A 13-Year-Old Girl in Trigeminy During Anesthesia for Outpatient Dental Surgery: A Case Report

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A 13-year-old girl presented to a private office for dental rehabilitation under general anesthesia. The patient had a previous uneventful anesthetic 5 years prior in the same office by another dental anesthesiologist. The patient was highly anxious and would not allow monitors placed prior to induction. After an uneventful mask induction with sevoflurane, nitrous oxide, and oxygen, monitors were placed and a 22-gauge intravenous cannula inserted. The initial rhythm on the electrocardiogram was trigeminy interspersed with normal sinus rhythm. The volatile anesthetic sevoflurane was discontinued immediately, and intravenous anesthesia was started but still the patient was consistently entering trigeminy. The patient was always hemodynamically stable and never hypoxic. An in-depth discussion of the case and discussion of ventricular dysrhythmias is presented.

Key Words: Pediatric cardiac arrhythmias; Trigeminy; Management of pediatric cardiac arrhythmias; Dental anesthesia.

The overall incidence of arrhythmias is 13.9 per 100,000 emergency room (ER) visits and 55.1 per 100,000 pediatric ER visits (children under 18 years of age).¹

Although infrequent, this case study report will focus on ventricular dysrhythmia, specifically *premature ventricular complexes* (PVCs) in the pediatric population. PVCs are a premature depolarization of the ventricles leading to early, less coordinated, systolic contraction of the ventricles and routinely have a compensatory pause that follows them. PVCs commonly occur in infancy, decline in incidence in early childhood, and become increasingly common again in adolescence and adulthood.²

The frequency of PVCs in the pediatric population undergoing outpatient general anesthesia for dental procedures has not been documented to date; however, the incidence of PVCs in the pediatric population under

20 years old is 0.5%.³ The significance of this case report is to inform anesthesiologists who provide outpatient general anesthesia about the case of a 13-year-old girl undergoing dental rehabilitation under general anesthesia who developed trigeminy in a private dental office setting and to promote effective management of children with similar findings on electrocardiogram (ECG).

CASE PRESENTATION

A 13-year-old girl presented to a private office dental facility as a referral for generalized dental care requiring general anesthesia. The patient was an extremely nervous and anxiety-ridden patient during the initial consult.

The medical history was reviewed in detail with the patient's mother. The patient was not interested in speaking to anyone during the consult (the mother had stated it was a miracle that she came to the consult!). The patient's past medical history was unremarkable from a

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cardiovascular or pulmonary perspective. The only pertinent findings were an apparent high level of anxiety and a potential for affected mental health with no formal diagnosis. The patient has an older sibling with early-onset schizophrenia. The patient also had questionable nasal patency issues. Furthermore, the patient had significant needle phobia. The patient was not seeing a psychiatrist or psychologist and was taking no medications. The patient had an allergy to penicillin (specifically a reaction to amoxicillin) and an unremarkable social history according to the mother, the patient being noncommunicative. There was no family history of anesthetic complications. Required dental treatment included complete examination, radiographs, extraction of 2 retained primary teeth, multiple restorations, and periodontal sanative therapy.

Past anesthetic history included a prior general anesthetic in the same dental office at age 6 with another dental anesthesiologist in the practice. The anesthesia record was unremarkable, and the patient tolerated the procedure well with stable hemodynamics (no cardiac abnormalities noted) and no intraoperative or postoperative complications.

On the day of treatment, the patient was extremely apprehensive. Thirty minutes before induction, the patient received 0.5 mg/kg of midazolam orally. The patient came into the treatment room accompanied by her mother and sat in the dental chair but would not allow monitors to be placed prior to induction. The patient allowed a mask induction in the sitting position; she had requested to not be reclined during induction. The inhalation sequence included a mixture of 50% nitrous oxide in oxygen (N_2O/O_2) and an incremental increase in sevoflurane from 2 to 8%. The patient tolerated the mask induction well, and the mother was asked to leave the treatment room. N_2O was discontinued for 100% O_2 delivery with sevoflurane. Standard monitors were placed immediately after induction. Initial readings were 99% oxygen saturation, noninvasive blood pressure (BP) 105/78 mm Hg, and a heart rate of 60 beats per minute with an irregular rhythm and no appreciated respiratory concerns. The rhythm immediately identified in lead II on the ECG was trigeminy or possible preatrial contraction with right bundle branch block every third beat. A 22-gauge intravenous (IV) catheter was placed immediately in the right hand with a normal saline infusion. At this point, the sevoflurane was immediately discontinued. The BP was recycled and shown to be 98/55 mm Hg with confirmed good peripheral pulses and regular capillary refill in the periphery. The patient was switched to an IV anesthetic to rule out sevoflurane as the cause of the arrhythmia as this was an apparent new finding for this patient. Fentanyl, 50 μ g intravenously, and 100 mg of propofol by IV bolus were administered. The patient

continued to have good peripheral perfusion, stable oxygen saturation, and normal spontaneous respiratory effort with full facemask delivering 100% O_2 . Despite the switch to IV anesthesia, the ECG continued to show trigeminy interspersed with normal runs of sinus rhythm. The defibrillator and emergency cart were brought into the room in case they were needed. The patient continued to maintain very stable hemodynamics throughout this period. A blood glucose concentration was obtained via fingerstick at this time and was 110 mg/dL (6.1 mmol/L). After another 8–10 minutes, the case was aborted and the patient woke up uneventfully. The patient had no chest pain, appeared to be appropriately alert and oriented for the immediate postanesthesia period, and continued to have stable hemodynamics despite still exhibiting the persistent arrhythmia mixed with some periods of normal sinus rhythm. Although elevated endogenous catecholamine levels due to anxiety may have been the cause of these perioperative findings, at this point, the normal saline infusion was continued and the decision was made to call emergency medical services to transfer the patient safely to the hospital with monitors for further investigation. The patient was in the ER for the remainder of the day. The dental anesthesiologist followed the patient to the hospital and delivered the case details to the attending pediatric ER physicians and nurses. The patient was very combative in the ER and uncooperative. Blood was drawn for a complete blood count and serum chemistry profile, and a 12-lead ECG was obtained. The patient continued to be in trigeminy dispersed with periods of normal sinus rhythm. Results of lab studies revealed a potassium level of 5.3 mEq/L. The patient was started on Kayexalate orally by the ER physician and sent home that night. She was scheduled for a follow-up visit with a pediatric cardiologist.

The patient underwent a cardiac Holter monitor from her family physician; results revealed the patient still had the arrhythmia. The patient was scheduled to see a pediatric cardiologist but due to psychological factors, she refused to go for the consult. The patient's mother continued to attempt to get her to see the cardiologist, with no success. The dental anesthesia office instructed the patient's mother that although the rhythm is most likely benign (the patient has been asymptomatic throughout this period with no dyspnea, chest pain, syncope, etc), a thorough assessment by the cardiologist is required to ensure maximum safety in the private practice arena for any future general anesthetics.

DISCUSSION

PVCs are not an uncommon finding in the healthy pediatric population. PVCs are even more common in

the arena of anesthesiology because multiple agents used in anesthesia practice have a direct effect (sympathomimetics, volatile anesthetics) or an indirect effect (light anesthesia, pain, ketamine) on the heart's conduction system. It is imperative that anesthesiologists understand the context of when PVCs during anesthesia can become life-threatening and require further investigation and referral.

Doniger¹ states

Although most children who have PVCs are otherwise healthy, PVCs can also be associated with congenital heart disease, mitral valve prolapse, prolonged QT syndrome, and cardiomyopathies (dilated and hypertrophic). Malignant origins include electrolyte imbalances, drug toxicities (eg, general anesthesia [author highlight], digoxin, catecholamines, amphetamines, sympathomimetics, and phenothiazines), cardiac injury, cardiac tumors, myocarditis (Lyme and viral diseases), hypoxia, and an intraventricular catheter.

History is a crucial factor in ascertaining which cardiac arrhythmias are significant or benign. Important elements include a history of palpitations, syncope, chest pain, and family history. A referral to a pediatric cardiologist is essential for cases where the patient is symptomatic, the diagnosis is new, or the ECG demonstrates a significant abnormality. A symptomatic child requires immediate referral to a hospital if a critical arrhythmia is possible.²

Holter monitors provide continuous rhythm recording from adhesive electrodes for a minimum of 24 to 48 hours. The monitors gather the following information: minimum/average/maximum heart rate, longest pause, the number of preatrial contractions and PVCs, and the number of runs of supraventricular tachycardia (SVT) and ventricular tachycardia (VT), including duration and rate. This is an extremely useful monitor for children who are asymptomatic with an irregular heart rate or beats, or for the symptomatic child to isolate the arrhythmia during onset of symptoms.²

Exercise testing is a noninvasive way to observe the effect of enhanced sympathetic drive in children with an ECG or Holter monitor abnormality. It can assess the significance of patients with frequent PVCs ($>8/\text{min}$) or significant PVC patterns (eg, bigeminy, trigeminy, polymorphic, >3 PVCs in a row). Generally, if the PVCs are suppressed with exercise, they are considered benign. If exercise worsens the frequency and leads to runs of VT, then the arrhythmia is significant and requires immediate attention. Sometimes, the test is equivocal if the PVCs remain but do not lead to VT or hemodynamic instability.²

Echocardiography is necessary when there is suspicion of cardiac structural disease, as noted above. Electrophysiological studies may be required to provide a more definitive etiology and diagnosis if the previous studies do not provide a diagnosis. Furthermore, some arrhythmias (certain SVTs and VTs) can be mapped and ablated during this invasive study.

PVCs can also appear regularly but be part of an organized rhythm called "ventricular parasystole." This involves a ventricular focus, which is not overdrive suppressed from the sinoatrial node, and therefore beats at its own inherent rate. Multifocal PVCs are a sign of impending disaster. If PVCs have different configurations in the same lead, the chances of developing a sustained dangerous (VT with pulse) or deadly (VT or ventricular fibrillation without a pulse) is higher.⁴

Anesthesia on its own contributes to the likelihood of seeing PVCs on the ECG. Anytime oxygenation is significantly affected, myocardial or conduction system hypoxia can lead to PVCs. Likewise, tachycardia and hypertension may lead to an imbalance between myocardial oxygen supply and demand leading to ectopic ventricular rhythms. Significant hypotension may also decrease myocardial oxygen supply. Hypercarbia as well can sensitize the myocardium to ectopic foci. Ischemic heart disease should always be considered but is unlikely in the pediatric population. Furthermore, many drugs used in anesthesia practice can either directly or indirectly trigger an ectopic ventricular focus and cause single or multiple PVCs. Importantly, the "occurrence of 6 or more PVCs per minute and repetitive or multifocal forms of ventricular ectopy, even if asymptomatic, indicates an increased risk of developing a life-threatening ventricular tachydysrhythmia."³

Anesthetic management of significant PVCs as noted above is to establish a differential diagnosis of possible causes and treat the most likely diagnoses once life-threatening presentations are addressed. The most common causes under anesthesia include myocardial oxygen imbalance due to significant hypertension, hypotension, or tachycardia, acidosis, electrolyte abnormalities, or various pro-dysrhythmic drugs—anesthetics and others. A defibrillator must be available and accessible. Beta-blockers are the most successful drugs for suppressing intermittent ventricular ectopy, especially when tachycardia with or without hypertension is present, and should be the first line of treatment, while lidocaine and amiodarone should be reserved for cases where PVCs progress to runs of VT, or persistent VT is causing hemodynamic instability. Early defibrillation or cardioversion are also indicated, of course, in the early management of significant persistent VT depending on presence of pulse.³ Where persistent ventricular ectopy is observed intraoperatively, the anesthesia provider

should monitor the patient during the recovery phase and consider urgent or early evaluation if dysrhythmia persists once anesthesia medications have been eliminated to a reasonable degree. If significant dysrhythmias are noted before induction, the case should be cancelled and cardiac consultation obtained prior to elective procedures.

CONCLUSION

Anesthesiologists are among the healthcare providers who are most likely to encounter cardiac rhythm abnormalities in the pediatric population. An ECG is one of the standard general anesthesia monitors and thus, every child under general anesthesia will have their cardiac conduction system monitored. There is no clear-cut guidance from the literature with an algorithm or protocol when arrhythmias are detected in the pediatric population. Another dilemma commonly faced in an outpatient dental anesthesia practice is uncooperative patient populations where it can be extremely difficult, if not impossible, to obtain even preoperative vital signs at the initial consult, let alone a preinduction ECG. It is essential to understand the causes of PVCs under anesthesia and be prepared to act appropriately.

The most common recommendation noted in the literature regarding irregular heart rates is that a determination of the underlying cause is critical. Some apparently benign rhythms can be life threatening. Primary care physicians, with a detailed history and

physical examination including 12-lead ECG, can diagnose the majority of arrhythmias. A referral to a pediatric cardiologist is necessary when a diagnosis cannot be made upon initial evaluation. Additionally, children who are symptomatic and/or their ECG demonstrates a significant abnormality should be evaluated urgently. Holter monitoring, exercise testing, echocardiography, and electrophysiological intervention are all available to aid in diagnosis.

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