



New Diagnosis of Postural Orthostatic Tachycardia Syndrome (POTS) in Pregnancy

Leela SP*, Reddy A, Atti V and Buschardt M

Midland Memorial Hospital, Texas Tech University Of Health Sciences Center, USA

***Corresponding author:** Leela Sharath Pillarisetty, Midland Memorial Hospital, 400 Rosalind Redfern Grover Parkway, 3rd Floor, Midland, Texas, USA, Tel: 79701 432 681-3100; Email: Drleelasharath@yahoo.com

Received Date: March 02, 2019; **Published Date:** March 13, 2019

Abstract

Introduction: Postural Orthostatic Tachycardia syndrome also known as POTS, is a syndrome of autonomic dysfunction and is characterized by marked increase in the heart rate without associated orthostatic hypotension, most of the existing literature on POTS are case reports and reviews on the patients who were already given the diagnosis of POTS even before attainment of pregnancy. Our case report and review is unique because it will discuss a patient who was newly diagnosed with POTS in pregnancy.

Case presentation: We present a case of 22 years old, primigravida with Postural Orthostatic Tachycardia Syndrome (POTS) which was unrecognized for seven years that continued in pregnancy and masked symptoms, making it difficult to diagnose. We also present the differential diagnosis of POTS and a review on why is it difficult to diagnose in pregnancy and its management.

Conclusion: The course of POTS in pregnancy is variable it does not increase the risk of complications in pregnancy and childbirth and also has no negative effects on the fetus or infant. POTS in pregnancy should not influence the mode of delivery. Vaginal delivery is still the safest route. POTS do not complicate the postpartum course or the choice for contraception.

Keywords: POTS; Pregnancy; Orthostasis; Tachycardia; Autonomic dysfunction

Abbreviations: POTS: Postural Orthostatic Tachycardia Syndrome; MFM: Maternal-fetal medicine; EFM: Electronic Fetal Monitoring.

Case Description

Introduction

Postural Orthostatic Tachycardia syndrome, also known as POTS, is a syndrome of autonomic dysfunction and is

characterized by a marked increase in the heart rate by greater than 30 bpm or heart rate greater than 120 bpm. The tachycardia must occur within 10 minutes of standing from a supine position and be without associated orthostatic hypotension [1]. POTS is particularly common in young patients between the ages of 15-30 years and is more frequently seen in females (4.5:1) [2]. The most common symptoms of POTS include; light-headedness, frequent syncopal episodes, fatigue, and weakness. Other

symptoms of autonomic dysfunction, such as; diarrhea, dyspnea, and chest pain may also be present. The symptoms of POTS are very similar to pregnancy symptoms, making its diagnosis particularly difficult during pregnancy [3]. Most of the existing literature on POTS is composed of case reports and reviews of reviews of patients given the diagnosis before pregnancy. This report represents a unique case because it discusses a patient who was newly diagnosed with POTS during pregnancy.

Case Report

Antepartum care

A 22-year-old Caucasian woman, at ten weeks of gestation, initially presented to her obstetrician with symptoms of; dizziness, palpitations, fatigue, and frequent syncopal episodes. Her dizziness and palpitations were worsened when going from the supine to standing position. Despite multiple multidisciplinary consultations, hospital admissions, and an ICU admission, she went undiagnosed for seven years.

Patient's initial vital signs were abnormal, with tachycardia ranging from 90-110's and elevated blood pressure ranging from 130-140/90's. Her initial prenatal labs were normal. She was severely symptomatic at the time of initial presentation, with a history of multiple syncopal episodes, tachycardia, and hypertension, so she was admitted to the hospital. A multidisciplinary team, consisting of Maternal-fetal medicine (MFM), intensivists, as well as, an obstetrician was responsible for her care. During her hospital stay, the patient underwent a thorough workup for orthostatic intolerance. A continuous Halter monitor was performed, which showed an increased heart rate of more than 30 beats per minute within 10 minutes of standing from the supine position. No significant changes in blood pressure were observed. The following tests were performed; CBC, CMP, HgA1c, thyroid studies, EKG, Chest X-ray, echocardiogram and 24-hour urine chemistries, in order to rule out other causes of orthostatic intolerance and postural tachycardia. Plasma catecholamine levels were measured and found to be normal. At 10-12 weeks gestation, the patient has prescribed metoprolol orally, once daily, which resulted in significant improvement in her symptoms. The patient was managed on metoprolol throughout her pregnancy and underwent induction of labor at 37 weeks gestation. She continues to follow closely with her obstetrician, cardiology, and MFM.

Intrapartum care

Due to worsening symptoms and unstable vital signs, MFM recommended early induction of labor. She was admitted to the labor and delivery unit at 37 weeks gestation and was induced using vaginal misoprostol. The fetus was monitored using continuous electronic fetal monitoring (EFM). She received epidural anesthesia without difficulty. Initial induction and labor progressed well. Upon complete dilation of the cervix, fetal heart tracings showed recurrent late decelerations, which were not improving with resuscitative measures. Subsequently, maternal bearing down resulted in a prolonged deceleration in fetal heart tracing, without improvement. The fetal head was found to be at -1 station and in persistent occiput posterior position. Due to fetal intolerance and malposition, the decision was made to perform an emergency cesarean section. She delivered a healthy female infant weighing 7 lbs. 3 oz., with Apgar scores of 9 and 10 at 1 and 5 minutes respectively.

Postpartum care

Her immediate postpartum course in the hospital was uneventful, and she continued metoprolol post-partum without any symptoms. She followed up with her obstetrician six weeks after delivery without any complaints and received an intrauterine device for contraception. She also continued to follow up with her primary care physician and cardiologist.

Discussion

The etiology of POTS is heterogeneous, and many causes have been identified without clear attribution [4]. Some of the factors which can exacerbate POTS include; heat exposure, physical exertion, heavy metal exposure, prolonged recumbence, menses, and certain drugs such as diuretics and vasodilators [3,4].

Symptoms of POTS

- a) Palpitations
- b) Chest discomfort
- c) Light-headedness
- d) Tremors
- e) Nausea and vomiting
- f) Mental clouding ('brain fog')
- g) Breathlessness
- h) Syncope (30%)

Diagnostic criteria for POTS [3]

- a. Increase in heart rate of > 30 bpm or HR > 120 bpm within 10 min of changing from supine to standing position.
- b. Symptoms lasting for more than 6 months.
- c. Symptoms worse with standing and better with recumbency.

Although not required for diagnosis, ruling out other causes of hypovolemic and a standing plasma Norepinephrine level >600 pg/ml (3.5nM), can both support a diagnosis of POTS.

POTS in pregnancy

It is indeed a challenge to diagnose POTS in pregnancy, as most of the symptoms of POTS mimic common pregnancy symptoms [3]. Pregnancy itself can cause; lightheadedness, syncope, palpitations, and dyspnea, which can hinder the diagnosis of POTS [3]. In pregnancy, numerous adaptations occur in the cardiovascular system to meet increasing demands. For example, blood volume increases by 45 % [3, 5]. Due to increases in both heart rate and stroke volume, the maternal cardiac output increases by 40 % [3, 5]. Peripheral vascular resistance also decreases in pregnancy, resulting in lower blood pressures with a nadir around 30-32 weeks gestation [6, 7]. All these regular physiological changes can preclude the diagnosis of POTS in pregnancy.

Most of the existing literature on POTS is composed of case reports and reviews of patients given the diagnosis before pregnancy. This report represents a unique case because it discusses a patient who was newly diagnosed with POTS during pregnancy.

One should have a high index of clinical suspicion to diagnose POTS in pregnancy. Although POTS is rare, it should be included in the differential diagnosis in any pregnancy complicated by recurrent syncopal episodes and orthostatic intolerance. Thorough clinical history, physical exam, as well as, other laboratory data are essential. A multidisciplinary team, consisting of an obstetrician, primary care physician and cardiologist is an essential part of the diagnosis and management of POTS in pregnancy.

Differential Diagnosis of POTS

- a. Arrhythmias
- b. Thyrotoxicosis
- c. Pheochromocytoma
- d. Conn's syndrome
- e. Preeclampsia
- f. HELLP Syndrome
- g. Cardiomyopathy

- h. Other causes of hypovolemia (anemia, dehydration)
- i. Neurocardiogenic syncope

POTS and neurocardiogenic syncope are both types of orthostatic intolerance, lead to very similar clinical manifestations, and are positional (can be diagnosed by table tilt) [8]. However, POTS is defined by tachycardia, while NCS is defined by a rapid decrease in heart rate and/or blood pressure. [8] These subtle differences can also lead to differences in treatment and are thus important. Both syndromes improve with recumbence.

Antepartum

The course of POTS in pregnancy is variable [9-12] [13]. The diagnosis of POTS does not have an increased risk of complications in pregnancy and childbirth [10,11,14,15,13] and has no adverse effects on the fetus or infant [9,10,11,15]. Patients who were not treated before pregnancy were less likely to have an exacerbation during pregnancy [11]. Most studies have concluded that POTS does not affect pregnancy-related complications [10,11,14,15]. In a retrospective chart review by Cecilia et al, there was no apparent association between pregnancy and POTS symptomatology, exacerbation, or treatment within their cohort of nine patients.

Intrapartum

POTS in pregnancy should not influence the mode of delivery [13]. Most women with POTS in pregnancy can undergo safe vaginal delivery without any complications [10,12,16]. Epidural analgesia is safe to administer in these patients [11]. In a review by Cecilia et al., seven patients delivered vaginally without complications, five of which received epidural anesthesia. Blood pressure and heart rate changes can lead to increased resistance in the pelvic blood vessels thereby decreasing uteroplacental perfusion. Valsalva maneuver during the second stage of labor can initially decrease blood pressure, and in later stages increase blood pressure and heart rate.

For these reasons, Mc Evoy et al. concluded that patient and fetus should be monitored continuously for hemodynamic changes during the second stage of labor. If hemodynamic variations occur and do not return to normal between contractions, avoidance of Valsalva through instrumental or cesarean delivery should be performed [16].

Postpartum

In the same review, by Cecilia et al., beta blocker treatment during pregnancy did not result in fetal bradycardia or intrauterine growth restriction. Another review by Kanjwal et al., reported that 69% of POTS

patients had stable symptoms during pregnancy, while 27% had an increase in their symptoms during pregnancy and continued to have symptoms in the postpartum period.

Management of POTS in pregnancy

The primary goal of treatment of POTS in pregnancy is to attain symptomatic relief through prevention of syncopal episodes, falls and injuries. This can be achieved through conservative approaches or with medications, as listed below in Table 1. In general, during the second stage of labor, passive descent is recommended in women with POTS, if labor is progressing as expected. This article supports a high index of suspicion in pregnant patients with symptoms of orthostatic intolerance, especially since these symptoms may be masked by those typically found in pregnancy. Although pregnancy-related complications are typically unaffected by the diagnosis of POTS [10,11,13,14], recognition of the diagnosis in pregnancy can decrease maternal morbidity. This is particularly true in severe cases. (Take home message).

Conservative management

- a. Compression stockings.
- b. 2. Volume expansion
- c. 3. Physical maneuvers

Disclosure of Interests

The authors have no conflict of interest to disclose.

Contribution to Authorship

All the authors were responsible for designing the case report, writing the manuscript, all the authors are responsible for the final manuscript.

Acknowledgements

We sincerely thank Texas Tech University Health Sciences Center of the Permian Basin and Dr. Natalia Schlabritz , Dr.Lori Stafford for their inputs, thoughts and contributions towards the case report.

References

1. Lide B, Sina Haeri S (2015) A Case Report and Review of Postural Orthostatic Tachycardia Syndrome in Pregnancy. *AJP Rep* 5(1): e33-6.
2. Glatter KA, Tuteja D, Chiamvimonvat N, Hamdan M, Park JK (2005) Pregnancy in postural orthostatic tachycardia syndrome. *Pacing Clin Electrophysiol* 28(6): 591-593.
3. Pramy N, Puliyahtinkal S, Sagili H, Jayalaksmi D, Reddi RP (2012) Postural orthostatic tachycardia syndrome complicating pregnancy: a case report with review of literature. *Obstet Med* 5(2): 83-85.
4. Benarroch EE (2012) Postural tachycardia syndrome: a heterogeneous and multifactorial disorder. *Mayo Clin Proc* 87(12): 1214-1225.
5. Monga M (1999) Maternal cardiovascular and renal adaptation to pregnancy. In: Creasy RK & Resnik R (Eds.), *Maternal-Fetal Medicine*. Philadelphia, W.B. Saunders, pp. 783-792.
6. Elkayam U, Gleicher N (1982) Cardiovascular physiology of pregnancy. In: Elkayam U, Gleicher N (Eds.), *Cardiac Problem in Pregnancy*. New York, Alan R. Liss Inc., pp. 5-26.
7. Ueland K (1976) Maternal cardiovascular dynamics. VII. Interpartum blood volume changes. *Am J Obstet Gynecol* 126(6): 671-677.
8. Kanjwal K, Sheikh M, Karabin B, Kanjwal Y, Grubb BP (2011) Neurocardiogenic syncope coexisting with postural orthostatic tachycardia syndrome in patients suffering from orthostatic intolerance: A combined form of a dysfunction. *Pacing Clin Electrophysiol* 34(5): 549-554.
9. Blitshteyn S, Poya H, Bett GC (2012) Pregnancy in postural tachycardia syndrome: clinical course and maternal and fetal outcomes. *J Matern Fetal Neonatal Med* 25(9): 1631-1634.
10. Kanjwal K, Karabin B, Kanjwal Y, Grubb BP (2009) Outcomes of pregnancy in patients with preexisting postural tachycardia syndrome. *Pacing Clinical Electrophysiol* 32(8): 1000-3.
11. Powless CA, Harms RW, Watson WJ (2010) Postural tachycardia syndrome complicating pregnancy. *The J Matern Fetal Neonatal Med* 23(8): 850-853.
12. Pramy N, Puliyahtinkal S, Sagili H, Jayalaksmi D, Reddi RP (2012) Postural orthostatic tachycardia syndrome complicating pregnancy: case report with review of literature. *Obstet Med* 5(2): 83-85.
13. Morgan, K, Choienta C, Tavener M, Smith A, Loxton D (2018) Postural Orthostatic Tachycardia Syndrome during pregnancy: A systematic review of

- the literature. *Autonomic Neuroscience* 215: 106-118.
14. Peggs KJ, Nguyen H, Enayat D, Keller NR, Al-Hendy A, et al. (2012) Gynecologic disorders and menstrual cycle lightheadedness in postural tachycardia syndrome. *Int J Gynaecol Obstet* 118(3): 242-246.
 15. Kimpinski K, Iodice V, Sandroni P, Low PA (2010) Effect of pregnancy on postural tachycardia syndrome. *Mayo Clin Proc* 85(7): 639-644.
 16. Torfs CP, Katz EA, Bateson TF, Lam PK, Curry CJR (1996) Maternal medications and environmental exposures as risk factors for gastroschisis. *Teratology* 54(2): 84-92.