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CASE REPORT

# ACUTE PANCREATITIS DURING PREGNANCY – A CASE REPORT

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## Abstract

**Introduction.** Studies report different incidence of acute pancreatitis (AP) during pregnancy, with a frequency rate between 1/1000 – 1/10 000 pregnancies.
**Case report.** We present the case of a 26-year-old primipara, 35 weeks pregnant woman, who came to our hospital through transfer from a municipal hospital with the diagnosis of acute pancreatitis (AP). On admission she presented nausea, vomiting, epigastric pain and impaired general state, symptoms that started 3 days before and rapidly worsened. In the context of acute pancreatitis and uncertain fetal status, keeping in mind the possible negative outcome as a consequence of fetal and maternal complications in acute pancreatitis, we decided that the patient should undergo immediate caesarean section.
**Conclusions.** There are no specific and well documented management protocols for acute pancreatitis in pregnancy.

**Key words:** acute pancreatitis, pregnancy, gallstones.

## Résumé

**La pancréatite aigue pendant la grossesse – rapport de cas et revue de la littérature**

Les études rapportent l'incidence variable de la pancréatite aiguë (AP) pendant la grossesse avec une incidence entre 1/ 1000 et 1 /10 000 grossesses.
**Présentation du cas.** Nous présentons le cas d'une femme âgée de 26 ans,primipare, enceinte depuis 35 semaines, qui a été transférée à notre hôpital d'un hôpital régional ,avec le diagnostic de pancréatite aiguë (PA). À l'admission, la malade présentait des nausées, des vomissements, des douleurs épigastriques et un malaise général; les symptômes avaient commencé il y a trois jours et se sont progressivement aggravés. Dans le contexte d'une pancréatite aiguë et d'un status fœtal incertain, compte tenu de la morbidité maternelle et fœtale associées aux complications possibles de la pancréatite, il a été décidé d'accomplir la naissance par chirurgie césarienne.
**Conclusions.** Il n'y a pas de protocoles spécifiques et bien documentés pour la pancréatite aiguë pendant la grossesse.

**Mots clés:** pancréatite aiguë, grossesse, calculs biliaires.

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## Introduction

Studies report different incidence of acute pancreatitis (AP) during pregnancy, with a frequency rate between 1/1000 – 1/10 000 pregnancies[1]. It is frequent in the 3rd trimester (50%) and in postpartum (38%), with higher frequency in multiparous women[2]. Acute pancreatitis in pregnancy is usually caused by gallstones – with frequencies that vary between 65% to 100%[3,4]. Studies show that other, rather common causes of pancreatitis outside pregnancy are less common in pregnant women – alcohol abuse, idiopathic causes or familial triglyceridemia. While older studies report high mortality rates, both maternal and fetal, with large variations, between 20% and 60%[5,6], more recent studies report far lower rates (0%-4.7%)[3,7], mostly due to the modern diagnostic techniques and the modern treatment of gallstones.

## Case report

We present the case of a 26 year old primipara, 35 weeks pregnant woman, who came to our hospital through transfer from a municipal hospital, with the diagnosis of acute pancreatitis (AP). At the moment of admission, she was presenting nausea, vomiting, epigastric pain and impaired general status, symptoms that started 3 days before and rapidly worsened. The patient had no history of previous illnesses. At the moment of her admission, the blood tests showed raised levels of serum amylase, lipase, aminotransferases and total bilirubin, mild anaemia, thrombocytosis and leucocytosis with neutrophilia, as seen in Table 1- Day 1.

Fetal ultrasound showed a fetus in breech presentation, with biparietal diameter – 84.3 mm, head circumference – 309.2 mm, abdominal circumference – 29.4 mm, femur length – 64.0 mm. The fetal weight was approximated at 2500 g, with fetal heart rate 120-125 beats per minute. The placenta was situated on the posterior wall of the uterus, with no sign of placental haematoma, maturation grade Granuum 1.

Abdominal ultrasound describes an enlarged pancreas, with a nonhomogeneous ecostructure, the gallbladder with thickened walls (6.5 mm), and multiple gallstones with diameters around 7 mm.

In the context of acute pancreatitis and uncertain fetal status, keeping in mind the possible negative outcome as a consequence of fetal and maternal complications in acute pancreatitis, we decided that the patient should undergo immediate termination of the pregnancy by emergency Caesarean section, in fetal and maternal interest. The surgical team included both obstetricians and surgeons. At surgical exploration of the upper abdomen, the palpation of the gallbladder, the biliary ducts and the pancreas were normal.

During the next 16 days, the patient was closely monitored. The blood test results, as showed above in Table 1, improved during her hospital stay. On admission, the amylase was 2674.00 UI/L and lipase 1413.20 U/L; at 16 days after admission, the levels dropped abruptly to 79.00 UI/L for amylase and 130.20 U/L for lipase; bilirubin levels began to decrease from the first day after surgery. At discharge, after 16 days of hospital stay, the patient was asymptomatic, with normal lab tests. At the 2 months re-evaluation, the lab results were completely normal, with no clinical signs of pancreatitis. The abdominal ultrasound showed a normal aspect of the pancreas and of the gallbladder.

Regarding the newborn, he was admitted to the Neonatal Intensive Care Unit (NICU) because of respiratory distress determined by prematurity and intrauterine distress. The evolution was satisfactory, being also discharged after 16 days.

## Discussion

The gallstones are the major cause for AP in pregnancy and also the etiological factor in our case. Pregnancy is a predisposing factor for gallstone and biliary sludge, due to the imbalance between increased cholesterol secretion and decreased secretion of bile acids and phospholipids[8], due to estrogenic impregnation and biliary hypertonia by elevated progesterone levels. Besides the fact that pregnancy itself is a predisposing factor, studies demonstrate that parity, per se, is also a predisposing factor[9]. Other risk factors for gallstone formation are obesity, increased leptin[10] but also weight loss[11].

Even though the symptomatology of AP during pregnancy is similar to that of nonpregnancy, with upper abdominal pain (either epigastric or the left upper quadrant pain, radiating to the back), nausea or vomiting, often associated with anorexia and fever; the diagnosis is usually difficult due to the entire panel of haematological changes. In order to diagnose AP and determine its severity, complete blood count, measurement of amylase and lipase, calcium, AST and LDH, and triglycerides levels are needed. Even though no clear protocol has been developed for the diagnosis of AP in pregnancy, studies show that concerning the amylase and lipase levels the threefold values are good predictors[12]. When taking into account the sensitivity and the specificity, lipase levels seem to correlate better with AP diagnosis that amylase levels (94% and 96% versus 83% and 88%)[13]. Abdominal ultrasound is the first line imaging technique, followed by magnetic resonance imaging (MRI), if the first one fails to deliver the necessary results[14].

The severity of AP is either established using Ranson and Balthazar criteria or the Atlanta

**Table 1.** Blood tests during hospital stay.

| *Test* | *Normal values* | *I.U.* | *Day 1* | *Day 2* | *Day 9* | *Day 16* |
|---|---|---|---|---|---|---|
| BAS# | 00.00 – 0.20 | *1000/uL | 0.05 | 0.05 | 0.024 | 0.07 |
| BAS% | 00.00 – 2.00 | % | 0.30 | 0.30 | 0.24 | 1.00 |
| EOS# | 00.00 – 0.70 | *1000/uL | 0.00 | 0.00 | 0.174 | 0.21 |
| EOS% | 00.00 – 5.00 | % | 0.00 | 0.00 | 1.44 | 3.10 |
| HCT | 36.00 – 48.00 | % | * 33.60 | * 29.40 | * 28.32 | * 32.60 |
| HGB | 11.70 – 15.00 | g/dL | * 10.50 | * 9.30 | * 9.02 | * 9.60 |
| LYM# | 1.50 – 4.50 | *1000/uL | * 0.87 | * 1.46 | 1.714 | 1.92 |
| LYM% | 20.00 – 45.00 | % | * 5.20 | * 8.20 | * 14.14 | * 28.30 |
| MCH | 27.00 – 32.00 | Pg | * 23.00 | * 24.10 | * 24.62 | * 23.40 |
| MCHC | 32.00 – 36.00 | g/dL | * 31.30 | * 31.60 | * 31.82 | * 29.40 |
| MCV | 80.00 – 100.00 | fL | * 73.50 | * 76.20 | * 77.32 | * 79.50 |
| MONO# | 0.20 – 1.00 | *1000/uL | 0.37 | * 1.02 | 0.944 | 0.43 |
| MONO% | 2.00 – 10.00 | % | 2.20 | 5.80 | 7.74 | 6.30 |
| MPV | 7.40 – 10.40 | fL | 10.40 | * 10.80 | 10.20 | 10.90 |
| NEU# | 2.00 – 8.00 | *1000/uL | * 15.48 | * 15.20 | * 9.324 | * 4.15 |
| NEU% | 50.00 – 75.00 | % | * 92.30 | * 85.70 | * 76.64 | * 61.30 |
| PDW | 11.50 – 16.50 | fL | 11.80 | 12.60 | | 13.30 |
| PLT | 150.00 – 450.00 | *1000/uL | * 474.00 | 385.00 | * 591.10 | * 511.10 |
| RBC | 4.20 – 5.60 | *10^6/uL | 4.57 | * 3.86 | * 3.66 | * 3.99 |
| WBC | 4.00 – 11.00 | *1000/uL | * 16.77 | * 17.73 | * 12.164 | * 6.78 |
| Serum Uric Acid | 2.40 – 6.50 | mg/dL | | | * 6.85 | |
| Serum Amylase | 28.00 – 100.00 | UI/L | * 2674.00 | *827.00 | * 147.00 | *79.00 |
| Conjugated Bilirubin | 00.00 – 0.30 | mg/dL | * 1.71 | * 0.60 | | |
| Total Bilirubin | 00.00 – 1.00 | mg/dL | * 1.84 | 0.74 | | |
| Total Serum Calcium | 8.30 – 10.20 | mg/dL | | 8.50 | | |
| CK | 26.00 – 174.00 | UI/L | 37.00 | | | |
| CK-MB | 00.00 – 26.00 | UI/L | 10.00 | | | |
| Serum Creatinine | 0.50 – 1.20 | mg/dL | 0.59 | | | |
| Glycaemia | 70.00 – 115.00 | mg/dL | 85.00 | 111.00 | | |
| Serum Lipase | 13.00 – 60.00 | U/L | * 1413.20 | *1152.80 | * 241.50 | * 130.20 |
| Serum Magnesium | 1.58 – 2.55 | mg/dL | | 1.78 | | |
| Sideremia | 37.00 – 145.00 | ug/dL | | | | |
| TGO | 00.00 – 34.00 | UI/L | * 108.00 | * 91.00 | 15.00 | 17.00 |
| TGP | 00.00 – 38.00 | UI/L | * 82.00 | * 83.00 | 11.00 | 13.00 |
| Triglyceride | 40.00 – 200.00 | mg/dL | | 154.00 | | |
| Serum Urea | 10.00 – 50.00 | mg/dL | 15.80 | | | |
| Serum Sodium | 135.00 – 148.00 | mmol/L | 141.00 | 141.00 | | |
| Serum Potassium | 3.60 – 5.20 | mmol/L | 4.33 | 4.17 | | |
| APTT (R) | 0.79 – 1.20 | Ratio | 0.92 | 1.06 | | |
| APTT s | 26.60 – 40.40 | Sec | 30.70 | 35.70 | | |
| INR | 0.90 – 1.20 | INR | 0.93 | 1.07 | | |
| PT | 11.60 – 15.30 | Sec | 12.20 | 13.60 | | |
| PT % | 75.00 – 120.00 | % | 110.00 | 91.00 | | |

**Table 2.** Strategy for the management of AP according to the gestational age.

| *First trimester* | *Second trimester* | *Third trimester* |
|---|---|---|
| ✓ conservative treatment<br>and<br>✓ laparoscopic cholecystectomy (during second trimester) | ✓ laparoscopic cholecystectomy | ✓ conservative treatment<br>or<br>✓ ERCP with biliary endoscopic sphincterotomy<br>and<br>✓ laparoscopic cholecystectomy (in the early postpartum period) |

classification, both of them non-specific in pregnancy[13]. There are authors who recommend the diagnosis of severe AP based solely on the presence of peripancreatic fluid or peripancreatic or pancreatic necrosis[15].

Researches concerning fetal outcome state the following: in case of „mild" AP, fetal loss is due to miscarriages and preterm labour, while in case of „severe" AP, the concerns are fetal death and stillbirth[16].

The management of pregnant women with AP is still surrounded by controversies, with no definite guidelines. Regarding the AP, Ducarme et al[14] suggest the following strategy (Table 2):

Antibiotherapy still remains a controversy, as the use of antibiotics in this pathology is a matter of history. Latest studies show that prophylactic antibiotherapy cannot reduce infected pancreatic necrosis or mortality in patients with acute necrotizing pancreatitis[17].

As for the recommended way of delivery, the Cesarean section is to be avoided if possible, and the vaginal birth should be encouraged, in order to limit the risk of superinfection necrosis associated with laparotomy. Even so, no matter the delivery mode, a multidisciplinary team consisting of a gastroenterologist, a surgeon and an obstetrician should be gathered.

## Conclusions

Even in the 21st century, there are no specific and well documented management protocols for acute pancreatitis in pregnancy. Even though using the guidelines for acute pancreatitis outside the pregnancy one shows promising results for the mother, we are still unable to predict the best time of delivery and the possible negative outcomes of prolonged fetal exposure to this maternal pathology.

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