

Case Report

## Transient Biliary Sludge in A Woman of 11 Weeks Gestation with Hyperemesis Gravidarum

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### Abstract

Nausea and vomiting during pregnancy is common, but hyperemesis gravidarum (HG) is a rare, occurring in approximately 0.1% of pregnancies. HG presents with severe and persistent nausea and vomiting, which can lead to dehydration, starvation ketosis and liver dysfunction, and predisposition the patient to biliary sludge/gallstone formation. We report the case of a 37-year-old G2P1 woman, who presented with HG and transient biliary sludge at 11 week gestation.

**Keywords:** Hyperemesis Gravidarum; Transient Biliary Sludge

### Abbreviations

HG: Hyperemesis Gravidarum

### Introduction

The reported incidence of hyperemesis gravidarum is 0.3 to 1.0%, and HG can lead to dehydration, starvation ketosis and liver dysfunction, and predisposition to biliary sludge/gallstone formation [1]. Asymptomatic gallstones are noted in 2-4% of pregnant women, and aside from acute appendicitis, acute cholecystitis is the second most common non-obstetrical indication for surgery in pregnant women [2-4]. The reported incidence of gallstone-related diseases in pregnant women is 0.05 to 0.33% [3]. In a prospective study, the cumulative incidence of new biliary sludge/stones or progression of baseline sludge to stones was 10.2% by four to six weeks postpartum [4]. Biliary sludge is a mixture of calcium, bilirubin crystals,

cholesterol crystals, calcium bilirubin pigment, and other particulate solids that have precipitated from bile. Biliary sludge has been reported with pregnancy and is associated with rapid weight loss, critical illness involving low or absent oral intake and the use of total parenteral nutrition (TPN). The clinical course of biliary sludge varies. Although it often vanishes, the sludge sometimes progresses to gallstones. It also might occur transiently and vanish after hydration and when oral intake is resumed in the above conditions. HG with transient biliary sludge is rarely reported. HG is typically of persistent and excessive vomiting with considerable ketonuria, greater than 5% weight loss. HG in pregnancy can also be secondary to a multiple gestation, molar pregnancy, cholecystitis, pyelonephritis, pancreatitis, hepatitis, and, hyperthyroidism [2]. There is a sig-

nificant association between *Helicobacter pylori* infection and hyperemesis gravidarum in hyperemetic pregnant patients [3].

## Case report

We report the case of a 37-year-old G2P1 woman in her 11th week of gestation who was admitted to the hospital with hyperemesis gravidarum and starvation ketosis (urine ketone 4+). Past history revealed a gallbladder polyp and previous hyperemesis gravidarum (HG) in her first uneventful pregnancy four years prior. Physical examination showed the following: BW 55 kg, BH 155 cm, body temperature 36.8 °C, blood pressure 113/75 mmHg, HR 91 bpm, and no jaundice but decreased skin turgor. Laboratory data revealed hematocrit 33.9%, hemoglobin 11.6 g/l, white-cell count 8100/uL, neutrophils 79.3%. She had lost 2 kg (57 kg to 55 kg) within seven days. Blood chemistry showed normal TSH, elevated aspartate aminotransferase (GOT) 62 U/L (normal 8-38), and alanine aminotransferase (GPT) 98 U/L (normal 4-44). After admission a pelvic ultrasonography confirmed a singleton pregnancy of 11 weeks. At hospital day 4, due to persistent upper abdominal pain and past history of gallbladder polyp, she was referred for trans-abdominal ultrasonography. Abdominal ultrasonography revealed an enlarged and over-distended gallbladder of 6.25x3.91 cm, half of which showed the presence of low-level echogenic material that shifted with position changes but without post-acoustic shadowing, indicative of biliary sludge rather than gallstone. The patient responded well to IV fluid administration and resumed a normal diet; concomitantly, her symptoms and liver dysfunction ameliorated. Approximately one week later, a subsequent abdominal ultrasonography revealed only gallbladder polyp, and no sludge was detected.



**Figure 1.** Abdominal ultrasonography findings showed an enlarged and over-distended gallbladder of 6.25x3.91 cm, half of which showed the presence of low-level echogenic material without post-acoustic shadowing.

## Discussion

This is the second report of the appearance/disappearance of biliary sludge association with hyperemesis gravidarum (HG) and liver dysfunction. Matsubara et al. (2011) reported two cases of HG, jaundice (one case), liver dysfunction, and transient biliary sludge [5]. Pregnancy itself has been reported to

increase the incidence of biliary sludge. Pregnancy increases the cholesterol saturation of bile and the rate of secretion of cholesterol. Pregnancy also decreases gallbladder motility with a net result of increased bile precipitation. HG with dehydration caused viscous bile and increased precipitation of bile. Biliary sludge has been detected in up to a third of pregnant women but does not usually cause symptoms [6]. It often vanishes, but sometimes progresses to gallstones [7]. HG may further enhance the formation of sludge. Physical examination of women with hyperemesis gravidarum is usually unremarkable. Additional upper abdominal ultrasonography imaging studies to evaluate the pancreas and/or biliary trees may be warranted if the patient's clinical presentation is atypical nausea and/or vomiting beginning after 9-10 weeks of gestation (as in this case). Both gallbladder stasis and stone/sludge formation can disappear after hydration and resumption of normal food intake, as in our and Matsubara's case reports. Reinstitution of oral food intake releases the sphincter of Oddi and induces strong gallbladder contractions, which then pushes the biliary sludge into the duodenum. It is still unclear whether the presence of biliary sludge during pregnancy is a risk factor for future gallstone formation.

In conclusion, HG with pregnancy-related physiologic changes might worsen the severity of hepatobiliary disease, such as cholecystitis. If a patient has HG with liver dysfunction and/or jaundice, an ultrasound of the gallbladder should be performed. In the presence of biliary sludge without signs of biliary tract obstruction, further invasive examinations, such as ERCP or gallbladder drainage should be avoided. Complications caused by biliary sludge include biliary colic, acute cholangitis, and acute pancreatitis; therefore, close follow-up of the clinical course is mandatory.

## Disclosure

None declared

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