Traditional naturopathic management of acute pancreatitis: A case study

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Abstract
Acute pancreatitis is a painful inflammatory disorder of variable aetiology causing abdominal pain, nausea, emesis, fever and elevated white blood cell count. It is characterised pathologically by pancreatic lytic enzymes activating the inflammatory cascade, causing acute inflammation and destruction of surrounding tissue. Medical intervention and investigation is paramount as the condition can be life threatening. A patient presented with acute and persistent abdominal pain, nausea and eructation with biomedical investigations diagnosing acute pancreatitis. Naturopathic management included the use of simple crude herbal medicines, phytochemically rich foods and traditional principles to manage gastrointestinal sequelae and inflammation. The patient responded quickly and had significantly reduced gastrointestinal pain within hours of initial treatment, and was largely asymptomatic after 72 hours of continuing naturopathic care. Traditional supportive herbal and naturopathic therapies may be an effective management strategy for acute pancreatitis in conjunction with close medical supervision.

Keywords: Acute pancreatitis, case report, case study, naturopathy, herbal medicine.

Introduction
Acute pancreatitis is a serious medical condition typically presenting to hospital emergency departments and carries with it a high mortality rate. It is considered a serious pathophysiological condition as once the pancreas is injured it can release lytic enzymes that can both enter the blood (causing severe shock) and also cause tissue digestion of the surrounding architecture. Over 80% of hospital admissions associated with acute pancreatitis are attributed to biliary tract disease and acute alcohol consumption, with the remaining causes being summarised in Table 1.1.

Table 1.1 Causes of Acute Pancreatitis 1-3

<table>
<thead>
<tr>
<th>Gallstones</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infections (Mumps, Coxsackie B virus)</td>
</tr>
<tr>
<td>Drugs (oestrogens, corticosteroids, azathioprine, furosemide, valproic acid)</td>
</tr>
<tr>
<td>Iatrogenic (ERCP, post-surgical)</td>
</tr>
<tr>
<td>Abdominal trauma</td>
</tr>
<tr>
<td>Hyperlipidaemia</td>
</tr>
<tr>
<td>Pancreatic tumours</td>
</tr>
<tr>
<td>Hyperparathyroidism</td>
</tr>
<tr>
<td>Scorpion bite</td>
</tr>
<tr>
<td>Cardiac surgery</td>
</tr>
<tr>
<td>Idiopathic</td>
</tr>
</tbody>
</table>

The incidence of acute pancreatitis is rising by 5% per year in the US and Europe, believed largely to be due to the increased presentation of biliary pancreatitis, with 1/5 of these patients developing necrotizing pancreatitis, which exhibits a 10-30% mortality rate. This high mortality rate is largely due to infection of the necrotic pancreatic tissue and represents the consequences of mucosal barrier failure, small bowel overgrowth and an inflammatory response leading to bacterial translocation of intestinal flora.

Acute pancreatitis should be a clinical consideration for all patients presenting with acute abdominal pain. Differential diagnoses associated with acute pancreatitis include perforated gastric or duodenal ulcer, dissecting aneurysm, appendicitis, diverticulitis, intestinal obstruction (strangulating), mesenteric infarction, biliary colic and an inferior wall myocardial infarction. Signs and symptoms commonly experienced include severe abdominal pain, which may radiate into the back in 50% of cases due largely to retroperitoneal involvement and can require parenteral analgesia. The pain is classically steady, boring and unrelenting. The patient appears acutely ill and is typically sweating and may have diminished sensorium. Nausea and emesis are common presentations with 20% of patients also experiencing upper abdominal distension, rigidity and tenderness upon physical examination. The patient may present with either hypertension or hypotension with associated tachypnoea. Fever may be present and investigations typically reveal a raised WBC count. While such cases are highly unlikely to present to naturopathic or herbal
The patient awakened at his normal waking time of 4:00 am. He put on percolated coffee and went out and sat for approximately 30 minutes in his outside hot tub. The temperature of the tub was 38.34°C (101 degrees Fahrenheit) and the outside temperature was -1°C (30 degrees Fahrenheit). The patient noticed a dull, constant pain in the epigastric region and upper left abdominal quadrant (4/10) at this time. The pain was associated with waves of nausea. The patient, who is a nurse practitioner, took Zofran ([ondansetron] 8mg oral disintegrating tablet) and then returned to his bedroom and took a nap without eating anything for breakfast. When he awakened an hour later the pain and nausea was more intense (7/10) and he had the urge to “burp” continuously in an effort to relieve the pressure. He took 30 mL of Mylanta (aluminium hydroxide 200mg, magnesium hydroxide 200mg & simethicone 20mg) without any improvement in symptoms. He was concerned he might be having an inferior wall myocardial infarction, so decided that at this point he needed medical assistance and chose not to drive 6 kilometres to the local hospital but rather 1 kilometre to one of the hospital satellite clinics nearby.

Shortly after arrival to the clinic the patient began vomiting green fluid that he described as not as bitter as one would expect after not eating for 12 hours. He was admitted to the clinic and an IV of normal saline was run and a blood draw completed. Presenting blood pressure was 108/60 with a pulse of 92bpm and a temperature of 37.2°C (99 degrees Fahrenheit). The patient was also dehydrated 4.5mcg per pressurized metered dose BID (q 12 hours) and an over the counter probiotic (20 billion organism count) BID since being on the ciprofloxacin.

The lab studies included a CBC (Full Blood Count), Comprehensive Metabolic Panel, serum amylase, lipase and Troponin 1. The results of the lab investigations were as follows:

<table>
<thead>
<tr>
<th>Haematology test results</th>
<th>Test</th>
<th>Test result</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>11.0</td>
<td>3.6 - 10.2 K / mm3</td>
<td></td>
</tr>
<tr>
<td>RBC</td>
<td>5.3</td>
<td>4.1 - 5.6 M / mm3</td>
<td></td>
</tr>
<tr>
<td>HGB</td>
<td>16.5</td>
<td>12.5 - 16.3 ggm / dl</td>
<td></td>
</tr>
<tr>
<td>MCV</td>
<td>91.3</td>
<td>73.0 - 96.2 fl</td>
<td></td>
</tr>
<tr>
<td>MCH</td>
<td>31.4</td>
<td>23.8 – 33.4 pg</td>
<td></td>
</tr>
<tr>
<td>MCHC</td>
<td>34.4</td>
<td>32.3 – 36.0 g / dl</td>
<td></td>
</tr>
<tr>
<td>RDW</td>
<td>13.1</td>
<td>12.1 – 16.2 %</td>
<td></td>
</tr>
<tr>
<td>MPV</td>
<td>7.9</td>
<td>7.4 – 11.4 fl</td>
<td></td>
</tr>
<tr>
<td>Platelet count</td>
<td>217</td>
<td>152 – 348 K / mm3</td>
<td></td>
</tr>
</tbody>
</table>

^ Values and measurements consistent with ranges set in the United States of America.
* Abnormal range detected
Based on these results and history, the attending physician diagnosed classic acute pancreatitis and discharged the patient with instructions including 1 week off work and no alcohol. No imaging or ultrasound was ordered to investigate for gallstones or pseudocyst and no medications were prescribed at this time with the patient being advised to return if symptoms did not ameliorate or if pain became excessive.

Following discharge, the patient was very sedated from the promethazine and had an altered state of consciousness, but managed to drive home to his residence. He slept for the next 36 hours only getting up to drink fluids and void. He got up feeling slightly improved but still complaining of upper left abdominal pain (6/10). Over the next 12 hours the patient developed intermittent abdominal cramps (pain 7/10) and diarrhea associated with high fever (39.5°C) and delirium. Treatment of the fever with 440 mg of naproxen sodium was hindered by lack of absorption due to hypermotility.

29/12/14

The patient was still quite ill and had frequent urges to defecate. He had frequent bouts of explosive diarrhea and hyper flatulence. It was at 10pm that evening that the patient came under the care of a naturopath who had arrived from Australia and was staying at his residence. Upon first examination, the patient was delirious and complained of chills and shaking. Physical examination revealed upper abdominal tenderness, rigidity (guarding) and extensive bloating, but did not show Cullen’s sign (umbilical bruising / oedema around the umbilicus) or Grey Turner’s sign (bruising of the flanks) which could have been suggestive of retroperitoneal haemorrhage. The naturopath monitored the patient throughout the night having only intermittent sleep.

30/12/14

Upon waking the next morning, the patient felt that the original abdominal pain had slightly improved (5/10), but that the diarrhea, intermittent nausea and excessive burping were still marked, along with transient gripping pain from hyper flatulence (6/10). The patient had not eaten anything in several days at this point but was staying hydrated.

Navigating herbal selection in a foreign country is difficult. In this city (population: 60,000) there exists one major health food shop which is largely an organic produce store but did have nutritional and herbal supplements. This being said, the Food and Drug Administration (FDA) does not test the quality of ingredients of natural products in the US, making finding suitable products for dispensation difficult due to lack of standardisation or quality testing. Given that ethanol was likely causative in this case, no ethanolic tinctures or fluid extracts were used, but rather crude herbs in their natural form.

**Investigations**

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**Treatment**

Falling back on traditional foundations, the naturopath employed simple naturopathic philosophies for patient management. Firstly, obstacles to cure such as coffee, stimulants, purified sugar and excessive, large fatty meals were discouraged in favour of a highly nutritious and phytochemically dense diet. To establish conditions...
for health, which is in line with the first therapeutic order, a simple vegetable soup was made with butter, onions, garlic, carrots, celery, cauliflower, broccoli and zucchini and was finished with yellow lentils in filtered water. The base of the soup was rich (3 heaped tablespoons) in Curcuma longa (turmeric, dried powder) for its anti-inflammatory effects, with in-vivo evidence suggestive that in a rat model of experimental pancreatitis, curcumin reduced inflammation via inhibition of NF-kappa-B and activator protein-1.9 Whilst turmeric can exert choleretic activity10 and gallstones had not been ruled out, the practitioner still opted for the importance of a strong anti-inflammatory effect to assist the pancreas and also rectify gastrointestinal (GIT) hypermotility. Further supportive actions of turmeric, including hepatoprotective, hypolipidaemic, antioxidative11 and carminative actions, were also desirable. This combined synergistically with the anti-inflammatory actions of Zingiber officinale (ginger, fresh rhizome x 1 heaped tablespoon) which also exhibits powerful antiemetic, carminative12 and spasmylytic actions.13 Allium sativum (garlic, fresh bulb x 3), like Zingiber officinale, was put in towards the end of cooking to preserve its volatile compounds and carminative, anti-inflammatory14 and antimicrobial effects. Other crude herbs employed within the soup included aromatic digestives and carminatives in the form of Coriandrum sativum (coriander, fresh chopped leaf & grated roots x 1 bunch and dried powder x 2 tablespoons), Cuminum cyminum (cumin, powdered seeds x 1 tablespoon), Illicium verum (star anise, whole fruit x 3), Foeniculum vulgare (fennel, crushed seeds x 1 tablespoon), Ellettaria cardamomum (true cardamom, whole pods x 5) and a small amount of Piper nigrum (black pepper, ground powder x 1/2 teaspoon). The soup was simmered gently for 2-3 hours until the lentils broke down, being stirred regularly.

At mid-afternoon, whilst the soup was being prepared, the patient consumed 2 tablespoons of Ulmus rubra (slippery elm, ground bark) mixed with enough water to form a drinkable solution, and followed by 2 glasses of water. This is in line with the third therapeutic order, supporting weakened or damaged organs, and the fifth order, addressing pathology using specific natural substances.15 Ulmus rubra exerts demulcent and nutritive actions, with large amounts of mucilage soothing irritation of mucous membranes within the gastrointestinal tract.14 This was followed 4 hours later with 2 heaped teaspoons of Plantago ovata (psyllium, dried husks) taken in a similar fashion but with less water in order to check the diarrhoea via its absorbptive properties.12 Both demulcent herbs were taken 2-3 hours away from any ingested medicines due to potential physicochemical interactions based on high mucilage content.16

Later that evening, the soup was salted to taste and served. Within an hour of having a small cup (250mL) of the soup, the patient reported considerable relief of upper abdominal pain (1/10 from 5/10) and complete resolution of the gripping pain (0/10 from 6/10) caused by hyper flatulence. The pre-existing nausea was also completely resolved. The patient retired early and slept for 10 hours after one last half dose (1 tablespoon) of Ulmus rubra and water.

Upon waking the next morning, a very small portion of scrambled eggs with powdered turmeric was served. With colour and energy returning to the patient, a gentle, meandering hike in the local nature reserve was taken to stimulate the vital force and encourage ambulation, and thus fulfilling the second therapeutic order of stimulating the self-healing mechanisms (vis medicatrix naturae).13 The hike was roughly 6km in length and 3 hours in duration, and the patient reported feeling tired but revitalized afterwards. He developed some loose stools later in the day and was treated again with Ulmus rubra bark. An infusion of Zingiber officinale radix (ginger, fresh rhizome) and Matricaria chamomilla flos (German chamomile, dried flowers) was also taken at this time for its anti-inflammatory and spasmylytic activity,17 with the patient’s bowel movements normalising within 3 hours and continuing to be normalised throughout his convalescence. The following day the patient complained of symptoms suggestive of his non-specific prostatitis, but the remainder of his original symptomatology had ameliorated.

Follow up

Consulting with his regular general practitioner (GP) 4 days later, the patient was symptom free and was organising to see an urologist regarding his non-specific prostatitis. The GP did not feel that ultrasound or follow up investigations were needed to monitor the patient’s pancreatitis based on his clinical presentation at this time.

Conclusion

There exists little evidence on potential naturopathic management strategies in the clinical and scientific literature for acute pancreatitis, which may largely be due to it being treated as a medical emergency. Nevertheless, when scientific or clinical evidence is lacking, one can rely upon traditional knowledge to support the patient’s symptoms, deficient body systems and general health. The author wishes to make clear that in no way does this undermine the necessity for close medical supervision in such circumstances as disease progression can be potentially fulminant if infection, necrosis or other sequelae develop.

Of clinical interest, follow up research on the use of probiotics in acute (severe) pancreatitis has suggested an increased risk of mortality.5,18 This is important to consider as many practitioners may consider prescribing probiotics in such a case due to it being beneficial to the gut. More research needs to be carried out in this area, however, until the underlying mechanism is revealed, prophylactic probiotics should be considered potentially unsafe.5
In conclusion, this case is a reminder that traditional naturopathic knowledge and philosophies still form a valid and useful source of evidence, and furthermore, how therapeutically valuable crude herbal medicines can be. This latter observation is easily forgotten and often overlooked in our current herbal profession, which is largely focused on commercially manufactured and standardised dosage forms.

References