Treatment and diagnosis of hyperlipidemia acute pancreatitis in pregnancy associated with pre-pregnancy obesity and diabetes: A case report

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Abstract. Hyperlipidemia acute pancreatitis (HLAP) is a specific type of pancreatitis mainly caused by elevated serum triglyceride (TG) levels. Therefore, knowledge of patients' medical history is crucial to the identification of those at high risk of HLAP. Diabetes and obesity are associated with high levels of triglycerides, a risk factor for the development of HLAP, which should be controlled before pregnancy. Moreover, HLAP is associated with additional diagnostic and management challenges related to hyperlipidemia (HL) and pregnancy. HLAP during pregnancy has a rapid onset and rapid progression, and complications are more likely to damage the function of multiple organs. HLAP is more common after 28 weeks of pregnancy, the cause is mostly high TG and the serum TG of the patient is often >1,000 mg/d1. Clinicians should be alert to the occurrence of server acute pancreatitis (AP). Therefore, clinicians need to identify and implement effective treatment in a timely manner to control the progression of HLAP during pregnancy and improve pregnancy outcomes. The present study reported the case of a 26-year-old pregnant patient who was hospitalized for epigastric pain at 35 weeks and 2 days of gestation. Medical and family history reported previous diagnoses of diabetes and obesity (weight before pregnancy, 103 kg; BMI, 36.40 kg/m^2). Laboratory tests demonstrated high levels of lipase and amylase, a notable systemic inflammatory response, HL, coagulopathy, hypoproteinemia and hyperglycemia. Abdominal ultrasonography demonstrated a hypoechoic pancreatic head. A clinical diagnosis of AP was confirmed using CT scanning. Initial interventions for HLAP included aggressive intravenous hydration, bowel rest, pain control and a combination of heparin and insulin. Lipid-lowering agents were administered to reduce serum lipid levels. Hemoperfusion and continuous renal replacement therapy were also used to rapidly counteract the elevated lipid levels. Antibiotics were administered in the present case because inflammatory markers such as leukocytes, neutrophils and C-reactive protein were elevated. The patient and newborn were discharged 11 days after hospitalization, with an improvement in maternal clinical health and the infant was healthy. When evaluating pregnant patients with pre-obesity and diabetes presenting with abdominal pain, obstetricians should consider HLAP. Timely diagnosis and multi-team precision treatment are effective for good outcomes for mother and baby.

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Abbreviations: AP, acute pancreatitis; APIP, AP in pregnancy; FG, fibrinogen; FDP, FG degradation product; CRP, C-reactive protein; WBC, white cell count; TG, triglycerides; TC, total cholesterol; LDL, low-density lipoprotein; ALB, albumin; ICU, intensive care unit; HLAP, hyperlipidemia acute pancreatitis

Key words: hyperlipidemia acute pancreatitis, pregnancy, obesity, diabetes

Introduction

Acute pancreatitis (AP) is an inflammatory process affecting the pancreas and is characterized by severe abdominal pain and increased levels of amylase and lipase (1). AP in pregnancy (APIP) is a rare complication (incidence between 1 in 12,000 and 1 in 1,000) that threatens the health of the patient and their offspring (2,3). APIP is associated with increased risk of prematurity, preeclampsia, post-partum hemorrhage, maternal death and fetal demise. In pregnant women with AP, delivery is associated with increased risk of requiring transfusions, developing venous thromboembolisms, acute respiratory

failure and disseminated intravascular coagulation (4,5). Hyperlipidemia (HL) AP in pregnancy has been reported to be associated with a maternal mortality rate of ~20% (6). HLAP can cause serious complications, such as fetal distress, intrauterine death, maternal multiple organ failure, abdominal compartment syndrome and pancreatic encephalopathy, when not treated in a timely manner (7).

HL has emerged as an important cause of APIP in the Chinese female population (8). Among Chinese women, HL is the second leading cause of APIP after cholelithiasis (6). HLAP is increased in patients with plasma triglyceride (TG) levels >1,000 mg/dl (11.3 mmol/l) compared with patients with plasma TG levels ≤1,000 mg/dl. Excess TGs are hydrolyzed by lipase enzymes excreted by pancreatic acinar cells to produce free fatty acids (FFAs). FFAs have a direct cytotoxic effect on acinar and vascular endothelial cells and elevated levels of FFAs trigger an inflammatory response (9). Local inflammation, triggered by the activation of pancreatic proteolytic enzymes within acinar tissues, may progress to systemic inflammation (10).

HLAP in pregnancy has been reported to be associated with a higher risk of severe AP (SAP) and poor maternal and fetal outcomes compared with AP due to other causes (11). TG levels have been demonstrated to be positively associated with AP severity, and a prospective multicenter study of 716 patients indicated a dose-dependent relationship between TG levels and AP severity, and high TG levels were associated with organ failure in patients with AP (12). An observational study of 54 pregnant patients with AP showed that HL is associated with more intrauterine fetal distress and worse fetal outcomes compared with AP from other etiologies (13). Severe pancreatitis is often complicated by varying degrees of multi-organ failure such as acute respiratory failure, acute renal failure, heart failure and arrhythmias, gastrointestinal bleeding, and pancreatic encephalopathy (3). SAP is a serious complication during pregnancy and the main cause of maternal and fetal death, with its fetal mortality rate at 10-30% (14). The 2012 revised Atlanta Classification classifies the severity of AP as follows: Mild (M) AP, patients without organ failure and local complications; moderately severe AP, patients with organ failure for <48 h or local complications; SAP, patients with organ failure for >48 h (15).

HL can originate from a primary (genetic) abnormality of lipid metabolism or it may be caused by other diseases that cause an overproduction of TG, such as diabetes, visceral obesity and pregnancy (16). HL is defined as >11.3 mmol/l or 5.6-11.3 mmol/l TG with chyle blood (17). Severe HL also increases the risk of AP. A previous study reported that, compared with a control group of individuals with <150 mg/dl TG, patients with 150-300 mg/dl TG have an AP hazard ratio of 1.5, whilst individuals with >500 mg/dl TG had a hazard ratio of 3.2. Moreover, the same authors reported a 4% increase in incidence of AP for every 1,000 mg/dl increase in serum TG (18).

HLAP in pregnancy usually occurs in pregnant individuals with preexisting abnormalities of lipid metabolism such as obesity, fatty liver disease and diabetes before pregnancy (16). The diagnosis of HLAP meets the AP diagnostic criteria: i) Persistent pain in the upper abdomen; ii) serum amylase and/or lipase concentrations are at least 3 times higher than the upper limit of normal; and iii) imaging findings of AP. AP is diagnosed if two of the three criteria are met. The following criteria should also be met for the diagnosis of HLAP: 1+3 or 2+3, where 1 indicates serum TG levels ≥11.3 mmol/l, 2 indicates serum TG levels between 5.65 and 11.3 mmol/l, and 3 indicates blood sample appeared chylous to exclude other causes of acute pancreatitis (19). However, early diagnosis of HLAP in pregnant patients can be delayed as its early clinical symptoms, such as abdominal pain, can be confused with abdominal pain caused by contractions in late pregnancy and abdominal pain caused by AP (20).

HLAP is mainly caused by elevated serum triglycerides and its treatment principles are similar to those of management of non-pregnant patients; however, multidisciplinary cooperation is required to ensure the safety of mother and baby. Specific treatment measures include nutrient solution support, fasting, gastrointestinal decompression, inhibition of pancreatic enzyme activity, suppression of pancreatic secretion, maintenance of insulin, heparin use and antibiotic use in infected patients (21).

As HLAP can lead to adverse maternal and infant outcomes, there is a need for early diagnosis and assessment of disease severity to improve the prognosis of HLAP in pregnancy. The present study reported on the early diagnosis and management of a case of HLAP in a pregnant patient with diabetes and obesity and emphasized the diagnostic and treatment challenges associated with this condition.

Case report

A 26-year-old patient at 35 weeks and 2 days of gestation of their first pregnancy was transferred from Zhenjiang Yangzhong County Hospital of Traditional Chinese Medicine (Zhenjiang, China) to Maternity and Child Health Hospital of Zhenjiang (Zhenjiang, China). They were hospitalized in March 2022 due to epigastric pain, which was dull with paroxysmal intensification. The patient presented with a body temperature of 36.8˚C, blood pressure at 138/88 mmHg, respiratory rate of 17 breaths/min and pulse at 78 beats/min. Physical examination of the abdominal pregnancy indicated a soft abdomen with tenderness under the xiphoid process and no rebound pain reported in the left upper abdomen. Bowel sounds were reduced to 1-2 times/min. Medical and family history reported diagnoses of diabetes and obesity. The patient's weight before pregnancy was 103 kg, with a BMI of 36.40 kg/m^2.

Laboratory test results at admission, including coagulation parameters, are presented in Table I. Levels of total cholesterol (TC), TG and low-density lipoprotein (LDL) cholesterol were increased at admission compared with their corresponding normal ranges and high-density lipoprotein cholesterol levels reached 9.89 mmol/l. In addition, blood samples appeared chylous. Other parameters were within normal ranges.

The patient presented with the following pregnancy measures: Uterine height, 41 cm; abdominal circumference, 120 cm; fetal orientation, left occiput anterior; fetal heart rate, 150 beats/min; head exposure. Abdominal ultrasonography using the emergency bedside B-ultrasound (GEE LOGIQ S8 ultrasound; parameters: MI 1.0 TIS 1.6 C1-5 abdominal mode) showed a hypoechoic pancreatic head (images not available). The Acute Physiologic Assessment and Chronic Health Evaluation II (APACHEII) scoring system was used, which

Table I. Laboratory tests of hyperlipidemia acute pancreatitis during pregnancy.

Main parameters	Admission day	Admission day-ICU	4 days-ICU	Discharge day	Normal range
White blood count, x10^9/l	13.27	12.99	11.56	6.35	4.00-10.00
Neutrophils, x10^9/l	11.23	8.94	9.77	6.61	1.80-6.30
Neutrophil ratio, %	84.6%	81.80	79.20	55.90	40.0-75.0
C-reactive protein, mg/l	105.90	112.30	148.20	4.30	0.00-8.00
Lipase, U/l	425.00	355.00	305.00	Untested	23.00-300.00
Amylase, IU/l	147.60	168.00	49.90	47.40	37.00-53.00
Uroamylase, IU/l	157.50	894.90	340.00	33.00	0.00-600.00
Total protein, g/l	128.00	81.80	45.90	56.30	60.00-83.00
ALB, g/l	34.80	26.00	23.00	34.70	37.00-53.00
GLOB, g/l	93.20	19.10	20.50	21.60	20.00-32.00
ALB/GLOB ratio	0.37	0.49	1.12	1.61	1.20-2.40
Lactic dehydrogenase, IU/l	212.00	345.00	194.00	193.00	103.00-227.00
Glucose, mmol/l	9.62	9.74	8.37	5.69	3.89-6.11
Creatinine, μmol/l	138.90	42.50	44.40	39.80	45.00-84.00
Magnesium, mmol/l	0.59	0.71	0.71	1.02	0.70-1.08
Total cholesterol, mmol/l	22.00	19.40	8.88	4.05	3.10-5.20
Triglycerides, mmol/l	26.30	27.50	6.86	5.66	0.40-1.70
Low density lipoprotein cholesterol, mmol/l	10.17	4.90	3.99	2.66	0.00-3.37
High density lipoprotein cholesterol, mmol/l	9.89	0.70	0.72	1.46	1.03-1.55
FG, g/l	5.76	8.39	9.60	3.88	2.38-4.98
FG degradation product, μg/ml	5.80	7.20	5.80	4.75	0.00-5.00
Antithrombin III, %	20.40	16.00	57.00	76.00	83.00-128.00
D-dimer, ng/ml	2447.00	2054.00	1385.00	1345.00	0.00-255.00

Normal ranges are based on pregnant women in Zhenjiang, Jiangsu, China. ICU, intensive care unit; ALB, albumin; GLOB, globulin; FG, fibrinogen.

includes an acute physiology score, chronic health score and age score. APACHEII scores range between 1 and 71 points in total, and the higher the score, the more severe the condition (22). The patient in the present study was rated with six points. For an AP diagnosis, at least two of the following three criteria need to be met: i) Abdominal pain in the upper abdomen at the onset of AP; ii) biochemical evidence of pancreatitis (serum amylase and/or lipase >3 times the upper limit of the normal range); and iii) typical abdominal imaging findings (19). A diagnosis of HLAP can then be made based on the following criteria established by the Chinese guidelines for HLAP: AP diagnosis and TG ≥11.3 mmol/l (>1,000 mg/dl) at the time of onset or TG between 5.65-11.3 mmol/l (500-1,000 mg/dl) without other underlying conditions, such as gallstone and alcoholism, or medications such as diuretics, hormonal drugs and antineoplastic drugs (19). Diagnosis of AP in the patient in the present study was based on symptoms, clinical findings (the patient was in epigastric pain, which was dull with paroxysmal intensification.), elevated TG, amylase and lipase and ultrasound examination.

Caesarean section was performed at 12 h later. The newborn was female, with a birth weight of 3,500 g and an Appearance, Pulse, Grimace, Activity and Respiration score at 1 and 5 min after birth of 9 and 10, respectively (23). The newborn was sent to the neonatal intensive care unit (ICU) due to the premature birth. Treatment for the newborn included: Warmth, monitoring of vital signs, prevention of nosocomial infections and developmental support. The newborn was healthy despite prematurity.

A 200-mLlfatty effusion was found in the abdominal cavity at the time of the caesarean section, a drainage tube was inserted for drainage and AP was confirmed by CT after the caesarean section. CT (64 rows of 128-slice Light Speed spiral CT; GE Healthcare; 64 rows of 128 layers; parameters: tube voltage, 120 kV; tube current, 350 mA; pitch, 0.9; matrix, 512X512; layer thickness, 5 mm; layer pitch, 5 mm; supine position) scanning after caesarean section demonstrated peri-pancreatic fat stranding and left anterior renal vein thickening (Fig. 1A and B) but no evidence of pancreatic necrosis. CT images of pancreatic cancer demonstrate pancreatic morphological variation, localized enlargement, loss of fat around the pancreas, narrowing of the pancreatic duct, dilation and compression of large blood vessels (24). Therefore, pancreatic cancer was excluded in this patient.

Following diagnosis of HLAP, the Pancreatic Surgery Group, Surgical Branch of Chinese Medical Association guidelines (19) state that treatment should continue to be performed with treatment routines such as aggressive hydration, analgesia and close observation. TG levels should be measured as early as possible and HL should be reduced

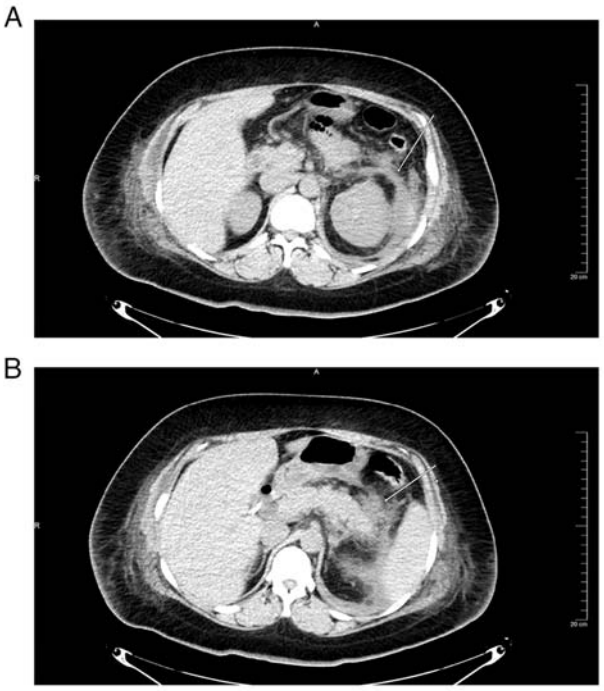

Figure 1. Abdominal computed tomography images demonstrating (A) peripancreatic fat stranding (arrow) and (B) left anterior renal vein thickening (arrow).

within 48 h from the onset of AP (25). After obstetric and ICU consultation, the patient in the present study was referred to ICU (15 h later) for further treatment. Following ICU admission, supportive care with fasting, fluid resuscitation, pain and blood sugar control, blood lipid-lowering drugs, anti-infective agents (once every 12 h: Ceftazidime, 2 g, cefoperazone sodium, 3 g and sulbactam sodium, 3 g) and inhibition of pancreatic enzyme secretion and activity (intravenous drip once every 8 h: Octreotide acetate, 0.3 mg and intravenous drip every 6 h: ulinastatin, 100,000 units). The Pancreatic Surgery Group, Surgical Branch of Chinese Medical Association guidelines state that patients with severe HLAP should continue treatment with lipid-lowering agents, including anti-HL drugs, insulin infusion, heparin and plasmapheresis treatments. The patient in the present study was administered fibric acid derivative bezafibrate (0.1 g, orally, three times/day), human insulin mixed injection (6 units, twice a day, subcutaneously) and low molecular weight calcium heparin (0.6 ml, every 12 h, subcutaneous injection). Plasmapheresis was utilized as a lipid- and inflammatory factors-reducing treatment, and three sessions of hemoperfusion (HP; 2 h; blood flow, 200-220 ml/min) and continuous renal replacement therapy (CRRT) were performed. A right internal jugular venous cannula was used for indwelling with a single-needle double-lumen catheter. Extracorporeal circulation CRRT treatment was performed using a blood filter connected in series with a HP device. Replacement fluid volume was 2,000 ml/h, blood flow was controlled at 200-220 ml/min and liquid clearance was 200 ml/min. Sodium bicarbonate was instilled at a uniform rate, the filter was replaced once at 24 h and treatment was continued for 3 days. The patient exhibited hypoproteinemia and ALB (10 g per day for 5 days) was administered. After 4 days in ICU, abdominal pain improved, amylase levels returned to normal, blood lipid levels (TC, 8.88 mmol/l; TG, 6.86 mmol/l; LDL-cholesterol, 3.99 mmol/l) markedly decreased and respiration and circulation were stable (Table I). However, WBC, neutrophils, NR and CRP levels indicated that there was still inflammation (Table I). Treatment continued after transfer to gastroenterology from ICU after 4 days with full improvement of clinical and laboratory parameters. The patient recovered after 11 days with laboratory tests demonstrating reduced serum TG levels and normalization of amylase, TC, LDL, glucose, WBC, neutrophils, NR and CRP levels (Table I). The patient was discharged from hospital after 11 days with AP symptoms improved and both the patient and infant were determined to be in good condition. The clinical course of the patient from hospitalization to discharge is demonstrated in Fig. 2.

The mother and baby were followed up at 42 days and 6 months postpartum and the mother and baby were healthy.

Discussion

APIP is an inflammatory syndrome that can occur following pancreatic injury, with a variable acute inflammatory response and numerous local (such as pancreatic abscess and pancreatic pseudocyst) and systemic (such as acute respiratory failure, acute renal failure, heart failure, gastrointestinal bleeding and pancreatic encephalopathy) complications (26). Obesity (defined as BMI >30 kg/m^2) can be characterized by a grade inflammatory state and has been reported to be harmful to the human body (27). It also increases the risk of insulin resistance and diabetes (28). The present case study demonstrated that the presence of obesity and diabetes pre-pregnancy are important risk factors in the development of APIP. Previous studies reported that a family history of HL and type 2 diabetes increased the risk of hyperlipidemic pancreatitis in pregnancy (29,30). Zeng *et al* (31) reported that mean platelet volume (MPV) was elevated in patients with HLAP, which has a certain predictive value for AP and disease severity during pregnancy. MPV is a blood parameter used for measuring platelet size and is an indicator of thrombocytic activity. Elevated MPV facilitates platelet adhesiveness and aggregation, which may lead to a high prothrombotic potential and impairment of pancreatic microcirculation in HL-induced SAP during pregnancy (32). In the present case, no MPV abnormalities were found.

Diabetes and obesity are also associated with elevated TG levels and increased concentrations of very-LDL and chylomicrons, which can lead to HLAP. It has been reported that TG levels >11.3 mmol/l (1,000 mg/dl) can trigger AP and associated complications (10). Pregnancy is associated with physiological increases in blood TC and TG, which are not associated with AP alone; however, a preexisting abnormal lipid metabolism may aggravate the increase in TG during pregnancy, leading to HLAP (10). Therefore, diabetes, overweight and obesity should be considered as potential cofactors in the etiology of HLAP. Uncontrolled pre-pregnancy obesity and diabetes are important risk factors for gestational HLAP (33), as demonstrated in the present case report. The patient had diabetes and obesity pre-pregnancy and became pregnant without controlling these conditions.

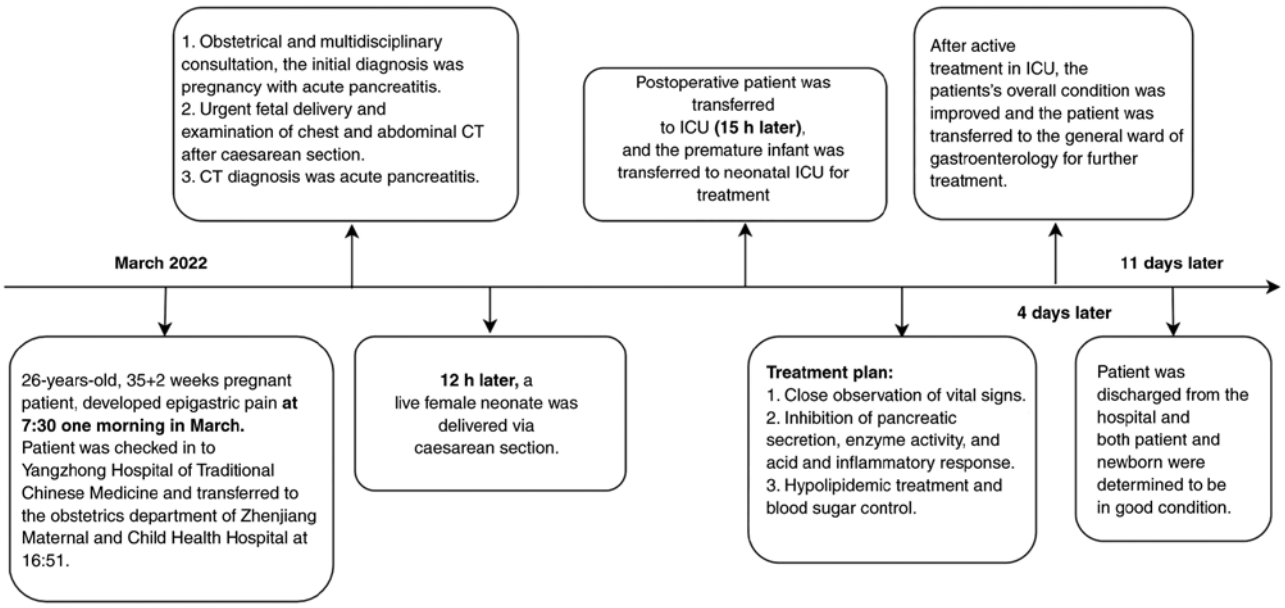

Figure 2. Complete clinical course of the patient from hospitalization to discharge.

Lipid-lowering therapy is particularly important in the treatment of HLAP, which includes the administration of lipid-lowering drugs and blood purification. In the present case, the patient was administered oral lipid-lowering drugs such as fibrate, non-oral lipid-lowering drugs, heparin and insulin, and underwent blood purification. This is consistent with other reports (34,35). Fibrates, a class of drugs belonging to category C medications, work to raise HDL and lower TG through the upregulation of lipoprotein lipase metabolism. In addition, non-oral drugs, such as heparin and insulin, can stimulate lipoprotein esterase activity and promote chylomicron degradation, thereby reducing blood lipids. The combination of heparin and insulin can be used as first-line therapy for severe HLAP (36). Several studies have reported that sequential hemofiltration therapy with HP can quickly decrease serum amylase and TG levels in patients with HLAP, effectively preventing pancreatic inflammation and necrosis, delaying disease progression and reducing case fatality (37,38). In the present study, amylase, lipase and TG were notably lower after HP-CRRT treatment, consistent with the aforementioned studies. Other clinically available lipid-modifying drugs include statins, niacin, resins and cholesterol absorption inhibitors. Drug treatment of diabetes includes oral drugs such as sulfonylureas, biguanide hypoglycemic drugs, α glucosidase inhibitors, insulin sensitizers and insulin therapy. Insulin preparations include animal insulin, human insulin and insulin analogues (39).

HLAP in pregnancy is associated with a much higher risk of SAP and is strongly associated with poor maternal-fetal outcomes compared with AP due to other causes (40). Apart from maternal complications such as acute renal failure, sepsis and acute respiratory distress syndrome, there are also potential fetus complications with HLAP. Risks to the fetus of HLAP in pregnancy include preterm, prematurity and in-utero fetus death. APIP usually presents with a sudden onset of severe epigastric pain that radiates to the back. Bowel sounds are reduced, and a positive Murphy's sign may be present. Early monitoring of the level of inflammatory indicators is important for the assessment of the severity and classification of AP. In the present case, the inflammation-related indicators, CRP, WBC, neutrophils and neutrophil ratio, continuously increased for several days. CRP is an acute-phase protein produced by the liver in response to inflammation. The main biological function of CRP is reported to be host defense against bacterial pathogens and clearance of apoptotic and necrotic cells (41). CRP is sensitive to the degree of inflammation and can markedly change with the degree of inflammation (42). Previous studies have reported that a systemic inflammatory state with abnormal indexes such as serum WBC, neutrophils, NR and CRP, is closely related to the severity of AP (43,44). In the early stage of AP, neutrophils are activated to produce numerous inflammatory factors such as IL-1β, IL-6 and IL-17, and oxygen-free radicals, leading to vascular endothelial cell damage and pancreatic microcirculation disturbance (45). At the same time, immune cells are recruited and activated to participate in the systemic inflammatory response, aggravating the damage to the pancreas and other organs such as the lung (46). AP itself causes elevated WBC, neutrophils and CRP, however this can change as the disease progresses. In MAP, WBC is only slightly elevated or unchanged. Activation of white blood cells is an important reason for the evolution of MAP to SAP. A previous study has reported that a systemic inflammatory state with abnormal indexes, such as serum WBC, neutrophils, NR and CRP, is closely associated with the severity of AP (47). The present case demonstrated that the levels of leukocytes, CRP and neutrophils change with the progression of HLAP disease. Therefore, the combined detection of CRP, leukocytes, neutrophils and neutrophil ratios is recommended to assess the severity of HLAP.

Fibrinogen (FG), also known as coagulation factor I, is a coagulation factor produced by the liver which serves an important physiological role in the coagulation process. Increased FG content is an independent risk factor for vascular thrombosis. D-dimer is the product of fibrinolytic hydrolysis after activation and crosslinking of fibrin monomers. D-dimer

is a marker used to assess hypercoagulability and thrombosis *in vivo* (48). FG degradation product (FDP) is a byproduct produced after FG and fibrin are decomposed by plasma elements, which reflects the intensity of fibrinolytic activity *in vivo* (49). In the present case, the levels of FG, D-dimer and FDP were increased, whereas antithrombin III levels were decreased. This suggested that the patient had abnormal coagulation which could cause disseminated intravascular coagulation and thrombosis. Additionally, a transient increase in TP and globulin and a persistent decrease in ALB were demonstrated. ALB is a plasma protein produced by the liver and the level of ALB can reflect the leakage of capillaries (50). SAP is often complicated with hypoalbuminemia and the mechanism may involve: i) Increased ALB extravasation; ii) digestive and absorption disorders; iii) decomposition increases. Several studies have reported that hypoalbuminemia serves a role as a booster in the pathological process of SAP (51,52). Elevated amylase and/or lipase are the diagnostic hallmarks of AP; however, amylase levels in HLAP may be reported as normal or low in >50% of patients (53). As reported in other studies (54,55), the patient in the present case had increased serum amylase and lipase levels which did not reach levels three times the upper limit of the normal range, as in patients with pancreatitis who had HL.

Chinese guidelines for the diagnosis and treatment of AP (19) do not recommend routine use of prophylactic antibiotics in patients with AP, consistent with the 2013 International Association of Pancreatology/American Pancreatic Association Acute Pancreatitis Guidelines (56) and the 2018 American Gastroenterological Association Institute Clinical Guidelines Committee (57). AP is a nonbacterial inflammation and prophylactic use of antibiotics is controversial; however, the appropriate use of antibiotics in the case of suspected infection is not controversial. In the present case, the patient's inflammatory indicators, leukocytes, CRP, WBCs and neutrophils, were very high and a bacterial infection was suspected. A puncture of the bacterial culture specimen was required for bacterial detection, but the patient and family did not agree to the procedure. Therefore, antibiotics were administered.

The clinical symptoms and findings from ultrasound and CT imaging in the present case are summarized as follows: i) The clinical symptoms of the present case are atypical as they manifested only as epigastric pain, without obvious nausea, vomiting and radiation pain; ii) Increased serum amylase and lipase levels were measured, but they did not reach levels three times the upper limit of the normal range; iii) Serum lipids were markedly elevated, especially TG, which was >11.3 mmol/l, and there were serum chyloform changes; iv) Inflammatory markers, such as leukocytes, neutrophils, and CRP, were markedly elevated; v) The patient had pre-pregnancy HL and diabetes mellitus; vi) Abdominal ultrasonography demonstrated a hypoechoic pancreatic head; vii) CT scan images demonstrated peripancreatic fat stranding and left anterior renal vein thickening.

To conclude, the present case study demonstrated that early and accurate diagnosis of the patient in the obstetrics department, timely fetal delivery, transfer to ICU and prompt treatment served key roles in the good maternal health and infant condition. Additionally, the treatment of HLAP in pregnancy requires multidisciplinary collaboration to develop personalized management plans. The treatment plan in the present case was developed after multidisciplinary discussions that included the obstetrics, ICU and gastroenterology departments.

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Availability of data and materials

All data generated or analyzed during the present study are included in this published article.

Authors' contributions

QW and BX obtained the clinical data. CH and XN performed the investigation. MG and LM conceived the methodology. JC and YX supervised the study. WC wrote the draft of the manuscript and XW wrote and reviewed the manuscript. JC and YX analyzed and interpreted the data. WC made substantial contributions to conception and design, acquisition, analysis and interpretation of data, and writing/editing/revising the manuscript. XW contributed to the conception and design of the study. WC and XN confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

The present study was approved by the ethics committee of the Maternity and Child Health Hospital of Zhenjiang (approval no. F201931; Zhenjiang, China). Informed consent was obtained from the patient.

Patient consent for publication

The patient who participated in the study provided written informed consent for the publication of any associated data.

Competing interests

The authors declare that they have no competing interests.

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