Pancreatitis In Pregnancy Secondary to Hyperparathyroidism and Hypercalcemia Treated by Parathyroidectomy

by Matthew J. Shellenberger, Robert Smith, David Schaefer and Chad Potteiger

A 21-year-old, G1, P0 at 30 weeks gestation, presented with a 1-day history of mid-epigastric abdominal pain radiating to her back with associated nausea and vomiting. She was found to have pancreatitis due to hypercalcemia and ultimately primary hyperparathyroidism. There is a lack of literature describing successful management when medical therapy is unsuccessful. We present a case of successful surgical parathyroidectomy in the third trimester after medical therapy had failed.

INTRODUCTION

Primary hyperparathyroidism is a rare etiology of hypercalcemic induced pancreatitis, causing anywhere from 0.4% to 1.5% of cases in the general population and up to 13% of cases during pregnancy (1–3). Several cases of hyperparathyroidism, hypercalcemia, and pancreatitis in pregnancy have been reported (4–8). In previously reported cases, the hypercalcemia was managed with medical intervention, usually with magnesium infusion (4,7). When magnesium proves ineffective, other agents such as phosphate-of-soda enemas, oral phosphates, calcitonin, and loops diuretics have been used with varied success (5,6). In the past mithramycin was used but is currently contraindicated secondary to teratogenic effects (3). Corticosteroids have also been used to decrease the absorption by the gastrointestinal tract, but have shown minimal effect when the hypercalcemia is secondary to hyperparathyroidism (3). Surgery is typically reserved until after delivery, but if necessary it is done during the second trimester in an effort not to disturb organogenesis during the first trimester and induction of labor during the third trimester (3,6). Our patient failed to improve with medical therapy necessitating surgical intervention during pregnancy. To date, a limited number of reported cases of parathyroidectomy during pregnancy have been reported to treat pancreatitis secondary to hypercalcemia. We report a case of pancreatitis secondary to hypercalcemia from hyperparathyroidism treated successfully with parathyroidectomy.

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CASE PRESENTATION

A 21-year-old, G1, P0 at 30 weeks gestation, presented with a 1-day history of mid-epigastric abdominal pain radiating to her back with associated nausea and vomiting. She denied fevers, or chills. Abdominal exam revealed good bowel sounds, epigastric tenderness, and voluntary guarding, without peritoneal signs. Obstetric exam was unremarkable. She denied the use of ETOH. Medications included prenatal vitamins. Lab studies revealed an amylase of 1907 U/L and a lipase of 2026 U/L. WBC count was 15.9 with a normal differential. Liver tests were normal. Triglycerides were 249 mg/dL. Abdominal ultrasound was normal. Ionized calcium was 1.41 mmol/L (1.13–1.32 mmol/L). Parathyroid hormone (PTH) was 82 pg/mL (6–40 pg/mL). Initial therapy with intravenous fluids and magnesium infusion failed to improve the hypercalcemia. Further medical therapy with diuretics and calcium binders was also unsuccessful. Therefore, she underwent parathyroidectomy. During surgery, rapid PTH monitoring revealed a rapid drop in the PTH from 258 to 87 picograms/mL after removal of a 1.4 cm parathyroid adenoma (Figures 1 and 2). Post-operatively her ionized calcium decreased to 1.0 mmol/L and her pancreatitis resolved. The patient recently delivered a healthy baby boy and has not had any further episodes of pancreatitis.

CONCLUSION

Pancreatitis in pregnancy secondary to hyperparathyroidism and hypercalcemia is a rare phenomenon often amendable to medical therapy. Pancreatitis during pregnancy is typically due to hyerlipidemia or biliary stones and occurs less than 13% of the time due to hyperparathyroidism and hypercalcemia (3,9–11). Our patient did have hypercalcemia and abdominal pain, but she lacked other previously described clinical clues of hyperparathyroidism in pregnancy, such as: she did not have hyperemesis, she had no history of kidney stones, and she had no history of miscarriages (3). The pathophysiology of pancreatitis in pregnancy is the same as in the non-pregnant patient. One of the most widely accepted theories is that calcium deposits in the pancreatic duct and causes the activation of trypsinogen to trypsin thus causing autodigestion of the pancreas (12). The degree of hypercalcemia in the mother must be monitored, as calcium is actively transported to the fetus, and parathyroid hormone is not transported across the placental barrier (8). Low fetal serum levels can lead to low fetal serum parathyroid hormone and complications after delivery secondary to this such as hypocalcemia, tetanus, and death (8). Fetal death rates have been sited up to 20%–30% in untreated mothers (3).

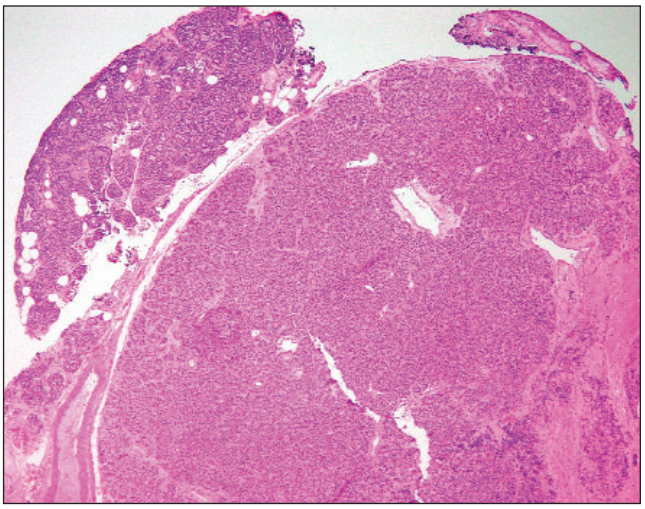

Figure 1. Parathyroid adenoma (40×)

There is a lack of literature describing successful management when medical therapy is unsuccessful. In one reported case, delivery of the infant at 36 weeks resulted in a successful outcome for both mother and

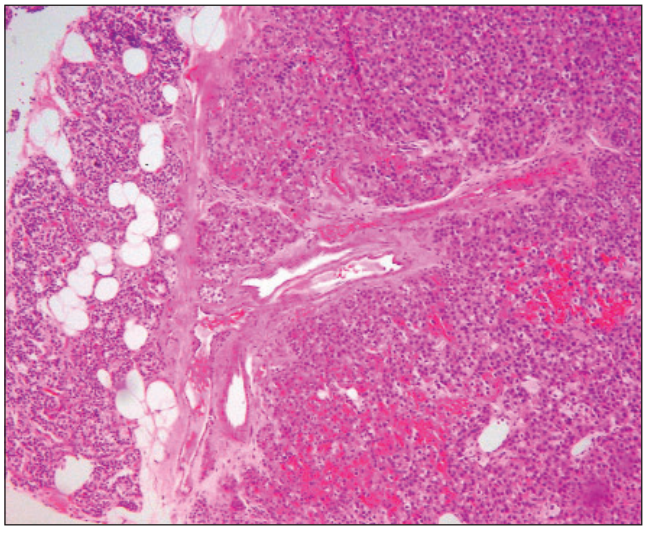

Figure 2. Parathyroid adenoma (100×)

infant (4). In our case, fetal viability and maturity precluded early delivery. If the child is able to be delivered, the mother must be monitored for hyperparathyroid crisis during the post partum period (3). This case illustrates the difficult clinical situation faced when conventional therapies for pancreatitis in pregnancy are unsuccessful. When medical therapies are exhausted, surgical therapy should be considered to improve maternal and fetal outcomes. ■

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