

# Hypertriglyceridemic Pancreatitis in Pregnancy - A Case Report

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Abstract: Acute pancreatitis is a rare event in pregnancy, occurring in approximately 3 in 10 000 pregnancies. The spectrum of acute pancraetitis in pregnancy ranges from mild pancreatitis to serious pancreatitis associated with necrosis, abscesses, pseudocysts, and multiple organ dysfunction syndromes. Acute pancreatitis is rare during first and second trimester (12%), usually occurring in the third trimester (50%) or early postpartum period (38%). The commonest identified causes of acute pancreatitis in pregnancy are gallstones (66%), alcohol (12%), hypertriglyceridemia (4%), idiopathic (17%). We present a case of 25-year-old primigravida who presented with severe epigastric pain and vomiting since five days. The patient was diagnosed with hypertriglyceridemic pancreatitis, which was managed conservatively. She recovered within several days and underwent a LSCS at term and delivered a healthy baby. Delayed diagnosis and treatment can allow for a greater prevalence of shock and sepsis in both the pregnant woman and fetus. Therefore, it is important to consider acute pancreatitis when a pregnant woman presents with upper abdominal pain, nausea and vomiting. Early diagnosis and proper therapeutic management help to reduce maternal mortality and fetal prematurity associated with acute pancreatitis during pregnancy.

*Keywords*: acute pancreatitis, gall stones, hypertriglyceridemia, pregnancy, serum amylase, serum lipase.

### 1. Introduction

Acute pancreatitisis a rare event in pregnancy, occurring in approximately 3 in 10000 pregnancies. The spectrum of acute pancreatitis in pregnancy ranges from mild pancreatitis to serious pancreatitis associated with necrosis, abscesses, pseudocysts, and multiple organ dysfunction syndromes (1). Acute pancreatitis is rare during first and second trimester (12%), usually occurring in the third trimester (50%) or early postpartum period (38%). The commonest identified causes of acute pancreatitis in pregnancy are gallstones (66%), alcohol (12%), hypertriglyceridemia (4%), idiopathic (17%). Hypertriglyceridemia is a rare cause of pancreatitis in pregnancy. This results from the increased estrogen effect of pregnancy and the familial tendency for some women toward high triglyceride levels.

### 2. Case report

A 25-year-old primigravida woman was admitted at 31 weeks of gestation with severe pain confined to the upper

abdomen, which was radiating to the back since 5 days and vomittings since a day before. On examination, pulse rate was 102/min, BP was 110/60 mmHg. Temperature was 100.20F and respiratory rate was 22/min. Per abdominal examination revealed epigastric tenderness with Uterus 32 weeks gravid size, relaxed. Fetal heart tones were at 140/min. Based on history and clinical examination, a differential diagnosis of pre eclampsia, acute appendicitis, red degeneration of fibroid, acute pancreatitis, gastroenteritis, bowel obstruction was made. Laboratory tests done on the day of admission showed a white blood count of 13,600/mm3, and a platelet count of 200,000. Liver function tests showed raised alkaline phosphatase - 112 mg/dl. Random blood sugar and renal function tests were within normal limits. Serum amylase was 388 IU/l (ref: 28-100 IU/l), lipase 1097 IU/l (ref: 13-60IU/l), calcium 8.7 mg/dl (ref: 8.5-11 mg/l), triglycerides 174 mg/dl (ref:<150 mg/dl), HDL Cholestrol 34 mg/dl (40-60 mg/dl), VLDL cholesterol 34.80 mg/dl (0.0-30.0 mg/dl) and uric acid 4 g/dl (ref: 2.4-6.7 mg/dl). Urine analysis was normal. Abdominal ultrasonography showed a single live intrauterine fetus of 31 weeks  $\pm$  2 days GA with normal cardiac activity and adequate liquor. The pancreatic image was obscured by bowel loops. Gallbladder and bile ducts were normal. The laboratory investigations, imaging studies along with history and clinical examination, confirmed the diagnosis as hypertriglyceridemic acute pancreatitis. She was managed by nil orally, intravenous fluids, analgesics, antiemetics and antacids with strict fetal monitoring. Patient improved with this treatment and discharged in a stable condition. Patient underwent a caesarean section at term in view of CPD. She delivered a healthy live baby of weight 3.9 kg. Patient was discharged on the 2nd postnatal day in good condition



Fig. 1. Causes of pancreatitis in pregnancy



## 3. Discussion and Conclusion

The common manifestations of acute pancreatitis include nausea, vomiting and abdominal pain. The diagnostic hallmark of acute pancreatitis is elevation of serum amylase and lipase levels. Abdominal ultrasound is the ideal imaging technique for detection of gallstones Maternal complications include ARDS, hypoglycemia, DIC, hyperglycemia, renal azotemia, hypocalcaemia. Increased preterm birth and perinatal morbidity. Local complications incorporate: acute peri/pancreatic fluid collections, pancreatic pseudocyst, acute necrotic collections, walled-off necrosis, gastric outlet dysfunction, splenic vein thrombosis, portal vein thrombosis and colonic necrosis (2). Management of pancreatitis in pregnancy, regardless of etiology, is with supportive treatments such as aggressive intravenously hydration, bowel rest, pain control, electrolyte monitoring, and respiratory support and nutritional support as needed. Plasmapheresis performed within 48 hours of presentation results in a significant reduction in triglyceride levels. Fibrates and niacin also reduce triglycerides level.

### References

- [1] Sreelatha S, Nayak Vedavathy, Nataraj, "Pancreatitis in pregnancy," Indian Journal of Clinical Practice, vol. 23, no. 4, September 2012.
- [2] Shiow Hung, Wei-Lun Tsai, Tzung-Jiun Tsai, Chih-Hsun Chu, Chun-Chin Sun, Hsin-Ju Cheng, Jenn-Kuen Lee, Hing-Chung Lam Hypertriglyceridemia-Associated Acute Pancreatitis in Pregnancy Ming -1, 4 Formos J. Endocrin Metab, vol. 4, no. 1.