

Comparative efficacy of octreotide and somatostatin in acute pancreatitis: a controlled trial of inflammatory markers and hospital length of stay.

Qunchao Zhu¹, Tian Jiang², Yan Li³, Sicong Jiang¹, Aifang Li⁴ and Chendong Ma¹

¹Department of Emergency Medicine, The First People's Hospital of Jiashan, Jiashan Hospital Affiliated of Jiaxing University, Jiashan, Zhejiang Province, China.

²Department of Thoracic Oncology, Jiangxi Cancer Hospital, Nanchang, Jiangxi Province, China.

³Department of Endocrinology, Jiujiang Hukou County People's Hospital, Hukou, Jiangxi Province, China.

⁴Department of Hematology, Jiamusi University Affiliated First Hospital, Jiamusi, Heilongjiang Province, China.

Keywords: Acute Pancreatitis; Octreotide; Somatostatin; Inflammation; Hospitalization.

Abstract. Acute pancreatitis is an acute pancreatic injury with multiple etiologies. Octreotide and somatostatin are commonly used treatments, but their clinical efficacy remains controversial. This study compares their effects on inflammatory markers and hospital length of stay. One hundred and twenty patients with acute pancreatitis admitted to The First People's Hospital of Jiashan between January 2022 and December 2024 were retrospectively included and divided into two groups based on treatment modality, namely the control group (somatostatin treatment) and the experimental group (octreotide treatment), with 60 cases in each group. Serum amylase (AMY), serum lipase (LPS), C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor- α (TNF- α), white blood cell count (WBC), serum albumin (ALB) content, procalcitonin (PCT), hospital stay duration, and the incidence of adverse reactions were assessed in both groups. Baseline data for the two groups were comparable, with no statistically significant differences ($p > 0.05$). After seven days of medication, compared with the control group, patients in the experimental group had lower AMY, LPS, CRP, IL-6, TNF- α , WBC, and PCT ($p < 0.05$), a shorter hospital stay ($p = 0.011$), and a lower incidence of adverse reactions ($p = 0.007$). ALB levels in the experimental group were significantly higher than those in the control group ($p = 0.039$). Compared with somatostatin, octreotide shows superior therapeutic effects in acute pancreatitis, alleviating inflammation more effectively, promoting recovery, improving clinical outcomes, and shortening hospital stay. These findings provide a scientific basis for optimizing clinical medication.

Eficacia comparada de octreótido y somatostatina en pancreatitis aguda: ensayo controlado sobre marcadores inflamatorios y duración de la estancia hospitalaria.

Invest Clin 2026; 67 (2): 189 – 204

Palabras clave: Pancreatitis Aguda; Octreótido; Somatostatina; Inflamación; Hospitalización.

Resumen. La pancreatitis aguda es una lesión pancreática de etiología múltiple. El octreótido y la somatostatina son tratamientos habituales para esta condición, aunque su eficacia clínica sigue siendo controvertida. Este estudio compara sus efectos sobre los índices inflamatorios y el tiempo de hospitalización. Se incluyeron retrospectivamente 120 pacientes con pancreatitis aguda ingresados en el First People's Hospital of Jiashan entre enero de 2022 y diciembre de 2024, distribuidos en dos grupos según el tratamiento recibido: grupo control (tratamiento con somatostatina) y grupo experimental (tratamiento con octreótido), con 60 casos en cada grupo. Se evaluaron en ambos grupos la amilasa sérica (AMY), lipasa sérica (LPS), proteína C reactiva (PCR), interleucina-6 (IL-6), factor de necrosis tumoral α (TNF- α), recuento de leucocitos (WBC), albúmina sérica (ALB), procalcitonina (PCT), duración de la estancia hospitalaria e incidencia de reacciones adversas. Los datos basales de ambos grupos no mostraron diferencias estadísticamente significativas, lo que confirma su comparabilidad ($p > 0,05$). Después de 7 días de medicación, en comparación con el grupo control, los pacientes del grupo experimental presentaron niveles más bajos de AMY, LPS, PCR, IL-6, TNF- α , WBC y PCT ($p < 0,05$); la duración de la estancia hospitalaria fue menor ($p = 0,011$); y la incidencia de reacciones adversas fue inferior ($p = 0,007$). Los niveles de ALB en el grupo experimental fueron significativamente más altos que en el grupo control ($p = 0,039$). En comparación con la somatostatina, el octreótido muestra una eficacia terapéutica superior en la pancreatitis aguda, aliviando la inflamación de forma más marcada, favoreciendo la recuperación, mejorando la eficacia clínica y acortando la hospitalización. Estos resultados aportan una base científica para optimizar el tratamiento farmacológico clínico.

Received: 27-01-2026 *Accepted:* 02-03-2026

INTRODUCTION

Acute pancreatitis arises from various etiological factors, leading to acute injury of pancreatic tissues, edema, hemorrhage, and necrosis, to name just a few ¹. It is an auto-digestive disease of pancreatic tissue caused by the abnormal activation of pancreatic enzymes and may lead to dysfunction of other organs ².

Acute pancreatitis usually occurs in adults, with an incidence of 5/100,000 to 30/100,000 per year. The incidence appears to be rising each year. In China, the leading cause of acute pancreatitis is cholelithiasis, with hypertriglyceridemia and excessive alcohol consumption as the next most common causes ³. Early control of the underlying cause can help alleviate the condition, improve prognosis, and prevent recurrence

of acute pancreatitis⁴. Based on severity, acute pancreatitis is classified into three types: mild, moderately severe, and severe. According to pathology, it can be categorized as interstitial edema type and necrosis type⁵. Acute pancreatitis may lead to functional impairment of one or more organs, with respiratory and renal impairment being the most common⁶. In patients with acute pancreatitis, elevated serum amylase (AMY) and lipase (LPS) levels are commonly observed on laboratory testing⁶. Originally isolated from hypothalamic extracts of pigs and sheep, somatostatin has a short biological half-life of approximately 3 minutes, which limits its duration of action in clinical therapy⁷. Common somatostatin analogs include somatostatin tetradecapeptide and octapeptide. Like somatostatin, somatostatin analogs effectively inhibit pancreatic enzyme secretion. However, unlike somatostatin, somatostatin analogs have a longer half-life, and their effects last for a relatively long time⁸. The specific mechanism of action is that, after medication use, somatostatin and its analogs, as the active ingredients, bind to somatostatin receptors on the patient's pancreatic cell surface⁹, which weakens the pancreas's exocrine function, interferes with the release of acetylcholine, and downregulates adenosine release and the activity level of adenylyl cyclase. These effects can help reduce pancreatic duct pressure, inhibit the infiltration of pancreatic fluid into pancreatic tissues, and thereby reduce pancreatic auto-digestive function. In the early stages of the condition, patients' platelet levels are abnormally low, but platelet activity is markedly elevated. Treatment with somatostatin or somatostatin analogs can effectively reduce the patient's platelet activity level and block the abnormal release of platelet-activating factors¹⁰. Abnormal release of platelet-activating factors thus exacerbates the systemic inflammatory response in patients and blocks their disease progression¹⁰. Octreotide is a widely used somatostatin analog that can significantly reduce the release

of growth hormone and pancreatic enzymes, fully relax Oddi's sphincter of the biliary tract, reduce pancreatic duct pressure, prevent pancreatic juice reflux, control pancreatic self-digestion, reduce the secretion of pro-inflammatory factors, and thus alleviate the body's inflammatory response¹¹. Octreotide is a cyclic octapeptide with greater physiological activity. It can inhibit the abnormal release of growth hormone and thyrotropin and reduce the release of gastric acid, glucagon, insulin, and pancreatic enzymes, thereby controlling gastrointestinal and pancreatic endocrine hormone pathology and ultimately inhibiting abnormal secretion in the intestinal tract.

In this study, the therapeutic efficacy of octreotide and somatostatin was compared in patients with acute pancreatitis, primarily using inflammatory markers such as AMY, LPS, C-reactive protein (CRP), interleukin-6 (IL-6), tumor necrosis factor- α (TNF- α), white blood cell count (WBC), and serum albumin (ALB), along with procalcitonin (PCT) and hospital stay duration for a comprehensive efficacy analysis. The aim was to compare the effects of octreotide and somatostatin in treating acute pancreatitis, to develop a more effective and personalized treatment plan for patients with acute pancreatitis, and to advance the discipline and contribute to the dual value of theory and practice.

PATIENTS AND METHODS

General information

This study retrospectively selected patients with acute pancreatitis admitted to The First People's Hospital of Jiashan from January 2022 to December 2024 as the research subjects, aiming to compare the efficacy of octreotide and somatostatin in treating acute pancreatitis. As shown in the experimental design flowchart in Fig. 1, a total of 132 cases were collected. After exclusions, 126 cases were included. Of these, two were lost, and four withdrew for personal reasons, leaving 120 cases for analysis.

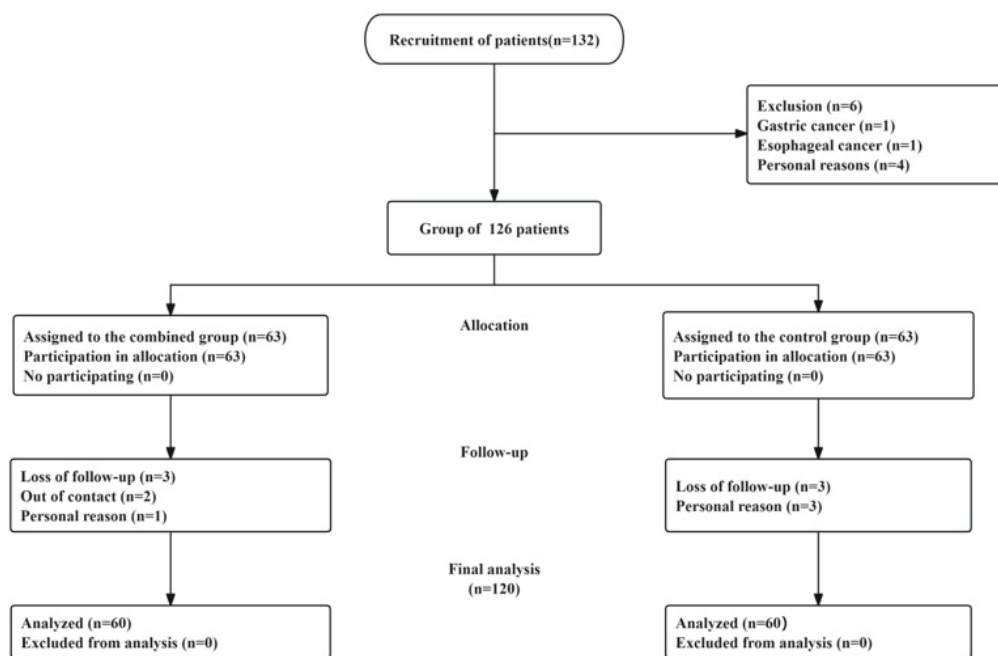


Fig. 1. Flowchart of experimental design.

These were divided into control and experimental groups based on treatment modality, with 60 cases in each group. Inclusion criteria: (1) patients with acute pancreatitis were diagnosed according to the Chinese Guide to Diagnosis and Treatment of Acute Pancreatitis in China (2021 Edition)¹², published by the Chinese Journal of Practical Surgery and written by the Pancreatic Surgery Group of the Surgical Branch of the Chinese Medical Association; (2) no other gastrointestinal or oncological diseases; (3) no history of allergy to the study drugs; (4) age 35-65 years; (5) complete clinical data and relevant examinations. Exclusion criteria¹³: (1) those with speech, mental, or psychological disorders that affect the smooth conduct of the study; (2) those with other digestive system malignant tumors; (3) patients with serious heart, liver, kidney, and lung problems; (4) female patients during pregnancy or breast-feeding; (5) a history of relevant drug treatment in the last four weeks; (6) those with serious infectious diseases.

Ethics statement

The clinical study followed the Declaration of Helsinki and other relevant ethical regulations, was reviewed and approved by the Hospital Ethics Committee, and the purpose, process, and potential risks of the study were explained in detail to the subjects or their proxies, with written informed consent obtained.

Mode of intervention

Both groups of patients received conventional treatment for acute pancreatitis. In practice, patients first underwent gastrointestinal decompression and fasting. Most patients had pain and required analgesic intervention as needed. Patients' basic vital signs were monitored to ensure that their water and electrolyte balance was maintained, and acid suppression and anti-infection treatment were provided. Meanwhile, patients received nutritional support based on their physical signs to support recovery, reduce symptoms, and prevent complications.

Patients in the control group received somatostatin as part of conventional treatment to inhibit pancreatic fluid secretion, administered by injection (trade name Sitanin, Laboratoires Serono S.A., H20090930, lyophilized powder, 250 μg). 250 μg of somatostatin was added to 500 mL of physiological saline, and the solution was infused intravenously. For patient ¹⁴, the infusion was delivered at 250 μg per hour, twice daily for 7 consecutive days.

Patients in the experimental group received octreotide, in addition to conventional treatment, to inhibit pancreatic fluid secretion. Octreotide acetate (trade name Zenith, Novartis AG, Switzerland, H20090948), a colorless, clear liquid (1 mL: 0.1 mg), was used. It was diluted with 250 mL of saline injection and administered via intravenous drip at 0.3 mg every 12 hours. The patients were medicated for 7 consecutive days ¹⁵.

At the same time as administering drug treatment to patients, it was necessary to pay rigorous attention to the patient's respiratory status and blood pressure fluctuations, to observe whether there were any adverse reactions to the medication, and to actively prevent them, to avoid patients from experiencing shock and other adverse reactions, and to ensure the patient's life safety.

Observation indicators

AMY, serum LPS, CRP, and ALB

Procoagulant tubes were used to collect 5 mL of venous blood from patients on an empty stomach as test samples. After centrifugation for 10 min at 3000 r/min using a Beckman Microfuge® 20R centrifuge, the serum was separated and measured using a Beckman Counter DXC800 automatic blood biochemistry analyzer.

IL-6, TNF- α , and PCT

The serum was collected as described above, and the enzyme-linked immunosorbent assay (ELISA) was used to detect IL-6, TNF- α , and PCT ¹⁶ in the serum using the

ELISA kits Beyotime PI330/PT518/PP790 from Shanghai Beyotime Biotechnology Co.

WBC count

A routine blood tube was used to collect 5 mL of fasting venous blood from the patients as a test sample, and a Beckman Vi-CELL BLU fully automated cell counter was used to perform the WBC count.

Hospital stay duration

Hospital stay duration is the total length of time a patient is hospitalized and an important indicator of the patient's condition and treatment effect ¹⁷.

Determine the admission time: the time when the patient completes admission procedures and officially becomes an inpatient.

Determine the discharge time: the point in time when the patient completes treatment and the nurse carries out the discharge instructions. The patient is considered to have completed treatment when the patient's condition is stable, with no obvious worsening symptoms, and vital signs are normal.

Calculate the total number of days: Subtract the discharge date from the admission date. The resulting number of days will be the total number of days in the hospital.

Attention to special circumstances: if there were cases such as a leave of absence or a temporary transfer, the calculation of hospitalization time should be adjusted accordingly.

Incidence rate of adverse reactions during treatment

The incidence rate of adverse drug reactions is the most important indicator for assessing drug safety and is used to measure the occurrence of adverse reactions during drug use. Calculating the incidence of adverse drug reactions can help doctors and patients better understand drug safety and make rational treatment decisions based on the risk of such reactions. In this study,

adverse conditions during the treatment of acute pancreatitis with somatostatin and octreotide mainly included abdominal pain, rash, nausea and vomiting, and dizziness and headache¹⁸.

The formula for calculating the incidence of adverse drug reactions is shown below:

Incidence of adverse reactions = (number of people with adverse reactions/total number of patients using the drug) * 100%

Sample size calculation methods

Sample sizes were determined using G*Power 3.1.9.7 to estimate the number needed to detect statistically significant differences. The calculation was based on the primary outcome, the Inflammation Indicator. According to previous studies¹⁴, IL-6 levels in patients with acute pancreatitis treated with somatostatin for 7 days were lower than in the healthy control group ($p=0.025$), with an effect size of 0.49. With a type I error rate (α) of 0.05 and 80% power, 53 patients were estimated to be needed in each group. To account for potential uncertainties, a total of 60 patients in the control group ($n=60$) and 60 in the experimental group ($n=60$) were ultimately selected for analysis in this study, and this sample size was considered sufficient to draw reliable conclusions.

Statistical Methods

SPSS version 28.0 was used to analyze the data. The data in this study were tested for normality. Baseline characteristics were described as counts and means ($\pm$ SD). AMY, LPS, CRP, IL-6, TNF- α , WBC, ALB, PCT, and hospital stay duration were expressed as means $\pm$ SD. An independent-samples test was used to compare two groups. The incidence of adverse events was expressed as [n (%)], and comparisons between the two groups were analyzed using the χ^2 test. All statistical tests were two-sided, and $p<0.05$ was considered statistically significant.

RESULTS

Comparison of baseline data between the two groups

Comparing the baseline data of the patients in the control and experimental groups, Table 1 provided evidence that no significant differences were observed between the two groups in age, BMI, gender, classification of acute pancreatitis, APACHE II scores, disease duration, and etiology of the disease^{16, 19} ($p=0.816, 0.662, 0.36, 0.714, 0.896, 0.504, 0.559, 0.609, 0.793, 0.697$), indicating that the groups were comparable before drug treatment.

Comparison of serum amylase and serum lipase between the two groups

As shown in Table 2, no significant difference was observed between the control and experimental groups in serum AMY and LPS levels on the day prior to dosing (AMY: 95% CI: -2.57-10.23, $p=0.239$; LPS: 95% CI: -2.78-12.17, $p=0.216$). After seven days of drug administration, these indices were significantly reduced in both groups (both $p<0.05$). In addition, compared with the control group (AMY: 44.85 ± 8.17 U/L; LPS: 42.48 ± 6.99 U/L), the experimental group (AMY: 42.15 ± 5.87 U/L; LPS: 39.80 ± 5.10 U/L) showed significantly lower levels (AMY: 95% CI: 0.13-5.28, $p=0.04$; LPS: 95% CI: 0.47-4.90, $p=0.018$), suggesting that, compared with somatostatin, octreotide can restore pancreatic function to normal more quickly in patients with acute pancreatitis.

Comparison of CRP between the two groups

As shown in Table 3, no significant difference in serum CRP levels was observed between the two groups on the first day of drug administration (95% CI: -3.50 to 5.01, $p=0.726$). After seven days of administration, CRP decreased in both the control and experimental groups (167.25 ± 11.62 mg/L vs.

Table 1. Baseline information of patients.

Indicator	Control group (n=60)	Experimental group (n=60)	95% CI		p	Effect size
			Lower	Upper		
Age	50.58±9.03	50.2±8.96	-2.87	3.64	0.816	0.02
BMI(kg/m ²)	22.62±1.7	22.76±1.80	-0.77	0.49	0.662	-0.04
Gender						
Male	35	30				
Female	25	30	0.681	2.878	0.36	0.084
Classification of Acute Pancreatitis						
Mild	33	31				
Severe	27	29	0.558	2.344	0.714	0.033
APACHE II scores	19.47±2.33	19.52±1.8	-0.80	0.70	0.896	-0.01
Disease duration (hours)	6.37±0.31	6.33±0.34	-0.08	0.16	0.504	0.06
Etiology						
Biliary	42	39	0.584	2.702	0.559	0.053
Alcoholic	8	10	0.281	2.107	0.609	-0.047
Hyperlipidemia	9	8	0.41	3.206	0.793	0.024
Other	3	4	0.158	3.443	0.697	-0.036

BMI: body mass index; APACHE II scores: acute physical and chronic health scores. Independent Samples t-test was used for continuous variables, and Chi-square Test was used for categorical variables.

Table 2. Comparison of serum amylase and serum lipase between the two groups.

Indicator	n	Time	Mean±SD		95% CI		p	Effect size
			Control group	Experimental group	Lower	Upper		
AMY (U/L)	60	1 day prior to dosing	261.53±17.59	257.70±17.83	-2.57	10.23	0.239	0.11
	60	7 days after dosing	44.85±8.17*	42.15±5.87*	0.13	5.28	0.04	0.19
LPS (U/L)	60	1 day prior to dosing	260.06±22.08	255.37±19.16	-2.78	12.17	0.216	0.11
	60	7 days after dosing	42.48±6.99*	39.80±5.10*	0.47	4.90	0.018	0.21

*p<0.05 vs 1 day prior to dosing. AMY: serum amylase; LPS: serum lipase. Independent Samples t-test was used for between-group comparisons, and Paired Samples t-test was used for within-group comparisons.

Table 3. Comparison of C-reactive protein between the two groups of patients.

Indicator	n	Time	Mean±SD		95% CI		<i>p</i>	Effect size
			Control group	Experimental group	Lower	Upper		
CRP (mg/L)	60	1 day prior to dosing	167.25±11.62	166.50±11.92	-3.50	5.01	0.726	0.03
	60	7 days after dosing	83.24±5.32*	80.34±7.4*	0.56	5.23	0.015	0.22

**p*<0.05 vs 1 day prior to dosing. CRP: C-reactive protein. Independent Samples *t*-test was used for comparisons between groups, and Paired Samples *t*-test was used for comparisons within groups.

83.24±5.32 mg/L, *p*<0.05; 166.50±11.92 mg/L vs. 80.34±7.4 mg/L, *p*<0.05), indicating that both somatostatin and octreotide can reduce inflammation in patients with acute pancreatitis. Compared with the control group (83.24±5.32 mg/L), CRP was significantly lower in the experimental group (80.34±7.4 mg/L) (95% CI: 0.56-5.23, *p*=0.015), suggesting that, compared with somatostatin, octreotide could normalize inflammation and tissue damage more quickly in patients with acute pancreatitis.

Comparison of IL-6, TNF- α , and PCT between the two groups

As shown in Table 4, there was no significant difference in the levels of IL-6, TNF- α , and PCT between the two groups on the first day before drug administration (*p*=0.222, *P*=0.392, *p*=0.546). After drug administration, the levels of these three markers decreased in both groups (all *p*<0.05). In addition, compared with the control group, the experimental group had lower levels of IL-6 (52.30±8.42 pg/mL vs. 55.36±8.03 pg/mL), TNF- α (40.07±5.74 ng/L vs. 42.71±7.66 ng/L), and PCT (33.80±4.70 ng/mL vs. 35.66±4.60 ng/mL), with 95% CIs of 0.08-6.03 (*p*=0.044), 0.19-5.09 (*p*=0.035), and 0.17-3.54 (*p*=0.031), respectively, suggesting that octreotide can normalize more quickly the degree of inflammation and infection in patients with acute pancreatitis compared to somatostatin.

Comparison of white blood cells between the two groups

As shown in Table 5, there was no significant difference in WBC between the two groups before drug administration (95% CI: -0.45 to 0.08; *p*=0.174). After drug administration, WBC decreased in both the control and experimental groups (13.76±1.39*10⁹/L vs. 12.55±1.39*10⁹/L, *p*<0.05; 13.94±0.69*10⁹/L vs. 11.94 ± 1.68*10⁹/L, *p*<0.05), indicating that both somatostatin and octreotide reduced leukocyte counts. Compared with the control group (12.55±1.39*10⁹/L), the experimental group's WBC (11.94±1.68*10⁹/L) decreased significantly (95% CI: 0.06-1.17; *p*=0.031), suggesting that, compared with somatostatin, octreotide could normalize infection and inflammatory responses in patients with acute pancreatitis more quickly.

Comparison of albumin between the two groups

As shown in Table 6, there was no significant difference in ALB levels between the two patient groups before drug administration (95% CI: -0.57 to 0.18; *p*=0.31). After drug administration, ALB levels increased in both groups (25.23±1.12 g/L vs. 36.99±1.94 g/L, *p*<0.05; 25.43±0.95 g/L vs. 38.47±5.12 g/L, *p*<0.05). Compared with the control group, ALB levels increased in the experimental group (36.99±1.94 g/L vs. 38.47±5.12 g/L; 95% CI: -2.89 to 0.07;

Table 4. Comparison of interleukin-6, tumor necrosis factor- α , and procalcitonin between the two patient groups.

Indicator	n	Time	Mean $\pm$ SD		95% CI		<i>p</i>	Effect size
			Control group	Experimental group	Lower	Upper		
IL-6 (pg/mL)	60	1 day prior to dosing	98.63 $\pm$ 9.59	100.65 $\pm$ 8.40	-5.28	1.24	0.222	-0.11
	60	7 days after dosing	55.36 $\pm$ 8.03*	52.30 $\pm$ 8.42*	0.08	6.03	0.044	0.18
TNF- α (ng/L)	60	1 day prior to dosing	81.61 $\pm$ 8.94	80.23 $\pm$ 8.71	-1.81	4.57	0.392	0.09
	60	7 days after dosing	42.71 $\pm$ 7.66*	40.07 $\pm$ 5.74*	0.19	5.09	0.035	0.30
PCT (ng/mL)	60	1 day prior to dosing	73.12 $\pm$ 5.63	73.78 $\pm$ 6.18	-2.79	1.48	0.546	-0.21
	60	7 days after dosing	35.66 $\pm$ 4.60*	33.80 $\pm$ 4.70*	0.17	3.54	0.031	0.35

**p*<0.05 vs 1 day prior to dosing. IL-6: interleukin-6; TNF- α : tumor necrosis factor- α ; PCT: procalcitonin. Independent samples t-test was used for between-group comparisons, and Paired Samples t-test was used for within-group comparisons.

Table 5. Comparison of white blood cells between the two groups of patients.

Indicator	n	Time	Mean $\pm$ SD		95% CI		<i>p</i>	Effect size
			Control group	Experimental group	Lower	Upper		
WBC (10 ⁹ /L)	60	1 day prior to dosing	13.76 $\pm$ 0.77	13.94 $\pm$ 0.69	-0.45	0.08	0.174	-0.12
	60	7 days after dosing	12.55 $\pm$ 1.39*	11.94 $\pm$ 1.68*	0.06	1.17	0.031	0.19

**p*<0.05 vs 1 day prior to dosing; WBC: white blood cells; Independent Samples t-test was used for between-group comparisons, and Paired Samples t-test for within-group comparisons.

Table 6. Comparison of serum albumin between the two groups of patients.

Indicator	n	Time	Mean $\pm$ SD		95% CI		<i>p</i>	Effect size
			Control group	Experimental group	Lower	Upper		
ALB (g/L)	60	1 day prior to dosing	25.23 $\pm$ 1.12	25.43 $\pm$ 0.95	-0.57	0.18	0.31	-0.10
	60	7 days after dosing	36.99 $\pm$ 1.94*	38.47 $\pm$ 5.12*	-2.89	-0.07	0.039	-0.19

**p*<0.05 vs 1 day prior to dosing. ALB: serum albumin. Independent Samples t-test was used for between-group comparisons, and Paired Samples t-test for within-group comparisons.

$p=0.031$), suggesting that, compared with somatostatin, octreotide could more quickly normalize nutrition and osmotic pressure in patients with acute pancreatitis.

Comparison of the hospital stay duration of patients in two groups

As shown in Table 7, the hospital stay duration for the control group was (11.03 ± 1.30) days, and for the experimental group it was (10.38 ± 1.46) days. The hospital stay duration for patients in the experimental group was evidently shorter than that of the control group (95% CI: 0.15-1.15; $p=0.011$), suggesting that, compared with somatostatin, octreotide could help patients with acute pancreatitis return to normal more quickly, shorten hospitalization time, and improve the quality of medical care.

Incidence of adverse reactions during treatment in the two groups

As shown in Table 8, during hospitalization, the control group had two patients with abdominal pain, three with skin rashes, five with nausea and vomiting, and three with dizziness and headache, for an overall

adverse reaction incidence of 21.7%. In the experimental group, one patient had nausea and vomiting, and two had dizziness and headache, for an adverse reaction incidence of 5%. These results suggest that, compared with somatostatin, octreotide for the treatment of patients with acute pancreatitis had a significantly lower incidence of adverse reactions (95% CI: 1.413-19.544; $p=0.008$).

DISCUSSION

Acute pancreatitis is mainly divided into two types: mild and severe. The mild type is mainly manifested by pancreatic edema, is mostly self-limiting, and usually resolves within a few days, with complete recovery to the pre-treatment state²⁰. In severe cases, complications such as pancreatic hemorrhage, peritonitis, pancreatic necrosis, and shock are common, posing a greater threat to patients' health. The prognosis of severe acute pancreatitis is usually poor, with a high mortality rate that can reach 10%-40%²¹. In recent years, with the continuous improvement of China's medical treatment technol-

Table 7. Comparison of hospital stay duration between the two groups (days).

Indicator	n	Mean $\pm$ SD		95% CI		<i>p</i>	Effect size
		Control group	Experimental group	Lower	Upper		
Hospital stay duration	60	11.03 $\pm$ 1.30	10.38 $\pm$ 1.46	0.15	1.15	0.011	0.23

An independent-samples t-test was used to compare groups.

Table 8. Comparison of incidence of adverse reactions between the two groups.

Indicator	n (%)		95% CI		<i>p</i>	Effect size
	Control group	Experimental group	Lower	Upper		
Abdominal pain	2	0				
Skin rash	3	0				
Nausea and vomiting	5	1	1.413	19.544	0.007	0.245
Dizziness and headache	3	2				
Adverse reactions	13(21.7%)	3(5%)				

Chi-square test was used to compare the groups.

ogy, a variety of new drugs have appeared in clinical practice, and their application has, to a certain extent, reduced morbidity and mortality in patients with severe acute pancreatitis, but some patients still die. The occurrence of acute pancreatitis is mainly due to the continuous release of pancreatic enzymes in the body, influenced by a series of intertwined factors, resulting in the activation of phospholipase A2, etc. Under the influence of the inflammatory response, the pancreas undergoes microcirculation abnormalities, accelerating pancreatic digestion, destroying the normal blood supply, and ultimately leading to pancreatic necrosis. Niu et al.²² proposed that phospholipase D2 (PLD2) plays a crucial regulatory role. In the medical management of patients with severe acute pancreatitis, comprehensive therapeutic approaches are typically used to reduce pancreatic secretion, inhibit pancreatic enzyme synthesis, improve clinical signs, and promote recovery.

AMY and LPS are two important laboratory tests for acute pancreatitis²³; the former is mainly due to the activation of pancreatic amylase by trypsinogen in the pancreas, which subsequently causes the elevation of AMY, and the latter is due to the activation of pancreatic lipase by lipaseogen in the pancreas; therefore, AMY and LPS are the characteristic indicators for the diagnosis of acute pancreatitis. Sun et al.²⁴, in a multicenter retrospective study, suggested that both somatostatin and octreotide could normalize serum AMY and LPS in patients with acute pancreatitis, and this article's research showed that octreotide could normalize pancreatic function more quickly than somatostatin. CRP, an acute-phase reactant synthesized by IL-6-stimulated hepatocytes, is one of the proteins mediating the inflammatory response in acute pancreatitis. The inflammatory response in acute pancreatitis and its concentration can reflect the presence or absence of inflammation and the intensity of the response in the organism²⁵. TNF- α , mainly

produced by monocytes and macrophages, can regulate immune function and enhance anti-infection capacity, and the multiple-organ damage caused by inflammation is closely associated with TNF- α ²⁶.

IL-6 plays an important role in the proliferation and activation of lymphocytes, and can promote the synthesis of CRP and the development of blood cells, but its concentration is too high, which can stimulate the granulocytes to release a large number of oxygen-free radicals and proteases, thus destroying the vascular endothelium and the endothelial cells. However, its high concentration can stimulate granulocytes to release large quantities of oxygen-free radicals and proteases, thereby destroying the vascular endothelium and aggravating organ damage. During inflammatory reactions, macrophages and monocytes in the liver, lymphocytes and endocrine cells in the lungs and intestinal tissues can synthesize and secrete PCT in response to bacterial endotoxin, TNF- α , and IL-6; these substances work together, leading to an apparent increase in the level of PCT in the blood circulation²⁷. The present study suggests that, in patients with acute pancreatitis, CRP, IL-6, TNF- α , and PCT levels in the blood were reduced more significantly in those treated with octreotide than in those treated with somatostatin, indicating that octreotide can normalize inflammatory responses and immune functions. Mao et al.²⁸, in a multicenter retrospective study, validated somatostatin and octreotide as widely used medications for acute pancreatitis and reported octreotide's superior therapeutic effect, which is more attuned to the results of this study. When germs invade the human body, leukocytes can pass through the capillary wall by deformation, concentrate at the site of germ invasion, surround and engulf the germs, and produce an inflammatory reaction, resulting in a higher leukocyte count than normal. ALB unequivocally expands blood volume and

preserves plasma colloid osmotic pressure, helps with blood transport and detoxification of body organs²⁹, and, in human metabolism, it can be used as a source of nitrogen to provide nutrients to our body tissues. Bhansali *et al.*³⁰ retrospectively studied 131 patients with infected pancreatic necrosis, and the use of octreotide increased the level of ALB, and reduced surgical complications, from the perspective of this study, it acts out the similitude to the results of the present study, in this study, compared to somatostatin, the patients with acute pancreatitis treated with octreotide had a significant reduction in the number of leukocytes, a significant increase in ALB, and a shorter hospitalization time, which suggests that octreotide can bring the inflammatory response and the osmotic pressure of the body in patients with acute pancreatitis back to a normal state more quickly.

In clinical practice, the most commonly used drugs for treating patients with acute pancreatitis are octreotide and somatostatin, which have similar pharmacological effects; octreotide is a synthetic octapeptide. When administered, octreotide mimics the action of endogenous somatostatin, inhibiting the release of inflammatory mediators and suppressing the activation of mononuclear phagocytes, thereby improving patients' hemodynamic status. In clinical practice, because the application of octreotide can inhibit a variety of enzymes in pancreatic tissue, it can not only control the digestion of pancreatic protease on its own, but also control and maintain the intestinal micro-ecological balance of the patient to avoid the generation of endotoxins, so as to improve the patient's intestinal environment, alleviate the patient's symptoms of abdominal pain and bloating, enhance the patient's intestinal and gastrointestinal peristalsis, inhibit the activity of platelets³¹, reduce bile reflux, ensuring patient comfort and promoting patient recovery. In the case of injectable somatostatin, this drug is syn-

thetic and belongs to the tetradecapeptide class; it is distributed in the human body's peripheral and central nervous systems, with higher concentrations in the gastrointestinal tract and the hypothalamus. Somatostatin can inhibit gastrin secretion³², and also inhibit gastric acid and pepsin secretion, improve the internal secretion function and external secretion function of the pancreas, maintain balance and coordination, thus reducing the inflammatory response of the patient, and promoting the recovery of the patient, but from the current clinical situation, there are certain adverse reactions in the application of this drug, which adversely affects the safety of the patient's use of the drug³³, leading to the limitation of the promotion of this drug in the clinic. Octreotide has a stronger effect and a longer maintenance time, and it can inhibit pancreatic enzymes and gastric acid, thus reducing the amount of pancreatic secretion, improving the blood and urine amylase levels of patients, promoting the improvement of gastrointestinal digestive function, and protecting the pancreatic parenchymal cell membrane of patients, so as to improve the effect of clinical application and promote the recovery of patients.

Although studies have shown that, in the treatment of acute pancreatitis, octreotide with a specific treatment regimen yields better therapeutic outcomes than somatostatin in controlling inflammation and shortening hospital stay, these studies still have the following limitations that may affect the generalizability of the results and their long-term application value: (1) this study provides preliminary single-center evidence comparing the efficacy of octreotide and somatostatin in the treatment of acute pancreatitis. However, limited by the inherent defects of the single-center and retrospective design, it still has deficiencies in external validity, bias control, and causal inference. (2) Lack of clear evidence of survival benefit; this study did not analyze the impact of octreotide on short-term mortality

or complication rate in patients with acute pancreatitis³⁴. (3) For severe acute pancreatitis, although octreotide may inhibit pancreatic juice secretion, there is currently no clear evidence that it is effective in blocking systemic inflammatory response or progressive multiple organ failure³⁵. (4) The medication cycle of octreotide in this study was only seven days, and its long-term medication risks remain unclear. (5) The localization of research data is insufficient, and the classification of disease severity is not clear. Future studies should adopt a multi-center prospective cohort design, expand the sample size, include more abundant confounding factors, conduct analyses targeting severe subgroups, clarify the details of drug use, and combine dynamic inflammation index monitoring with long-term follow-up data to further verify the efficacy difference between octreotide and somatostatin, so as to provide more reliable evidence to support clinical medication.

In conclusion, octreotide in the therapy of the acute pancreatitis patients has a better effect, which is conducive to the enhancement of the body's immune function, the improvement of the patient's AMY and LPS, the body's inflammatory response, osmolality returns to normal faster, shortens the patient's hospital stay, improves the quality of medical care, and provides a scientific basis for the optimization of clinical therapeutic medication regimens.

Acknowledgment

None.

Funding

None.

ORCID ID of the authors

- Qunchao Zhu (QZ):
0009-0004-6987-6091
- Tian Jiang (TJ):
0009-0002-5083-5500

- Yan Li (YL):
0009-0002-4819-7941
- Sicong Jiang (SJ):
0000-0001-8974-6770
- Aifang Li (AL):
0009-0008-7880-6078
- Chendong Ma (CM):
0009-0003-3787-5468

Author's contributions

QZ, CM: Developed and planned the study, conducted experiments, and interpreted the results. Edited and refined the manuscript, with a focus on critical intellectual contributions. TJ, YL: Participated in collecting, assessing, and interpreting the data. SJ, AL: Made significant contributions to data interpretation and manuscript preparation.

Conflicts of interest

The authors declare no financial conflicts of interest.

Consent to publish

The manuscript has not been published previously and is not under consideration by any other journal. All authors have approved the content of the paper.

Consent to participate

We obtained a signed informed consent form from every participant.

Ethic approval

This study was approved by the Ethics Committee of The First People's Hospital of Jiashan.

Data availability statement

The data that support the findings of this study are available from the corresponding author, upon reasonable request.

REFERENCES

1. Mederos MA, Reber HA, Girgis MD. Acute Pancreatitis: A Review. *JAMA*. 2021; 325(4): 382-90. <https://doi.org/10.1001/jama.2020.20317>.
2. Beyer G, Habtezion A, Werner J, Lerch MM, Mayerle J. Chronic pancreatitis. *Lancet*. 2020; 396(10249): 499-512. [https://doi.org/10.1016/S0140-6736\(20\)31318-0](https://doi.org/10.1016/S0140-6736(20)31318-0).
3. Yang AL, McNabb-Baltar J. Hypertriglyceridemia and acute pancreatitis. *Pancreatolgy*. 2020; 20(5): 795-800. <https://doi.org/10.1016/j.pan.2020.06.005>.
4. Saini J, Marino D, Badalov N, Vugelman M, Tenner S. Drug-Induced Acute Pancreatitis: An Evidence-Based Classification (Revised). *Clin Transl Gastroenterol*. 2023; 14(8): e00621. <https://doi.org/10.14309/ctg.0000000000000621>.
5. Sohail Z, Shaikh H, Iqbal N, Parkash O. Acute pancreatitis: A narrative review. *J Pak Med Assoc*. 2024; 74(5): 953-8. <https://doi.org/10.47391/JPMA.9280>.
6. Gardner TB. Acute Pancreatitis. *Ann Intern Med*. 2021; 174(2): ITC17-ITC32. <https://doi.org/10.7326/AITC202102160>.
7. Horváth IL, Bunduc S, Fehérvári P, Váncsa S, Nagy R, Garmaa G, et al. The combination of ulinastatin and somatostatin reduces complication rates in acute pancreatitis: a systematic review and meta-analysis of randomized controlled trials. *Sci Rep*. 2022; 12(1): 17979. <https://doi.org/10.1038/s41598-022-22341-7>.
8. He C, Liu J, Xu L, Sun F. The Effect of Percutaneous Catheter Drainage Combined with Somatostatin on Inflammation and Plasma Thromboxane 2, Prostacyclin I2 Levels in Patients with Severe Pancreatitis. *Georgian Med News*. 2024; 352/353(7/8): 278-283.
9. Ampofo E, Nalbach L, Menger MD, Laschke MW. Regulatory Mechanisms of Somatostatin Expression. *Int J Mol Sci*. 2020; 21(11):4170. <https://doi.org/10.3390/ijms21114170>.
10. Khedr A, Mahmoud EE, Attallah N, Mir M, Boike S, Rauf I, et al. Role of octreotide in small bowel bleeding. *World J Clin Cases*. 2022; 10(26): 9192-9206. <https://doi.org/10.12998/wjcc.v10.i26.9192>.
11. Huang ZY, Gong H, Tang CW, Wang MJ, Wang R. Remission of organ failure in patients with predicted severe acute pancreatitis treated by somatostation, octreotide and cyclooxygenase-2 inhibitors. *Pancreatolgy*. 2024; 24(1): 24-31. <https://doi.org/10.1016/j.pan.2023.12.006>.
12. Chinese Pancreatic Surgery Association CSoSCMA. Guidelines for diagnosis and treatment of acute pancreatitis in China (2021). *Zhonghua Wai Ke Za Zhi*. 2021; 59: 578-587. <https://doi.org/10.3760/cma.j.cn112139-20210416-00172>.
13. Cao JP, Piao XH, Zhu LX, Feng PF. Xuebijing and somatostatin against acute pancreatitis: A systematic review and network pharmacology. *Medicine (Baltimore)*. 2024; 103(50): e40964. <https://doi.org/10.1097/MD.00000000000040964>.
14. Sliwinska-Mosson M, Marek G, Grzebieniak Z, Milnerowicz H. Relationship between somatostatin and interleukin-6: A cross-sectional study in patients with acute pancreatitis. *Pancreatolgy*. 2018; 18(8): 885-891. <https://doi.org/10.1016/j.pan.2018.09.013>.
15. Bao W, Kong R, Wang N, Han W, Lu J. PPAR-Alpha Agonist Fenofibrate Combined with Octreotide Acetate in the Treatment of Acute Hyperlipidemia Pancreatitis. *PPAR Res*. 2021; 2021: 6629455. <https://doi.org/10.1155/2021/6629455>.
16. Zheng XL, Li WL, Lin YP, Huang TL. Computerized tomography-guided therapeutic percutaneous puncture catheter drainage-combined with somatostatin for severe acute pancreatitis: An analysis of efficacy and safety. *World J Gastrointest Surg*. 2024; 16(1): 59-66. <https://doi.org/10.4240/wjgs.v16.i1.59>.
17. Rivera-Suazo Y, Vázquez-Frias R. Factors associated with hospital length of stay in children with acute pancreatitis. *Rev Gastroenterol Mex. (Engl Ed)*. 2023; 88(1): 4-11. <https://doi.org/10.1016/j.rgmxen.2021.05.016>.

18. **Zhu LX, Chen Y, Chen XF, Sheng N, Feng PF.** Systematic Review and Meta-analysis of the Clinical Efficacy of Octreotide in Combination with Ulinastatin in the Treatment of Acute Pancreatitis. *Drugs R D.* 2025; 25(3): 195-207. <https://doi.org/10.1007/s40268-025-00518-5>.
19. **Chen ZP, Huang HP, He XY, Wu BZ, Liu Y.** Early continuous blood purification affects TNF-alpha, IL-1beta, and IL-6 in patients with severe acute pancreatitis via inhibiting TLR4 signaling pathway. *Kaohsiung J Med Sci.* 2022; 38(5): 479-485. <https://doi.org/10.1002/kjm2.12497>.
20. **Zerem E, Kurtcehajic A, Kunosić S, Zerem Malkoćević D, Zerem O.** Current trends in acute pancreatitis: Diagnostic and therapeutic challenges. *World J Gastroenterol.* 2023; 29(18): 2747-2763. <https://doi.org/10.3748/wjg.v29.i18.2747>.
21. **Hong W, Pan J, Goyal H, Zippi M.** Acute pancreatitis infection: Epidemiology, prevention, clinical characteristics, treatment, and prediction. *Front Cell Infect Microbiol.* 2023; 13: 1175195. <https://doi.org/10.3389/fcimb.2023.1175195>.
22. **Niu JW, Zhang GC, Ning W, Liu HB, Yang H, Li CF.** Clinical effects of phospholipase D2 in attenuating acute pancreatitis. *World J Gastroenterol.* 2025; 31(2): 97239. <https://doi.org/10.3748/wjg.v31.i2.97239>.
23. **Wang L, Qi X, Tian F, Li H, Zhao H, Lv J, et al.** Diagnostic value of hematological parameters in acute pancreatitis. *Ann Palliat Med.* 2020; 9(5): 2716-2722. <https://doi.org/10.21037/apm-20-160>. 2716-2722. doi: 10.21037/apm-20-160. Epub 2020 Jul 23. PMID: 32787347.
24. **Sun C, Li Z, Shi Z, Li G.** Current diagnosis and treatment of acute pancreatitis in China: a real-world, multicenter study. *BMC Gastroenterol.* 2021; 21(1): 210. <https://doi.org/10.1186/s12876-021-01799-1>.
25. **Potempa M, Hart PC, Rajab IM, Potempa LA.** Redefining CRP in tissue injury and repair: more than an acute pro-inflammatory mediator. *Front Immunol.* 2025; 16: 1564607. <https://doi.org/10.3389/fimmu.2025.1564607>.
26. **Ashrafizadeh M.** Cell Death Mechanisms in Human Cancers: Molecular Pathways, Therapy Resistance and Therapeutic Perspective. *J Can Biomol Therap.* 2024; 1(1): 17-40. <https://doi.org/10.62382/jcibt.v1i1.13>.
27. **Velissaris D, Zareifopoulos N, Lagadinou M, Platanaki C, Tsiotsios K, Stavridis EL, et al.** Procalcitonin and sepsis in the Emergency Department: an update. *Eur Rev Med Pharmacol Sci.* 2021; 25(1): 466-479. https://doi.org/10.26355/eurrev_202101_24416.
28. **Mao X, Yang Z.** Current usage status of somatostatin and its analogs and trypsin inhibitors: a real-world study of 34,654 Chinese adult patients with acute pancreatitis. *Ann Palliat Med.* 2021; 10(2): 1325-1335. <https://doi.org/10.21037/apm-19-363>.
29. **Ullah A, Shin G, Lim SI.** Human serum albumin binders: A piggyback ride for long-acting therapeutics. *Drug Discov Today.* 2023; 28(10): 103738. <https://doi.org/10.1016/j.drudis.2023.103738>.
30. **Bhansali SK, Shah SC, Desai SB, Sunawala JD.** Infected necrosis complicating acute pancreatitis: experience with 131 cases. *Indian J Gastroenterol.* 2003; 22(1): 7-10. PMID: 12617444.
31. **Li ZF, Xu MY, Zhang DH, Gao TT, Gao Z, Li H.** Effects of ulinastatin combined with octreotide on blood coagulation function, inflammatory factors and amylase in patients with severe acute pancreatitis. *J Biol Regul Homeost Agents.* 2020; 34(6): 2147-2151. <https://doi.org/10.23812/20-362-L>.
32. **Chen F, Xu Y, Wang Z.** Ulinastatin combined with somatostatin enhances disease control and modulates serum inflammatory factors in patients with severe pancreatitis. *Am J Transl Res.* 2023; 15(9): 5797-5807. PMID: 37854214.
33. **Dou H, Kan Y, Xu Z, Wang Z, Zheng C.** Effect of probiotics combined with Ulinastatin and Somatostatin in the treatment of severe acute pancreatitis. *Pak J Med Sci.* 2024; 40(8): 1729-1734. <https://doi.org/10.12669/pjms.40.8.9744>.

-
34. Rykina-Tameeva N, Samra JS, Sahni S, Mittal A. Non-Surgical Interventions for the Prevention of Clinically Relevant Postoperative Pancreatic Fistula-A Narrative Review. *Cancers (Basel)*. 2023; 15(24):5865. <https://doi.org/10.3390/cancers15245865>.
 35. Nickel F, Anthony Wise P. Acute pancreatitis and multiple organ failure-Who beats the odds? *United European Gastroenterol J*. 2021; 9(2): 137-138. <https://doi.org/10.1002/ueg2.12056>.