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Case Report

Ventricular tachycardia and seizure in hyperemesis gravidarum

Jadhav Rahuldeo S[1], Kumar Sushil[2], Kapur Anupam[3], Tugnait Pragati[4]

[1]PG Resident [2]HOD, [3]Classified Specialist [4]Graded Specialist
Department of Obstetrics and Gynaecology, INHS Asvini, Naval Hospital, Mumbai – 400 005.

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Introduction:

Nausea and vomiting are common (prevalence 70% and 59% respectively) during the first trimester of pregnancy. Persistent vomiting becomes hyperemesis when the patient is unable to maintain adequate hydration. Fluid and electrolyte as well as nutritional status may be jeopardized as a result of the severity and duration of the problem[1]. Complications and hyperemesis include weight loss, dehydration, acidosis from malnutrition, alkalosis from hypokalemia, muscle weakness, electrocardiogram abnormalities, and tetany[2]. Hypokalemia is a known association with hyperemesis as well as known cause of ventricular tachycardia. To date, there has not been a reported case of ventricular tachycardia in association with hyperemesis gravidarum in PubMed and Cochrane database.

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Correspondence:
Dr. Tugnait Pragati, MD
Graded Specialist, Obstetrics and Gynaecology
INHS Asvini, Naval Hospital,
Mumbai – 400 005, Mobile – 9869176479
E-mail : cdrsushilkumar@rediffmail.com
rahulmsgn@yahoo.com

Case report

A 25-year-old gravida 3, para 2 presented with 13 weeks amenorrhea and 4 weeks of nausea, vomiting and ptyalism. She did not drink alcohol. A diagnosis of hyperemesis gravidarum was made. She was admitted and managed with intravenous infusion and metaclopramide. She improved enough to be discharged 6 days later. She was readmitted 4 weeks later with persistent vomiting and inability to tolerate intake of either solids or fluids. The woman was well oriented and afebrile. Examination revealed a pulse of 74 per minute. Blood pressure was 100/60mmHg. The buccal mucus membranes were dry and there was no clinical evidence of jaundice. Physical examination was otherwise unremarkable.

Laboratory evaluation revealed hypokalemia (2.4 mEq/L; normal 3.5 to 5.5 mEq/L), hyponatremia (109 mEq/L; normal- 130-145 mEq/L), ketonuria (++), and impaired liver function (bilirubin- 0.83 mg/dL; normal- 0.3 to 1.0 mg/dL ; aspartate aminotransferase – 103 U/L, normal 0-35 U/L; alanine aminotransferase – 87 U/L, normal- 0-35 U/L). Ultrasound examination revealed a single viable fetus compatible with dates.

On the third day of admission she had recurrent generalized clonic tonic convulsions. Intravenous infusion of

phenobarbitone was started. We found progressive fall in oxygen saturation along with spasticity in lower limbs. Hence she was placed on mechanical ventilation. One day later she developed hypotension. So she was given ionotropic support. Her pulse was 130/minute and blood pressure 86/70 mmHg. She was comatose. Pupils were reactive and symmetrical with no papilledema on fundoscopy. There was no nystagmus. Bilateral planters were extensors. Cranial nerves were intact.

On the 4th day of admission she had sustained ventricular tachycardia with hypotension and evidence of Torsade de pointes on cardiac monitor. It was reverted to sinus cardiac rhythm with synchronized DC shock of 150 joule, lignocaine and intravenous magnesium sulfate. She had recurrent episodes of polymorphic ventricular tachycardia on the 5th and 6th day of admission for which repeated synchronized DC shock was given and it reverted to sinus rhythm.

She aborted spontaneously on the 5th day of admission. Ultrasonography of the pelvis was suggestive of retained products of conceptus. She was weaned off the ventilator on the 6th day of admission with optimal correction of electrolyte deficiency. She was hemodynamically stable without ionotropic support. Elective evacuation of retained products was done on the 10th day of admission under general anesthesia.

Discussion

This is a first case report of ventricular tachycardia complicating hyperemesis gravidarum. This will remind readers of the possible severe consequences of hyperemesis gravidarum. Many serious complications of hyperemesis gravidarum have been reported including Wernicke's encephalopathy, central pontine myelinolysis, convulsions[4], coma, Marchiafava bignami disease[5], retinal damage[6], hepatic dysfunction, coagulopathy[2], renal damage, Mallory-Weiss syndrome, aspiration pneumonia, esophageal rupture and pneumomediastinum.

In our patient the disease was evidently precipitated by prolonged vomiting. Acute deterioration was probably triggered by infusion of carbohydrates in the form of intravenous fluids[4]. The recurrent generalized clonic tonic convulsion was attributed to hyponatremia. Polymorphic ventricular tachycardia was attributed to hypokalemia. Rapid correction of hyponatremia may result in central pontine myelinolysis. Hence chronic hyponatremia should be corrected slowly[3]. Other electrolyte abnormalities particularly hypocalcemia and hypomagnesemia may contribute to seizure[4].

References :

1. Nelson-Piercy C, Swiet M. Corticosteroids for the treatment of hyperemesis gravidarum. *Br J Obstet Gynaecol 1994;01:1013-5.*

2. Robinson JN, Banerjee R, Thiet MP. Coagulopathy secondary to vitamin K deficiency in hyperemesis gravidarum. *Obstet Gynecol 1998;92:673-5.*

3. Bergin PS, Harvey P. Wernicke's encephalopathy and central pontine myelinolysis associated with hyperemesis gravidarum. *BMJ 1992;305:517-8.*

4. ReesJH, Ginsberg L, Schapira AHV. Two pregnant women with vomiting and fits. *Am J Obstet Gynecol 1997;177:1539-40.*

5. Hillbom M, Pyhtinen J, Pylvanen V et al. Pregnant, vomiting and coma. *Lancet 1999;353:1584*

6. Tesfaye S, Achari V, Yang YC et al. Pregnant, vomiting and going blind. *Lancet 1998;352:1594*