Changes of Serum TNF-α in Acute Pancreatitis Patients and Its Clinical Significance

Wenrui Yang*
Department of General Surgery, The People's Hospital of Pinglu District, Shuozhou, Shanxi 036800, China

ABSTRACT Objective: The aimed of this study is to investigate the changes in serum levels of tumor necrosis factor (TNF-α) in the patients with acute pancreatitis treated with octreotide and its clinical significance. Method: Total of 65 patients of acute pancreatitis were selected as a case study, in which 30 patients with mild acute pancreatitis (MAP) and 35 severe acute pancreatitis (SAP) patients were treated with octreotide. 60 healthy subjects as control group and 65 case group was subjected to performed double antibody sandwich enzyme-linked immunosorbent assay (ELISA) to detect the serum levels of TNF-α. Results: The serum TNF-α level in the case group was (12.67 ± 3.45) pg/mL and the control group was (1.56 ± 0.57) pg/mL. Case group was significantly higher than control group (p < 0.05). Serum level of acute pancreatitis (AP) before treatment was (8.96 ± 2.12) pg/mL. After treatment, SAP group was (17.34 ± 4.56) pg/mL, MAP group was significantly lower than SAP group, and the difference was statistically significant (p < 0.05). Conclusion: The serum levels of TNF-α in patient with acute pancreatitis were significantly higher than those of normal healthy people, and their serum level was closely related to the severity of illness.

1. Introduction
Acute pancreatitis (AP) is very a common critical disease in clinical due to various reasons cited intestinal trypsin activation leads to local pancreas tissue inflammation, also can be associated with other organ function [1]. Mild acute pancreatitis (MAP) is one of the most common clinical symptoms of acute pancreatitis. About 30% of untreated MAP could develop to severe acute pancreatitis (SAP) that is critically ill and the prognosis was not completely known for the pathogenesis of acute pancreatitis. However, a number of studies showed that cytokines and inflammatory factors play an important role in the development of acute pancreatitis [2]. The paper presented reliable diagnostic data for early diagnosis, treatment and prognosis of acute pancreatitis (AP) in 65 patients with acute pancreatitis (TNF-α).

2. Materials and methods
2.1. Clinical data
From January 2013 to January 2015, a total of 65 patients of acute pancreatitis receiving treatment, and there were 40 males and 25 females. The mean age was 45.5 ± 13.5 years old (range: 23–67 years). The admission criteria should be consistent with the diagnosis and treatment of acute pancreatitis. Guide to the exclusion of other diseases of the patients with acute abdomen. The patients were not drug therapy and anti-infection drugs allergy. Etiology: 35 cases of gallstone disease, 28 cases of severe drinking, 2 cases of high blood lipids. According to the standard of acute pancreatitis: 30 cases of mild acute pancreatitis (MAP), 35 cases of severe acute pancreatitis (SAP). As a control, 60 healthy subjects in our hospital were selected as the control group, and there were 38 males and 23 females with the mean age of 45.5 ± 14.5 years (aged 26–68 years). Age and gender were not significantly different between control group and case group.

2.2. Treatment method
The 65 cases of diagnosed acute pancreatitis patients were
given Beijing Connaught Pharmaceutical Co., Ltd. the production of acetate octreotide 0.6 mg intravenous infusion in the treatment, the patients with abdominal pain and abdominal distension wait for a symptom to be alleviated, reduced to 0.3. Co-treatment such as fasting, water deprivation, anti-acid and anti-inflammatory symptomatic support treatment, parenteral nutrition support treatment, maintain water and electrolyte balance, to have occurred in patients with liver damage given liver and reducing enzyme treatment for two weeks.

2.3. Detection method
5 mL of peripheral blood was extracted from acute pancreatitis patients after 24 h admission and the control group blood was withdrawn during their physical examination day with the similar volume. The blood sample was run under centrifugation at 4000 rpm for 10 minutes for fractionation. The separated serum was stored in the refrigerator at -80 ºc. Double antibody sandwich ELISA method was employed to detect the serum TNF-α in the participants.

2.4. Observation index
To observe the serum TNF-α level of the patients in the treatment group and the control group before treatment. To observe the serum TNF-α level before and after treatment in the patients with SAP.

2.5. Statistical analysis
Statistical Analysis was performed by SPSS version 15.0 software. Data were measured by $p < 0.05 + s (x)$, the count data were analyzed by $x^2$ test, and the data were statistically analyzed by $t$ test.

3. Results
3.1. Comparison of serum TNF-α level in untreated patients and the control group
The level of serum TNF-α in patients was (12.67 ± 3.45) pg/mL, and the control group was (1.56 ± 0.57) pg/mL and the case group was significantly higher than that of the control group. There were significant differences between the two groups ($p < 0.05$). (Table 1)

### Table 1. Comparison of serum TNF-α level in patients with acute pancreatitis before treatment and control group.

<table>
<thead>
<tr>
<th>Group</th>
<th>Cases</th>
<th>Serum TNF-α (pg/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case group</td>
<td>65</td>
<td>12.67 ± 3.45</td>
</tr>
<tr>
<td>Control group</td>
<td>60</td>
<td>1.56 ± 0.57</td>
</tr>
<tr>
<td>$t$</td>
<td>-</td>
<td>145.39</td>
</tr>
<tr>
<td>$p$</td>
<td>-</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

3.2. Comparison of serum TNF-α levels before and after treatment in the case group
Before treatment, the level of serum TNF-α in the case group was (2.12 ± 3.45) pg/mL, after treatment, the difference was statistically significant, that is, $p < 0.05$, before and after treatment, the difference was statistically significant. (Table 2)

### Table 2. Comparison of serum TNF-α level before and after treatment in the case group.

<table>
<thead>
<tr>
<th>Group</th>
<th>Cases</th>
<th>Serum TNF-α (pg/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before treatment</td>
<td>65</td>
<td>12.67 ± 3.45</td>
</tr>
<tr>
<td>After treatment</td>
<td>65</td>
<td>5.78 ± 2.12</td>
</tr>
<tr>
<td>$t$</td>
<td>-</td>
<td>24.24</td>
</tr>
<tr>
<td>$p$</td>
<td>-</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

4. Discussion
Acute pancreatitis is a common clinical emergency. Its pathogenicity due to intrapancreatic trypsin activation from variety of disease which caused by pancreatic tracheal swelling, hemorrhage and necrosis. The primary clinical symptoms were abdominal pain, fever and nausea and vomiting. Clinical diagnosis could be performed by blood test. The specific symptoms include the following five points: first, in the extreme fatigue or binge eating after the attack, the main symptoms of the upper left abdomen pain, mild pain and gradually increased, classis signs of shock may appear in more severely ill patients. Second, frequent nausea and vomiting symptoms and the symptoms will change as an increase in the severity of the disease. Fourth, fever. Since this disease belongs to the inflammatory disease, it will induce temperature rise. Mild pancreatitis body temperature will be at 39 ºc or lesser and able to reduce fever. The body temperature could reach to 40 ºc and it could be the presence of sepsis. Fifth, necrotic pancreatitis and inflammatory fluid exudation could reach the abdominal subcutaneous tissue, the dissolution of subcutaneous fat causing blood vessels rupture and local skin appear blue purple. Purple colour may appear in the lower abdomen and the navel. In complications, it may lead to systemic complications, including renal failure, acute respiratory failure, sepsis, high blood glucose or heart failure and other symptoms. The performance of acute pancreatitis in patients with mild edema and the disease can be cured by the body itself. Some patients showed pancreatic necrosis and then lead to infection or even shock that lead to death. The factors causing pancreatitis in the current academic community has not been determined, generally considered the following seven factors: first, mechanical obstruction factor. Certain patients may suffer from bile reflux caused by roundworm, stones, etc., into pancreatic acinar and even lead to pancreatic gland rupture, so that the trypsin pancreatic inflammation. Second, alcohol consumption factors. Long term alcohol consumption of people prone to pancreatitis, large amounts of alcohol will promote the secretion of pancreatin and the trypsin will cause pancreatic inflammation. Moreover, presence
of high protein food and alcohol could easily lead to pancreatitis. Third, vascular factors. Stenosis or obstruction of the pancreatic periphery may result in ischemic necrosis of the pancreas. Fourth, trauma factor. Pancreatic trauma does not have suitable treatment, will lead to the rupture of the pancreatic duct, pancreatic fluid spills leading to inflammation. Fifth, bacterial infection. The presence of bacteria or viruses in the blood and the blood flow through the pancreatic tissue could cause pancreatitis. Sixth. Other disease factors. High cholesterol may cause pancreatitis. Seventh, other factors. Such as genetic and drug allergies, etc.

Mild pancreatitis patients only show pancreatic edema and self-limited disease associated with little or no distant organ dysfunction, however it could develop into SAP [3]. Clinical practice showed that SAP disease has rapid changes and the mortality rate could be as high as 40%. At present, it is considered that the occurrence of acute pancreatitis is related to many factors. Pathogenic factors; pancreatic enzymes are activated, pancreatic juice secretion increased, pancreatic duct pressure increased, large gland cell rupture, pancreatin is abnormal activation leading to the leakage of large amount of trypsin that results in pancreatic auto-digestion. In addition, cytokines activated and various inflammatory mediators in the development of acute pancreatitis. In the early stage of acute pancreatitis, pancreatic enzymes and cytokines may enter the peritoneal cavity. It can be absorbed into the blood through many channels, so that the mononuclear macrophages activated and release a variety of cytokines and inflammatory mediators, and causing multi organ failure leading to a series of clinical symptoms and signs. In the present study, the serum level of patients in the case group was significantly higher than that in the control group before treatment, the difference was statistically significant, that is, \( p < 0.05 \).

In summary, TNF-α levels were elevated in the serum of patients with acute pancreatitis, and were closely correlated with the severity of the disease by octreotide treatment can reduce the serum TNF-α levels. Therefore, serum TNF-α levels can be as acute pancreatitis patient assessment and evaluation of curative effect, an important indicator of the prognosis evaluation [4] and octreotide in the treatment of patients with acute pancreatitis, remarkable curative effect, it is worth of clinical application.

References