Case Report

Hyperparathyroidism complicating pregnancy: A diagnostic challenge?

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ABSTRACT

Primary hyperparathyroidism (PHPT) is a rare etiology of hypercalcemia-induced pancreatitis, contributing about 0.4% to 1.5% of cases in the general population and up to 13% of cases during pregnancy. PHPT that occurs during pregnancy is a challenging diagnosis as the physiological changes in calcium homeostasis mask the symptoms of hypercalcemia. PHPT during pregnancy often remain undiagnosed and untreated, and may result in serious clinical implications for the mother and fetus. Most clinicians consider surgery within the second trimester of pregnancy as the treatment of choice in this group of patients. This article refers to a case of a 24-year married woman in whom PHPT was diagnosed for the first time in postpartum period. She succumbed to complications on Day 20 postpartum. Pathological findings revealed metastatic calcification in lungs, pancreas and uterine vessels, chronic pancreatitis and renal cortical necrosis.

KEY WORDS: Hypercalcemia, pregnancy, primary hyperparathyroidism

Introduction

Primary hyperparathyroidism (PHPT) is a rare etiology of hypercalcemia-induced pancreatitis, accounting for 0.4% to 1.5% of cases. To date, less than 200 cases with PHPT diagnosed during pregnancy have been described.\(^{[1]}\) Asymptomatic presentation and rarity of PHPT in the reproductive age group makes its diagnosis during pregnancy challenging.\(^{[2]}\) Laboratory findings are also masked by pregnancy-induced changes in calcium homeostasis.\(^{[3]}\) Pregnant females with PHPT often experience a clinically overt disease which makes correct diagnosis imperative. If left untreated, it may pose a significant risk to the mother and fetus in the form of postpartum maternal hypercalcemia, preterm delivery and fetal hypocalcemia that develops late.\(^{[4]}\)

Case Report

A 24-year woman was referred on Day 15 postpartum to our hospital with complaints of pain in abdomen, vomiting, fever and decreased urine output. She had delivered a preterm 1.9-kg female child in a referral hospital and her previous delivery was uneventful. During her pre-, intra- and postnatal period, she had history of repeated upper respiratory tract infections. On examination, she was febrile (103°F), pale, with a dry tongue, tachycardia and blood pressure of 140/60 mmHg. Right-sided lung crepitations were present. Tenderness and guarding were present in the epigastric region. The uterus was bulky (12 weeks) and antverted on per vaginal examination. Ultrasonography (USG) revealed increased echogenicity in kidneys and a slightly enlarged pancreas. A USG on Day 2 of admission showed an enlarged liver, bilateral enlarged kidneys with a poor corticomedullary ratio. On Day 6 of admission, a CT scan revealed enlarged pancreas consistent with acute pancreatitis, cortical nephrocalcinosis with acute renal failure. She had no history of gestational diabetes mellitus, hypo/hyperthyroidism, alcohol use, OC pill use, gallstones or drug intake. Initial laboratory investigations (Day 2) revealed amylaseaemia (220 U/A; normal values <150 U/A) and lipasaemia (646 U/A; normal values <150 U/A), increased leucocyte count (14,300/mm\(^3\)) and a hematocrit of 23.4%. Hemoglobin was 7.9 gm% and platelet count was 249 x 10\(^9\)/cmm. Lipid profile was normal, serum creatinine was 8 mg/dl and urea was 133 mg%. Calcium level was 11.56 mg/dl (normal value 8.5-10.1 mg/dl). Ionized calcium was 1.60 mmol/l (1.12-1.32 mmol/l). Phosphorus was 7.5 mg% (2.4-5 mg %). Prothrombin time (PT) - 22.1 sec (normal-12.5 sec), International normalized ratio (INR) - 1.76, activated partial thromboplastin time (APTT) - >60 sec (normal value 22-30). Acute pancreatitis with chronic renal disease with sepsis with disseminated intravascular coagulation (DIC) was diagnosed. On 17\(^{th}\) postpartum day parathyroid hormone was done and found to be raised at a value of 581 pg/ml (normal:15-65 pg/ml). Also her lipase was raised to 1018 U/l. And platelet count was reduced 56,000/cmm (on 17\(^{th}\) day itself). On Day 20, her PT
was 101.5 and INR was 7.8 and she died the same day. Postmortem examination revealed bilateral renal cortical necrosis [Figures 1 and 2], few whitish firm areas in lungs and chalky white firm areas in pancreas [Figure 3]. Histopathology revealed extensive calcification in lungs [Figure 4], pancreas [Figure 5] and uterine vessels [Figure 6]. Pancreas also showed interstitial fibrosis, inflammation [Figure 7] and peripancreatic fat necrosis.

Discussion

Gestational PHPT often goes undetected, and by the time of diagnosis, a majority of women have endured one or more failed pregnancies. This is because of the physiological changes in calcium homeostasis during pregnancy like
maternal blood volume expansion, hypoalbuminemia and increased fetal calcium requirements mask the symptoms of hypercalcemia. This happened with our patient. Maternal and fetal complications include nephrolithiasis, osteitis fibrosa cystica, pancreatitis, hypercalcaemic crisis, spontaneous miscarriage, intrauterine growth retardation, premature delivery, neonatal hypocalcaemia, fetal polyhydramnios and fetal death. Incidence of maternal and fetal complication is 67% and 80%, respectively. Interestingly, the frequency of pancreatitis in pregnancy-related hyperparathyroidism is higher (7-13%) than in nonpregnant (1-2%). PHPT causes pancreatitis by several mechanisms but most probably through hypercalcemia. In fact, persistent hypercalcemia might increase calcium concentration in pancreatic juice, and activate trypsinogen to trypsin causing pancreatic ductal and parenchymal damage leading to pancreatitis. Hypercalcemia also decreases the volume of pancreatic juice and causes protein plugs in the pancreatic duct which obstruct the flow leading to pancreatitis. Hypercalcemia causes vasoconstriction and pancreatic duct narrowing. Some believe that parathyroid hormone itself acts as toxin causing local thromboendarteritis. All these might result in pancreatic tissue necrosis. Our patient developed complications like pancreatitis and renal failure due to hypercalcemia as evinced by higher serum calcium levels and metastatic calcification in lung, pancreas and uterine vessels, and DIC in the postpartum period. Suspicion of PHPT and an early diagnosis can thus greatly benefit and help avert maternal and fetal morbidity.

### References


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