

Anesthetic Consideration In A Patient With Postural Orthostatic Tachycardia Syndrome

S Ramamoorthy, V Reddy, P Dhar

Citation

S Ramamoorthy, V Reddy, P Dhar. *Anesthetic Consideration In A Patient With Postural Orthostatic Tachycardia Syndrome*. The Internet Journal of Anesthesiology. 2009 Volume 27 Number 2.

Abstract

This rare case report highlights the hemodynamic variability associated with Postural orthostatic tachycardia syndrome (POTS) in a patient with splenic injury secondary to trauma and offers a safe anesthetic practice in general. Tachycardia is usually a predominant manifestation of hypovolemia in patients who have lost significant amount of extracellular fluid and at the same time tachycardia could be the only symptoms of POTS under general anesthesia. Hence it is essential to include tachycardia associated with POTS as part of the differential diagnosis.

INTRODUCTION

Postural orthostatic tachycardia syndrome (POTS) is an autonomic disorder of chronic orthostatic intolerance with two subtypes based on pathophysiology (1). Even though the etiology is unclear, this syndrome affects 500,000 patients in United States, with preponderance in younger women (1,2,3). The first type, partial dysautonomia form (90% of cases), manifests as tachycardia in the upright position with symptoms of orthostatic intolerance such as lightheadedness, nausea, chronic fatigue, dependent edema, and acrocyanosis. The second type, hyperadrenergic form (10% of cases), manifests as increased serum norepinephrine levels, tremulousness, anxiety, and an exaggerated response to beta-adrenergic stimulation. Diagnostic criteria for POTS include a heart rate increase of at least 30 beats per min or a rate that exceeds 120 beats per min that occurs within the first 10 min of standing or head-up tilt. A heart rate increase of more than 30 beats per min in response to an infusion of isoprenaline 1 microgram per min is also used to confirm the diagnosis (1). In POTS, failure of the peripheral vasculature to vasoconstrict appropriately during hypovolemia is compensated by an increase in heart rate and blood pressure (1,4,5). However, this mechanism may not fully compensate for the lack of vasoconstriction resulting in hypoperfusion of the brain and other organs. Furthermore, at times there may be overcompensation, resulting in uncontrolled tachycardia and hypertension. Because of the variability in presentation and subtype, and relative resistance to medical therapy, POTS represents a particularly difficult disease to diagnose, more so in trauma patients, who often encounter big fluid

losses.

CASE REPORT

A 17 year old male weighing 57 kg, with past medical history of attention deficit hyperactivity disorder (ADHD), with history of abdominal trauma secondary to motor vehicle accident was discharged in a stable condition after being treated conservatively for a Grade 4 splenic injury. He was readmitted 8 days after initial trauma with complaints of severe abdominal pain, lightheadedness, dizziness associated with profuse diaphoresis. After initial evaluation, the patient was found to be severely anemic with a hematocrit of 25% which had been 44% at his discharge from the hospital. The patient was tachycardic with the heart rate in the range of 110-118 beats per minute and a systolic blood pressure in the range of 108-130 mm Hg and diastolic blood pressure in the range of 61-70 mm Hg. He was emergently brought to the operation room for an exploratory laparotomy after receiving 1L of lactated ringer's in the emergency department.

In the operating room, the patient was placed under standard monitoring including pulse oximetry, electrocardiogram, non-invasive blood pressure measurement and end tidal CO₂ tracing. At the initial recording, the heart rate was noted to be 118 bpm and blood pressure was 118/62 mm Hg. A Rapid sequence induction was initiated with administration of Etomidate 16 mg, Succinylcholine 120 mg, and fentanyl 50 micrograms. The patient was intubated with 8.0 cuffed endotracheal tube. Anesthesia was maintained with sevoflurane in a 50% O₂ and air mixture, and additional

muscle relaxation was obtained by administration of rocuronium. After induction of anesthesia, a left radial artery catheter, nasogastric tube and esophageal temperature probe were placed. Thermoregulation was achieved with forced air warmer.

Exploratory laparotomy revealed an injury to spleen which included superficial laceration in the inferior pole and the rupture in the superior pole of the spleen. Splenectomy was performed and hemostasis was achieved. Even though the patient's blood pressure remained stable intraoperatively his heart rate varied from 102 to 120 bpm. Blood investigations obtained intraoperatively at the beginning of the procedure showed a hematocrit of 19%, base deficit of 6, lactate value of 2.46 mmol/L. The patient's estimated blood loss was 1.5 L and urine output was 200 ml during the procedure. The patient received 3L of crystalloids, 4 units of Packed Red Blood cells and 2 units of Fresh frozen plasma. Repeat lab values obtained at the end of the procedure showed a hematocrit of 34 %, base deficit of 3.5 and a lactate value of 1.5 mmol/L. The procedure lasted for 45 min during which time, the patient received fentanyl 150 micrograms and morphine 14 mg intravenously. At the end of surgery, neuromuscular blockade was adequately reversed and the patient's trachea was extubated. At wake up the patient was normothermic, appeared comfortable without any complaints of pain, was spontaneously breathing adequately but he continued to remain tachycardic in the range of 110 -120 bpm. Based on the blood investigations and clinical presentation, it was deemed that the patient was appropriately fluid resuscitated and pain was optimally controlled. Patient was transferred to surgical intensive care unit in a stable condition for post operative monitoring and evaluation.

However, in the surgical intensive care unit, the patient continued to remain tachycardic with heart rate ranging from 130-140 bpm, stable blood pressure, normal lactate and base deficit levels. At this point, after careful reevaluation of his medical history, it was discovered that the patient is a known case POTS syndrome. This was confirmed by direct questioning of the patient. The patient also stated that in order to compensate for POTS, he was advised by his primary care physician to consume large amounts of fluid which often was in the range of 10-12 L per day. The patient received 6 L of crystalloids and 1 L of colloid on post- op day 1 and 7 L of crystalloid on post-op day 2. In spite of large volume of fluid intake, there was no clinical or radiological evidence of fluid overload. During this period of

time his hemtocrit was stable, urine output was adequate and there was no concern for any occult blood loss. The patient's pain was well controlled with Morphine PCA. Het was advanced to regular diet and tolerated ambulation. The patient remained tachycardic throughout his hospital stay and was discharged home under stable condition on the second postoperative day. On a follow up phone call at home, the patient was recuperating well. The patient admitted to persistence of his baseline symptoms which included tachycardia with occasional chest palpitation and dizziness, otherwise reported doing well.

DISCUSSION

A review of the literature identified three cases reports regarding anesthetic management of patients with POTS. The review of each of the case reports delineated anesthetic management in pregnant woman undergoing elective cesarean section (^{6,7,8}). So far, we believe that, no known cases of POTS have been reported in pediatric male patients involved in trauma with injuries to major organs such as spleen. In this case report we highlight the tachycardia and hemodynamic variability associated with POTS and offer a safe anesthetic practice in POTS patients.

The majority of patients with POTS are described as having a partial dysautonomia, with the predominant pathophysiology being an inability to increase peripheral vascular resistance when moving to an upright posture, resulting in an excessive compensatory postural tachycardia (^{1,4,5}). The patient discussed in this report belongs to the above mentioned group of patients. Disproportionate venous pooling leads to an exuberant baroreceptor response with increased sympathetic stimulation and a persistent tachycardia, which often manifests as symptoms of tremulousness and anxiety on standing.

Differential diagnosis of tachycardia during surgery, include a wide variety of etiologies including hypovolemia, hyperthermia, nociceptive stimulation, pulmonary embolism, pharmaceutical drugs administered and anaphylaxis. In this case, patient was appropriately fluid resuscitated as per normal lactate, base deficit levels and adequate urine output. Thermoregulation was optimal as patient remained normothermic throughout the procedure. With administration of fentanyl 150 micrograms and morphine 14 mg for a 45 minute operation, we believe that adequate levels of pain control was obtained. This was confirmed by lack of complaints of pain by the patient at the end of surgery. With a normal A-a gradient, and a stable PaO₂,

Sao₂ and ETCO₂ levels, pulmonary embolism would be an unlikely etiology of tachycardia. Often, sustained severe tachycardia is not caused by induction agents and anaphylaxis presenting only as tachycardia is unlikely.

In this report, we present a rare cause of tachycardia and delineate the general principles in the management of general anesthesia in a patient with POTS. Induction agents like thiopental by releasing histamine and ketamine by sympathomimetic response could exacerbate tachycardia. Propofol, and etomidate appears to be safe agents. Succinylcholine is often associated with bradycardia, whereas non-depolarising agents notably pancuronium, with its atropine like anticholinergic effect, could worsen tachycardia. Deep reverse Trendelenberg position should be avoided as this may worsen venous pooling. Desflurane could induce rebound tachycardia. Anesthesia induced hypotension is better treated with alpha adrenergic agonist like phenylephrine (⁹). Less commonly, POTS is characterized by heightened sensitivity to b-adrenergic agonists. Hence ephedrine, an indirect sympathomimetic agent should be avoided (⁹). Pain control needs to be optimized either with neuroaxial, regional or patient controlled narcotic based techniques. If neuroaxial blockade is considered, it is essential to avoid epinephrine containing local anesthetics, which may worsen the symptoms in a subgroup of POTS who are sensitive to catecholamines or increase the heart rate. Regional procedures with local anesthetics could be used as an effective adjuvant for pain control. Some of the other anesthetic considerations include maintaining normocarbida in these patients as hypercarbida

and subsequent respiratory and metabolic acidosis can exacerbate tachycardia associated with POTS. Also, care should be taken to avoid hyperthermia in this population

Finally, good elicitation of medical history is critical in the identification of POTS, since tachycardia for various above discussed reasons is a common presentation in trauma patients.

References

1. Kanjwal Y, Kosinski D, Grubb B.P. The postural orthostatic syndrome: definitions, diagnosis and management. *Pacing Clin Electrophysiol* 2003; 26:1747–57.
2. Robertson D. The epidemic of orthostatic tachycardia and orthostatic intolerance; *Am J Med Sci* 1999; 317: 75–77.
3. Benrud-Larson L.M, Dewar M.S, Sandroni P, et al. Quality of life in patients with postural tachycardia syndrome. *Mayo Clin Proc* 2002; 277: 531–537.
4. Carew S, Connor M.O, Cooke J, Conway R, Sheehy C, Costelloe A, and Lyons D. A review of postural orthostatic tachycardia syndrome; *Europace* 2009; 11: 18–25.
5. Raj S.R. Postural Tachycardia Syndrome (POTS): Pathophysiology, Diagnosis & Management; *Indian Pacing Electrophysiol J* 2006; 6(2): 84–99.
6. McEvoy M.D, Low P.A, Hebbar L. Postural Orthostatic Tachycardia Syndrome: Anesthetic Implications in the Obstetric Patient; *Anesth Analg* 2007; 104: 166–167.
7. Corbett W.L, Reiter C.M, Schultz J.R, Kanter R.J and Habib A.S. Anaesthetic management of a parturient with the postural orthostatic tachycardia syndrome: a case report. *British Journal of Anaesthesia* 2006; 2: 196–199.
8. Glatter K.A, Tuteja D, Chiamvimonvat N, et al. Pregnancy in postural orthostatic tachycardia syndrome. *Pacing Clinical Electrophysiol* 2005; 28: 591–593.
9. Lee A, Ngan Kee W.D, Gin T. A quantitative, systematic review of randomized controlled trials of ephedrine versus phenylephrine for the management of hypotension during spinal anesthesia for cesarean delivery; *Anesthesia Analgesia* 2002; 94: 920–926.

Author Information

Saravanan Ramamoorthy, M.D

Department of Anesthesiology, Milton. S. Hershey Medical Center, Pennsylvania State University

Venugopal Reddy, M.D

Department of Anesthesiology, Milton. S. Hershey Medical Center, Pennsylvania State University

Padmani Dhar, M.D

Department of Anesthesiology, Milton. S. Hershey Medical Center, Pennsylvania State University