Hyperemesis gravidarum with acute liver injury and positive feto-maternal outcome

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ABSTRACT

This is a case report of Hyperemesis gravidarum (HG) with a distinctly abnormal elevated ALT and AST values, in association with intractable nausea and vomiting. A 26-year-old woman G2P1A0 with a 9-week gestational age and no significant past medical history, presented with intractable nausea and vomiting despite medication use for three weeks. Despite administration of Vitamin B6, the symptoms persisted. During this period, the patient was unable to tolerate solids or liquids, and experienced progressive weakness, fatigue, and non-radiating epigastric pain. The patient was admitted to the Brooklyn Hospital Center Family Medicine service and found to have notable electrolyte disturbances, and remarkable transaminitis, with alanine aminotransferase (ALT) and aspartate aminotransferase (AST) values of 3400 IU/L and 717 IU/L respectively. All alternative diagnoses were ruled out. Ultimately, transaminitis resolved and mother an baby are doing well. This case report describes the typical clinical presentation of HG with acute liver injury and positive feto-maternal outcome.

Keywords: Acute liver injury, Benign transaminitis, Hyperemesis gravidarum, Pregnancy

INTRODUCTION

Hyperemesis gravidarum (HG) was first documented by Antoine Dubois, the head obstetrician to Napoleon Bonaparte in 1852, as “pernicious vomiting of pregnancy” [1]. HG is extremely unique in that it is a common presentation on the severe end of the spectrum of nausea and vomiting experienced by many women, yet studies have not concluded a firm pathogenesis, leading to varying epidemiological factors and incidence rates. Approximately 50–80% of pregnant women experience nausea, and an estimated 50% suffer from retching [2]. HG, in which persistent vomiting is associated with a 5% or more loss of pre-pregnancy body weight, dehydration, and ketosis, occurs in up to 3% pregnancies [3]. Up to half of hospitalized women have abnormal liver enzymes. Aminotransferase levels may rise up to 200 IU/L but are generally below 300 IU/L, [3] and alkaline phosphatase may rise to twice the normal value. Both direct and indirect bilirubin values may rise to 4 mg/dL, [4] and serum amylase and lipase levels may rise up to five times the normal values.

The diagnosis of nausea and vomiting during pregnancy, is a clinical diagnosis without distinct criteria, and will vary on presentation, however it is important...
to rule out life threatening differential diagnosis like Intrahepatic Cholestasis do Pregnancy (ICP), Acute Fatty Liver of Pregnancy (AFLP), HELLP syndrome or Acute Hepatitis just to mention a few. Most women will experience the onset of HG within five to six weeks of gestation, and the symptoms will usually dissipate by around twenty weeks of gestation [5].

CASE REPORT

A 26-year-old G2P1A0 African American female at nine weeks gestation with no medical history presented to the Emergency Department upon referral from the outpatient OB-GYN office at The Brooklyn Hospital Center for further evaluation of intractable nausea and vomiting. She complained of weakness, fatigue, and non-radiating epigastric pain without improvement despite medication use, including Vitamin B6, for three weeks. During her previous pregnancy, the patient also experienced nausea and vomiting to a lesser extent. The patient did not have any known drug or food allergies. Her family history was not significant for any medical disorders, and she denied history of smoking, alcohol, or illicit drug use.

On examination, the patient was a young, well-nourished, ambulating female oriented to person, place and time. Her physical examination was notable only for slight weakness and minimal epigastric pain on palpation. No other abnormalities were noted on physical exam. Laboratory analysis on admission showed an elevated BUN/Cr ratio of 35, hyponatremia (134 mmol/L) and hypochloremia (80 mmol/L) which was corrected by administration of Ringer Lactate 2L in 4 hours, Thiamine 100mg, and started maintenance intravenous fluid with D5NS at 150 mL/h and supplemental potassium. The patient also received and IV pushes of Metoclopramide 5 mg q6h/day and Famotidine 20mg IVPB q12h for 24 hours. Of note, the LFTs were exceedingly abnormal with AST and ALT values of 717 IU/L and 3400 IU/L respectively, which were indicative of potential liver damage as acute phase reactants. RUQ ultrasonography showed distended gallbladder with biliary sludge with no inflammation or signs of acute cholecystitis. HELLP syndrome was ruled out, as her platelets and haptoglobin were within reference range. Gastroenterology ruled out acute viral or autoimmune hepatitis, drug-induced liver injury, and hepatic ischemia. Laboratory values included in the work up were Hepatitis profile including HEV, Ceruloplasmin, Anti-Smooth antibody, ASA and Tylenol levels, Hemoglobin electrophoresis, and STD panel; all of them were within normal limits and/or negative.

The liver chemistries subsequently down-trended with no other interventions than controlling the hyperemesis and maintaining appropriate electrolytes balance and hydration. Patient’s symptoms improved, she was eventually able to tolerate oral intake by her fourth day of admission, and was subsequently discharged. Outpatient consult with Gastroenterology recommended no further intervention due to normalization of liver enzymes and normal liver functions studies, one month after the HG episode.

Patient delivered a healthy newborn via C-Section at 36 weeks of gestation due to preterm premature rupture of membranes in early labor, with no other complications. Currently mother and baby are doing well with unremarkable postpartum course.

DISCUSSION

Nausea and vomiting are very common complaints in the first trimester of pregnancy. HG usually indicates severe vomiting associated with dehydration and electrolyte abnormalities which prompt hospitalization in some cases [5]. HG usually resolves spontaneously around sixteen weeks, or during the second trimester. The exact pathogenesis is unclear, but has been concluded that it is multifactorial in nature, and a clinical diagnosis of exclusion. While more common in younger women, it has been found that there are no clear epidemiological factors or incidence rates that are definitive for diagnosing HG [6, 7].

Liver dysfunction demonstrated by elevated serum aminotransferases (LFTs) is noted in 15–50% of patients with HG [5]. There is no known etiology of the elevation, but it usually resolves with concurrent improvement in emesis. However, the extent of elevation is usually up to two to three times the upper limit of normal, and rarely as high as 1000 IU/L. There is a direct proportional relationship between the severity of vomiting and the derangement of liver enzymes. Liver enzyme levels above the upper limit of normal are extremely rare, and since there has not been a previous case with appropriate treatment or follow up, the approach was unique and prolonged, until the patient was able to tolerate PO intake, and demonstrated a down-trending hepatic panel. Serum amylase and lipase levels are elevated in roughly 10–15% patients, and can increase up to five times the upper limit of normal. The increase in these enzymes are not specific, and since their origin can be variable, there has been no concluded correlation between their elevation and their role in evaluation and diagnosis of HG.

The evaluation of pregnant women with the symptomatology of HG, includes a vast laboratory and radiological analysis, spanning from CBC and BMP to ultrasonography, excluding the necessity for liver biopsy. Electrolyte derangements include hypochloremic metabolic alkalosis, due to excessive emesis and hypokalemia. In some cases, ketosis can occur if caloric intake is minimal and appetite is decreased secondary to nausea and vomit. The management of HG includes correcting the dehydration, electrolyte abnormalities, and minimizing fetal effects of maternal nausea and vomiting. Treatment modalities are targeted towards specific
symptomatology; nausea alone, nausea with vomit and dehydration, nausea with vomit without dehydration, and so forth. In regards to this case, the patient’s nausea and dehydration due to vomiting were approached with initial IV fluids and Metoclopramide, whereas standard guideline algorithms suggest initial approach to nausea with vomiting and dehydration with Ondansetron as the primary anti-emetic [1]. Hospital admission is generally appropriate for women with HG symptoms persisting after re-hydration and IV anti-emetic treatments in the emergency setting. The decision to admit or discharge must be individualized, depending on severity of the disease, other recurring or new onset of symptoms, and patient preference.

In this report, we described a patient with HG with intractable nausea and vomiting, weakness, fatigue, non-radiating epigastric pain, and significantly elevated LFTs. After the nausea and vomiting persisted despite treatment for three weeks with conventional management, the patient was referred to the Brooklyn Hospital Center’s emergency department and symptomatically treated with IV fluids and anti-emetic therapy. Significant laboratory chemistry derangements were found in liver panel profile which prompted to more detailed work up looking for the most common courses of acute liver failure in pregnancy. All of which were ruled out and made us to conclude it was a case of HG were AST and ALT had 18 and 60 folds increased respectively. Most importantly, with the appropriated management, there were no sequels for the mother nor the newborn.

CONCLUSION

Hyperemesis gravidarum is a diagnosis of exclusion and persistent symptoms is prompted. Further evaluation to rule out more serious entities. Nevertheless, HG with acute liver injury treated and monitored appropriately can have a positive outcome.

REFERENCES


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Author Contributions

Ricardo Alonso – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published
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The corresponding author is the guarantor of submission.

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Consent Statement

Written informed consent was obtained from the patient for publication of this case report.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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