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The Impact of Prophylactic Antibiotic Use in Emergency Treatment of Acute Pancreatitis on the Incidence of Infectious Pancreatic Necrosis

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Abstract: *Objective*: To analyze the value of prophylactic antibiotic use in reducing the incidence of pancreatic necrosis in patients with emergency acute pancreatitis (AP). *Methods*: A total of 70 AP patients who sought medical attention from January 2024 to January 2025 were randomly divided into groups by drawing lots. Group A received prophylactic antibiotic intervention, while Group B received conventional intervention. *Results*: Group A demonstrated superior outcomes compared to Group B in terms of therapeutic efficacy, duration of symptoms, inflammatory factors, symptom scores, and complication rates, with $p < 0.05$. *Conclusion*: Prophylactic antibiotic treatment in emergency AP patients leads to a decrease in inflammatory factor levels, alleviation of symptoms, a reduction in the incidence of infectious pancreatic necrosis, and is safe and effective.

Keywords: Prophylactic antibiotics; Acute pancreatitis; Infectious pancreatic necrosis

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1. Introduction

Acute pancreatitis (AP) accounts for a relatively high proportion of digestive emergencies and poses a significant threat to patients.

It is associated with the activation of pancreatic enzymes within the pancreas triggered by multiple etiological factors, leading to hemorrhage, edema, and necrosis of pancreatic tissue, thereby reducing the patient's quality of life.

The typical pathological features of acute pancreatitis (AP) include fever, nausea, vomiting, and elevated pancreatic enzymes. If AP is mild, the typical pathological feature is pancreatic edema, with most patients having a favorable prognosis and the condition being self-limiting. However, if AP is severe, it can increase the mortality rate [1].

Additionally, AP itself is a sterile inflammatory lesion. If bacteria infect during the progression of the disease, it can rapidly exacerbate pancreatic inflammation and increase the difficulty of diagnosis and treatment, making early treatment extremely important. Prophylactic antibiotic therapy is a commonly used treatment regimen for AP, but reports on relevant antibiotic use lack comprehensiveness, and there are few studies on the reduction of the

risk of infectious pancreatic necrosis in AP patients by antibiotics [2].

This article explores the value of prophylactic antibiotic therapy using a sample of 70 AP patients treated from January 2024 to January 2025.

2. Materials and methods

2.1. Materials

Seventy AP patients were treated from January 2024 to January 2025 and were divided into groups by lottery drawing.

The baseline data of AP patients in Group A were compared with those in Group B, with $p > 0.05$, as shown in **Table 1**.

Table 1. Analysis of baseline data for AP

Group	n	Age range (years)	Age (years, Mean ± SD)	Duration range (days)	Duration (days, Mean ± SD)	Male [n (%)]	Female [n (%)]
A	35	26–77	56.81 ± 2.42	8–42	33.21 ± 2.14	20 (57.14)	15 (42.86)
B	35	26–78	56.79 ± 2.39	8–41	33.18 ± 2.12	21 (60.00)	14 (40.00)
Statistic (χ^2/t)			0.0348		0.0589	0.0589	
p-value			0.9724		0.9532	0.8083	

2.2 Inclusion and exclusion criteria

2.2.1. Inclusion criteria

(1) Laboratory tests, imaging examinations, and clinical manifestations suggest pancreatitis
(2) Signing of informed consent
(3) Normal organ function
(4) Initial diagnosis, with no prior surgical or pharmacological treatment before enrollment

2.2.2. Exclusion criteria

(1) Organ lesions
(2) Drug contraindications
(3) Chronic diseases such as hyperglycemia and hypertension

2.3. Treatment methods

Group A received intravenous treatment with Piperacillin and Tazobactam for Injection (a single dose of 4.5 g, twice daily) + Levofloxacin (a single dose of 0.5 g, once daily) + Ornidazole (a single dose of 0.5 g, twice daily). The medication was administered for 7 days.

Group B followed medical advice to fast and reduce gastrointestinal pressure. Based on the patient's physiological state, medications such as octreotide and proton pump inhibitors were administered to inhibit pancreatic secretion. Fluid replacement and nutritional support were provided as appropriate, along with analgesics and anti-shock drugs. Organ function was monitored, and amylase levels, blood routine tests, and abdominal CT scans were reviewed to target and prevent adverse indicators. Treatment lasted for 7 days.

2.4. Statistical research

Data was processed using SPSS 23.0, with the chi-square (χ^2) test and percentages used to describe count data, and the *t*-test and mean ± standard deviation ($\overline{x} \pm s$) used to describe measurement data. Statistical differences were considered significant when $p < 0.05$.

3. Results

3.1. Efficacy

The efficacy of AP in Group A was higher than that in Group B, with $p < 0.05$. As shown in **Table 2**.

Table 2. Efficacy (n, %)

Group	Markedly effective	Effective	Ineffective	Effective rate
Group A (n = 35)	24 (68.57)	10 (28.57)	1 (2.86)	34 (97.14)
Group B (n = 35)	18 (51.43)	11 (31.43)	6 (17.14)	29 (82.86)
χ^2-value				3.9383
p-value				0.0464

3.2. Duration of symptoms

The duration of AP symptoms in Group A was shorter than that in Group B, with $p < 0.05$. As shown in **Table 3**.

Table 3. Duration of symptoms ($\overline{x} \pm s$)

Group	Abdominal distension (days)	Abdominal pain (days)	Nausea & Vomiting (days)	Hospital Stay (days)
Group A (n = 35)	2.21 ± 0.25	1.91 ± 0.42	1.82 ± 0.33	18.71 ± 0.91
Group B (n = 35)	3.11 ± 0.36	2.73 ± 0.68	2.69 ± 0.49	23.66 ± 1.48
t-value	12.1482	6.0697	8.7125	16.8556
p-value	0.0000	0.0000	0.0000	0.0000

3.3. Serum inflammatory factors

After medication, the levels of WBC, CRP, and PCT in AP patients in Group A were all lower than those in Group B, with $p < 0.05$. As shown in **Table 4**.

Table 4. Serum inflammatory factors ($\overline{x} \pm s$)

Group	WBC ($\times 10^9$/L)		CRP (mg/L)		PCT (μg/L)	
	Before	After	Before	After	Before	After
Group A (n = 35)	14.28 ± 1.81	10.33 ± 0.48	259.43 ± 8.73	81.46 ± 4.82	11.58 ± 1.42	5.11 ± 0.81
Group B (n = 35)	14.29 ± 1.79	11.15 ± 0.81	259.61 ± 8.71	98.63 ± 6.99	11.62 ± 1.41	6.25 ± 0.99
t-value	0.0232	5.1524	0.0864	11.9635	0.1183	5.2725
p-value	0.9815	0.0000	0.9314	0.0000	0.9062	0.0000

3.4. Symptom scores

After medication, the symptom scores of AP patients in Group A were lower than those in Group B, with $p < 0.05$. As shown in **Table 5**.

Table 5. Symptom scores ($\overline{x} \pm s$)

Group	Abdominal pain (score)		Nausea & vomiting (score)		Fever (score)		Abdominal distension (score)	
	Pre-treatment	Post-treatment	Pre-treatment	Post-treatment	Pre-treatment	Post-treatment	Pre-treatment	Post-treatment
Group A (n = 35)	2.51 ± 0.46	0.88 ± 0.21	2.53 ± 0.41	0.87 ± 0.26	2.48 ± 0.32	0.78 ± 0.21	2.42 ± 0.33	0.79 ± 0.18
Group B (n = 35)	2.52 ± 0.49	1.39 ± 0.37	2.54 ± 0.44	1.42 ± 0.38	2.49 ± 0.33	1.42 ± 0.29	2.41 ± 0.31	1.46 ± 0.33
t	0.0880	7.0919	0.0984	7.0669	0.1287	10.5747	0.1307	10.5448
p	0.9301	0.0000	0.9219	0.0000	0.8980	0.0000	0.8964	0.0000

3.5. Complications

The complication rate in Group A was lower than that in Group B, with $p < 0.05$, as shown in **Table 6**.

Table 6. Complication rate (n, %)

Group	Anastomotic leak	Bleeding	Infected pancreatic necrosis	Incidence rate
Group A (n = 35)	0 (0.00)	0 (0.00)	1 (2.86)	1 (2.86)
Group B (n = 35)	3 (8.57)	1 (2.86)	3 (8.57)	7 (20.00)
χ^2-value	-	-	-	5.0806
p-value	-	-	-	0.0242

4. Discussion

Acute pancreatitis (AP) is associated with the activation of pancreatic enzymes in the human body, leading to the gradual digestion of pancreatic tissue. It is prevalent among adults and carries a high risk of organ dysfunction. Analyzing the etiology of AP, it is related to biliary obstruction, biliary tract infections, and other factors. After onset, bile refluxes into the pancreatic duct, increasing the activity of pancreatic enzymes and thus triggering AP. It is also associated with excessive alcohol consumption; alcohol continuously stimulates the body upon entering, increasing the secretion of pancreatic enzymes, which manifests as pancreatic duct obstruction and sphincter spasm. Additionally, it is linked to hyperlipidemia; continuous deposition of lipids in the pancreatic microvasculature leads to vascular obstruction and damage to pancreatic cells, which can also induce AP.

Furthermore, AP is also related to multiple factors such as daily medication use, iatrogenic procedures, metabolic diseases, trauma, and infections. After the onset of AP, the typical symptom is abdominal pain, mostly confined to the upper abdomen, with intense pain that can radiate to the back. The pain intensifies especially after a heavy meal or excessive alcohol consumption, and it can be alleviated by leaning forward or bending over. As AP progresses, patients develop symptoms such as nausea and vomiting, and the pain does not subside after vomiting gastric contents. In severe cases, the vomitus contains coffee-ground material or bile, and inflammatory

factors inhibit intestinal nerves, leading to intestinal paralysis, characterized by decreased bowel sounds.

Some patients with AP have severe conditions and may present with a series of systemic manifestations, such as fever during the onset (high fever in cases of secondary infection); after progressing to severe AP, the body releases a large number of inflammatory factors, resulting in hypotension, slowed pulse, and pallor; when complicated by biliary obstruction, jaundice may occur; after being complicated by ARDS, patients may develop hypoxemia due to respiratory distress [3].

The main hazards of AP to the human body are summarized as follows:

(1) Pancreatic necrosis

Inflammatory lesions and ischemia of pancreatic tissue can lead to pancreatic necrosis, progressing to necrotizing AP, which requires surgical debridement.

(2) Peripancreatic fluid collection

Increased inflammatory exudate increases pressure on adjacent blood vessels and intestines, which can lead to venous thrombosis and intestinal obstruction. In cases complicated by pseudocysts, it can cause abdominal hemorrhage and infection.

(3) Acute kidney injury

Continuous release of inflammatory factors in AP patients stimulates renal tubules and renal blood vessels, which can damage renal function and prolong the course of the disease.

(4) Circulatory system failure

Long-term inflammatory stimulation induces dilatative shock and myocardial suppression, increasing the risk of inadequate tissue perfusion and hypotension, with a minority of patients developing organ failure.

(5) Liver function impairment

Inflammation stimulates the hepatobiliary system, elevating transaminase levels, which can lead to secondary liver failure and further damage liver function.

(6) Abnormal coagulation indicators

Inflammation in AP patients stimulates the coagulation system, inducing disseminated intravascular coagulation, which increases the risk of adverse conditions such as thrombosis and hemorrhage.

Therefore, treatment should be initiated as early as possible after the onset of AP, with active prevention and control of complications to reduce the incidence of infectious pancreatic necrosis. During routine medication management, fasting as prescribed by the doctor is implemented for gastrointestinal decompression, along with the administration of drugs to inhibit pancreatic secretion, neurotrophic agents, analgesics, and anti-shock medications. Regular follow-up examinations of various indicators as per medical advice can control the progression of AP but cannot effectively manage inflammation. The prophylactic use of antibiotics in the treatment of AP patients can reduce the incidence of infections in severe cases. By inhibiting the progression of inflammation and reducing pancreatic tissue necrosis, it protects intestinal barrier function, decreases the number of intestinal flora, and also reduces adverse events such as pancreatic abscesses and peripancreatic infections [4]. Additionally, some high-risk AP patients may experience organ failure and extensive pancreatic necrosis, limit the effectiveness of routine treatment and potentially increasing the risk of mortality. Prophylactic antibiotic use can reduce the risk of multiple organ failure and systemic inflammatory response.

Based on the data analysis in this article, the prophylactic use of antibiotics as prescribed by the doctor demonstrates excellent efficacy and shortens the duration of symptoms. The reasons for this are as follows: the prophylactic use of piperacillin-tazobactam, which is effective against anaerobic bacteria and Gram-positive/

negative bacteria, can rapidly penetrate pancreatic tissue and is suitable for the treatment of pancreatic infections. However, the dosage and duration of administration should be adjusted based on the patient's physiological state during actual medication use. The active ingredient in levofloxacin inhibits bacterial DNA synthesis, providing broad-spectrum antibacterial effects. Ornidazole inhibits anaerobic bacteria and enhances the overall management of AP [5]. During the actual medication treatment period, it is essential to guide AP (acute pancreatitis) patients in adjusting their diet reasonably. For instance, if abdominal pain occurs during disease flare-ups, patients should follow medical advice to fast, thereby avoiding food stimulation of the pancreas and reducing digestive fluid secretion, which in turn lowers the pancreatic burden. Once the abdominal pain subsides or eases and all indicators stabilize, patients can follow medical advice to consume liquid foods such as rice water and warm water. After AP patients' conditions stabilize, they should still avoid high-fat foods like fried dough sticks, fried chicken, and cream, and they should eat small, frequent meals to avoid consuming large amounts of food in a short period. It is recommended that patients engage in exercises such as Tai Chi or walking for about 30 minutes daily to maintain a normal weight range. Daily abstention from smoking, alcohol, beverages, and strong tea is advised, along with maintaining a calm mindset.

Another set of data indicates that the inflammatory factor indicators of patients decrease after the prophylactic use of antibiotics. Analyzing the reasons, after the onset of AP, the WBC (white blood cell) count in patients increases, which is directly proportional to the progression of AP. However, an isolated increase in the WBC count cannot serve as a definitive diagnostic basis for AP and requires comprehensive analysis with other indicators. Generally, the WBC count in mild AP patients ranges from 10 to 15×10^9/L, while in moderate to severe AP patients, it is $\geq 15 \times 10^9$/L. CRP (C-reactive protein) is an inflammatory marker that rapidly increases when the body develops AP and reaches its peak 48–72 hours later, assisting physicians in predicting the severity of AP. Generally, a CRP level > 150 mg/L in mild AP patients indicates a critical condition and poor prognosis. PCT (procalcitonin) is a protein polypeptide substance with extremely low levels in a healthy body and extremely high levels in severely infected individuals. Generally, the PCT level in mild AP patients is around 0.5–2 ng/L, and a PCT level > 10 ng/L in a few AP patients suggests concurrent sepsis [6].

After the prophylactic use of antibiotics in this study, the levels of the aforementioned inflammatory factors decreased, suggesting that the rational use of antibiotics can rapidly control the progression of inflammation. Moreover, antibiotics can reduce the incidence of infectious pancreatic necrosis, further lowering inflammation levels. Another set of data indicates that prophylactic antibiotic use can alleviate symptoms of acute pancreatitis (AP). The analysis suggests that prophylactic antibiotics effectively prevent and control infections, particularly reducing conditions such as infectious necrosis and peripancreatic infections, which are beneficial for prognosis. However, it should be noted that while prophylactic treatment of AP with piperacillin-tazobactam, levofloxacin, and ornidazole does not directly alleviate AP-related symptoms, controlling the condition through anti-infection pathways can indirectly reduce patient discomfort. The final set of data demonstrates that prophylactic antibiotic therapy for AP can reduce AP-related complications, particularly lowering the rate of infectious pancreatic necrosis. Nevertheless, clinical studies have yielded varying results regarding the prevention of complications through prophylactic antibiotic use. Some scholars argue that prophylactic antibiotics may increase the rate of antibiotic misuse and the risk of multidrug-resistant bacteria, while others believe that prophylactic antibiotic therapy for AP can achieve excellent outcomes and reduce adverse events such as infections.

In the study by Gu Shen and colleagues, targeted therapy and prophylactic antibiotic therapy were administered to AP patients separately, revealing that after prophylactic antibiotic intervention, patients

experienced a lower complication rate and improved quality of life [7]. A thorough analysis of the factors influencing complications in patients with acute pancreatitis (AP) reveals that these are not solely determined by a single factor, namely clinical medication. Rational selection of antibiotics, along with adjustments in the timing and duration of administration, can reduce the incidence of drug resistance and ensure therapeutic efficacy in AP patients.

However, during the actual prophylactic use of antibiotics, bacteria in the intestinal tract may infiltrate the abdominal cavity, increasing the risk of extra-pancreatic infections. Blind selection of antibiotics can lead to dysbiosis, heighten the risk of excessive fungal proliferation, and even result in multidrug resistance.

Therefore, during the prophylactic use of antibiotics for AP treatment, the following considerations should be noted:

(1) Strictly control antibiotic indications

Antibiotics should only be administered to AP patients when there are clear indications for their use, to avoid antibiotic abuse.

(2) Scientifically select the type of antibiotics

It is recommended to use broad-spectrum antibiotics for prophylactic purposes and subsequently adjust the antibiotic regimen based on the patient's condition management. Additionally, when selecting antibiotic types, preference should be given to drugs with excellent pancreatic tissue penetration, enabling rapid penetration through the blood barrier.

(3) Regulate the timing of antibiotic administration and treatment duration

Prophylactic antibiotics should be administered as early as possible in the initial stage of AP, but not for prolonged periods. It is advisable to control the treatment duration to around 7 days. During the actual use of antibiotics, close attention should be paid to fluctuations in the patient's condition and the safety of antibiotic use should be analyzed. In this study, after the prophylactic use of antibiotics, the complication rate in AP patients decreased. However, due to the limited number of AP samples included, there may be biases in predicting infectious pancreatic necrosis.

Therefore, future studies should include a larger number of AP samples and conduct multicenter investigations to explore the safety of prophylactic antibiotic use and its value in reducing infectious pancreatic necrosis events.

5. Conclusion

In summary, during the prophylactic use of antibiotics in AP patients, levels of inflammatory factors decrease, symptoms are alleviated, and therapeutic efficacy is excellent, making it worthy of promotion.

Disclosure statement

The author declares no conflict of interest.

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