Case Report

Pancreatic Pseudocyst After Acute Organophosphate Poisoning

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Abstract

Acute organophosphate poisoning (OP) shows several severe clinical symptoms due to its strong blocking effect on cholinesterase. Acute pancreatitis is one of the complications associated with acute OP, but this association still may not be widely recognized. We report here the case of a 73-year-old man who had repeated abdominal pain during and after the treatment of acute OP. Hyperamylasemia and a 7-cm pseudocyst in the pancreatic tail were noted on investigations. We diagnosed pancreatic pseudocyst that likely was secondary to an episode of acute pancreatitis following acute OP. He was initially treated with a long-term intravenous hyperalimentation, protease inhibitors and octreotide, but eventually required surgical intervention, a cystgastrostomy. Acute pancreatitis and hyperamylasemia are known to be possible complications of acute OP. It is necessary to examine and assess pancreatic damage in patients with acute OP.

Key words: organophosphate poisoning, acute pancreatitis, pancreatic pseudocyst, hyperamylasemia, cystgastrostomy

Introduction

Organophosphate compounds are widely used as agricultural chemicals, but they have a high degree of toxicity for humans. Because these agents have a strong blocking effect on cholinesterase, toxic exposure results in several kinds of severe clinical symptoms which are caused by an increase in cholinergic activities. Though hyperamylasemia and acute pancreatitis is not rare complications of acute organophosphate poisoning (OP)\textsuperscript{4)–9)}, the association between acute pancreatitis and acute OP still may not be widely recognized in general. We present here a case of a pancreatic pseudocyst associated with acute OP. In this case, because of the occurrence of a severe pancreatic pseudocyst as a complication of acute pancreatitis, he was initially treated medically with a long-term intravenous hyperalimentation, protease inhibitors and octreotide but eventually required surgical intervention, a cystgastrostomy. Therefore, it is important to examine and assess pancreatic damage in patients with acute OP.

Case Report

A 73-year-old man was admitted to our hospital because of repeated upper abdominal pain. In October 2001, he was found in
a state of coma in his home and was transferred to the local emergency hospital. He had respiratory arrest, sweating, hyper-salivation, pin-point pupils, and a foul smell on his breath. The laboratory findings in the emergency hospital (Table 1) showed a decreased level of cholinesterase, 8 U/l (normal range 203–460 U/l), hyperamylasemia, 2000 U/l, and hyperglycemia, 366 mg/dl. After admission to the hospital, organophosphate compounds were detected in the patient’s urine and gastric juices. Because of these clinical symptoms and laboratory findings, he was diagnosed with acute OP. The reason why he took the organophosphate was unclear. He was successfully treated by atropine and pralidoxime iodide (PAM), however, during this period the cause of hyperamylasemia was not fully examined. During the admission and after discharge from this hospital, he continuously complained of upper abdominal pain. So, he visited his family doctor’s clinic and his family physician pointed out a pseudocyst in the pancreatic tail. He was admitted to our hospital for further management in February 2002.

Physical examination revealed tenderness in upper abdomen. He had no history of alcohol abuse, pancreatitis, diabetic mellitus, and medications induced pancreatitis prior to the poisoning episode. Laboratory data (Table 2) showed slight pancytopenia and an increase in serum pancreatic amylase, 105 U/l (normal range 10–65 U/l), but serum lipase and elastase-1 were within the normal range. Abdominal ultrasonography revealed a 7-cm pseudocyst in the pancreatic tail (Fig. 1). Abdominal computed tomography showed a pseudocyst in the pancreatic tail (Fig. 2). Endoscopic retrograde pancreatography showed that the tail lesion of the main pancreatic duct was narrowed and that a contrast medium from this lesion leaked into the pseudocyst (Fig. 3). From these findings, we diagnosed a pancreatic pseudocyst in the pancreatic tail that likely was the end-result of an episode of acute pancreatitis, and we consider that he suffered pancreatic damage from acute pancreatitis that was failed to notice at the time of acute OP and later resulted in pseudocyst formation. We started a medical treatment with an intravenous hyperalimentation and protease inhibitors. Though this management was continued for one month, reduction in the cyst was not observed. So with the purpose of inhibiting the pancreatic exocrine secretion, we treated with octerotide, a somatostatin analogue, at the dose of 200 µg/day for next one month. Though the hyperamylasemia was in a normal range, the size of the pseudocyst was unchanged. Therefore, we performed endoscopic ultrasonography-guide pseudocyst drainage from the gastric wall, but this trial was not successful. Lastly, the patient underwent the surgical operation for cystgastrostomy. After the surgical operation, the pseudocyst was rapidly reduced (Fig. 4). The patient had no postsurgical complications and no recurrence of acute pancreatitis or a pancreatic pseudocyst.
Table 2 Laboratory data on admission to our hospital

<table>
<thead>
<tr>
<th>Urinalysis</th>
<th>Serum chemistry</th>
<th>Pancreatic Enzyme</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein (−)</td>
<td>T.P. 7.0 g/dl</td>
<td>p-Amylase 105 U/l</td>
</tr>
<tr>
<td>O.B. (−)</td>
<td>Alb 3.8 g/dl</td>
<td>Lipase 26 U/l</td>
</tr>
<tr>
<td>Sugar (−)</td>
<td>T.Bil 0.6 mg/dl</td>
<td>Elastase-1 191 ng/dl</td>
</tr>
<tr>
<td>Stool</td>
<td>AST 32 U/l</td>
<td></td>
</tr>
<tr>
<td>O.B. (−)</td>
<td>ALT 18 U/l</td>
<td></td>
</tr>
<tr>
<td>Blood cell count</td>
<td>LDH 188 U/l</td>
<td></td>
</tr>
<tr>
<td>WBC 3080 /μl</td>
<td>ALP 237 U/l</td>
<td></td>
</tr>
<tr>
<td>RBC 363×10⁴/μl</td>
<td>γ-GTP 13 U/l</td>
<td></td>
</tr>
<tr>
<td>Hb 11.1 g/dl</td>
<td>LDH 188 U/l</td>
<td></td>
</tr>
<tr>
<td>Ht 33.9 %</td>
<td>BUN 13 mg/dl</td>
<td></td>
</tr>
<tr>
<td>Plt 12.1×10⁴/μl</td>
<td>Cr 0.82 mg/dl</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Glucose 103 mg/dl</td>
<td></td>
</tr>
<tr>
<td></td>
<td>CRP 0.08 mg/dl</td>
<td></td>
</tr>
</tbody>
</table>

Fig. 1 Abdominal ultrasonography revealed a 7-cm pseudocyst in the pancreatic tail and detected a hyperechoic structure in the cyst that was suspected septum and debris in the cyst.

Fig. 2 Abdominal computed tomography revealed a pseudocyst in the tail region of the pancreas.

Fig. 3 Endoscopic retrograde pancreatography revealed narrowing and a leakage of the contrast medium from the tail lesion of the main pancreatic duct (black arrow). The pseudocyst was slightly enhanced (white arrowhead). Most of the pancreatic duct branches were irregularly dilated.

Fig. 4 After the surgical operation for cystgastrostomy, the pseudocyst in the pancreatic tail was reduced.
Discussion

Organophosphate compounds are frequently used as insecticides in agricultural areas. They are considered more dangerous to humans than any other agricultural chemicals. Due to their high degree of toxicity, clinical manifestations include hypersalivation, abdominal pain, nausea, vomiting, diarrhea, abnormal defecation and urination, muscle fasciculation, bradycardia, and hypotension. In severe cases, seizures, respiratory failure, shock, and death may result. In this case, the patient when first admitted to the local emergency hospital showed typical severe manifestations resulting from acute OP. The detection of organophosphate compounds in the patient’s urine and gastric juices led to the diagnosis. Immediate treatment with atropine for acute OP, which can block the activity of acetylcholine, was dramatically effective, but the hyperamylasemia and repeated abdominal pain were not fully examined and not treated at that time.

Since Dressel et al. described the first case report of acute pancreatitis and pancreatic pseudocyst as a complication of acute OP in 1979, hyperamylasemia and acute pancreatitis as a complication of organophosphate intoxication have been infrequently addressed. The several possible pathogenic mechanisms suggested that the pancreatic insult in organophosphate intoxication is excessive cholinergic stimulation of the pancreas and the Oddi’s muscle. This excessive stimulation shows an increase of pancreatic exocrine secretion and contraction of Oddi’s muscle, and then leads to pancreatic ductular hypertension. Moreover, organophosphate like echothiophate, which inhibit the two cholinesterase isoenzymes in human pancreas (acetylcholinesterase and butyrylcholinesterase), further increase the sensitivity of the pancreas to acetylcholine. Although these phenomena seem to cause acute pancreatitis associated with acute OP, in our patient the initial pulmonary arrest might have added hypoxic insult to the pancreas, and contribute to the development of overt pancreatic damage. Previous reports showed that acute hypoxemia might have caused non-specific salivary-type hyperamylasemia. It is likely to be the possibility that the initial conditions in our patient, especially pulmonary arrest and hypersalivation, might have induced the salivary-type hyperamylasemia, however, the appearance of a pancreatic pseudocyst after acute OP was strongly supported the existence of pancreatic damage in our patient.

In several reviews reported the incidence of hyperamylasemia and acute pancreatitis in adult patients of acute organophosphate poisoning. These data show that hyperamylasemia is a common complication of acute OP. And acute pancreatitis is com-

Table 3 Reports of the incidence of hyperamylasemia and acute pancreatitis in adult patients of acute organophosphate poisoning

<table>
<thead>
<tr>
<th>Total number of patients in OP</th>
<th>Hyperamylasemia</th>
<th>Acute pancreatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dagli &amp; Shaikh (1983)</td>
<td>75</td>
<td>42 (56%)</td>
</tr>
<tr>
<td>Lee WC (1998)</td>
<td>121</td>
<td>44 (36.4%)</td>
</tr>
<tr>
<td>Sahin I (2002)</td>
<td>47</td>
<td>15 (31.9%)</td>
</tr>
<tr>
<td>Martin Rubi JC (1996)</td>
<td>506</td>
<td>ND</td>
</tr>
<tr>
<td>Brahmi N (2005)</td>
<td>70</td>
<td>ND</td>
</tr>
</tbody>
</table>

OP: Organophosphate Poisoning, ND: not determined.
Pancreatic pseudocyst after acute OP

paratively a rare complication at the rates of 0.6–12.8% in acute OP. On the other hand, Weizman and Sofer reported the incident rate of acute pancreatitis after acute OP in children. This report showed higher rate, 29.4% (5/17), than the adult’s rates and suggested that the infant pancreatic acinar cell and/or Oddi’s muscle are likely to be more sensitive than adult’s one to organophosphate.

Acute pancreatitis is comparatively a rare complication in acute OP, however, its severities and clinical courses are unclear. Dagli and Shaikh suggested that acute OP could cause mild and transient pancreatitis because the elevated amylase levels returned to normal within 3–4 days. But other case reports showed severe necrotizing pancreatitis caused by acute OP. And, interestingly, Lankisch et al. reported two cases of painless acute hemorrhage pancreatitis subsequent to organophosphate intoxication. From these reports, it might be clearly suggested that acute pancreatitis associated acute OP shows several patterns of clinical findings. Meanwhile, acute OP could also cause parotitis, which was shown hyperamylasemia without elevation of serum lipase level. So, it is necessary to be carefully assessed pancreatic damage with the analysis of pancreatic enzymes, especially, fractioning pancreatic amylase, lipase, and abdominal imaging in patients of acute OP.

In the case presented here, the clinical course of the patient showed a high degree of serum amylase level after acute OP, repeated upper abdominal pain and the complication of the pancreatic pseudocyst. From these episodes, we speculated that this patient might have complicated acute pancreatitis, though he was not assessed on the severity of the pancreatitis. As a result of the lack of treatment for the acute pancreatitis, he might have developed the complication of the severe pancreatic pseudocyst.

In conclusion, hyperamylasemia is common with acute OP and patients should be closely monitored for the development of acute pancreatitis during the management of acute poisoning and patients with ongoing or recurrent abdominal discomfort should be evaluated further for pancreatic damage.

Acknowledgment

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References

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有機リン中毒後の膲仮性囊胞

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田中 雅夫3、中野 逸郎3、名和田 新1、高柳 涼一1)

有機リンは強力な抗コリンエステラーゼ活性をもつため、様々な重篤な中毒症状を示す。
急性有機リン中毒の合併症のひとつとして急性脳炎を発症することがあるが、その関係につ
いてはいまだ広く認識されているとは言い難い。今回、我々は急性有機リン中毒後に難治性
の膲仮性囊胞を発症した一例を経験したので文献的考察を含めて報告する。症例は 73 歳、男
性。2001年 10月に急性有機リン中毒にて救急病院に搬送され、アミログン、PAM の使用に
て救命された。同病院の入院時血液検査にて高アミラーゼ血症を認め、さらに入院中からしば
しば上腹部痛を認めるも放置されていた。同病
院退院後、上腹部痛続くため、近医受診。膲に
囊胞性病変認め、精査加療目的にて 2002 年 2
月当科紹介入院となった。入院時血液検査にて
高アミラーゼ血症を認め、各種画像検査にて脳
尾部に約 7 cm の仮性囊胞を認めた。急性有機
リン中毒から急性脳炎を合併した結果、膲仮性
囊胞を発症したと考えた。経食の上、長期間の
中心静脈栄養、蛋白分解酵素阻害剤、アミロ
オチドにて治療を行うも囊胞の縮小は認めず、
結果的に胃囊胞吻合術を必要とした。急性有機
リン中毒に合併する高アミラーゼ血症と急性
脳炎は文献的にも必ずしも稀ではなく、急性有
機リン中毒患者の診療の際には障害の十分
な評価も必要であると考えられた。