Timing of cholecystectomy after acute severe pancreatitis in pregnancy

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Abstract

Acute pancreatitis is one of the most common diseases of the gastrointestinal tract and is usually caused by gallstones; its occurrence in pregnancy is rare. Cholecystectomy for biliary pancreatitis during pregnancy is unavoidable, but its timing is controversial. We herein present the case of a patient who underwent termination of pregnancy due to deteriorated acute severe pancreatitis during the 27th week of gestation. Cholecystectomy was performed because of the relapse of acute biliary pancreatitis 10 days after being discharged. The interval from pancreatitis to cholecystectomy varies with its severity; in mild pancreatitis the interval may be one week, but in severe cases it maybe up to three weeks. Because pancreatitis may relapse during this interval, as occurred in the present case, a better solution for the timing of cholecystectomy must be sought.

Keywords: acute pancreatitis, biliary, cholecystectomy, gallstone, pregnancy, timing

Introduction

Acute pancreatitis is classified as mild, moderate, and severe. Mild pancreatitis is the most common presentation and does not involve organ failure or any local or systemic complication, usually subsiding in about one week. In mild acute pancreatitis, cholecystectomy should be performed during the same admission. Moderate acute pancreatitis is defined by the presence of transient organ failure (i.e. lasting less than 48 h), local complications and/or exacerbation of co-morbidity. With persistent organ failure (lasting more than 48 h), acute pancreatitis is considered as severe (1). For patients who develop severe acute biliary pancreatitis, especially with necrotising pancreatitis, a complex decision must be made regarding the timing of cholecystectomy (2). In these patients, cholecystectomy should either be delayed in case of prolonged admission, performed along with surgical treatment for pancreatic necrosis, or conducted following hospital discharge, as was the case herein (3).

Case report

A 17-year-old female patient, without any family history of gallstones or any underlying haemolytic disease, presented with epigastric and right upper quadrant (RUQ) pain associated with nausea and vomiting. Clinical findings on presentation were: blood pressure, 100/60 mmHg; Pulse Rate (PR): 88/min and Oral Temperature (OT): 37 °C. Upon abdominal examination, a midline caesarean section scar with generalised tenderness was found. Tenderness was more in the RUQ and epigastrium with guarding detected. Her surgical history included a caesarean section 19 days prior, during the 27th week of gestation of her first pregnancy, following two days of severe epigastric pain accompanied by nausea and vomiting. Magnetic resonance cholangiopancreatography (MRCP) on the day of the caesarean section showed acute pancreatitis with ascites and left pleural effusion with gallstone and a normal biliary tree. The abdominal computed tomography (CT) scan with IV and oral contrast showed the same findings as the MRCP after five days. She was discharged and followed-up by the surgical outpatient department for the appropriate timing of laparoscopic cholecystectomy. She had no pain until the day when she was readmitted and her abdominal ultrasonography showed a contracted gallbladder with several gallstones and a normal biliary tree. She was diagnosed with mild biliary pancreatitis; following MRCP, she underwent laparoscopic cholecystectomy and was discharged.

Discussion

Cholecystectomy, after an appropriate interval, is necessary following an episode of acute biliary pancreatitis; however, its relapse
may occur during the recommended interval. This was the case for our patient, who presented with relapsed acute pancreatitis 10 days after being discharged, as indicated by severe and constant upper abdominal pain and the laboratory test results (Table 1), which included two out of three criteria for acute pancreatitis (2). Further, because abdominal CT or magnetic resonance imaging (MRI) tests should be performed in patients who have not improved after 48–72 h (2), MRCP was performed. MRCP findings showed mild ascites, a swollen pancreas with associated surrounding fat stranding, a contracted gallbladder with several filling defects (small gallstones), and a normal biliary tree, thus confirming acute pancreatitis (1). Because acute pancreatitis was considered to be mild due to the absence of organ failure and local or systemic complications, laparoscopic cholecystectomy was performed during the same admission. The patient’s original presentation of acute pancreatitis was considered severe due to persistent systemic inflammatory response syndrome lasting more than 48 h, and led to termination of pregnancy (4).

Acute pancreatitis in pregnancy (APIP) can be caused by gallstones, alcohol abuse, idiopathic hyperlipidaemia, and, less commonly, hyperparathyroidism, trauma, medication and fatty liver of pregnancy. Hyperlipidaemia was ruled out following a triglyceride level of 217 mg/dL. Management of gallstone pancreatitis is controversial (5). The principle of therapeutic regimens for APIP is in accordance with that for non-pregnant women. Termination of pregnancy and conservative management of acute pancreatitis has been proved to be effective (6). Contrary to aggravating severe acute pancreatitis, moderate acute pancreatitis does not indicate the need for pregnancy termination nor caesarean section. A pregnancy termination is performed to remove organ compression without taking the foetus into consideration. For APIP, most scholars advocate non-surgical treatment except when there is i) pancreatic abscess or infected effusion; ii) an association with other serious complications such as gastrointestinal perforation; or iii) a deterioration after active treatment for 2 to 3 days (4). Endoscopic retrograde cholangiopancreatography is indicated in severe biliary pancreatitis with cholangitis and/or with evidence of common bile duct obstruction (7); none of these were found in our patient.

Termination of pregnancy was considered due to an accompanying differential diagnosis of concomitant partial Hemolysis Elevated Liver Enzymes Low Platelet (HELLP) masquerading the picture. There is currently no consensus for the diagnosis of partial HELLP; if two out of the four criteria for the diagnosis of complete HELLP present, namely the presence of haemolysis, an

### Table 1: Laboratory parameter

<table>
<thead>
<tr>
<th></th>
<th>30.09.2013</th>
<th>04.10.2013</th>
<th>5.10.2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC (PMN %)</td>
<td>8700 (76%)</td>
<td>8900 (62%)</td>
<td>12200 (86%)</td>
</tr>
<tr>
<td>Hb (g/dL)</td>
<td>10.6</td>
<td>10.7</td>
<td>11.4</td>
</tr>
<tr>
<td>Plt (mm³)</td>
<td>454000</td>
<td>293000</td>
<td>328000</td>
</tr>
<tr>
<td>AST (IU/L)</td>
<td>500</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>ALT (IU/L)</td>
<td>325</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>LDH (IU/L)</td>
<td>497</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Total Bilirubin (mg/dL)</td>
<td>3.2</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Indirect Bilirubin (mg/dL)</td>
<td>0.8</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Amylase (IU/L) blood</td>
<td>502</td>
<td>73</td>
<td>–</td>
</tr>
<tr>
<td>urea (mg/dL)</td>
<td>7</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Cr (mg/dL)</td>
<td>0.9</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>PT (sec)</td>
<td>12.1</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>PTT (sec)</td>
<td>30</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Alp (sec)</td>
<td>1500</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

**Abbreviation:** WBC = white blood cell; Hb = Hemoglobin; Plt = Platelet; AST = aspartate aminotransferase; ALT = alanine transaminase; LHD = lactate dehydrogenase; Cr = creatinase; PT = prothrombin time; PTT = partial prothrombin time; Alp = alkaline phosphate.
elevated level of lactate dehydrogenase, total bilirubin > 1.2 mg/dL and an elevated alanine aminotransferase greater than two-fold, then partial HELLP is diagnosed (8). After more than 48 h from the onset of pain, she developed a blood pressure of 135/80, a platelet count fall to 53,000, an increment in lactate dehydrogenase (LDH) to 2171 mg/dL, a schistocyte of 3.5%, polychromasia of 2% with haemoglobin of 9.5 g/dL (vs. 12.3 at admission) and bilirubin of 2.2 mg/dl; therefore, the possibility of partial HELLP was considered. Based on the recommendations advising postponement of cholecystectomy to at least 3 weeks following the presentation of acute severe pancreatitis due to the risk of increased infection, her cholecystectomy was postponed (3). It is noteworthy that, based on a previous study, her pregnancy could have continued to term (9).

Conclusion
Because the unavoidable recommended delay in cholecystectomy after a severe acute biliary pancreatitis presentation may result in its relapse, as in the case herein, endoscopic retrograde cholangiopancreatography and sphincterotomy may help the follow-up during this interval, although these are not yet recommended.

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Conception and design, analysis and interpretation of the data, critical revision of the article for the important intellectual content, final approval of the article, provision of study materials or patient: AZ, MTB, AM
Drafting of the article: AZ, MTB
Collection and assembly of data: AM

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