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Case Report

Hypertriglyceridemia Induced Acute Pancreatitis in Pregnancy: A Case Report

Anjali Subedi Adhikari,[a,c] Sonam Gurung[b,c]

ABSTRACT

Introduction: Acute pancreatitis is a rare event in pregnancy. Hypertriglyceridemia induced acute pancreatitis accounts for the second most common cause in pregnancy. This rare event has a high maternal and fetal mortality of 20% and 50% respectively. **Case report**: A 21-year-old woman, G2P0+1 at 26 weeks period of gestation presented to OBGYN emergency in a state of shock with history of sudden onset of severe epigastric pain and multiple episodes of vomiting for one day and absent fetal movement for six hours. Immediate fluid resuscitation was done. Her reports showed increased hematocrit, leukocytosis, serum lipase and amylase elevated to > 200U/L. Ultrasonography showed bulky pancreas with intrauterine fetal death. With the diagnosis of acute pancreatitis with fetal demise, she was managed conservatively in intensive care unit by fasting, nasogastric aspiration, intravenous fluids, antibiotics, analgesics and heparin. She was intubated on the third day for increasing tachypnea. Her lipid profile showed elevated triglyceride> 1000 mg/dl and was started on oral hypolipidemic drugs. Pregnancy was terminated vaginally by misoprostol and was discharged on 19th day. Conclusion: Hypertriglyceridemia induced acute pancreatitis in pregnancy has an increased maternal and fetal complication.

Key words: Acute pancreatitis, Hypertriglyceridemia, Pregnancy

INTRODUCTION:

Acute pancreatitis (AP) is a rare event in pregnancy, incidence ranging from 1in 1000 to 1 in 3333 pregnancies.[1]Hypertriglyceridemia induced AP accounts for the second most common cause in pregnancy, gallstone being the first one.[2]

Hypertriglyceridemia is a well known phenomenon of pregnancy occurring due to physiologic changes in sex hormone levels. Gestational hypertriglyceridemia-induced acute pancreatitis occurs in pregnant women usually with preexisting abnormalities of lipid metabolism.[3]

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a - Lecturer
b - Resident
c - Department of Obstetrics and Gynecology, Manipal Teaching Hospital, Pokhara, Nepal.

Corresponding Author:
Anjali Subedi Adhikari
E-mail: anzee739@gmail.com
ORCID: https://orcid.org/0000-0002-9809-6180

This rare event has a very profound effect in pregnancy with high maternal and fetal mortality (20% and 50% respectively.[4] With the advancement in diagnosis, intensive treatment and neonatal care, the mortality has significantly decreased to 0%-3%. [5]

CASE REPORT:

A 21-year-old woman, G_2P_{0+1} at 26 weeks period of gestation presented to OBGYN emergency with complaints of sudden onset of severe epigastric pain and multiple episodes of vomiting for one day with no history of fever and jaundice. She also complained of absent fetal movement for six hours, however there was no history of lower abdominal pain, vaginal leaking, bleeding or hypertension. She was booked at a private hospital and had history of admission for hyperemesis gravidarum at 17 weeks. She had been managed conservatively with

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intravenous (IV) fluids and antiemetic medications. She had one spontaneous abortion one year back. She had no history of diabetes, dyslipidemia, gall stone in the past and no family history suggestive of hypertriglyceridemia.

At the time of examination, she was ill looking, drowsy and dehydrated. Her blood pressure was not recordable and she was tachypneic and tachycardic. On abdominal examination, there was generalized distension with tenderness more on epigastrium. On obstetric examination, uterus was 26 weeks and relaxed. However fetal heart sound could not be heard. Pelvic examination confirmed she was not in labor. Chance of abruption was ruled out clinically and by ultrasonography (USG). She was kept on oxygen, catheterized and immediately resuscitated with IV colloids and crystalloids successfully. Blood investigations were sent.

Reports showed Hb was 17.2 gm%, total count was raised to 14000/ mm^3, amylase and lipase raised to 734 U/L and 395 U/L respectively. Arterial blood gas analysis revealed metabolic acidosis. Liver function test, renal function test and blood sugar were within normal limits. USG of abdomen showed bulky pancreas (Fig.1), obstetric scan confirmed intrauterine fetal death (IUFD) and chest X-ray showed diffuse bilateral lung infiltrates (Fig. 2). So, diagnosis of acute pancreatitis with septic

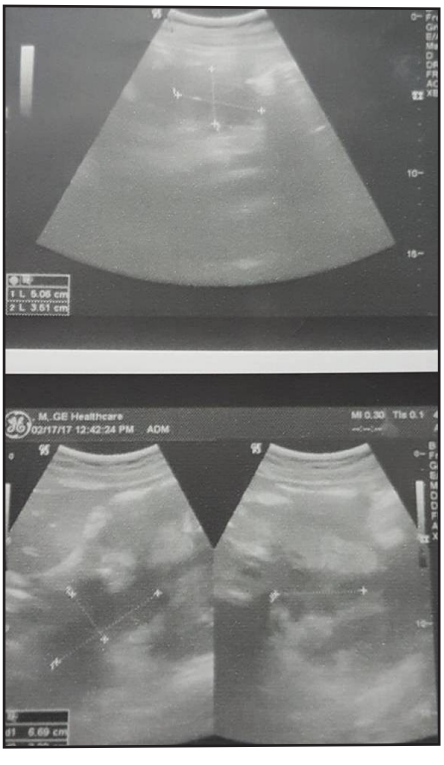

Figure 1. USG showing swollen pancreas.

shock with IUFD was made. She was shifted to Intensive Care Unit (ICU) where central venous line was accessed to monitor her fluid balance. She was kept nil by oral with nasogastric tube aspiration and managed with oxygen, IV fluids, ionotropes, analgesics, low molecular weight heparin and antibiotics.

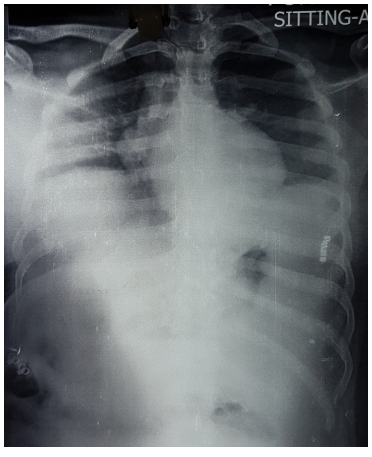

Figure 2. Chest X-Ray showing bilateral lung infiltrates.

However, on her second day, as she got tachypneic, she was intubated. Pregnancy was terminated by using repeated doses of vaginal misoprostol. Lipid profile was sent which showed Total cholesterol-284mg% (<200) Triglyceride (TGL)-1329mg% (<150) HDL-15mg% (30-60) LDL-46(<100) VLDL-266(<40). So, it was confirmed that it was hyper triglyceride induced acute pancreatitis.

After two days, patient was extubated and was started on hypolipidemic drug rovastatin and was advised for fat free diet. She was improving both clinically and biochemically but on day seven, she again developed tachypnea and was reintubated. Her chest X-ray showed bilateral diffuse lung infiltrates and her lipid profile showed TGL of 483 mg% which had decreased significantly. She was extubated the next day and fenofibrate was added. Patient then improved gradually and was discharged after nineteen days on oral hypolipidemic drugs to follow up regularly for lipid levels.

DISCUSSION:

Hypertriglyceridemia induced acute pancreatitis in pregnancy is a rare event accounting to 1%-7% of gestational pancreatitis.[6] It is usually common in women with pre-existing abnormal lipid metabolism. However, our case had no significant

medical or family history of dyslipidemia. In pregnancy due to increased estrogen, there is increased production of triglyceride rich lipoprotein and decrease in clearance of triglyceride due to suppression of lipoprotein lipase activity in liver and adipose tissue. The level of triglyceride is maximum in third trimester and may increase by two to four folds than in non-pregnant level. So the incidence of pancreatitis is more common in third trimester (52%).[7,8]

When the levels of TGL are high, there is hydrophilic degradation leading to formation of cytotoxic fatty acid that destroys the acinar cells and vascular endothelium of pancreas. Along with this, increased chylomicrons increase the viscosity of blood leading to capillary obstruction. This in turn leads to ischemia, acidosis and activation of trypsinogen.

These physiological changes might have been exacerbated by underlying abnormal lipid metabolism which was undiagnosed prenatally leading to pancreatitis in our case.

Diagnosis of AP is difficult in pregnancy. The common symptoms of pancreatitis like epigastric pain, nausea, vomiting of various degrees usually mimic common ailments of pregnancy and this can delay in diagnosis and treatment, which can further increase maternal and fetal morbidity and mortality. However, it may also present with severe manifestations like metabolic acidosis, sepsis and shock like in our case. But obstetric causes like preeclampsia, abruptio placenta, obstetric cholestasis, acute fatty liver of pregnancy, and uterine rupture should be ruled out.[9]

For the diagnosis, significant rise in amylase and lipase > 200U/L respectively has higher sensitivity and specificity. USG further aids in the diagnosis which is safe in pregnancy. However, if USG is inconclusive, MRI is preferred over CT scan due to adverse effects of ionising radiation of CT scan to the live fetus.

Management of pancreatitis in pregnancy is multidisciplinary involving obstetrician, surgeon, physician, anesthesiologist and radiologist. The management protocol in pregnancy is not different from that of non-pregnant population. It includes supportive treatment including fasting, IV fluids, analgesics, antibiotics, parenteral nutrition and fat free diet once acute state is treated. Specific to hypertriglyceridemia induced pancreatitis, insulin and low molecular weight heparin has to be used as they increase the activity of lipoprotein lipase and lower the TGL levels immediately. Hypolipidemic drugs take weeks to lower down the increased TGL levels. The TGL level also decreases after the delivery of baby and placenta. In cases not responding to above management, plasmapheresis can be done.

There is no standardized guideline published concerning the most effective way for delivery in women with AP during pregnancy to reduce maternal and neonatal mortality and morbidity. The decision depends on the gestational age and the severity of AP. When vaginal delivery is possible, it is preferable to limit the risk of superinfection necrosis associated with laparotomy used for cesarean sections. However, pregnancy should be terminated by caesarean delivery as soon as possible in case of TGL induced pancreatitis because of the significantly increased risk of maternal and fetal mortality.[9]

Our case presented at 26 weeks period of gestation with IUFD, so pregnancy was terminated by medical induction of labor, which also aided in fall of triglyceride levels.

Maternal and fetal complications are high in acute pancreatitis. Maternal morbidity and mortality are common due to complications of pancreatitis itself like hypovolemic shock, acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC) and rarely is due to preeclampsia, eclampsia secondary to AP.[10] Our case presented with hypovolemic shock. Otherwise with early diagnosis, good supportive treatment in present scenario, the complications are dismal.

The common fetal complications are abortion if present in first trimester, preterm labor and intrauterine fetal death (IUFD). Our case presented with IUFD as mother had developed shock with metabolic acidosis.

During the literature search, few case reports of pregnancy complicated by hypertriglyceridemia induced AP were found. In case reports by Gupta et al.[10] and Shreelatha et al.[7], patients presented at third trimester with acute epigastric pain with stable vitals and live pregnancy which is a common presentation. However, our case presented in mid-

trimester in shock with metabolic acidosis with IUFD, which is atypical presentation.

In contrary, Jeon et al.[5] published a case report where patient presented at 22 weeks with twin pregnancy with mild epigastric pain but the patient deteriorated and died within 24 hours of admission due to necrosis of pancreas secondary to hypertriglyceridemia induced AP, which is a dreadful complication.

However, our case gradually improved with conservative management despite severe symptoms and this may be due to absence of secondary complications of AP.

CONCLUSION

Hypertriglyceridemia induced gestational pancreatitis, a rare event, is associated with increased maternal and fetal morbidity and mortality. Its management always requires a multidisciplinary approach, early diagnosis and treatment for better outcome.

Conflict of interest: Authors declare that no competing interest exists.

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