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CASE REPORT

Successful Treatment with Sulbactam-Durlobactam for Severe Acute Pancreatitis Complicated by Extensively Drug-Resistant *Acinetobacter baumannii* Infection at Multiple Sites: A Case Report

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Background: The management of severe acute pancreatitis (SAP) complicated by multi-site infections with extensively drug-resistant *Acinetobacter baumannii* (XDR-AB) represents a critical therapeutic challenge. This report describes, to our knowledge, an early successful application of sulbactam-durlobactam in an SAP patient with concurrent XDR-AB infections in the abdomen, biliary tract and lungs.

Case Presentation: A 36-year-old female with hypertriglyceridemic SAP developed infections at multiple sites, with cultures consistently yielding XDR-AB. After the failure of multiple last-line antimicrobial regimens, therapy was switched to sulbactam-durlobactam combined with meropenem. This change was followed by a substantial clinical improvement: body temperature normalized, infection biomarkers decreased significantly and microbiological clearance of XDR-AB from all sites was achieved, leading to successful liberation from mechanical ventilation and discharge from the ICU.

Conclusion: This case demonstrates that sulbactam-durlobactam can be a highly effective therapeutic option for achieving both clinical and microbiological cure in complex SAP cases with multi-site XDR-AB infections that are refractory to conventional treatment regimens. It contributes early real-world evidence supporting a new therapeutic strategy for managing such life-threatening, complex infections.

Keywords: severe acute pancreatitis, *Acinetobacter baumannii*, drug resistance, sulbactam-durlobactam, case report

Introduction

Severe acute pancreatitis (SAP) is a critical illness with high mortality, often driven by secondary infections.[1,2] The pathophysiology of SAP—involving a systemic inflammatory storm, subsequent immunoparalysis, gut barrier dysfunction with bacterial translocation and pancreatic necrosis—creates a high risk for multi-site infections.[3] This vulnerability is compounded by the global rise of carbapenem-resistant Gram-negative bacteria, which poses a severe therapeutic challenge in SAP.[4] Traditional regimens like polymyxin and tigecycline, while possessing some activity, are often suboptimal due to limitations such as uneven tissue distribution and nephrotoxicity.[5]

Sulbactam-durlobactam is a novel β-lactamase inhibitor combination. Durlobactam, a novel diazabicyclooctane β-lactamase inhibitor, potently inhibits Ambler class A, C and D β-lactamases. This action protects sulbactam from hydrolysis and can synergize with other β-lactams, collectively restoring activity against resistant *Acinetobacter baumannii*. This agent was approved by the US Food and Drug Administration in 2023 for treating hospital-acquired bacterial pneumonia and ventilator-associated bacterial pneumonia caused by carbapenem-resistant *Acinetobacter baumannii* (CR-AB).[6] However, robust real-world data on its clinical effectiveness and optimal application in the specific and complex scenario of SAP complicated by multi-site extensively drug-resistant *Acinetobacter baumannii* (XDR-AB) infections are currently lacking.

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This report describes the successful use of sulbactam-durlobactam in a patient with hypertriglyceridemic SAP (HTG-SAP) (Figure 1) and concurrent multi-site XDR-AB infection who had refractory to conventional regimens. This case provides early evidence supporting the use of sulbactam-durlobactam as a potent salvage therapy for complex, multi-site XDR-AB infections refractory to conventional regimens.

Case Presentation

This case report was prepared in accordance with the CARE guidelines (Supplementary File 1).[7] A 36-year-old female with a body mass index of 33.7 kg/m^2 was admitted for persistent mid-upper abdominal pain. Her history included hyperlipidemia and fatty liver. Initial labs showed leukocytosis (11.17×10^9/L), elevated amylase (368 U/L), lipase (1794 U/L), and hypertriglyceridemia (7.93 mmol/L). Abdominal CT confirmed acute pancreatitis (Figure 2). The diagnosis was acute pancreatitis, hyperlipidemia, and fatty liver. Her condition rapidly deteriorated with soaring triglycerides (53.55 mmol/L), rising lipase (2958 U/L), hypocalcemia and hypoxemia, prompting immediate ICU transfer.

Initial Management and Emergence of Multi-Site Infection

In the ICU, she received continuous renal replacement therapy (CRRT) and empiric cefoperazone-sulbactam. Three plasma exchange sessions lowered triglycerides to 4.98 mmol/L. However, evidence of multi-site infections emerged. Repeat CT showed increased peripancreatic exudation, suggesting peritonitis (Figure 2). Percutaneous drainage yielded bloody and dark brown ascitic fluid. On day 3, severe acute respiratory distress syndrome (ARDS) developed, requiring mechanical ventilation. Microbiological cultures were sent from sputum, bronchoalveolar lavage fluid, ascites and bile.

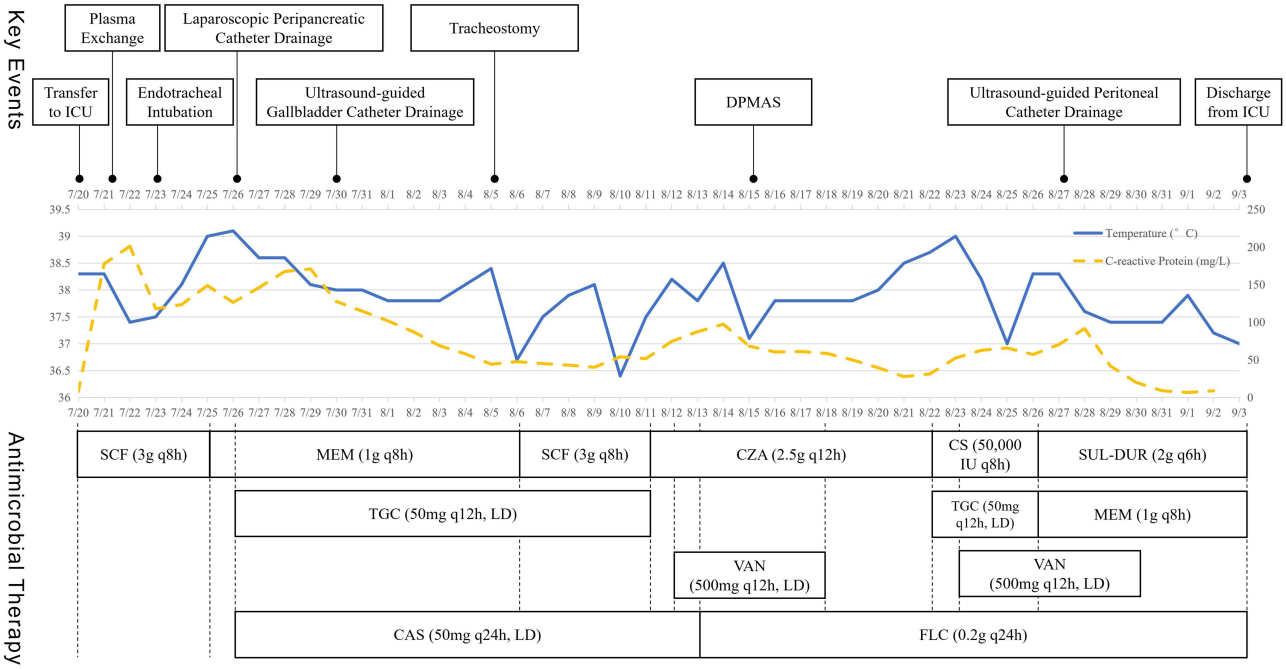

Figure 1 Timeline of Key Clinical Events and Therapeutic Interventions During ICU Stay.

Notes: The upper section of the figure chronologically depicts major clinical milestones, including admission to ICU, therapeutic procedures (eg, plasma exchange, drainage procedures), and respiratory support events (eg, endotracheal intubation, tracheostomy). The lower section illustrates the sequential administration of antimicrobial and antifungal agents, using standard abbreviations. Vertical dashed lines in the lower panel mark the time points of major antimicrobial regimen changes. The Y-axis plots serum C-reactive protein (CRP, mg/L) levels, reflecting the trajectory of the systemic inflammatory response, while the X-axis represents time and is correlated with the patient's body temperature (°C).

Abbreviations: ICU, Intensive Care Unit; LD, Loading Dose; SCF, Cefoperazone-Sulbactam; MEM, Meropenem; CZA, Ceftazidime-Avibactam; CS, Colistin Sulfate; SUL-DUR, Sulbactam-Durlobactam; TGC, Tigecycline; VAN, Vancomycin; CAS, Caspofungin; FLC, Fluconazole.

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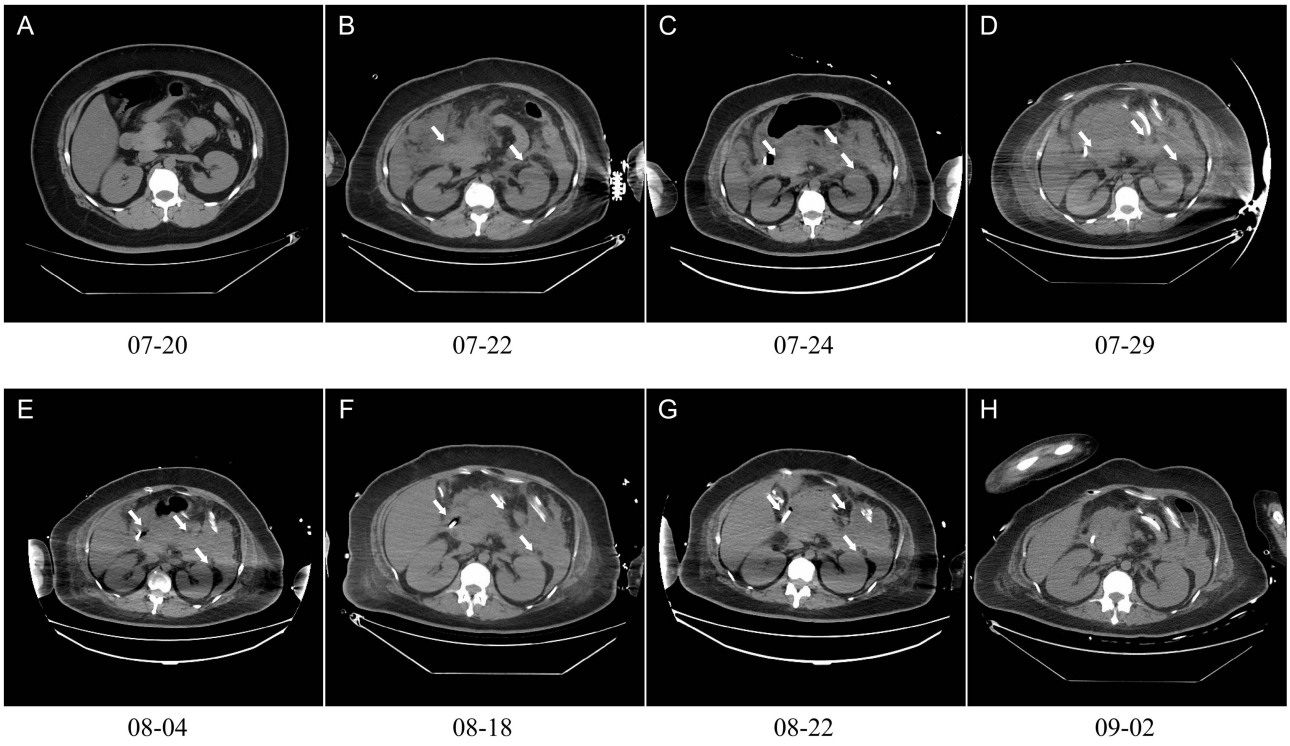

Figure 2 Comparative Serial Axial CT Images at the Level of the Renal Hilum.
Notes: (**A**–**H**) Serial axial non-contrast CT images at the level of the renal hilum obtained on July 20, 22, 24, 29, August 4, 18, 22 and September 2, 2025 respectively. White arrows in (**B**–**G**) highlight key pathological features, including progressive peri-pancreatic infiltration and fluid collections. This series illustrates the dynamic clinical course: (**A**) obtained at initial admission, shows early signs of pancreatitis. By day 3 (**B**), a rapid and significant progression of peri-pancreatic infiltration and fluid collections is evident. Following intensive medical and surgical intervention, subsequent images demonstrate gradual resolution. By day 44, on the eve of ICU discharge (**H**), although edema of the pancreatic head remains notable, the extensive peri-pancreatic exudation has been largely controlled.

Escalating Antimicrobial Challenges and Microbiological Findings

Due to persistent fever and elevated infection biomarkers (procalcitonin (PCT) 4.38 ng/mL, C-reactive protein (CRP) 177.6 mg/L), antimicrobial therapy was escalated to meropenem. Laparoscopic necrosectomy and drainage were performed for worsening abdominal condition. As detailed in Table 1, microbiological data revealed a succession of carbapenem-resistant *Klebsiella pneumoniae* (CR-KP) and CR-AB, which later exhibited an XDR-AB, from abdominal

Table 1 Selected Microbiological Culture Identification and Drug Susceptibility Testing Results

Specimen	Report Date	Culture Result	MIC (mg/L)									
			MEM	IMP	CAZ	FEP	TZP	SCF	AMK	LVX	MIN	PB
Ascites	08/07	*K. pneumoniae*, CR-KP	≥16	≥16	≥32	≥32	≥128/4	≥64/32	≤4	≥8	=8	N/A
	08/14	*A. baumannii*, MDR-AB, CR-AB	≥16	N/A	≥32	≥32	≥128/4	≥64/32	≥64	≥8	≤4	≤2
	09/04	No bacterial growth										
Bile	08/12	*A. baumannii*, MDR-AB, CR-AB	≥16	≥16	≥32	≥32	≥128/4	≥64/32	≥64	≥8	≤4	≤2
	08/26	No bacterial growth										
	09/03	No bacterial growth										
Sputum	08/22	*A. baumannii*, CR-AB	≤1	=8	≤4	=8	≤4/4	=16/8	≥64	=1	≤4	≤2
	09/02	*A. baumannii*, MDR-AB, CR-AB	=4	≥16	≤4	=16	=16/4	≥64/32	≥64	=1	≤4	≥4
	09/04	Normal oropharyngeal flora										

Abbreviations: CR-KP, carbapenem-resistant *Klebsiella pneumoniae*; MDR-AB, multi-drug resistant *Acinetobacter baumannii*; CR-AB, carbapenem-resistant *Acinetobacter baumannii*; MIC, Minimum Inhibitory Concentration; MEM, Meropenem; IMP, Imipenem; CAZ, Ceftazidime; FEP, Cefepime; TZP, Piperacillin-tazobactam; SCF, Cefoperazone-sulbactam; AMK, Amikacin; LVX, Levofloxacin; MIN, Minocycline; PB, Polymyxin B; N/A, Not available.

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and respiratory sites between early and late August. Anti-infective regimens were repeatedly adjusted based on culture and susceptibility results (Table 1), sequentially employing tigecycline, ceftazidime-avibactam, and colistin. Despite these targeted efforts, clinical and biomarker evidence of persistent sepsis indicated uncontrolled abdominal infection.

Initiation of Novel Agent and Clinical Turnaround

On August 27, with recurrent fever (38.3°C), leukocytosis (21.87×10^9/L), and ongoing culture positivity for XDR-AB, therapy was switched to sulbactam-durlobactam combined with meropenem. This intervention marked a clinical turning point. Body temperature and infection biomarkers normalized promptly (CRP decreased from 70.4 mg/L to 8.9 mg/L; PCT fell to <0.10 ng/mL). Microbiological clearance of XDR-AB from all sites was confirmed by September 4. Consequently, oxygenation improved, allowing liberation from mechanical ventilation, and abdominal drainage decreased. After one week of combination therapy, the infection was controlled, and the patient was transferred from the ICU on September 3.

Discussion

The diagnostic and therapeutic course of this patient exemplifies the extreme challenges and potential breakthroughs in managing HTG-SAP complicated by XDR Gram-negative bacterial infections.

Underlying Host Factors and the Pathogenesis of Infection

This young female patient had a background of hyperlipidemia and fatty liver and SAP was triggered by hypertriglyceridemia. HTG-SAP often follows a more severe course. Its pathophysiology involves massive pancreatic enzyme activation, a systemic inflammatory storm and immune dysfunction, creating conditions predisposing to secondary infections.[8] This case demonstrated typical features of SAP-associated infections, namely sequential colonization or infection of multiple sites—abdomen (peripancreatic, ascites), biliary tract and respiratory tract—forming a complex "network of infection foci." The early appearance of pancreatic necrosis and peritonitis, rapidly progressing to severe ARDS requiring mechanical ventilation, highlights the severity and complexity. Although comprehensive management, including CRRT, plasma exchange and surgical debridement/drainage, was implemented early, infection remained uncontrolled, reflecting the collapse of host defense mechanisms in such patients, providing an opportunity for colonization and invasion by resistant bacteria.

The Therapeutic Impasse of Multi-Drug Resistant Infections

The anti-infective course in this case was particularly challenging. Typically, carbapenems, β-lactam/β-lactamase inhibitor combinations, fifth-generation cephalosporins, oxazolidinones and some aminoglycosides show good activity against infections complicating acute pancreatitis.[3] Therefore, during empirical treatment, we continuously evaluated and escalated the regimen, sequentially using broad-spectrum, potent agents like cefoperazone-sulbactam, meropenem, tigecycline and ceftazidime-avibactam, yet with poor efficacy. This could be partly attributed to poor antibiotic penetration into deep-seated abdominal infections, particularly those enclosed within necrotic tissue. Furthermore, microbiological evidence obtained later revealed a more intractable problem: mixed infection with CR-KP and XDR-AB. This aligns with previous reports identifying *Klebsiella pneumoniae* and *Acinetobacter baumannii* as predominant carbapenem-resistant pathogens in SAP.[9] Studies, including one by Chen et al on AP-related infections, have also noted that the emergence of resistant pathogens limits treatment options and can severely impact prognosis.[10] Thus, we considered the presence of these resistant organisms as the primary reason for the prolonged failure to control the infection.

In interpreting these microbiological findings, a critical distinction was made between infection and colonization based on specimen source and clinical context. In this patient, *A. baumannii* isolated from all sites was considered indicative of active infection. This judgment was based on composite criteria: isolation from sterile abdominal/biliary fluids, and for the respiratory tract, concurrent purulent secretions on bronchoscopy, systemic signs of infection (fever, elevated CRP) and the absence of an alternative septic focus. This confirmed a state of disseminated, multi-site infection, underscoring the clinical challenge and validating the goal of microbiological clearance from all compartments.

After obtaining etiological evidence, we escalated therapy based on drug susceptibility and tNGS results, using ceftazidime-avibactam (targeting CR-KP) and later the potent combination of tigecycline plus polymyxin (targeting XDR-AB).[11,12] However, even with these currently considered most powerful antimicrobial weapons, the patient's

clinical and microbiological response remained poor after a minimum treatment period of 4 to 11 days, with persistence of sepsis signs and ongoing culture positivity. The suboptimal performance of polymyxin, in particular, may be attributed to pharmacokinetic variability and unpredictable drug exposure in a patient undergoing continuous renal replacement therapy, compounding the inherent challenge of achieving effective concentrations in deep-seated, necrotic infections.

The Rationale and Efficacy of Sulbactam-Durlobactam in Complex Infections

The therapeutic strategy was guided by the Infectious Diseases Society of America (IDSA) 2024 guidance, which recommends sulbactam-durlobactam in combination with imipenem-cilastatin or meropenem as a preferred regimen for CR-AB infections, a recommendation supported by in vitro synergy data aiming to optimize treatment efficacy.[13,14] Meropenem was selected for this combination based on its favorable safety profile—particularly a lower risk of neurotoxicity compared to imipenem—and its reliable activity against potential co-pathogens such as Pseudomonas aeruginosa.[15] Durlobactam, a novel diazabicyclooctane β-lactamase inhibitor in the combination, exerts potent inhibition against class A, C and D carbapenemases. Its synergy with sulbactam and β-lactam antibiotics (eg, meropenem) creates a triple synergistic effect.[16,17] Crucially, the β-lactamase inhibitors restore the efficacy of β-lactam drugs against β-lactamase-producing pathogens. This mechanism not only effectively targets CR-AB, as in this case, but could also theoretically retain activity against some enzyme-producing CR-KP strains. However, clinical evidence for this indication remains limited, and the drug is not currently approved for CR-KP infections. In our patient, the observed microbiological clearance may have been primarily attributable to its potent activity against CR-AB. In contrast, traditional drugs like polymyxin and tigecycline often struggle to achieve effective therapeutic concentrations simultaneously at all critical infection sites due to issues like poor tissue penetration or low serum levels.[18] The observed rapid clinical and microbiological response following its initiation suggests a key advantage: the ability to achieve effective bactericidal concentrations at multiple, disparate infection sites—a feat often unattainable with older agents like polymyxin or tigecycline due to unfavorable pharmacokinetics.

Regarding drug safety, liver and kidney impairment are common during SAP. Traditional antimicrobials often carry risks of hepatorenal toxicity. During prolonged anti-infective courses, these side effects can limit drug use and exacerbate organ dysfunction.[19] Sulbactam-durlobactam, as a β-lactam derivative, generally has a favorable safety profile. In this patient, who already had liver injury and required ongoing CRRT, its good safety and tolerability were crucial for completing a full course of potent anti-infective therapy. A Phase III clinical trial has confirmed that for patients with CR-AB causing pneumonia, sulbactam-durlobactam demonstrated comparable efficacy to polymyxin-based regimens but with a significantly lower incidence of nephrotoxicity ($P < 0.001$).[20]

Implications and Limitations

This case is among the first reports successfully using sulbactam-durlobactam to rescue a patient with SAP and multi-site XDR-AB infection. It provides crucial, early real-world evidence for infectious disease and critical care specialists facing the challenge of XDR-AB in SAP. It suggests that for complex resistant Gram-negative infections failing conventional combination therapy, especially when the pathogen is carbapenemase-producing *Acinetobacter baumannii*, sulbactam-durlobactam can be a powerful salvage option. It is crucial to emphasize that the success of sulbactam-durlobactam was built upon adequate surgical drainage and organ function support.[21] The drug's mechanism involves killing actively growing bacteria, while surgical drainage removes the massive bacterial reservoir within biofilms and necrotic tissue, reducing the inoculum. Together, they form a successful model combining "chemical eradication" and "physical clearance". This case underscores that in SAP infection control, any potent antimicrobial must be integrated with active surgical intervention and meticulous organ support to achieve maximum efficacy.

This case has limitations. It is a single-center, retrospective report and the successful outcome was achieved on the foundation of comprehensive treatments like surgical drainage and organ support. Furthermore, detailed strain homology analysis and deeper resistance mechanism studies were not performed. Potential confounders such as heteroresistance or site-specific pharmacokinetic variability in SAP patients were not assessed and may represent unmeasured confounders that could influence outcomes. Future larger-sample clinical studies are needed to further confirm its efficacy, optimize dosing regimens and explore resistance evolution.

Conclusion

For the clinical challenge of SAP complicated by multi-site, extensively drug-resistant *Acinetobacter baumannii* infection, the novel β-lactamase inhibitor combination sulbactam-durlobactam demonstrates breakthrough therapeutic potential when conventional regimens fail. This case confirms that this agent can effectively clear resistant bacteria from multiple sites, including the respiratory tract, abdomen and biliary system, achieving both clinical and microbiological cure, thereby providing a new key weapon for managing such critically ill patients.

Data Sharing Statement

All data generated or analyzed during this study are included in this published article.

Ethics Approval and Consent to Participate

This study was approved by the Institutional Ethics Committee of the 967 Hospital, Joint Logistics Support Force of PLA (Approval No: PLA967-GC2025-32). This approval included consent for publication of anonymized case details. Written informed consent was obtained from the patient's legal guardian (father) for publication of this case report, as the patient was bedbound and had limited communication capacity during the consent process.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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