Case Report

Ventricular tachycardia and seizure in hyperemesis gravidarum

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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 problem¹. Complications and hyperemesis include weight loss, dehydration, acidosis from malnutrition, alkalosis from hypokalemia, muscle weakness, electrocardiogram abnormalities, and tetany². 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.

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

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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 inotropic 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 inotropic 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 myeli-

nolysis, convulsions⁴, coma, Marchiafava bignami disease⁵, retinal damage⁶, hepatic dysfunction, coagulopathy², 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 fluids⁴. 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 slowly³. Other electrolyte abnormalities particularly hypocalcemia and hypomagnesemia may contribute to seizure⁴.

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