Original Research Paper

Volume - 11 | Issue - 11 | November - 2021 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Gynaecology

MANAGEMENT OF HYPERTRIGLYCERIDEMIA INDUCED ACUTE PANCREATITIS IN PREGNANCY AT A TERTIARY CARE CENTRE: A CASE REPORT

Dr. Pushpawati Thakur	Associate Professor, Department of Obstetrics and gynaecology, AIIMS RAIPUR.
Dr. Pragati Trigunait*	Assistant Professor, Department of Obstetrics and Gynecology, AIIMS MANGALAGIRI. *Corresponding Author
Dr. Sagarika Majumdar	Assistant Professor, Department of Obstetrics and Gynecology, Dr Vasantrao Pawar Medical college Hospital and Research.
Dr. Pavan B C	Senior Resident, Department of Obstetrics and Gynecology, Kodagu Institute of Medical sciences and Teaching Hospital.
Dr. Vinita Singh	Associate Professor, Department of Obstetrics and Gynecology, AIIMS RAIPUR.
Dr. Sarita Rajbhar	Assistant Professor, Dept. of Obsetrics and Gynecology.

ABSTRACT The incidence of acute pancreatitis during antenatal period is relatively low, but it can lead to maternal and foetal morbidity as well as mortality. In this case report, the authors have described a 32 year old antenatal female, who had presented with history of pain abdomen with nausea and vomiting in 34th week period of gestation , who was diagnosed as a case of acute pancreatitis based on radiological findings and lab reports. There were multiple challenges associated with diagnosis and management of the patient which has been discussed in this case report. The Patient was successfully managed and delivered a healthy child with 1.67 kg weight by spontaneous breech delivery.

KEYWORDS : High risk Pregnancy, Pancreatitis in Pregnancy.

INTRODUCTION:-

The incidence of Acute Pancreatitis is relatively rare in the ante-natal period and it is estimated to affect 0.03-0.09% of pregnancies worldwide (1). Acute pancreatitis is triggered by activation of pancreatic trypsinogen followed by autodigestion which leads to cellular disruption, proteolysis, edema, hemorrhage, and necrosis (2). Pregnancy related hematological and biochemical alterations influence the interpretation of diagnostic tests and assessment of severity of AP (3). The spectrum of AP in pregnancy ranges from mild pancreatitis to serious pancreatitis associated with necrosis, abscesses, pseudocysts, and multiple organ dysfunction syndromes. (4)

The most common misdiagnosis of pancreatitis in the first trimester is hyperemesis. In women presenting with severe nausea and vomiting in the first trimester, consider obtaining amylase, lipase levels, and liver function tests, which when elevated are diagnostic for pancreatitis (5) Biliary tract diseases are the commonest cause, of which gallstones cause 70% of cases (as in non-pregnant women). Hyperlipidemia (usually Hypertriglyceridemia) is the next common etiology for AP. Hyperlipidemic pancreatitis is more dangerous than biliary pancreatitis. The majority occur in third trimester and can be associated with Pre-eclampsia and HELLP syndrome (6).

Abdominal ultrasound, CT scan, EUS, and MRCP are the available imaging studies in diagnosing a biliary etiology for AP. Diagnostic ERCP is to be avoided whenever possible owing to the associated risks including bleeding, perforation, pancreatitis, fetal radiation, while abdominal ultrasound, MRCP and EUS do not carry these risks. The general management of AP in pregnancy is supportive (5)

Fortunately, if treated early, pre-term labour can be avoided, and the incidence of recurrent attacks can be minimized (7). This case report describes a 32-year-old antenatal female, who was diagnosed with acute pancreatitis and various challenges associated with diagnosis and management of the patient.

CASE PRESENTATION

A 32 year old antenatal female, Gravida 2 Para 1 Living 1(G2P1L1), at 33 weeks 1 day of gestation had presented with complaints of sudden onset upper abdominal pain for past 1 day. The pain was constant in intensity and duration, radiating to the back, with no known aggravating and relieving factors and associated with nausea and vomiting (3 episodes). There was no history of hypertriglyceridemia in family. There was no history of previous Gallstones in past. There was no history of alcohol abuse, surgery, diabetes mellitus, infection, or any iatrogenic sources like diuretics, antibiotics, anti-hypertensive agents. The course of pregnancy was uncomplicated and there was no significant past or family history.

On examination, she looked exhausted and in agony, conscious and oriented, mild pallor, no icterus, no edema or lymphadenopathy. Her pulse was 108/min, regular, normal in rhythm, volume and character, BP was 138/70 mmHg, right upper arm, in supine position, Temperature- 99.6oF (oral), Respiratory Rate -24 breaths per minute. SpO2- 99% on room air and BMI was 33.3 kg/m2.

Per abdominal examination revealed mild tenderness in the epigastric region. Uterus was 32 weeks gravid size, relaxed, breech presentation. FHR – 148/min and regular.SFH-32 cm and AG- 93 cm. Systemic examination was otherwise normal.

Per Vaginum – Cervix – 1.5 – 2 cm dilated, 10-20% effaced, membranes present, breech presentation, show present. Pelvis was adequate on clinical pelvic examination. NST was reactive. Urine dipstick for proteins was 3+ and ketones were negative. All necessary blood investigations were sent, and results are as summarized in Table 1.

She had raised amylase and lipase levels which were indicative of acute pancreatitis, also she had raised triglycerides levels which pointed towards hypertriglyceridemia induced pancreatitis.

PARAMETERS	VALUE
HAEMOGLOBIN	10.9g%,
TOTAL LEUCOCYTE COUNT	15,400/uL (N- 87%, L- 6%, M- 6%, E-1%, B-1%)
PLATELET COUNT	1,82,000/Ul
BLOOD GLUCOSE	146mg/dl
LFT - ALP	123 units/L
RFT- UREA	49.2 mg
S. CREATININE	1.22 mg/dL
URIC ACID	10.5mg/dL
Na+	137 meq/L
K+	4.50 meq/L
Cl-	97 meq/L
S. CALCIUM	8.5 mg/dL
S.LDH	447 U/L

S. AMYLASE	1767 units/L
S. LIPASE	10,007 units/L
FLP- TGs	689 mg/dl

USG Abdomen was suggestive of Acute Pancreatitis (bulky body and tail of pancreas, heterogenous in echotexture and peripancreatic collection along the tail. No gall bladder distention/sludge was noted. Mild pleural effusion present.

ANC Sonogram showed-SLIUG, 33+2 weeks of gestation. FHR – 145bpm, severely reduced liquor with LVP- 1.5cm and EFBW – 1921g. Normal Doppler study.

Management:

The patient was explained about the high-risk nature of pregnancy with guarded prognosis and put on NPO. She was kept under close monitoring with intravenous fluids and intravenous antibiotics, Foley's catheterization, nasogastric aspiration with Ryle's tube and urine input /output monitoring, with vigilant foetal heart monitoring. She was given Injection Betamethasone in view of anticipated preterm labour.

There was spontaneously progress of labour and about 16 hours following admission, she delivered vaginally a boy baby weighing 1.67 kg by spontaneous breech delivery. Baby cried immediately but was shifted to NICU for observation in view of prematurity and very low birth weight.

Since her abdominal pain persisted following delivery with fever, a CECT scan was done which showed Severe Acute Necrotizing Pancreatitis with Early stage of splenic vein thrombosis.

The patient was put on higher order antibiotics and supportive treatment. She responded well and was discharged on day 9 with complete recovery.

DISCUSSION:

On literature search, evidence shows hypertriglyceridemia induced pancreatitis during pregnancy is establish fact. Hypertriglyceridemia during pregnancy occur due to abnormalities in lipid metabolism as a result of physiological changes during pregnancy. In our case, patient was a case of pregnancy induced hypertriglyceridemia landing up in severe pancreatitis. She responded well to conservative line of management with good maternal and foetal outcome.

Physiological hypertriglyceridemia in pregnancy rarely exceeds 300 mg/dL. The levels of triglycerides that cause acute pancreatitis should be >1000 mg/dL, seen in women with pre-existing disorders of lipid metabolism. (8)

Hypertriglyceridemia may occur in familial chylomicronaemia syndromes where hypertriglyceridemia is exacerbated by the pregnancy, leading to fatal complications such as acute pancreatitis. Chylomicrons are triglyceride-rich lipoprotein particles. They are present in the circulation when triglycerides are >10 mmol/L (900 mg/dl). These are large enough to occlude the pancreatic capillaries, leading to ischemia and subsequent acinar structural alteration, and also a release of pancreatic lipase. Enhanced lipolysis leads to an increased concentration of free fatty acids, which results in the release of inflammatory mediators and free radicals culminating in inflammation, edema, and necrosis.

Fibrates are the mainstay of therapy. They reduce plasma triglyceride levels by 50% and raise HDL cholesterol by 20%. They also decrease VLDL secretion and also increase lipolysis of plasma triglyceride. Other therapies which are useful are statins and omega-3-fatty acids. Statins reduce the cholesterol by inhibiting hydroxyl methyl glutaryl CoA reductase. Omega-3-fatty acids (eicosapentaenoic and docosa hexanoic acid) reduce plasma triglycerides by 20% when used in combination with other triglyceride lowering therapies.

Other treatment modalities include plasma exchange and lipid aphaeresis, insulin and heparin, and lipoprotein lipase gene therapy (9).

CONCLUSION:

During pregnancy it is important to diagnose causes of acute pancreatitis along with multidisciplinary management to reduce maternal and fetal mortality and morbidity. Pancreatitis due to hypertriglyceridemia is not uncommon during pregnancy in present scenario. Early diagnosis and prompt treatment may reduce complication related to it. (10) The treatment of pancreatitis in pregnancy should be conservative as far as possible with delaying the definitive treatment until delivery of the baby.

Conflict Of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

REFERENCES:-

1. Tesic-Rajkovic S, Radovanovic-Dinic B. Acute pancreatitis in pregnancy: From etiopathogenesis to therapy. Acta Medica Medianae. 2020;59(3):41-7.
2. Chemsi H, Kamal N. Acute pancreatitis and pregnancy: A rare and severe association. International Journal of Medical Reviews and Case Reports. 2020;4(5):96-7.
3. Luo L, Zen H, Xu H, Zhu Y, Liu P, Xia L, He W, Lv N. Clinical characteristics of acute pancreatitis in pregnancy: experience based on 121 cases. Archives of gynecology and obstetrics. 2018 Feb;297(2):333-9.
4. Yalcin Bahat P, Turan G, Aslan Cetin B. Abruptio placentae caused by hypertriglyceridemia-induced acute pancreatitis during pregnancy: case report and literature review. Case reports in obstetrics and gynecology. 2018 Sep 5;2018.
5. Cruciat G, Nemeti G, Goidescu I, Anitan S, Florian A. Hypertriglyceridemia triggered acute pancreatitis in pregnancy–diagnostic approach, management and follow-up care. Lipids in health and disease. 2020 Dec;19(1):1-6.
6. Tang M, Xu JM, Song SS, Mei Q, Zhang LJ. What may cause fetus loss from acute pancreatitis in pregnancy: analysis of 54 cases. Medicine. 2018 Feb;97(7).
7. Popescu I, Banacu M, Popescu M, Pacu I, Gheorghiu D, Davitoiu B, Socea B, Dimitriu MC. ACUTE PANCREATITIS DURING PREGNANCY–.vol. 52, no. 4, pp. 471-474 December 2017
8. Inayat F, Zafar F, Baig AS, Chaudhry NA, Aslam A, Khan ZH, Iqbal MJ. Hypertriglyceridemic pancreatitis treated with insulin therapy: a comparative review of 34 cases. Cureus. 2018 Oct;10(10).
9. Kandemir A, Coşkun A. Treatment of hypertriglyceridemia-induced acute pancreatitis with therapeutic plasma exchange in 2 pregnant patients. Journal of Obstetrics and Gynaecology. 2019 Jul 4;39(5):702-4.
10. Abdullah B, Kathiresan Pillai T, Cheen LH, Ryan RJ. Severe acute pancreatitis in pregnancy. Case reports in obstetrics and gynecology. 2015 Jan 5;2015.