure 2) and an echocardiogram were performed.

ECG: Isolated PVCs with left bundle branch block, normal QT and Tp-Te intervals.

Echocardiogram: No structural or functional abnormalities. After 2 h of uneventful stay, the patient was moved to the ward and, 12 h later, was finally discharged pending a Holter monitoring and a stress test, which finally were not carried out because of patient´s decision.

Discussion

Ventricular ectopy in children can be a marker for serious cardiac pathology. It may be associated with structural heart disease, cardiomyopathy and long QT syndrome, all of which increase the risk of Ventricular Tachycardia (VT) and sudden death and demand follow-up echocardiogram, Holter monitoring and exercise stress test. In healthy children we must differentiate: 1) PVCs disappearing with exercise and/or originating from the left ventricle (right bundle branch block morphology), which generally subside during childhood, needing no follow-up. 2) PVCs not disappearing with exercise and/or originating from the right ventricle (left bundle branch block morphology), which don´t usually subside, requiring monitoring as they could be the first sign of an arrhythmogenic right ventricular cardiomyopathy. In structurally normal hearts, a higher burden of ectopy (24 h-PVC%) has been correlated to a higher risk of left ventricle dilation or systolic dysfunction [1-8].

Perioperative ventricular ectopy in healthy children is relatively rare. It can be secondary to adrenergic stimulation (anxiety, pain, superficial anaesthesia), hypoxemia, hypercarbia, acidosis, hypotension, hypothermia, electrolyte disturbance or myocardial ischemia. It´s noteworthy that certain anaesthetics and other perioperative medications (Table 1) can induce a long QT syndrome and potentially lead to ventricular ectopy and tachycardia-*torsade de pointes*-, especially in patients with congenital or acquired long QT syndrome. Long QT is not by itself a risk factor of *torsade de pointes*, being the electrophysiological substrate of this arrhythmia an increased transmural dispersion of repolarization, measured in ECG as

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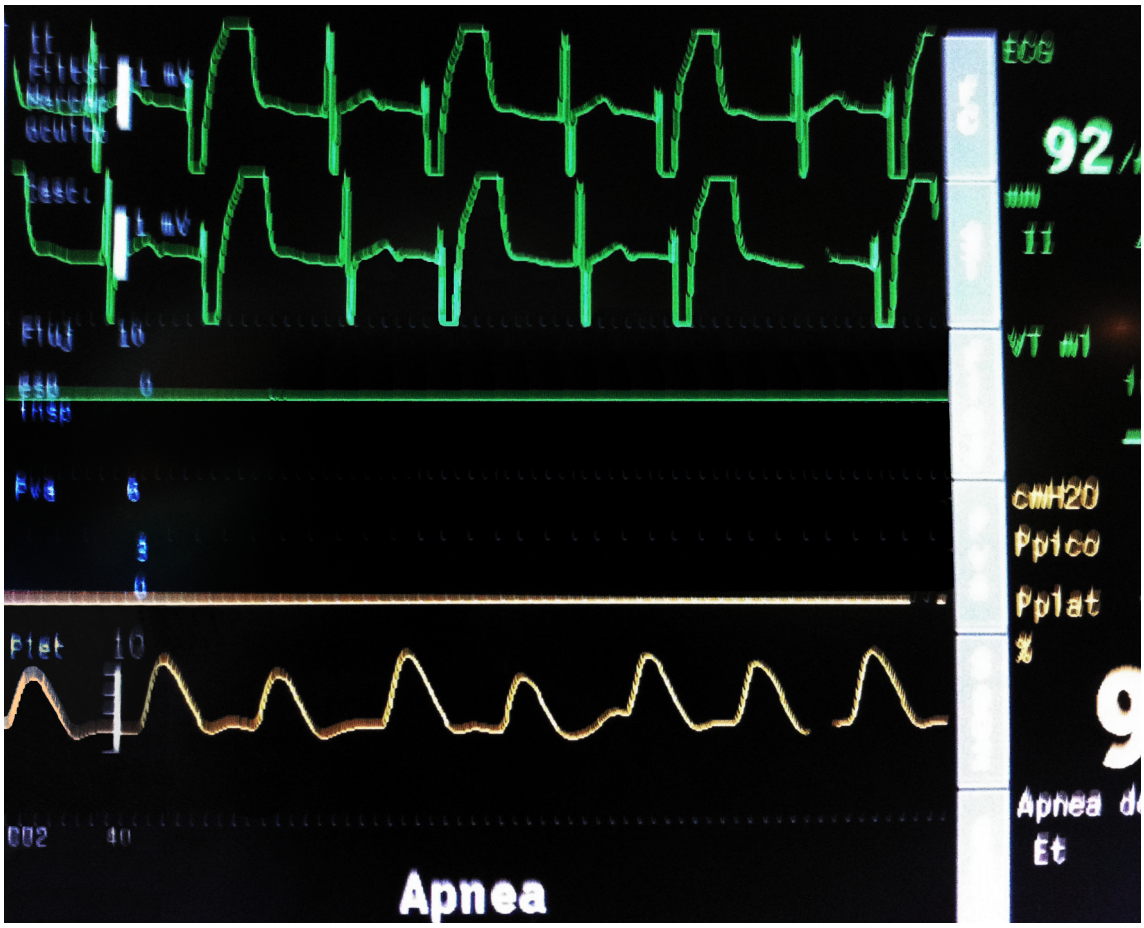

Figure 1: Ventricular bigeminy in multiparameter monitor.

	Perioperative drugs affecting QT interval prolongation
✓	Anesthetics: Halogenated agents, Thiopental
✓	Opiods: Sufentanil, Methadone
✓	NSAIDs: Ketorolac
✓	Neuromuscular blockers: Succinylcoline
✓	Anticholinesterases: Neostigmine
✓	Anticholinergics: Atropine, Glycopyrrolate
✓	Adrenergics: Epinephrine, Ephedrine
✓	Antiemetics: Ondansetron, Droperidol
✓	Antibiotics: Piperacilin/Tazobactam, Ampicilin/Sulbactam, Cefoxitine

Table 1: Perioperative drugs affecting QT interval prolongation.

a prolonged interval between the peak and the end of T wave (Tp-Te). Among the medication listed in Table 1, sevoflurane and ondansetron are remarkable (given its widespread use in pediatric anesthesia) because both drugs prolong QT interval but not Tp-Te interval, not increasing the risk of *torsade de pointes* in healthy children [9-15].

Anesthetic management of ventricular ectopy in healthy children must be based on exhaustive vigilance and hemodynamic monitoring, particularly during periods of enhanced sympathetic activity (induction and especially emergence); adequate anesthesia and analgesia; and maintenance of normoxemia, normocarbia, normothermia and normovolemia. It´s best to avoid bradycardia as well as agents that decrease heart rate and prolong QT interval, as all of them could trigger ventricular ectopy or VT; likewise, caution should be taken when combining drugs influencing repolarization. Sevoflurane, nitrous oxide and propofol are considered good options as anesthetic agents, given their low arrhythmogenicity and side effect profile. Atropine should be contemplated for bradycardia-dependent ventricular ectopy. However, prophylactic antiarrhythmics are not recommended in these patients [15-19].

With regard to our case, in the patient´s medical history we found he suffered from obesity (BMI > 95th percentile), being classified as ASA-I when perhaps should have been scored as ASA-II. Obesity may be associated to increased heart rate, hypertension and left ventricular hypertrophy, all of which may lead to ventricular ectopy and tachycardia [20,21]; however, postoperative echocardiogram revealed normal left ventricular dimension or wall thickness. We also found that the child lost his mother–due to cancer-few months before the surgery. Emotional distress can be associated to sympathetic/parasympathetic imbalance, coronary arterial vasoconstriction or even increased QT dispersion, all of which may predispose to arrhythmia [22,23]. Perioperative adrenergic stimulation, causing potential ectopic activity, was offset with midazolam premedication for anxiety; sevoflurane induction to reach an optimal anesthetic level; and ilioinguinal/iliohypogastric block for analgesia. Ventricular ectopy related to perioperative medication could have been provoked by sevoflurane induction, so it was promptly decreased to lower concentrations for anesthetic maintenance, with no ECG changes. And also by atropine, which was administered to treat a borderline sinus bradycardia that could have lead to ventricular ectopy, with no positive outcome or even worsening in ECG tracing, since the pattern turned into a bigeminal rhythm.

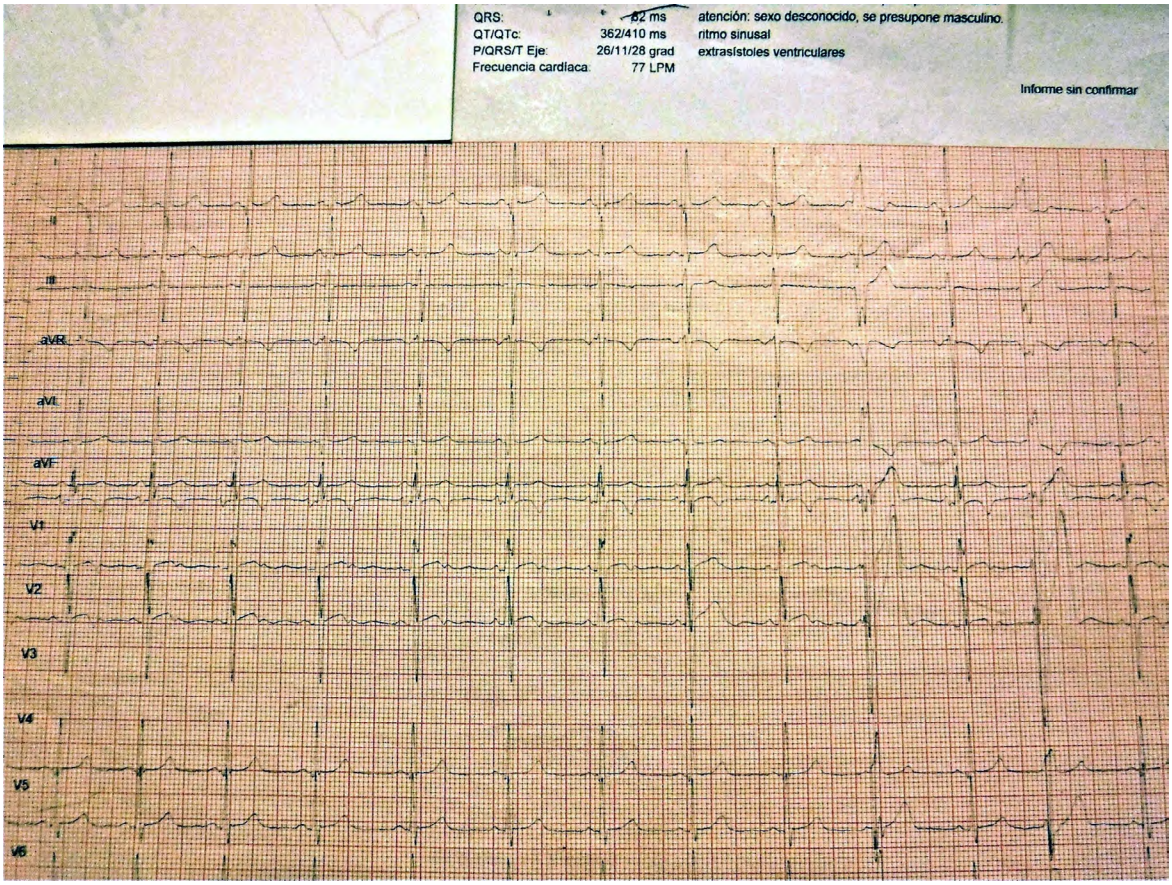

Figure 2: Isolated PVCs with left bundle branch block in 12-lead ECG.

In conclusion, we consider that the reported event turned out to be an atropine-induced ventricular bigeminy by treating bradycardia-dependent ectopy, in a child with a structurally normal heart and a background of emotional distress. This case highlights the importance of anesthetic management of perioperative ventricular ectopy in children, summarizing two main key points:

- Exhaustive vigilance and monitoring, especially during induction and emergence.
- Avoid arrhythmogenic triggers, with particular caution on perioperative medication.

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