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Case Series

Maternal and perinatal outcomes in acute pancreatitis complicating pregnancy: a case series and review of literature

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ABSTRACT

Acute pancreatitis in pregnancy (APIP) is uncommon but can be a serious maternal and obstetric emergency. Its symptoms may resemble common pregnancy-related complaints, making early diagnosis challenging, particularly in the third trimester. We report three cases of APIP presenting in the third trimester. Case 1 was a 23-year-old G2P1L0 at 31 weeks who presented with severe epigastric pain, vomiting, and preterm labour. Markedly elevated amylase and lipase, along with imaging findings of acute necrotizing pancreatitis with limited pancreatic necrosis, extensive peripancreatic inflammation, collections, mild ascites, ileus, and left pleural effusion, confirmed the diagnosis. Fetal distress subsequently developed, necessitating emergency caesarean delivery. She required postoperative intensive care and ventilatory support. Case 2, a multigravida at 33+5 weeks with recurrent pancreatitis, fetal growth restriction, and anemia, presented with abdominal pain and reduced fetal movements. She improved with conservative management and later underwent induction at 35+5 weeks, resulting in a live preterm vaginal delivery. Case 3, a primigravida at 31+5 weeks with gestational hypertension and intrahepatic cholestasis, presented with epigastric pain radiating to the back and shoulder. Elevated pancreatic enzymes confirmed pancreatitis. She responded well to conservative treatment and subsequently delivered vaginally. All patients received bowel rest, intravenous fluids, electrolyte correction, analgesia, antibiotics, and close maternal-fetal monitoring. Maternal survival was 100%, and all pregnancies resulted in live preterm births. Early recognition and multidisciplinary management are crucial for favourable outcomes in APIP.

Keywords: Acute pancreatitis, Pregnancy, Necrotizing pancreatitis, Preterm birth, Maternal outcome, Perinatal outcome, Case series

INTRODUCTION

Acute pancreatitis in pregnancy (APIP) is an uncommon but potentially life-threatening medical and obstetric emergency, with a reported incidence ranging from 1 in 1,000 to 1 in 12,000 pregnancies.[1,2] It occurs most frequently during the third trimester and the early postpartum period and is associated with significant maternal and perinatal morbidity when diagnosis or treatment is delayed. Gallstone disease is the leading cause, followed by hypertriglyceridemia, while less common etiologies include hypercalcemia, medications, infections, trauma, and idiopathic disease.[1,3]

Diagnosis can be challenging because symptoms such as nausea, vomiting, and abdominal discomfort overlap with normal pregnancy-related complaints. Persistent epigastric pain radiating to the back, together with elevated serum pancreatic enzymes and supportive imaging findings, is essential for diagnosis. Management is primarily conservative and includes aggressive intravenous fluid resuscitation, analgesia, nutritional support, correction of metabolic abnormalities, and close maternal-fetal surveillance through a multidisciplinary approach.

Advances in diagnostic imaging, intensive care, and obstetric management have substantially improved

maternal survival; however, preterm birth and fetal morbidity remain important concerns, particularly in severe disease [4,5]. We present a case series of three pregnant women with acute pancreatitis, highlighting variations in clinical presentation, management, and maternal and neonatal outcomes, while emphasizing the importance of early recognition and multidisciplinary care.

CASE SERIES

Case 1

A 23-year-old woman, gravida 2 para 1 living 0 (G2P1L0), at 31 weeks of gestation with chronic hypertension and preterm labour pain was referred to our centre with epigastric pain of two days' duration associated with one episode of nausea and vomiting since that morning, along with lower abdominal pain for one day.

On presentation, her vital signs were blood pressure 134/90 mmHg and pulse rate 104/min. Abdominal examination revealed a uterus of 32-week size with a cephalic, irritable presentation, and regular fetal heart sounds at 130 beats/min. Per-vaginum examination confirmed early labour.

She was managed with strict vital-sign and input-output charting alongside conservative treatment for pancreatitis. Investigations revealed liver function tests (SGOT/SGPT) of 48/79 U/L and serum amylase/lipase of 568/820 U/L, which subsequently rose to 2352/4086 U/L. Serum creatinine was 1.11 mg/dL, which rose to 1.36 mg/dL before falling to 1.08 mg/dL. Ultrasonography of the whole abdomen showed an enlarged liver with altered echotexture; the pancreas was diffusely bulky (anteroposterior diameter of head 4 cm), homogeneous, and hyperechoic, without cyst or calcification, with peripancreatic echogenicity consistent with inflammation.

Contrast-enhanced computed tomography (CECT) of the chest and abdomen showed diffuse pancreatic enlargement with small patchy areas of non-enhancing necrotic tissue involving less than 30% of the gland, extensive acute peripancreatic inflammatory changes, and acute indeterminate peripancreatic collections involving the lesser sac, anterior pararenal space, paracolic gutters, and pelvis. Extra-pancreatic complications included adynamic ileus involving the proximal small and large bowel, with mild ascites and a left-sided pleural effusion. There was mild prominence of the common bile duct with smooth distal intrapancreatic narrowing and upstream biliary dilatation, likely secondary to inflammatory compression. These findings were consistent with acute necrotizing pancreatitis with limited (<30%) pancreatic necrosis, corresponding to a modified CT severity index (CTSI) of 8/10 (severe).

Surgical and medical opinions were obtained. She was kept nil per oral with strict input–output and vital-sign charting, and serum electrolytes were monitored 12-hourly. Antenatal corticosteroid (dexamethasone) prophylaxis for fetal lung maturity was initiated. The patient went into spontaneous labour with leaking per vaginum, and an emergency lower-segment caesarean section (LSCS) was performed for acute fetal distress. She was shifted to the intensive care unit postoperatively and remained intubated on mechanical ventilation for two days. She received calcium correction for hypocalcaemia. Ultrasonography of the chest confirmed a left-sided pleural effusion that was not amenable to tapping. Oral intake was gradually resumed after 48 hours. Blood pressure was controlled with telmisartan 40 mg once daily and amlodipine 10 mg once daily after 48 hours of a nitroglycerin (NTG) infusion. Intravenous antibiotics were continued for five days, and she was ultimately discharged in stable condition. Serial investigations are summarised in Table 1.

Case 2

A gravida 4 para 1 living 1 abortus 2 (G4P1L1A2) woman at 33+5 weeks of gestation with recurrent pancreatitis, a fetus small for gestational age (SGA), and moderate anemia presented with abdominal pain followed by persistently decreased fetal movements, subsequently reassessed at 35+5 weeks. Medical and surgical opinions were obtained, and she was managed conservatively with nil-per-oral status, intravenous fluids, and antibiotics, alongside strict vital-sign and input-output charting. She improved with conservative management. In view of persistently decreased fetal movements, labour was induced with a Foley catheter followed by prostaglandin gel and oxytocin augmentation. She had a preterm vaginal delivery of a live neonate weighing 1.8 kg; both mother and baby were discharged in stable condition. Serial investigations are summarised in Table 2.

Table 1: Serial laboratory trend, case 1.

Parameters	6 Jun	10 Jun	12 Jun	14 Jun
Hb/TLC/platelet count	9.6/13,900/2.93 l	8.7/18,950/2.62 l	7.9/26,200/2.17 l	8.6/33,000/2.71 l
LFT (SGOT/ SGPT)	54/41	32/79	52/ 27	34/21
KFT (Blood urea/ S. creatinine)	25.9/0.86	52/0.7	34.1/0.43	31.3/0.34
Serum calcium (mg/dL)	9.28	5.3	2.23	–
Serum amylase/ lipase (U/L)	568 / 820	2352/4086	190/132	–

Table 2: Serial laboratory trend, case 2.

Parameters	16 May 2026	24 May 2026	25 May 2026
Hb/ TLC/ Platelet count	9.6 / 9,400 / 2.3 l	9.9 / 8,900 / 1.73 l	9.8 / 7,700 / 2.5 l
Blood urea/ S. creatinine	15.7 / 0.91	17.6 / 0.83	17 / 0.52
ALT/ AST	14 / 27	25 / 14	12 / 26
Serum amylase (U/L)	197	239	-
Serum lipase (U/L)	463	739	-

Case 3

A primigravida at 31+5 weeks of gestation with gestational hypertension and intrahepatic cholestasis of pregnancy (IHCP) presented with epigastric pain radiating to the back and shoulder. Surgical opinion was obtained; serum amylase/lipase was elevated at 227/635 U/L. Ultrasonography of the whole abdomen showed the pancreas obscured by overlying bowel gas. The patient was kept nil per oral and managed conservatively with intravenous fluids and intravenous antibiotics.

Serum electrolytes remained within normal limits throughout admission. She improved symptomatically, with electrolytes remaining normal and amylase/lipase levels showing a falling trend. Oral intake was gradually resumed from day 2. She subsequently went into preterm labour and delivered a live male neonate weighing 1.65 kg. Serial investigations are summarised in Table 3.

Table 3: Serial laboratory trend, case 3.

Parameters	20 Jun	23 Jun	24 Jun
Hb/TLC/ platelet count	12.4/9,500/1.57 l	10.4/ 9,600/ 1.32 l	10.1/ 10,000/1.5 l
LFT (SGOT/ SGPT)	64/48	80/ 71	42/ 50
KFT (Blood urea/ S. creatinine)	25.3/ 0.97	20.9/ 0.69	14/ 0.5
Serum amylase/ lipase (U/L)	227/ 635	82/ 197	65/ 132

DISCUSSION

A comparative summary of the clinical, biochemical, and outcome parameters of the 3 cases is presented in Table 4.

Table 4: Comparative analysis of the three cases.

Parameters	Case 1	Case 2	Case 3
Maternal age and parity	G2P1L0	G4P1L1A2	Primigravida
Gestational age at presentation	31 weeks	33+5 weeks (delivered at 35+5 weeks)	31+5 weeks
Associated obstetric disorders	Chronic hypertension, preterm labour	Small for gestational age (SGA), moderate anemia	Gestational hypertension, intrahepatic cholestasis of pregnancy (IHCP)
Presenting symptoms	Severe epigastric pain, nausea, vomiting, labour pains	Abdominal pain followed by decreased fetal movements	Epigastric pain radiating to back and shoulder
Severity of pancreatitis	Severe acute necrotizing pancreatitis	Mild recurrent pancreatitis	Mild acute pancreatitis
Suspected etiology	Biliary / inflammatory compression (CBD narrowing on CT)	Recurrent pancreatitis (likely biliary / idiopathic)	Not clearly established (likely biliary)
Peak serum amylase (U/L)	2352	239	227
Peak serum lipase (U/L)	4086	739	635
Imaging findings	Bulky pancreas, <30% pancreatic necrosis, extensive peripancreatic collections, mild ascites, left pleural effusion, ileus	No pancreatic necrosis reported	Pancreas obscured on ultrasonography; no necrosis reported
Systemic complications	Acute kidney injury, hypocalcemia, pleural effusion, ileus, ICU admission	None	None

Continued.

Parameters	Case 1	Case 2	Case 3
Management	Conservative treatment, NPO, IV fluids, electrolyte correction, antibiotics, ICU care, multidisciplinary management	Conservative treatment with IV fluids, antibiotics, NPO, fetal surveillance	Conservative treatment with IV fluids, antibiotics, NPO, fetal surveillance
Need for intensive care	Yes (ventilatory support for 2 days)	No	No
Mode of delivery	Emergency LSCS for acute fetal distress	Induced vaginal delivery	Spontaneous preterm vaginal delivery
Indication for delivery	Acute fetal distress during spontaneous preterm labour	Persistent decreased fetal movements	Spontaneous preterm labour
Neonatal outcome	Live preterm neonate	Live preterm neonate (1.8 kg)	Live preterm male neonate (1.65 kg)
Maternal outcome	Recovered after ICU stay; discharged stable	Discharged stable	Discharged stable

All three patients presented in the third trimester, the period of highest reported incidence of APIP. One patient (33%) developed severe necrotizing pancreatitis requiring intensive care, whereas two (67%) had mild disease with rapid clinical improvement. Two of the three cases had an associated hypertensive disorder of pregnancy, and all three underwent preterm termination of pregnancy; one required caesarean delivery for acute fetal distress in the setting of decreased fetal movements. Maternal survival was 100% and neonatal survival was 100%, demonstrating favourable outcomes with timely multidisciplinary management despite significant maternal morbidity in one case.

This distribution of severity is consistent with contemporary pooled data. In the meta-analysis by Hughes et al encompassing 823 patients drawn from the concurrent literature, 249 patients (30.3%) had severe pancreatitis and 467 (56.7%) had mild disease closely mirroring the one-in-three rate of severe disease observed in the present series.[6] Luo et al in the largest single-centre cohort of 121 cases, similarly found that disease severity was the principal determinant of maternal and fetal death ($p<0.01$) reinforcing that the intensive care admission and modified CT severity index of 8/10 recorded in case 1 placed her in the highest-risk category described in the literature.[3]

The suspected etiologies in this series-inflammatory biliary compression in case 1, and unconfirmed but probable biliary causes in cases 2 and 3-reflect the biliary-predominant pattern reported in South Asian case series such as those of Kumari et al and Magudapathi et al and contrast with East Asian cohorts in which hypertriglyceridemia predominates: Zhang et al ten-year Beijing series found hypertriglyceridemia in 56.2% (18/32) of patients compared with gallstones in 28.1% (9/32) while Luo et al reported a closer balance of gallstone (36.4%) versus hypertriglyceridemic (32.2%) disease.[3,4,7,8] No patient in the present series had documented hypertriglyceridemia, consistent with the biliary-predominant etiological profile more typical of Indian cohorts.

The 100% maternal and neonatal survival achieved in this series compares favourably with, and indeed exceeds, pooled contemporary benchmarks. Hughes et al reported a pooled maternal mortality rate of 2.8% (95% CI 1.5-5.1) and a fetal mortality rate of 12.3% (95% CI 5.7-24.7) across 823 patients, while Luo et al recorded maternal and fetal mortality rates of 3.3% (4/121) and 11.6% (14/121), respectively, in their cohort.[3,6] The Beijing series by Zhang et al which like the present series recorded no maternal or fetal deaths among its 32 patients, further demonstrates that with early recognition and multidisciplinary intensive care, near-zero mortality is achievable even when hypertriglyceridemia-related severe disease predominates.[4] These converging data support the conclusion that the historic maternal mortality of over 30% and fetal mortality of up to 50% once associated with APIP have been substantially overcome in modern obstetric and critical care settings, irrespective of the predominant local etiology.[7,5]

All three pregnancies in the present series ended in preterm delivery, in keeping with the well-documented association between APIP and both iatrogenic and spontaneous preterm birth. Luo et al reported that 72.7% of their moderate-to-severe APIP patients required caesarean section to safely terminate gestation, comparable to the single caesarean delivery-performed for acute fetal distress-required in this series.[3] Tang et al studying 96 spontaneously occurring cases at a single US academic centre, found that adverse fetal outcomes clustered among women who developed pancreatitis earlier in gestation, whereas third-trimester onset, as in all three of the present cases, was more often complicated by maternal systemic derangements such as disseminated intravascular coagulation rather than pregnancy loss.[5] The overlap between APIP and hypertensive disorders of pregnancy observed in cases 1 and 3 of this series is likewise recognised in the literature, including by Kumari et al and in the comprehensive review by Mądro, both of which caution that pancreatitis can closely mimic HELLP syndrome and severe preeclampsia-a diagnostic overlap

that was directly relevant to the assessment of cases 1 and 3.[7,9]

APIP is an uncommon but potentially life-threatening medical emergency that poses significant diagnostic and therapeutic challenges for obstetricians. Although the reported incidence ranges from approximately 1 in 1,000 to 1 in 5,000 pregnancies, it remains one of the most important non-obstetric causes of acute abdomen during pregnancy. Improvements in diagnostic imaging, intensive care management, and multidisciplinary obstetric care have markedly reduced maternal mortality over recent decades, although fetal morbidity related to prematurity continues to be significant.

According to Williams Obstetrics (26th edition), pancreatic disorders in pregnancy are uncommon but require prompt recognition because delayed diagnosis may result in maternal organ dysfunction, preterm delivery, fetal compromise, and increased perinatal morbidity.[10] Early diagnosis and aggressive supportive management remain the cornerstone of successful treatment.

Epidemiology

Acute pancreatitis occurs predominantly during the third trimester and the early postpartum period, though the precise distribution varies across published series. In the largest single-centre cohort reported to date, Luo et al reviewed 121 cases of APIP managed at a gastroenterology department in China and identified the third trimester as the period of peak presentation.[3] A ten-year, three-centre retrospective study from Beijing by Zhang et al which screened 194,723 pregnancies across three tertiary hospitals, identified only 32 confirmed cases of APIP, an incidence consistent with the widely cited range of 1 in 1,000 to 1 in 12,000 pregnancies.[1,4,2] A large meta-analysis pooling 4,034 Chinese patients from 154 studies found that the proportion of cases rose progressively across gestation, from 4.7% in the first trimester to 25.2% in the second and 63.2% in the third while a separate meta-analysis of maternal and fetal outcomes by Hughes et al pooling 823 patients from the concurrent international literature, found that 64.9% (548 patients) presented in the third trimester.[6,11] First-trimester disease is relatively uncommon but is associated with higher fetal loss owing to spontaneous abortion and severe maternal illness. The recent Indian case series by Kumari et al. similarly reported that most patients presented during late pregnancy, emphasizing that physiological and hormonal changes during the third trimester increase susceptibility to biliary disease and pancreatitis; all three patients in the present series likewise presented between 31 and 34 weeks of gestation.[7]

Etiology

Gallstone disease and hypertriglyceridemia together account for the substantial majority of reported cases, although their relative contribution varies markedly by population. In the 121-case cohort of Luo et al gallstone disease accounted for 36.4% of cases and hypertriglyceridemia for 32.2%, and hypertriglyceridemic APIP was independently correlated with a higher rate of local complications ($p=0.012$).[3] By contrast, the ten-year Beijing series of Zhang et al found hypertriglyceridemia to be the dominant cause, present in 56.2% (18 of 32) of patients compared with gallstones in 28.1% (9 of 32), and demonstrated that hypertriglyceridemia-induced disease carried a significantly higher risk of severe pancreatitis ($p=0.025$) and correlated positively with disease severity ($p=0.039$).[4] Pregnancy-induced estrogen excess increases cholesterol secretion into bile, whereas progesterone decreases gallbladder contractility, resulting in biliary stasis and gallstone formation.[12]

Hypertriglyceridemia is consistently reported as the second, and in some East Asian cohorts the leading, cause of APIP.[1,4] Pregnancy normally causes a two- to three-fold rise in serum triglyceride concentrations, but women with underlying lipid metabolism disorders may develop marked hypertriglyceridemia capable of precipitating pancreatitis. Other uncommon causes include alcohol consumption, hyperparathyroidism, medications, trauma, infections, autoimmune disorders, and idiopathic disease; no patient in the present series had documented hypertriglyceridemia, consistent with the biliary-predominant profile more typical of South Asian series.[7,8]

The 2026 Indian case series further highlighted that biliary disease remains the predominant precipitating factor, although rare vascular complications, including arterial thrombosis, have occasionally been described in severe pancreatitis owing to systemic inflammatory and hypercoagulable states.[7,13]

Pathophysiology

Pregnancy predisposes women to pancreatitis through several physiological mechanisms. Increased estrogen enhances biliary cholesterol saturation, while progesterone-induced smooth-muscle relaxation delays gallbladder emptying. These changes promote gallstone formation and transient obstruction of the ampulla of Vater, resulting in activation of pancreatic digestive enzymes within the pancreatic parenchyma.

Hypertriglyceridemia-induced pancreatitis develops through hydrolysis of triglycerides into toxic free fatty acids, causing endothelial injury, pancreatic ischemia, acinar cell damage, and activation of inflammatory cytokines. These inflammatory mediators may progress to systemic inflammatory response syndrome (SIRS), multiorgan dysfunction, and pancreatic necrosis in severe disease. Luo et al provided a biochemical correlate of this cascade, demonstrating that serum calcium level was significantly and inversely correlated with disease severity ($p<0.01$), consistent with the marked hypocalcaemia observed in case 1 of the present series, whose corrected calcium fell to 2.23 mg/dL at the height of her illness.[3]

Clinical presentation

The hallmark symptom is severe epigastric pain radiating to the back.[1,5] Associated manifestations include nausea, vomiting, abdominal tenderness, fever, abdominal distension, and occasionally ileus. In the cohort of Luo et al abdominal pain (86.8%) and vomiting (73.6%) were the two most frequent presenting symptoms, while the Beijing series by Zhang et al reported abdominal pain in an even higher proportion, 93.7% (30 of 32 patients)- figures that mirror the presentation of all three cases in this series, each of whom presented with severe abdominal or epigastric pain, and two of whom (Cases 1 and 2) also had nausea or vomiting.[3,4] Clinical diagnosis may be difficult because symptoms overlap with several pregnancy-specific disorders, including HELLP syndrome, acute fatty liver of pregnancy, severe preeclampsia, gastroesophageal reflux disease, and biliary colic. Kumari et al emphasized that pancreatitis should be considered whenever pregnant women present with persistent upper abdominal pain associated with elevated pancreatic enzymes, particularly during the third trimester.[7]

Diagnosis

Diagnosis is established when at least two of the following three criteria are fulfilled: (i) characteristic abdominal pain; (ii) serum amylase and/or lipase levels greater than three times the upper limit of normal; and (iii) characteristic findings on imaging.

Serum lipase possesses higher sensitivity and specificity than serum amylase.[7,14] Laboratory evaluation should additionally include complete blood count, liver function tests, renal function tests, serum calcium, serum triglycerides, inflammatory markers, and coagulation profile.

Ultrasonography is recommended as the first-line imaging modality because it is safe during pregnancy and can identify gallstones and biliary dilatation.[7,9] When ultrasonography is inconclusive, magnetic resonance cholangiopancreatography (MRCP) offers excellent visualization without ionizing radiation. Computed tomography is generally reserved for complicated or severe pancreatitis because of fetal radiation exposure. Endoscopic retrograde cholangiopancreatography (ERCP) is primarily therapeutic rather than diagnostic and is reserved for persistent biliary obstruction or cholangitis.

Severity assessment

The revised Atlanta classification categorizes acute pancreatitis into mild, moderately severe, and severe disease according to the presence and duration of organ failure and local complications.[14] Mild pancreatitis usually resolves with conservative treatment and carries an excellent maternal and fetal prognosis. Severe pancreatitis may result in pancreatic necrosis, infected collections, respiratory failure, renal failure, shock, disseminated intravascular coagulation, and multiorgan dysfunction, requiring intensive care management. In the meta-analysis by Hughes et al severity was documented in 823 pooled patients, of whom 467 (56.7%) had mild disease and 249 (30.3%) had severe pancreatitis-a distribution closely matching the one-in-three rate of severe, ICU-requiring disease observed among the three cases in this series.[6]

Management

Management of acute pancreatitis during pregnancy is largely conservative and similar to that in non-pregnant patients. The primary objectives are stabilization of the maternal condition, prevention of complications, treatment of the underlying cause, and continuous fetal surveillance.

Initial treatment consists of aggressive intravenous crystalloid resuscitation, bowel rest during the acute phase, adequate analgesia, antiemetics, correction of electrolyte abnormalities, oxygen supplementation, and nutritional support. Early enteral nutrition is preferred over prolonged total parenteral nutrition whenever feasible, as it preserves intestinal integrity and reduces infectious complications.

Routine prophylactic antibiotics are not recommended in uncomplicated pancreatitis, because current evidence demonstrates no reduction in mortality or infected pancreatic necrosis.[9,14] Antibiotics should be reserved for documented infection or infected pancreatic necrosis.

For gallstone pancreatitis, laparoscopic cholecystectomy is ideally performed during the second trimester once acute inflammation resolves.[9,15] In a systematic review focused specifically on this clinical dilemma, Al Samaraee and Bhattacharya highlighted that conservative management alone carries a substantial risk of early recurrent attacks, and recommended that laparoscopic cholecystectomy be performed within the index admission, or within two weeks of the attack, in patients fit for surgery, reserving ERCP with sphincterotomy for those with obstructive choledocholithiasis or those unfit for surgical intervention.[15] None of the three patients in the present series underwent cholecystectomy or ERCP during the index admission, reflecting the absence of confirmed cholelithiasis on imaging in all three cases.

Maternal and fetal outcomes

Historically, maternal mortality exceeded 30% and fetal mortality approached 50%.[5,7] Advances in diagnostic techniques, multidisciplinary intensive care, fetal monitoring, and neonatal support have dramatically improved outcomes. In the meta-analysis by Hughes et al., pooling 823 patients from the concurrent literature, the pooled maternal mortality rate was 2.8% (95% CI 1.5-5.1) and the pooled fetal mortality rate was 12.3% (95% CI 5.7-24.7), with maternal survival improving further in studies published between 2016 and 2020 (mortality 2.4%) compared with 2010-2015 (mortality 3.3%).[6] Luo et al reported maternal and fetal mortality rates of 3.3% (4/121)

and 11.6% (14/121), respectively, in their 121-patient cohort, with disease severity significantly correlated with the risk of death ($p<0.01$), and noted that 72.7% of moderate-to-severe cases required caesarean delivery to safely terminate gestation.[3] By contrast, the more recent Beijing series of Zhang et al recorded no maternal or fetal deaths among 32 patients despite hypertriglyceridemia-related disease predominating, and a Chinese meta-analysis of 4,034 patients found pooled maternal mortality of 1.8% and fetal mortality of 10.2%.[4,11]

Nevertheless, acute pancreatitis remains associated with significant obstetric complications, including spontaneous preterm labour, fetal growth restriction, fetal distress, stillbirth, and neonatal prematurity. Tang et al in a cohort of 96 spontaneously occurring cases from a single US academic centre, found that adverse fetal outcomes, including fetal loss and preterm delivery, occurred predominantly among women who developed pancreatitis earlier in gestation, while three of their third-trimester patients instead developed disseminated intravascular coagulation.[5] Severe necrotizing pancreatitis continues to carry higher maternal morbidity because of pancreatic necrosis, systemic inflammatory response syndrome, sepsis, respiratory failure, acute kidney injury, and thromboembolic complications, as illustrated by case 1 of the present series, who required two days of mechanical ventilation and developed acute kidney injury and hypocalcaemia in addition to her pleural effusion and ileus.

The recent Indian case series by Kumari et al in which maternal outcomes were favourable and neonatal outcomes were uneventful across the reported cases, similarly demonstrated favourable maternal recovery following multidisciplinary management but emphasized that pancreatitis may mimic HELLP syndrome, severe preeclampsia, and acute fatty liver of pregnancy, underscoring that early diagnosis is essential for improving maternal and neonatal outcomes.[7]

CONCLUSION

Acute pancreatitis in pregnancy is uncommon but carries meaningful maternal and perinatal risk, particularly when it presents in the third trimester with features that overlap other obstetric emergencies. This series illustrates the spectrum of disease, from mild, self-limiting pancreatitis managed conservatively to severe necrotizing pancreatitis requiring intensive care and emergency delivery. A high index of clinical suspicion, prompt biochemical and imaging confirmation, and coordinated multidisciplinary management involving obstetrics, medicine, surgery, and critical care are essential to optimize outcomes. In this series, timely recognition and supportive care resulted in 100% maternal survival and 100% neonatal survival despite preterm delivery in all cases, reinforcing that favourable outcomes are achievable even in severe disease when management is prompt and coordinated.

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