Short report

Use of ERCP in the diagnosis of internal pancreatic fistula

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SUMMARY Two cases of internal pancreatic fistula, causing pancreatic ascites, were demonstrated by the use of ERCP. This new technique is a useful way of establishing the diagnosis, and treatment may in the future be determined by the ERCP findings.

Massive ascites occurring as a complication of pancreatic disease is a rare condition (Cameron et al., 1967; Lazaro et al., 1969), and its pathogenesis has not been fully explained (Dowowitz et al., 1974), although Cameron et al. (1969) believe that disruption of the pancreatic duct is an aetiological factor.

We describe two cases of pancreatic ascites in which internal pancreatic fistulae were demonstrated preoperatively using endoscopic retrograde pancreatography (ERCP) (Bohlman et al., 1976).

Case 1

A 31-year-old African male was admitted to a peripheral hospital in July 1975. He complained of progressive, painless abdominal distension, immediate post-prandial vomiting, and a 14 kg weight loss. Three months previously he had received treatment for pulmonary tuberculosis. He had been drinking for the past 10 years.

The patient was cachetic with marked tense ascites. As there was no jaundice or hepatosplenomegaly, tuberculous peritonitis was suspected. Various investigations were carried out and the results obtained are set out in the Table.

Despite repeated paracentesis the ascites accumulated rapidly. The patient was then submitted to surgery on 2 October 1975 and 5000 ml of fluid were aspirated from the peritoneal cavity. There were numerous nodules on the peritoneum and omentum and a solid nodular mass was situated on the body of the pancreas. A biopsy of the omentum showed extensive fat necrosis without evidence of malignancy.

No further surgery was undertaken.

The patient was then transferred to Baragwanath Hospital, Johannesburg, where he remained cachetic and the ascites reappeared. A diagnosis of pancreatic ascites was made, based mainly on the high protein and amylase content of the ascitic fluid (Table).

ERCP was then carried out by one of us (M.S.) and this revealed the presence of an advanced degree of chronic pancreatitis. There was a leak of contrast from the tail of the pancreas into the ascitic fluid (Figure).

A diagnosis of ascites secondary to an internal pancreatic fistula was made, and conservative management, with nasogastric aspiration and alcohol-free parenteral hyperalimentation, was therefore instituted.

When the patient was examined six weeks later the ascites had almost completely disappeared and his general condition had improved markedly. Follow-up showed no recurrence of the ascites and a dramatic improvement of the general condition.

Case 2

A 42-year-old African female presented with a history of painless swelling of the abdomen for four months, a history of heavy ethanol consumption, and marked weight loss. On examination there was a tense ascites. Following paracentesis the fluid accumulated rapidly and, again, the high protein and amylase content of the fluid suggested pancreatic ascites (Table).

ERCP was carried out (M.S.) and this showed the features of chronic pancreatitis, with a leak of contrast into the lesser sac from a ruptured pseudocyst.
Use of ERCP in the diagnosis of internal pancreatic fistula

Table  Special investigations in two patients with pancreatic ascites

<table>
<thead>
<tr>
<th>Case 1</th>
<th>Case 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>At peripheral hospital</strong></td>
<td><strong>At Baragwanath</strong></td>
</tr>
<tr>
<td>Routine blood tests</td>
<td><strong>At Baragwanath</strong></td>
</tr>
<tr>
<td>Blood sugar (mmol/l) (mg%)</td>
<td>Normal</td>
</tr>
<tr>
<td>Serum albumin (mmol/l) (g %)</td>
<td>28.5 (2.85)</td>
</tr>
<tr>
<td>Liver function tests</td>
<td>Normal</td>
</tr>
<tr>
<td>Serum amylase (SC)</td>
<td>900 (N = 8 - 38)</td>
</tr>
<tr>
<td>(g/l)</td>
<td></td>
</tr>
<tr>
<td>Ascitic fluid</td>
<td>Slightly raised alk. phosphatase</td>
</tr>
<tr>
<td>Protein (g/l)</td>
<td>36/0</td>
</tr>
<tr>
<td>(g %)</td>
<td>(3-6)</td>
</tr>
<tr>
<td>Amylase (SC)</td>
<td>1000</td>
</tr>
<tr>
<td>Bacilli</td>
<td>&gt;1000</td>
</tr>
<tr>
<td>Malignant cells</td>
<td>-ve</td>
</tr>
<tr>
<td>Tine test</td>
<td>-ve</td>
</tr>
<tr>
<td>Barium meal</td>
<td>Normal</td>
</tr>
<tr>
<td>Intravenous cholangiogram</td>
<td>Normal</td>
</tr>
<tr>
<td>Liver biopsy</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Figure  The typical changes of chronic pancreatitis are evident in the pancreatogram. Free leakage of contrast is seen at the tail.

The patient, who was never fit enough for surgery, died 13 days after a standard course of conservative management was started. The ERCP findings were confirmed at necropsy.

Discussion

The diagnosis of pancreatic ascites is not based on pathognomonic findings (Donowitz et al., 1974), but the findings of a high level of amylase and protein in the ascitic fluid is strongly suggestive, provided other causes for ascites can be ruled out. Marked weight loss, a high serum amylase, and low serum protein are also commonly found, and Smith et al. (1973) recommended that a paracentesis should be performed in such patients to exclude the condition.

Pancreatic ascites is often due to alcoholism or trauma (Donowitz et al., 1974), and the basic abnormality appears to be a disruption of the pancreatic duct, with or without the formation of a pseudocyst (Cameron et al., 1967).

While these patients are poor surgical risks, there may be some indication for early operation in the trauma cases, but not in the alcoholic type (Donowitz et al., 1974). In the latter group conservative management is advised for four to six weeks, and if this fails surgery may be considered (Smith et al., 1973).

As far as the present authors are aware, only one case of pancreatic ascites, confirmed by ERCP has been described (Bohlman et al., 1976). ERCP, apart from establishing the diagnosis, can be useful in planning the best method of treatment.

References


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