Maternal and Perinatal Outcomes of Acute Pancreatitis During Pregnancy

Gebelikte Görülen Akut Pankreatitin Maternal ve Perinatal Sonuçları

Aytül Çorbacıoğlu Esmer, Mehmet Özsürmeli, İbrahim Kalelioğlu

Department of Gynecology and Obstetrics, Istanbul Faculty of Medicine, İstanbul University, İstanbul, Turkey

ABSTRACT

Objective: To investigate the maternal and perinatal mortality and morbidity due to acute pancreatitis during pregnancy by reviewing our experience over a ten-year period.
Methods: In this retrospective study, hospital charts were reviewed for every pregnant women with pancreatitis who was admitted between January 2003 and July 2012.
Results: A total of 14 women had acute pancreatitis within this period with a frequency of 1 in 1451 births, and 57.2% of cases were classified as mild pancreatitis. Two maternal deaths occurred (14.2%) and the etiology of pancreatitis in these cases was hypercalcemia due to hyperparathyroidism and PIH. The frequency of preterm delivery was 61.5%. In the mild pancreatitis group, more than half of the deliveries (4/7) occurred beyond 37 weeks, while almost all deliveries (5/6) occurred prematurely in the severe pancreatitis group. The cause of preterm delivery was spontaneous preterm labor and fetal distress in most of the cases. No fetal or neonatal deaths were observed.
Conclusion: This study emphasizes the progressive and fulminant course in severe disease, especially in hypercalcemia and PIH induced pancreatitis. Acute pancreatitis causes preterm delivery in most of the cases, which is a result of fetal compromise and premature uterine contractions associated with the severity of the disease. *(Gazi Med J 2012; 23: 133-7)*

Key Words: Acute pancreatitis, pregnancy, maternal mortality, perinatal mortality

Received: 09.10.2012 **Accepted:** 22.11.2012

ÖZET

Amaç: Kliniğimizin on yıllık deneyimini gözden geçirerek akut pankreatit nedeniyle gelişen maternal ve perinatal morbidite ve mortaliteyi araştırmayı amaçladık.
Yöntemler: Bu retrospektif çalışmada, Ocak 2003-Temmuz 2012 tarihleri arasında kliniğimize başvurmuş olan ve pankreatit tanısı alan tüm gebelerin dosyası incelendi.
Bulgular: Belirtilen süre zarfında akut pankreatit 1/1451 sıklıkla toplam 14 gebede görüldü. Hastalığın başladığı ve doğumun gerçekleştiği ortalama gestasyonel hafta sırasıyla 31.4±4.9 ve 35.5±3.2 idi. Pankreatitin nedeni olguların %50'sinde (7/14) safra taşı, %28.5'inde (4/14) hipertrigliseridemiydi. Geri kalan üç olguda ise etiyoloji hiperkalsemi, preeklampsi ve idiyopatikti. Olguların %57.2'si (8/14) hafif pankreatit olarak sınıflandırıldı. Maternal ölüm iki olguda gerçekleşti (%14.2) ve bu olgularda pankreatitin etiyolojisi hiperparatiroidizme sekonder hiperkalsemi ve preeklampsiydi. Preterm doğumun sıklığı %61.5 idi. Hafif pankreatit olgularında doğumların yarısından çoğu (4/7) 37. gebelik haftasından sonra gerçekleşirken, ağır pankreatit olgularının hemen hepsinde (5/6) prematür doğum meydana geldi. Olguların çoğunda preterm doğumun nedeni spontan preterm eylem ve fetal distres idi. Fetal veya neonatal ölüm meydana gelmedi.
Sonuç: Bu çalışma, şiddetli ve özellikle hiperkalsemi ve preeklampsiye sekonder gelişen pankreatitin progresif ve fulminan seyrini vurgulamaktadır. Akut pankreatit olguların büyük çoğunluğunda, hastalığın şiddetiyle ilişkili olarak ortaya çıkan fetal distres ve prematür uterin kontraksiyonlar nedeniyle preterm doğuma yol açmaktadır. *(Gazi Med J 2012; 23: 133-7)*

Anahtar Sözcükler: Akut pankreatit, gebelik, maternal mortalite, perinatal mortalite

Geliş Tarihi: 09.10.2012 **Kabul Tarihi:** 22.11.2012

Address for Correspondence / Yazışma Adresi: Dr. Aytül Çorbacıoğlu Esmer, Department of Gynecology and Obstetrics, Istanbul Faculty of Medicine, İstanbul University, İstanbul, Turkey Phone: +90 542 311 92 40 E-mail: aytulcorbacioglu@gmail.com

doi:10.5152/gmj.2012.31

INTRODUCTION

Acute pancreatitis during pregnancy is a rare disease having an incidence between 1/1000 and 1/4000 births (1). It is associated with gallstone disease in 40-66% of patients, as lodging or impaction of a stone or microlitiasis in the ampulla of vater initiates the premature activation of intracinar trypsinogen to trypsin (2). 5-56% of acute pancreatitis in pregnancy develops due to hypertriglyceridemia (3). During pregnancy the concentration of serum cholesterol increases by 50% and triglyceride by two to four times (4). However, physiological hypertriglyceridemia in pregnancy rarely exceeds 300 mg/dL and acute pancreatitis is induced by levels higher than 1000 mg/dL (11.4 mmol/L), mostly in women with pre-existing disorders of lipid metabolism (5). The other causes are alcohol consumption, medications, fatty liver of pregnancy, trauma and idiopathic (6). Moreover, hypercalcemia of any cause, including hyperparathyroidism, can lead to acute pancreatitis. The deposition of calcium in the pancreatic duct and calcium activation of trypsinogen within the pancreatic parenchyma are suggested mechanisms (7). Pregnancy induced hypertension (PIH) also rarely causes acute pancreatitis due to the microvascular abnormalities involving splanchnic circulation which results in pancreatic ischemic changes (8-10).

The predominant symptom of acute pancreatitis is midepigastric pain radiating to the back which is accompanied by midepigastric tenderness, nausea, vomiting and fever (6). The course of pancreatitis during pregnancy is usually mild and self-limited but can be rapidly progressive and fulminant due to severe complications such as pancreatic necrosis, generalized peritonitis, acute respiratory distress syndrome (ARDS), disseminated intravacular coagulopathy (DIC) and multiple organ failure (2, 6). Maternal and perinatal mortality were reported to be as high as 20% and 50% respectively in prior reports, but as a result of improvement in laboratory tests and imaging techniques leading to early diagnosis, the rate of maternal and perinatal mortality has been reported to be <1% and 0-18% respectively in recent studies (6, 11).

In this study we aimed to investigate the maternal and perinatal morbidity and mortality due to acute pancreatitis during pregnancy by reviewing our experience over a ten-year period.

METHODS

In this retrospective study, hospital charts and computerized medical records were reviewed for every pregnant women with pancreatitis admitted to the Istanbul Faculty of Medicine Department of Obstetrics and Gynecology during the period between January 2003 and July 2012. All cases were treated and followed up by a multi-diciplinary team consisting of obstetricians, general surgeons, gastroenterologists, anesthesiologists and endocrinologists. The infomation on maternal age, obstetric history, gestational age at onset and delivery, etiology, management, complications, the length of stay, maternal and fetal outcomes was collected.

The etiology of pancreatitis was determined based on the history, ultrasound and laboratory studies. Biliary pancreatitis was diagnosed when gallstones or sludge were detected in the biliary tree or the gallbladder. Hyperlipidemic pancreatitis was diagnosed when the serum triglyceride level was above 1000 mg/dL. Pancreatitis secondary to PIH was diagnosed when other causes of acute pacreatitis were excluded in the presence of preeclampsia with or without HELLP syndrome. Pancreatitis due to hypercalcemia was diagnosed when the serum total calcium level was higher than 10.5 mg/dL. Cases were classified as idiopathic when an etiologic factor could not be found with diagnostic tests.

Severity of acute pancreatitis was evaluated by Ranson criteria (12). Ranson criteria include age >55 years, (2) white blood cell count >16000 cells/mm^3, blood glucose >10 mmol/L (200 mg/dL), serum AST >250 IU/L, serum LDH >350 IU/L at admission and serum calcium <2 mmol/L (8 mg/dL), hematocrit fall >10%, pO_2 <60 mmHg, BUN increased by ≥1.8 mmol/L (≥5 mg/dL) after IV fluid hydration, base deficit >4mEq/L, sequestration of fluids >6 L at 48^{th} hour of admission. Acute pancreatitis was defined as mild when two or fewer criteria were present and severe when three or more criteria were present.

RESULTS

Over the study period, 20325 women were delivered and 14 women had acute pancreatitis among this population, with a freqeuncy of 1 in 1451 births. The demographic and clinical features of the cases are shown in Table 1. The age of patients ranged between 23 and 36 with a mean of 28.4±4. 35.7% (5/14) of patients were nulliparous, while 64.3% (9/14) were parous. 21.4% (3/14) of patients were in the second and 78.6% (9/14) of patients were in the third trimester of pregnancy at the time of diagnosis. The mean gestational age at onset was 31.4±4.9 and the mean gestational age at delivery was 35.5±3.2 weeks. The median time interval between these dates was 1.5 (1-110) days. The median length of stay in hospital was 7 (3-70) days. The cause of pancreatitis was gallstone in 50% (7/14) and hypertriglyceridemia in 28.5% (4/14) of cases. The etiology of pancreatitis in the remaining three cases was hypercalcemia, PIH and idiopathic. 57.2% (8/14) of the cases had a Ranson score of less than three and were classified as mild pancreatitis. Four of these eight cases had biliary pancreatitis, while three had hyperlipidemic and one had idiopathic pancreatitis.. Among the remaining six (42.8%) cases with severe pancreatitis, four had a Ranson score of 3-4 (three biliary and one hyperlipidemic pancreatitis) and two had a Ranson score of 7-8 (pancreatitis secondary to hypercalcemia and PIH).

Of the seven cases with biliary pancreatitis, five (case 4, 5, 6, 7 and 10) were managed conservatively including bowel rest, intravenous fluid hydration and pain relief. One patient (case 2) underwent therapeutic endoscopic retrograde cholangiopancreatography (ERCP) at 32 weeks of gestation after the detection of choledecholithiasis by ultrasound. One patient (case 14) was managed conservatively at 27 weeks of gestation when cholelitiasis was observed with a normal biliary duct. Three weeks later, pancreatitis recurred and she underwent laparoscopic cholecystectomy with no complications. One case (case 4) was complicated with hyperglycemia which necessitated insulin therapy. In one case (case 7) a pseudocyst developed after delivery and was treated by percutaneous drainage and antibiotic.

All four patients with hyperlipidemic pancreatitis had familial hypertriglyceridemia and the mean serum triglyceride level was 4266.7±2240.5 mg/dL. Plasmapheresis was performed in all of these patients with a goal to maintain a triglyceride level below 1000 mg/dL. Two (case 3 and 8) underwent plasmapheresis just after the delivery for the treatment of pancreatitis. It was performed eight times in case 9 and four times in case 13 throughout gestation in order to prevent the recurrence of pancreatitis, as well as for the treatment of acute pancreatitis.

Table 1. The demographic and clinical characteristics of patients.

No	G	P	GAO	GAD	Etiology	Ranson score	Treatment	Complications	Mode of delivery	Apgar	Birth weight
1.	3	1	35+0	38+4	İdiopathic	2	Conservative	-	NSD	7 / 7	3240
2.	3	0	32+6	33+6	Gallstone	4	ERCP	-	C/S	8 / 10	2140
3.	3	2	35+3	35+3	HTG	2	Plasmapheresis, ICU	-	C/S	9 / 10	3400
4.	2	1	26+1	40+0	Gallstone	2	Conservative	Hyperglycemia	NSD	9 / 10	2890
5.	1	0	36+4	36+4	Gallstone	4	Conservative	-	C/S	7 / 9	2670
6.	1	0	31+0	39+0	Gallstone	2	Conservative	-	C/S	8 / 9	2350
7.	3	2	29+5	30+0	Gallstone	2	Conservative, TPN, ICU, percutaneous drainage of pseudocyst	Pseudocyst	C/S	8 / 10	1460
8.	1	0	37+2	37+5	HTG	0	Plasmapheresis	-	C/S	7 / 10	2480
9.	4	2	30+0	36+1	HTG	0	Plasmapheresis	-	C/S	8 / 10	2880
10.	2	1	37+1	37+1	Gallstone	4	Conservative	-	NSD	9 / 10	3200
11.	4	0	29+0	29+2	Hypercalcemia	7	Hemofiltration, calcitonin, TPN, ICU	ARDS, ARF, death	C/S	3 / 5	1120
12.	3	1	36+1	36+1	PIH	7	Laparotomy, TPN, ICU	Necrosis, ARDS, DIC, ARF, hyperglycemia, death	C/S	5 / 8	2390
13.	3	1	20+2	33+5	HTG	3	Plasmapheresis	-	C/S	7 / 8	2140
14.	3	2	27+0	Ongoing	Gallstone	2	L/S cholecystectomy	-	-	-	-

G: Gravidity, P: Parity, GAO: Gestational age at onset, GAD: Gestational age at delivery, HTG: Hypertriglyceridemia, PIH: Pregnancy induced hypertension, ERCP: Endoscopic retrograde cholangiopancreatography, TPN: Total parenteral nutrition, ICU: Intensive care unit, ARDS: Acute respiratory distress syndrome, DIC: Disseminated intravascular coagulopathy, ARF: Acute renal failure, NSD: Normal spontaneous delivery, C/S: cesarean section

Four women required intensive care unit (ICU) admission (case 3, 7, 11 and 12). Two maternal deaths occurred (14.2%) and the etiology of pancreatitis in these cases was hypercalcemia due to hyperparathyroidism and PIH. In the first patient (case 11) the serum concentration of total calcium and parathormone was 23.2 mg/dL and 1833 pg/mL, respectively at the time of admission. She had no previous history of hyperparathyroidism or hypercalcemia. Since the hypercalcemia was refractory to saline hydration and calcitonin, hemofiltration was required to reduce calcium levels. The patient died due to ARDS and acute renal failure which developed on the third day of admission. Therefore, we could not investigate the cause of hyperparathyroidism. In the second patient (case 12) who underwent cesarean section with an indication of HELLP syndrome and unfavorable cervix, a diagnosis of pancreatitis was made by laboratory tests and computerized tomography (CT) the day after delivery. As the severe abdominal pain persisted, laparotomy was performed to rule out the other complications of cesarean section and pancreatic necrosis was diagnosed at the time of surgery. Necrosis was considered to be sterile (confirmed by cultures later), thus, a prophylactic drain was inserted and imipenem prophylaxis was started. The clinical course of the patient deteriorated rapidly within three days, progressing to multiple organ failure and death.

At the time of writing, one patient (case 14) was at 34 weeks of gestation and doing well. The frequency of preterm delivery was 61.5% (8/13). The cause of preterm delivery was premature rupture of the membranes (case 13), preterm labor (cases 2 and 7), fetal distress (case 3, 5, 9 and 11), and HELLP syndrome (case 12). Of the 7 mild pancreatitis cases, 4 deliveries occurred beyond 37 weeks and only one delivery occurred before 34 weeks. Of the 6 severe pancreatitis cases, only one delivery occurred beyond 37 weeks and 3 occurred before 34 weeks. Of the 13 deliveries, 3 (23%) were vaginal and 10 (76.9%) were cesarean. The indications for cesarean section were fetal distress (case 3, 5, 6, 8, 9 and 11), footling breech presentation (case 2), dystocia (case 13), previous cesarean section (case 7), and HELLP syndrome and unfavorable cervix (case 12). No fetal or neonatal deaths were observed.

DISCUSSION

In this study, the frequency of pancreatitis was 1 in 1451 births, being consistent with the literature. Pancreatitis is reported during any trimester, but in more than half of the cases it occurs in the third trimester (13). Our findings were in agreement with this data, since in 78.6% of our patients, pancreatitis was diagnosed during the third trimester of pregnancy. The etiology of acute pancreatitis was gallstone in half and hypertriglyceridemia in almost a quarter of the cases, which was in line with the literature. However, unlike previous reports, we did not have any alcohol-related cases, which might be a result of cultural difference. On the other hand, our series included two very rare etiologies, namely hyperparathyroidism and PIH.

In the present study, the maternal mortality rate was higher than the recent reports. This might be a result of infrequent etiologies with unfavorable prognosis which constitute a higher proportion of cases in this study compared with the other series (14, 15). In contrast to the biliary and hyperlipidemic etiologies, the scarcity of clinical experience with the course and management of pancreatitis

secondary to hyperparathyroidism and PIH may have contributed to the high rate of mortality in these cases. In patients with hyperparathyroidism, together with high levels of serum calcium, pancreatic inflammation is severe and the complication rate is high (16). The clinical course of our patient with hyperparathyroidism deteriorated rapidly and progressed to multiple organ failure within a few days. Preeclampsia is associated with alteration in pancreatic vasculature leading to pancreatitis, which may result in organized pancreatic necrosis, as with the case in this study (10). Necrotizing pancreatitis is the severe form of acute pancreatitis and its natural course progresses in two phases (17). The first phase, which lasts 14 days, is characterized by the systemic inflammatory response syndrome (SIRS) resulting in organ dysfunction, while the second phase is dominated by sepsis-related complications due to the infection of pancreatic necrosis (17). None of the prophylactic intravenous antibiotics, except for imipenem, have been shown to be effective in reducing infected pancreatic necrosis and mortality in patients with acute necrotizing pancreatitis (18, 19). Percutaneous aspiration is recommended for the patients with suspected infected necrotic pancreas, while laparotomy and necrosectomy are performed once the diagnosis of infection in necrosis is established (18, 20). Since the necrosis was sterile in our patient, we started imipenem prophylaxis, but the patient was lost due to multiple organ failure within a few days.

The incidence of preterm delivery and perinatal death in pregnancies complicated with pancreatitis is higher than for the general population and most of the adverse perinatal outcomes are related to prematurity (11). Our rate of preterm delivery (61.5%) was much higher than the studies of Eddy et al. and Hernandez et al. where the corresponding rates were reported to be 31.2% (27/84) and 11.7% (4/34) (14, 15). However, the proportion of severe disease was not noted in these studies. In our study, nearly half of the patients had severe pancreatitis, and in contrast to the mild pancreatitis group, where more than half of the deliveries (4/7) occurred beyond 37 weeks, in the severe pancreatitis group almost all deliveries (5/6) occurred prematurely. This finding is consistent with the study of Geng et al. where 6 fetal losses, 7 preterm deliveries and 5 term deliveries were recorded among 18 severe pancreatitis patients (21). Also in the study of Sun et al., (22) the term delivery rate in the mild acute pancreatitis group was found to be significantly higher than in the severe acute pancreatitis group. In light of these findings, prematurity seems to be related with the severity of pancreatitis and the high rate of preterm delivery in our study can be explained by the high rate of severe disease. In the present series, the majority of preterm deliveries occurred because of fetal distress and spontaneous preterm labor. Hypovolemic shock, hypercoagulable state and inflammation due to pancreatitis can cause a significant decline in placental perfusion leading to fetal distress. Also, pancreatitis may directly irritate the uterus and lead to premature contractions (21). Furthermore, early delivery is considered when homeostatic conditions cannot be achieved rapidly, because termination of pregnancy lowers intrabdominal pressure, facilitates resuscitation of the mother, allows more aggressive treatment and may prevent fetal distress enhancing fetal survival (4, 22). Despite the high incidence of prematurity in our study no perinatal death was observed, which can be attributed to the close fetal monitorization and the high quality neonatal care.

Spontaneous vaginal delivery is the preferred route of delivery in women with pancreatitis, because cesarean section may cause penetration of the accumulated fluids resulting in contamination, intraperitoneal leakage and sepsis (23). Also, hypotension during cesarean delivery can exacerbate pancreatitis due to hypoxia (4). However, in case of a large pseudocyst, cesarean section is recommended to prevent rupture (24). In our series, the majority of fetuses (76.9%) were delivered via cesarean section and the indication was fetal distress in most of the cases. This high rate of fetal distress and cesarean section may be explained by the high rate of severe disease which causes a decrease in placental perfusion, as mentioned previously.

There are several limitations of the present study. Firstly, the frequency of pancreatitis in our cohort does not exactly reflect the incidence in the population, because our unit is a tertiary referral center where a multi-diciplinary approach is available. The other limitation is the small size of the study group which is a result of the rarity of the diesase. On the other hand, the strength of this study comes from the variety of different etiologies including the very rare causes such as PIH and hyperparathyroidism.

CONCLUSION

This study emphasizes the progressive and fulminant course in severe disease, especially in hypercalcemia and PIH induced pancreatitis. Acute pancreatitis causes preterm delivery in most of the cases, as a result of fetal compromise and premature uterine contractions associated with the severity of the disease.

Conflict of interest

The authors declare that there are no conflicts of interest.

REFERENCES

1. Whitten AE, Lorenz RP, Smith JM. Hyperlipidemia-associated pancreatitis in pregnancy managed with fenofibrate. Am J Obstet Gynecol 2011; 117: 517-9.
2. Hua-ping L, Ya-jaun H, Xuan C. Acute pancreatitis in pregnancy: a 6-year single center clinical experience. Chin Med J 2011; 14: 2771-5.
3. Stimac D, Stimac T. Acute pancreatitis during pregnancy. Eur J Gastroenterol Hepatol 2011; 23: 839-44.
4. Choy CMY, Tam WH, Leung TN. Gestational hyperlipidaemic pancreatitis. BJOG 2002; 109: 847-8.
5. Crisan LS, Steidl ET, Rivera-Alsina ME. Acute hyperlipidemic pancreatitis in pregnancy. Am J Obstet Gynecol 2008; 198: 57-9.
6. Papadakis EP, Sagianni M, Mikhailidis DP, Mamopoulos A. Acute pancreatitis in pregnancy: an overview. European J Obstet Gynecol Reprod Biol 2011; 159: 261-6.
7. Al-Haddad M, Wallace MB. Diagnostic approach to patients with acute idiopathic and recurrent pancreatitis, what should be done? World J Gastroenterol 2008; 14: 1007-10.
8. Badja N, Troche G, Zazzo JF, Benhamou D. Acute pancreatitis and preeclampsia-eclampsia: a case report. Am J Obstet Gynecol 1997; 176: 707-9.
9. Opatrny L, Michon N, Ray E. Preeclampsia as a cause of pancreatitis: a case report. J Obstet Gynaecol Can 2004; 26: 594-5.
10. Parmar MS. Pancreatic necrosis associated with preeclampsia-eclampsia. JOP 2004; 5: 101-4.
11. Altun D, Eren G, Cukurova Z, Hergünsel O, Yasar L. An alternative treatment in hypertriglyceridemia-induced acute pancreatitis in pregnancy: Plasmapheresis. J Anaesthesiol Clin Pharmacol 2012; 28: 252-4.
12. Ranson JH, Rifkind KM, Roses DF, Fink SD, Eng K, Spencer FC. Prognostic signs and the role of operative management in acute pancreatitis. Surg Gynecol Obstet 1974; 139: 69-81.

13. Nanda S, Gupta A, Dora A, Gupta A. Acute pancreatitis: a rare cause of acute abdomen in pregnancy. Arch Gynecol Obstet 2009; 279: 577-8.
14. Hernandez A, Petrow MS, Brooks DC, Banks PA, Ashley SW, Tavakkolizadeh A. Acure pancreatitis and pregnancy: A 10-year single center experience. J Gastrointest Surg 2007; 11: 1623-7.
15. Eddy JJ, Gideonsen MD, Song JY, Grobman WA, O'Halloran P. Pancreatitis in pregnancy: a 10 year retrospective of 15 midwest hospitals. Obstet Gynecol 2008; 112: 1075-81.
16. Smith MD, Pawlak M, Pantanowitz DP, Botha RJ. Hyperparathyroidism and chronic pancreatitis. S Afr J Surg 1999; 37: 12-4.
17. Büchler MW, Gloor B, Müller CA, Friess H, Seller CA, Uhl W. Acute necrotizing pancreatitis: Treatment strategy according to the status of infection. Annals of Surgery 2000; 232: 619-26.
18. Ducarme G, Chatel P, Alves A, Hammel P, Luton D. Management of necrotizing pancreatitis in the third trimester of pregnancy. Arch Gynecol Obstet 2009; 279: 561-3.
19. Villatoro E, Mulla M, Larvin M. Antibiotic therapy for prophylaxis against infection of pancreatic necrosis in acute pancreatitis. Cochrane Database Syst Rev 2010; CD002941.
20. Amico EC, Canedo LF, Machado CC, Faria SG, Vivas DV. Conservative treatment of pancreatic necrosis with suggestive signs of infection. Clinics 2005; 60: 429-32.
21. Geng Y, Li W, Sun L, Tong Z, Li N, Li J. Severe acute pancreatitis during pregnancy: Eleven years experience from a surgical intensive care unit. Dig Dis Sci 2011; 56: 3672-7.
22. Sun L, Li W, Geng Y, Shen B, Li J. Acute pancreatitis in pregnancy. Acta Obstet Gynecol Scand 2011; 90: 671-6.
23. Hua-dong Q, Jian-guo Z, Chuan-le L, Xiao-ming Z. Three cases of hyperlipidemic pancreatitis in late pregnancy. Chin Med J 2004; 117: 314-5.
24. Eddy JJ, Lynch GE, Treacy DE. Pancreatic pseudocysts in pregnancy: a case report and review of the literature. J Perinatol 2003; 23: 69-72.