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Case Report

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Pulseless Electrical Activity and Perioperative Cardiac Arrest Due to Undiagnosed and Asymptomatic Hypothyroidism During Outpatient Surgery in an Adolescent

Josiane Kerbage[a], Olamide O. Dairo[a, b], Lauren Ketchum[a], Craig Smith[c], Joseph D. Tobias[a, b, d]

Abstract

Commonly identified risk factors for perioperative cardiac arrest in pediatric-aged patients include cardiac surgery, younger age, comorbid conditions including pulmonary hypertension and cyanotic congenital heart disease, and emergency surgery. Although medication-related etiologies formerly predominated, the elimination of halothane from anesthetic care has resulted in a shift in etiology to hemodynamic events related to blood loss or hyperkalemia associated with the rapid administration of blood products. Rarely, cardiac arrest can be sudden and unexpected without an identified pre-existing etiology in an otherwise apparently healthy patient. We present an 18-year-old adolescent who experienced pulseless electrical activity (PEA) and cardiac arrest following anesthetic care for an outpatient orthopedic procedure, who was eventually diagnosed with hypothyroidism. The potential etiologies of PEA and cardiac arrest during anesthesia are reviewed, components of successful resuscitation discussed, and an outline for the investigative workup presented.

Keywords: Cardiac arrest; Pediatric anesthesiology; Hypothyroidism

Introduction

Intraoperative cardiac arrest is a rare yet devastating complication of anesthesia in children with an estimated incidence of 2.9 per 10,000 cases in non-cardiac surgery [1]. The Perioperative Cardiac Arrest (POCA) Registry was formed in 1994 to study the etiologies and outcomes from perioperative cardiac arrest during anesthetic care in infants and children [2]. This registry has identified several broad categories which may be responsible for perioperative cardiac arrest including cardiovascular events, medication-related events, respiratory events, and equipment problems [2, 3]. Patient-related factors responsible for perioperative cardiac arrest include younger age, pre-existing comorbid conditions including congenital heart disease or pulmonary hypertension, and emergency surgery [2-4].

Although the POCA registry initially identified medication-related cardiac arrest, specifically halothane, as the most common etiology, as halothane has been replaced in anesthetic practice by sevoflurane, the most likely etiologies of perioperative cardiac arrest have shifted to blood loss and hyperkalemia associated with the rapid administration of blood and blood products. Rarely, cardiac arrest can be sudden and unexpected without an initially obvious etiology in an otherwise apparently healthy patient. We present an 18-year-old adolescent who experienced a sudden postoperative cardiac arrest following anesthetic care for an outpatient orthopedic procedure. Postoperative evaluation led to the diagnosis of previously undiagnosed and relatively asymptomatic hypothyroidism.

The review and presentation of this case followed the guidelines set by the Institutional Review Board of Nationwide Children's Hospital (Columbus, OH, USA) and was conducted in compliance with the ethical standards of the responsible institution on human subjects as well as with the Helsinki Declaration.

Case Report

Investigations

The patient was an 18-year-old adolescent who presented to a free-standing outpatient surgery center for removal of hardware from the left arm and elbow. Past medical history was relatively unremarkable. She was status post open reduction and internal fixation of the left ulna following a fracture 6 months prior. The patient also had a history of Raynaud's disease. During the preoperative examination, there was some difficulty with obtaining an accurate pulse oximeter reading which was attributed to vasoconstriction from Raynaud's. Her heart rate

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[a]Department of Anesthesiology & Pain Medicine, Nationwide Children's Hospital, Columbus, OH, USA
[b]Department of Anesthesiology & Pain Medicine, The Ohio State University College of Medicine, Columbus, OH, USA
[c]Department of Orthopedic Surgery, Nationwide Children's Hospital, Columbus, OH, USA
[d]Corresponding Author: Joseph D. Tobias, Department of Anesthesiology & Pain Medicine, Nationwide Children's Hospital, Columbus, OH 43205, USA. Email: Joseph.Tobias@nationwidechildrens.org

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(HR) was 50 beats/min, which was noted to be similar to her HR for prior procedures. The parents reported a history of a low resting HR, which had been discussed with the patient's primary care physician previously. This had been attributed to her being a healthy teenager and no further workup was pursued. Her preoperative blood pressure (BP) was 123/71 mm Hg, the room air oxygen saturation was 95%, and the temperature was 36.6 °C (97.9 °F). The remainder of the physical examination was unremarkable. The only laboratory evaluation obtained was a negative urinary pregnancy test. The patient was assigned an American Society of Anesthesiologists physical status class 1.

Diagnosis and treatment

The patient was transported to the operating room and routine American Society of Anesthesiologists Monitor were placed. Upon arrival to the operating room, there was difficulty obtaining an effective reading from the pulse oximetry when placed on either hand, and therefore, the monitor was placed on an ear lobe and a reading of 100% was obtained. The electrocardiogram (ECG) pads were repositioned and replaced several times, due to low voltage. With an adequate BP, a functioning pulse oximeter on the ear lobe, and a continuous ECG, anesthesia was induced with propofol (200 mg) and hydromorphone (1 mg) through a pre-existing intravenous cannula. A laryngeal mask airway (LMA) was placed, and maintenance anesthesia was provided by sevoflurane (expired concentration 0.9-1.2%) in air and oxygen. Intraoperatively, the HR varied from 40 to 75 beats/min with a BP of 80 - 90/40 - 50 mm Hg. The ECG voltage remained low and there was a decrease in the amplitude of the pulse oximeter waveform. Given concerns with the low HR and issues concerning the amplitude of the ECG and pulse oximeter, it was decided not to infiltrate the surgical bed with a local anesthetic agent, and ondansetron was not administered. Additional intraoperative medications included cefazolin (1 g) for prophylaxis against surgical site infections, dexamethasone (4 mg) for prevention of postoperative nausea and vomiting, and ketorolac (27 mg). Intraoperative fluids included 1,600 mL of lactated Ringers. Following completion of the surgical procedure, the LMA was removed, and the patient was transported to the post-anesthesia care unit (PACU) with spontaneous respirations and an oxygen saturation of 100%. Shortly after arrival at the PACU, the patient was noted to be pale and mottled. The peripheral pulse was thready and difficult to palpate. Heart sounds were faint on auscultation. Ventilation was immediately assisted by bag-valve-mask and cardiopulmonary resuscitation initiated according to standard advanced cardiac life support (ACLS) guidelines. The resuscitation lasted approximately 25 min during which time the patient received epinephrine (total of four doses or 4 mg) and glycopyrrolate (0.4 mg). The continuous ECG demonstrated a sinus rhythm throughout the resuscitation with periods of absence of palpable pulses and no evidence of cardiac function thereby diagnostic for pulseless electrical activity (PEA). The trachea was reintubated after the administration of propofol (40 mg) and succinylcholine (40 mg). An arterial cannula was placed, and an initial arterial blood gas revealed pH 7.28, partial pressure of oxygen 89 mm Hg, partial pressure of carbon dioxide 48 mm Hg, and base deficit -4.2. The lactate was 3 mmol/L (normal: 0.5 - 2.2 mmol/L). The sodium, potassium, ionized calcium, hemoglobin, and hematocrit were unremarkable. Pulses were palpable during chest compressions, and after 25 min of resuscitation, return of spontaneous circulation (ROSC) was obtained. After ROSC, the patient started breathing spontaneously and followed commands. Midazolam (2 mg) was administered to provide amnesia and anxiolysis. The patient was admitted to the pediatric intensive care unit (PICU).

Follow-up and outcomes

Following admission to the PICU, the patient's hemodynamic and respiratory status stabilized. A 12-lead ECG showed bradycardia and low voltage. An echocardiogram revealed a large globally distributed pericardial effusion with normal biventricular systolic function. Although the pericardial effusion appeared to be chronic without evidence of tamponade, she was transported to the cardiac catheterization suite, and a percutaneous pericardial drain was placed. A total of 850 mL of fluid was removed. Her trachea was extubated later than the day in the PICU. Further investigation revealed mild normocytic anemia. A viral respiratory panel from a nasal swab was positive for coronavirus disease 2019 (COVID-19). Evaluation for thyroid disease showed a thyroid-stimulating hormone (TSH) level of 95.218 IU/mL (normal: 0.4 - 4 IU/mL) and a free T4 level less than 0.5 ng/mL (normal: 0.7 - 2.1 ng/mL) confirming the diagnosis of hypothyroidism. The adrenocorticotrophic hormone (ACTH) stimulation test was normal. Further testing including thyroid peroxidase (TPO) antibodies and anti-thyroglobulin antibodies confirmed a diagnosis of Hashimoto's thyroiditis. On further questioning, there was a history obtained of cold hands, dizziness, and a general sense of feeling cold. To further investigate the potential impact of the associated COVID-19 infection, infectious disease consultation was obtained. Cardiac magnetic resonance imaging (MRI) demonstrated a mildly thickened and enhanced pericardium, suggestive of pericarditis. The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) cycle threshold values were high, suggesting a low viral load indicative of a past infection which was not impacting the current clinical course of the patient. Therefore, no pharmacologic therapy was recommended by the infectious disease team. Based on an evaluation of the infectious disease, endocrinology, and rheumatology consultations, it was concluded that the etiology of the pericardial effusion and the cardiac arrest were primary hypothyroidism related to Hashimoto's thyroiditis. The patient was started on replacement therapy with oral thyroxine and discharged home on postoperative day 5.

Discussion

In adolescence and throughout adult life, the most common

etiology of hypothyroidism remains Hashimoto's thyroiditis, an autoimmune disorder resulting from dysregulation of the immune system, leading to lymphocytic infiltration of the thyroid parenchyma and destruction of the functioning tissue [5]. The disorder is characterized by the development of autoantibodies to thyroid peroxidase, the enzyme that catalyzes the production of thyroxine [6]. Clinical signs and symptoms in infants and children are nonspecific including constipation, poor growth, cold intolerance, lethargy, and dry skin. In adolescents and adults, patients are frequently asymptomatic or manifest symptoms that are insidious and gradual in onset including fatigue, weight gain, and cold intolerance that are attributed to other etiologies [7, 8]. The diagnosis of hypothyroidism is confirmed by laboratory evaluation of free T4 and TSH values. Treatment includes replacement therapy with levothyroxine (1 - 2 µg/kg/day in adults) [8].

Hypothyroidism can have a significant impact on cardiac function with hemodynamic effects including decreased cardiac output, increased systemic vascular resistance, and decreased arterial compliance [9]. In severe circumstances when left untreated, the decrease in stroke volume, the impaired cardiac muscle relaxion, and the decrease in the HR can result in heart failure and even cardiac arrest [10, 11]. Arrythmias including bradycardia, heart block, and torsade de pointes have been reported. Although uncommon, cardiac arrest primarily related to hypothyroidism has been reported in adults with myxedema coma due to undiagnosed hypothyroidism [11-14]. Intraoperative cardiac arrest potentially due to severe undiagnosed hypothyroidism was reported during a bronchoscopy in a 68-year-old man with multiple comorbid conditions including atrial fibrillation treated with amiodarone that resulted in hypothyroidism [15]. However, after further investigation, the primary etiology of the cardiac arrest was determined to be a myocardial infarction and not hypothyroidism. In pediatric-aged patients, an intraoperative cardiac arrest was reported during general anesthesia for radiologic imaging in a 5-year-old child with Williams syndrome and hypothyroidism [16]. However, the arrest was attributed to coronary artery involvement related to Williams syndrome and not hypothyroidism.

In our patient, we noted a deterioration in cardiac function upon arrival to the PACU following anesthetic care. Initially, the patient appeared pale and mottled with thready and difficult to palpate pulses. During resuscitation, the ECG demonstrated a sinus rhythm with periods of an absence of a palpable pulse and no evidence of cardiac function thereby leading to a diagnosis of PEA and cardiac arrest. When making a diagnosis of PEA, it is possible that there is some residual cardiac output or function without clinical evidence such as pulses or a pulse oximeter waveform, indicate of an extremely low cardiac output state. Regardless, whenever there is evidence of inadequate cardiac output, cardiac arrest or PEA, resuscitation is indicated according to standard ACLS guidelines.

The current case highlights the potential for previously undiagnosed hypothyroidism to present as a sudden cardiac event during perioperative care. It also highlights the need for a thorough evaluation to consider and identify uncommon etiologies for cardiac arrest once other more common causes are excluded. Although generally asymptomatic, our patient presented with subtle signs of hypothyroidism, which were identified during careful retrospective evaluation including a low HR and cool extremities with difficulties obtaining pulse oximetry plethysmography. Previous investigators have reported an association of Raynaud's phenomenon with hypothyroidism [17-19]. Sipila et al reported a 29-year-old woman with Raynaud's disease and undiagnosed hypothyroidism who developed a myocardial infarction despite normal coronary arteries [17]. Vasospasm from Raynaud's disease was suggested as the likely etiology of coronary ischemia. Others have suggested that Raynaud's disease may be an early clinical sign of hypothyroidism [18, 19]. Additional intraoperative concerns which retrospectively may be consistent with hypothyroidism, a low cardiac output state or the pericardial effusion included continued bradycardia during the surgical procedure, low voltage on the ECG (likely due to the pericardial effusion), low amplitude on the pulse oximeter plethysmograph, and a lower-than-expected requirement for sevoflurane intraoperatively.

Echocardiography following the cardiac arrest noted a large pericardial effusion. Although the echocardiogram did not demonstrate tamponade physiology, the presence of a pericardial effusion may have been partially responsible for the cardiac arrest during the perioperative period. Pericarditis and pericardial effusions have been reported in association with and even as a presenting sign of hypothyroidism in children and adults [20-22].

In summary, we present an unexpected and sudden development of PEA and cardiac arrest occurring during perioperative care, which was eventually attributed to undiagnosed hypothyroidism. Subtle preoperative clinical signs included a low resting HR, a low ECG amplitude, and cool extremities that resulted in problems obtaining a pulse oximetry reading. During the subsequent workup, a pericardial effusion was noted. Laboratory evaluation eventually resulted in the diagnosis of Hashimoto's thyroiditis and hypothyroidism. Regardless of the etiology, the basic tenets of resuscitation during perioperative cardiac arrest include strict adherence to ACLS guidelines including prompt airway management, the rapid administration of epinephrine, and high-quality chest compressions. Treatable although less likely etiologies such as electrolyte and metabolic disturbances, adrenal insufficiency, and anaphylaxis should be considered.

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None to declare.

Conflict of Interest

None to declare.

Informed Consent

Informed consent was obtained for anesthetic care and the use of deidentified information for publication.

Author Contributions

JK: preparation of initial, subsequent, and final drafts. OOD: review of final draft, perioperative care of patient. LK: review of final draft, perioperative care of patient. JDT: concept, review of all drafts.

Data Availability

Any inquiries regarding supporting data availability of this study should be directed to the corresponding author.

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