Introduction

Pancreatitis is a pancreatic inflammation that includes two types, acute and chronic [1]. Acute Pancreatitis (AP) includes a wide range of diseases, from its low-risk symptoms to a fulminant process with multiple organ damage and high mortality [2].

The incidence of AP has increased over the past 20 years [3]. Common symptoms of AP include nausea and vomiting, and the abdominal pain that mostly spreads to the back. AP has different causes, some of which include: gallstones, alcohol consumption, hypercalcemia, hyperlipidemia, idiopathic, autoimmune, and anatomic (pancreas divisum), infection, tumors and drug-induced. AP can be recurrent. Multiple and recurrent attacks of AP can lead to chronic pancreatitis, which can ultimately lead to pancreatic insufficiency. AP has various complications, one of the most severe complications is necrotizing pancreatitis, which is due to tissue ischemic and necrosis and has high mortality [1]. Here we describe the case of a young woman with acute necrotizing pancreatitis induced by Ginseng compounds administration. Necrotizing pancreatitis has a very poor prognosis and is associated with high mortality (15%), which, if infected, can reach up to 30% to 39%, which is the major cause of mortality [4]. Intervention is generally required for infected pancreatic necrosis and less commonly in patients with sterile necrosis who are symptomatic (gastric or duodenal outlet or biliary obstruction). Fever, elevated white blood cell count, and abdominal pain suggest infection of this fluid, and percutaneous aspiration is confirmatory. Percutaneous drainage and IV administration of antibiotics should be instituted if infection is present.

Case Presentation

The patient is a 29-year-old woman that sent to Sina Hospital, Tehran University for further evaluation and treatment from Ilam. The patient’s problems began with the fever and chills intermittently, and then 2 days later, suddenly suffered abdominal pain in the epigastrium, which was persistent and progressive, gradually spreading to the entire abdomen and not radiating anywhere. The patient’s pain was worse with eating and it was not associated with defecation and 2 days later, the patient developed another symptom. The symptoms included: non-bloody watery diarrhea, nausea and vomiting, and anorexia, and during the last (3-9) months weight loss was 15 kg. There is no history of this problem in the past.

Her past medical history was otherwise negative and she had no allergies. She denied alcohol intake or tobacco use. She has a two-time history of cesarean section, which was last two years ago.

The only drug used recently was weight gain that contains Ginseng compounds that only consume 10 of them and discontinued with the onset of symptoms.

In the family history, cousin’s aunt, he has pulmonary tuberculosis.
On examination she was febrile (T=39.5), with a heart rate of 140 beats/min, blood pressure was 100/70 mmHg and respiratory rate of 18/min.

Abdominal examination revealed tenderness and generalized rebound tenderness in epigastrium. There were non-palpable masses and bowel sounds were normal. Lung examination had bilateral rales.

In Ilam, a CT scan is performed for the patient, in which the free fluid in the abdomen was reported, with a suspicion of perforated appendicitis, patient was under laparotomy that there was an obvious ascites and seeding that the drain was embedded and the omentum and the multiple point sampling that has been examined twice by the pathologist, which has not been malignancy, also, the sample was examined once by a pathologist in Tehran, which was not been malignancy (Figure 1).

After surgery, 2 days were treated with Imipenem, the patient’s leukocytosis was 25000, and bilateral pleural effusion was found and the respiratory distress was found, so transferred to the ICU, and in consultation with the professor of Surgery in Sina Hospital in Tehran has provided double-sided chest tube and has been sent to Tehran by Intubation.

The patient was admitted to Tehran for about 17 days in ICU, who had 10 days of intubation and had 5 days of C-pap, and was extubated and transferred to the ward after 2 days, during which time she was treated with antibiotics. The patient’s leukocytosis was from 25000 to 13000, and transferred to the ward.

According to the history and physical examination findings, differential diagnosis was made for the patient, which was done to achieve the final diagnosis of paraclinical measures; including, complete lab tests, abdominopelvic ultrasound, CXR, CT scan, endoscopy, colonoscopy, and EUS were performed.

Complete Blood Count (CBC diff) was at its onset of 24000 leukocytosis with 80% PMN, rising progressively in the course of the disease, and the highest level of 25000, which was found to be lowered with antibiotic therapy and reached 6160 on the day of discharge.

Patient’s differential diagnosis including: Rheumatologic disorders, TB, brucellosis, malaria and in general infectious diseases, lymphoma, malignancy, pancreatic dysfunction (necrotizing pancreatitis, pancreatic cancer, and autoimmune pancreatitis), thyroid disorders.

For rheumatologic disorders, rheumatology tests were performed completely, all of which were negative. Also, rheumatologic examinations (lymphadenopathy, hair pull test) were performed, which was negative. The patient has no symptoms such as arthritis, arthralgia, oral ulcer and malar rash, hair loss, rash, dry eye, dry mouth, photophobia, psychosis, seizure, and the history of abortion.

PPD test was performed for tuberculosis according to positive ADA (7,4,32), which was negative and the patient was treated with anti-TB drugs (INH, Ethambutol) for 14 days with an infectious professor’s advice, with no change in the patient’s symptoms. The patient had no symptoms including: coughing, hemoptysis, night sweats.

Brucellosis tests were negative. For malaria PBS, it was normal. Viral markers were negative.

For lymphoma and malignancy, tumor markers were checked to be negative, did not have lymphadenopathy and night sweats, and also with suspicion of gastrointestinal cancers, inflammatory bowel disease, endoscopy and colonoscopy were performed. Colonoscopy was normal. Endoscopy was highly suggestive for gastric cancer which was taken biopsy, was chronic gastritis and ulceration, and malignancy was rejected, due to in consistency of endoscopic findings with pathological outcomes, endoscopy was performed again, which, as before, was suspected of stomach cancer, and biopsy, showed only
a ulceration (Figure 2).

About pancreatic dysfunction, amylase and lipase, liver tests, albumin, and urine analysis were checked several times, the procedure was as follows, and the microscopic hematuria was present in three examinations. Other study performed for the evaluation of the pancreas was CT scan (Figure 3), which included pancreatic inflammation that CT scan with pancreatic cut was performed for a more accurate evaluation which revealed a pancreatic tail and body inflammation and brief duct dilatation. According to the patient’s CT scan results, EUS was performed, with a normal pancreas reported, and an emphasis on the atypical Crohn’s disease (Figure 4). Thyroid and renal tests were normal.

Discussion

The most common causes of AP are gallstones alcohol and hyperlipidemia respectively, however, nearly 20% of cases it is idiopathic [5]. The patient described in this report did not drink alcohol and did not have gallstone and had normal liver function test, and normal lipid profile. Thus excluding other etiologies for the AP that in this case was drug-induced (Ginseng compounds).

The diagnosis of AP requires the patient to present with abdominal pain consistent with AP (acute onset of a severe constant epigastric pain which often radiates through to the mid back) and the elevation of serum amylase or lipase (>3 times upper limit of normal). Imaging (usually by contrast enhanced CT scanning) is only required for the diagnosis of AP when these diagnostic criteria are not met [6].

Recovery from AP is now expected, with mortality less than 10%, which reflects improvements in the treatment of complications and intensive care management.

A third of patients with AP develop complications and a quarter of these will die of them. Severe acute pancreatitis is associated with fluid collections and tissue necrosis in and around the pancreas. In this case complication of acute pancreatitis was necrosis.

Necrosis increases the morbidity and mortality risk of AP because of its association with organ failure and infectious complications.

As such, patients with necrotizing pancreatitis may need admission to the Intensive Care Unit (ICU), nutritional support, antibiotics, and radiologic, endoscopic, or surgical interventions.

Nearly 20% of patients with AP develop necrotizing pancreatitis, of which approximately 25% to 70% will develop to infected necrosis [7,8].

Necrotizing pancreatitis is a complication of AP. It may happen when AP is untreated, or treatment is ineffective.

No predisposing factors have been validated to increase the risk of development of pancreatic necrosis, however, the most common etiology of necrotizing pancreatitis is gallstones (40% to 48%), followed by alcohol consumption (24% to 27%).

Two thirds of necrotic pancreatic collections are sterile and will resolve with conservative management, while the remainder will become infected and will require further intervention.

Initial management of necrotizing pancreatitis is a supportive treatment that includes: fluid resuscitation, pain control, resting the gastrointestinal tract [9].

In the setting of necrotizing pancreatitis, the necrosis most commonly involves both pancreatic and peripancreatic fat tissue (75% to 80%).

An early contrast-enhanced Computed Tomography (CT) may fail to reveal necrosis within the first few days; however, a contrast enhanced CT after the first week revealing non enhancing pancreatic parenchyma is considered to be pancreatic necrosis.
Effectiveness of prophylactic antibiotic and antifungal therapy, which prevents infected necrosis, has been questionable. Recent studies, conducted to evaluate the effectiveness of this therapy, have, almost, proven its weaknesses. This therapy is no more recommended by the American College of Gastroenterology and the Infectious Diseases Society of America no longer recommends it. In patients with concomitant cholangitis or extra pancreatic infection, this therapy, is the only case that can be is recommended to be given [10].

If a patient is predicted to have severe pancreatitis or not expected to resume oral intake within 5 to 7 days, early enteral nutrition is recommended. Enteral nutrition most commonly involves bedside or endoscopic placement of a nassojejunal feeding tube and collaboration with a nutritionist to determine protein caloric requirements [10].

However, for patients with higher rates of infection, multi organ dysfunction and failure, and death, total parenteral nutrition is suggested instead of enteral nutrition [10].

The first-line intervention for acute necrotizing pancreatitis is intravenous hydration with isotonic and pH-balanced crystalloid solution such as Lactated Ringer’s [11].

Having provided no supportive data, recent studies have proven that the use of prophylactic antibiotics in cases of severe AP cannot be an effective solution. For patients with severe AP complications, organ failure, and mortality, early enteral nutrition should be the supplanted medication [11].

This case is an atypical presentation of AP with a severe attack leading to pancreatic necrosis. We experienced a case of AP associated with normal serum enzyme levels. Patient was diagnosed based on clinical and radiological evidence. She was successfully treated with intravenous fluids, antibiotic therapy, and radiologic external percutaneous drainage with clinical and laboratory improvement. The importance of this case report is the unlikely presentation of AP. We believe that more research is needed to determine the exact proportion of AP patients who first present with normal serum lipase, since similar cases have been seen in case reports.

**Conclusion**

Patients with AP are susceptible to a wide spectrum of diseases: alcohol abuse, cholelithiasis, infection, lesions, tumors, hypercalcemia, hyperlipidemia, and drug-induced. These diseases range from mild self-limiting symptoms to a fulminant process with multiple organ failure and high mortality. The etiologic factors contributing to AP include a wide range with some being rare. Choledolithiasis or sustained alcohol abuse is the factor which attributes to, approximately, 80% of cases [12-14].

Having stopped the drugs, the symptoms resolved in this patient; this shows that the drug-induced pancreatitis is probably present in her [15]. In various studies, Ginseng compounds have had different effects on the pancreas that in this patient, lead to acute necrotizing pancreatitis.

**References**