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CASE REPORT

Brucella Infection Associated with Abdominal Aortic Rupture and Retroperitoneal Hematoma: A Case Report

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Background: Involvement of large vessels by *Brucella* infection, particularly leading to abdominal aortic rupture, is exceptionally rare in clinical practice. However, it constitutes a life-threatening condition associated with high mortality rates. Early identification of the infectious etiology combined with timely surgical intervention is crucial for improving patient outcomes.

Case Presentation: A 63-year-old male was admitted to the emergency department due to sudden-onset lower back pain. Computed tomography angiography (CTA) confirmed the diagnosis of abdominal aortic rupture accompanied by a massive retroperitoneal hematoma. Emergency endovascular aortic repair with a stent graft was performed to control the hemorrhage. Postoperatively, the patient continued to experience unexplained fever and fatigue. Routine infection and immunological screenings returned negative results. Subsequent continuous blood culture identified *Brucella* melitensis, leading to a definitive diagnosis of ruptured infectious aortic aneurysm caused by brucellosis. A standard anti-*Brucella* regimen (doxycycline combined with rifampin) was promptly initiated. The patient showed marked improvement in infectious symptoms and was subsequently discharged.

Conclusion: *Brucella* infection may be associated with abdominal aortic rupture, although the exact pathological mechanism remains uncertain. The rupture could be attributable to either an infectious aneurysm or aortitis with bacteremia. Clinicians should consider brucellosis in patients with unexplained aortic pathology, especially those from endemic areas. Prompt microbiological diagnosis and multidisciplinary management are essential.

Keywords: *Brucella*, abdominal aortic rupture, retroperitoneal hematoma, case report

Background

Brucellosis is a zoonotic infectious disease caused by bacteria of the genus *Brucella*. Its clinical manifestations are diverse, predominantly featuring nonspecific symptoms such as fever, profuse sweating, fatigue, and osteoarticular pain.[1] The pathogen is a Gram-negative, facultative intracellular bacterium capable of evading host immune clearance and persisting within the reticuloendothelial system, leading to chronic or relapsing infection. While the disease most commonly affects the osteoarticular, reproductive, and hepatobiliary systems, its hematogenous dissemination can result in rare yet severe vascular complications.[2,3]

Infectious aortic aneurysm represents a life-threatening complication of brucellosis. Although its incidence is low, it is associated with high mortality. When *Brucella* invades the vascular wall, it can induce local inflammation, intimal damage, and medial destruction, ultimately leading to aneurysm formation and potential rupture.[4] The abdominal aorta is relatively less frequently involved, likely due to its specific anatomical and hemodynamic characteristics. However, once an aneurysm ruptures, resulting in a retroperitoneal hematoma, the condition often deteriorates rapidly with a high risk of hemorrhage, posing significant diagnostic and therapeutic challenges.

It is important to distinguish, based on contemporary classifications, between primary bacterial aortitis (direct infection of an intact aortic wall) and secondary infection of a pre-existing degenerative aneurysm. The former may

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lead to focal wall destruction and rupture without a true saccular aneurysm, whereas the latter represents superinfection of an already weakened vessel. This distinction has implications for diagnosis and management, yet it often cannot be made without histopathological examination. In this case, we consider both possibilities.

Currently, reports on abdominal aortic rupture possibly caused by *Brucella* are exceedingly scarce, suggesting a potential gap in clinical awareness. Given the insidious nature, rapid progression, and potential lethality of this complication, heightened clinical vigilance is paramount for early diagnosis and the implementation of a multidisciplinary management strategy encompassing targeted antimicrobial therapy and urgent vascular surgical intervention. This article reports a pathogen-confirmed case of abdominal aortic rupture with retroperitoneal hematoma, in which *Brucella* infection was identified as a possible etiological factor, aiming to provide a reference for the clinical management of such rare and critical emergencies.

Case Presentation

A 63-year-old male was admitted to the emergency department due to "sudden onset lower back pain for 4 hours." The patient experienced abrupt, severe, and persistent lower back pain during physical labor 4 hours prior to admission, accompanied by marked fatigue, night sweats, and generalized myalgia. Past medical history included bilateral knee arthroplasty for osteoarthritis three years ago, with satisfactory postoperative recovery. The patient denied a history of chronic conditions such as hypertension, diabetes, or heart disease, as well as any known history of infectious disease exposure.

Admission Examination and Initial Management: Vital signs on admission were as follows: temperature 36.5°C, pulse 78 beats/min, respiratory rate 20 breaths/min, and blood pressure 156/92 mmHg. Physical examination revealed a slightly distended abdomen without tenderness, rebound tenderness, or guarding. No distinct mass was palpable. Tenderness and percussion pain over the lumbar region were positive. Bilateral femoral, popliteal, dorsalis pedis, and posterior tibial pulses were symmetric and strong. Laboratory tests showed an elevated neutrophil percentage (85.7%) and decreased hemoglobin level (95 g/L). Emergency computed tomography angiography (CTA) of the thoracoabdominal aorta demonstrated a localized rupture in the distal segment of the abdominal aorta associated with a massive retroperitoneal hematoma (Figure 1A).

Emergency Intervention and Postoperative Management: Following the definitive diagnosis, the patient immediately underwent emergency endovascular aortic repair with a stent graft under general anesthesia. The procedure was performed successfully, sealing the rupture site. Two units of suspended red blood cells were transfused intraoperatively to correct anemia, and blood samples were collected for bacterial culture. Postoperatively, the patient was transferred to the intensive care unit (ICU) and received empirical antibiotic therapy (cefuroxime 3.0 g, q12h, intravenous infusion). A follow-up CTA on postoperative day 1 confirmed the stent graft was in a satisfactory position with no significant enlargement of the hematoma (Figure 1B).

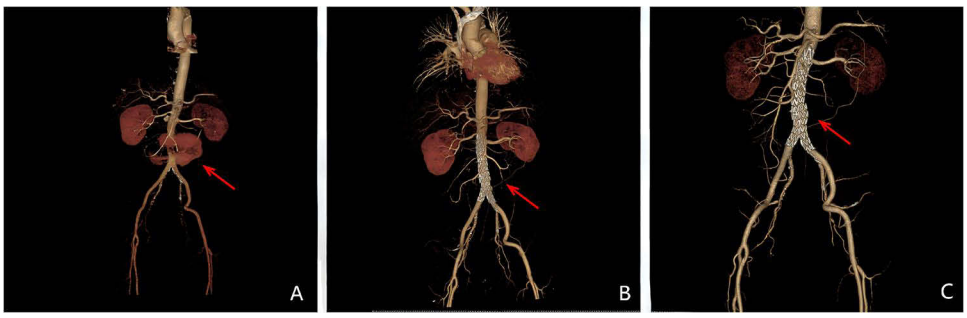

Figure 1 Presents serial computed tomography angiography (CTA) findings of the aorta. (**A**) shows the preoperative CTA image, where a red arrow indicates the site of hemorrhage. (**B**) demonstrates the postoperative CTA image, with a red arrow indicating satisfactory placement of the stent. (**C**), obtained one month after discharge, reveals significant regression of the retroperitoneal hematoma, characterized by marked reduction in both volume and density, with the red arrow pointing to the area of resolution.

Postoperative Clinical Course and Etiological Investigation: The patient continued to experience recurrent low-grade fever (irregular pattern), fatigue, night sweats, and myalgia postoperatively. The initial blood culture report was negative. Given the persistent signs of systemic infection and lack of response to conventional antibiotic therapy, a systematic workup was performed to exclude potential causes including malignancy (relevant tumor markers negative), tuberculosis (T-SPOT and imaging not supportive), autoimmune diseases (rheumatic immunological markers negative), syphilis, HIV, and viral hepatitis (Table 1). During this period, dynamic monitoring of infection markers revealed persistently elevated neutrophil percentage and procalcitonin levels, alongside progressively decreasing lymphocyte and eosinophil percentages (Table 2). At this point, epidemiological history was re-evaluated in detail; however, the patient still denied any definite history of livestock contact. Additional venous blood samples for culture were collected from both upper and lower limbs on postoperative days 2 and 3.

Definitive Diagnosis and Targeted Therapy: On postoperative day 3, both sets of blood cultures yielded growth of *Brucella melitensis*. The antibiotic regimen was promptly adjusted to minocycline (100 mg, bid, oral) combined with rifampin (900 mg, qd, oral). Following the initiation of targeted therapy, the patient's body temperature rapidly normalized, with marked alleviation of fatigue, night sweats, and myalgia. Corresponding improvement was observed in laboratory parameters: the neutrophil percentage and procalcitonin level gradually decreased to the normal range, while the lymphocyte and eosinophil percentages recovered to normal levels (Table 2). The patient's condition stabilized, and he was discharged with an adjusted medication regimen of doxycycline (100 mg, bid, oral) combined with rifampin (900 mg, qd, oral), with instructions to complete the full course of treatment.

Follow-up: The patient returned for a follow-up visit one month after discharge, reporting complete resolution of all previous symptoms. Laboratory tests indicated that all infection markers had returned to normal ranges (Table 2). Abdominal CTA demonstrated significant absorption and reduction in the size of the retroperitoneal hematoma, with decreased density (Figure 1C). The stent graft was patent without evidence of endoleak or migration.

Table 1 Analysis of Infection Indicators

		Detection Time												
	Reference Range	**Preoperative**	**D0**	**D1**	**D2**	**D3**	**D4**	**D5**	**D6**	**D7**	**D9**	**D10**	**D11**	**Month.After.Surgery**
Temperature (°C)	36-37.3	37.3	37.1	37.2	38.0	38.3	37.5	37.4	36.5	37.1	37.0	36.5	36.5	36.4
White blood cell count (×10^9/L)	3.5–9.5	8.55	7.74	5.05	3.57	7.04	7.02	7.23	7.87	9.07	7.18	5.24	5.24	5.67
Neutrophil percentage (%)	40.0–75	85.7	82.8	88.9	85.4	78.1	77.8	78.1	81.2	80.1	75.8	69.8	69.8	66.5
Lymphocyte percentage (%)	20.0–50.0	8.4	9.7	7.7	11.2	13.9	15.0	15.9	13.7	13.7	15.9	20.4	20.4	22.2
Eosinophil percentage (%)	0.4–8.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.3	0.3	0.8	1.0	1.0	3.2
Procalcitonin (PCT)	<0.250	0.41	0.38	0.42	0.38	0.44	0.31	0.22	0.16	0.11	-	-	-	0.10

Notes: Table 1 summarizes the analysis of infection-related laboratory indicators throughout the clinical course. Body temperature is axillary temperature; infection indicators are all collected from venous blood. Cefuroxime was used for anti-infective treatment during D0-D2. Minocycline + rifampicin were used for anti-infective treatment during the period after D3.

Table 2 Results of Microbiologic and Serologic Testing

Variable	**Result**	**Reference Range**
Tumor markers		
Alpha-fetoprotein	1.03	0-7.00 (ng/mL)
Carcinoembryonic antigen	3.0	0-4.70 (ng/mL)
Carbohydrate antigen 125	4.80	0-35 (U/mL)
Carbohydrate antigen 153	8.77	0-25 (U/mL)
Carbohydrate antigen 199	7.99	0-39 (U/mL)

(*Continued*)

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Table 2 (Continued).

Variable	Result	Reference Range
Mycobacterium tuberculosis related indicators		
Acid fast staining	Continuous observation of 300 different fields of view, no acid-fast bacilli were found	
Mycobacterium tuberculosis complex nucleic acid	Negative	Negative
Tuberculosis-infected T cell negative control	1.10 (pg/mL)	
Tuberculosis infection T cell stimulation level	2.10 (pg/mL)	
Tuberculosis-infected T cell positive control	650.50 (pg/mL)	
Stimulates T cells - background N levels	1.00 (pg/mL)	0-14 or N<4
Tuberculosis infection T cell test results	Negative	Negative
Immune-related indicators		
Antinuclear antibody spectrum	Negative	Negative
Anti-cyclic citrullinated peptide antibody	0.95 (RU/mL)	<5.00
Anti-neutrophil cytoplasmic antibody (cANCA)	Negative	
cANCA	Negative	
pANCA	Negative	
aANCA	Negative	
Protease 3	0.56 (RU/mL)	<20
Myeloperoxidase (MPO)	0.80 (RU/mL)	<20
Acquired Immune Deficiency Syndrome (AIDS) and Syphilis		
AIDS	Negative	
Syphilis	Negative	
Blood culture		
Intraoperative inspection	Negative	
First postoperative	Negative	
Second postoperative	*Brucella melitensis*	
Third postoperative	*Brucella melitensis*	

Notes: Table 2 details the results of microbiologic cultures and serologic testing performed during the patient's evaluation.

Discussion

Brucella infection of the abdominal aorta leading to aneurysm formation is extremely rare in clinical practice, yet it represents one of the most severe vascular complications of brucellosis.[5] Such infected aneurysms are characterized by rapid progression and a high propensity for rupture. Once rupture occurs, it results in retroperitoneal hematoma, and death in such cases is typically caused by hemorrhagic shock. Consequently, the cornerstone of clinical management lies in early recognition and emergent surgical intervention. Postoperatively, close monitoring of hemoglobin levels and hemodynamic status is essential for detecting delayed hemorrhage.

Current understanding of the clinical presentation is largely derived from case reports. This case demonstrates that symptoms may result from the combined effects of systemic brucellosis and the local impact of the aneurysm. Patients may present with non-specific systemic symptoms such as fever, night sweats, fatigue, and myalgia,[6,7] or the initial symptoms may be dominated by acute back pain caused by aneurysm expansion or rupture. Notably, in this case, despite rupture, distal arterial pulses remained normal. This suggests that the pathological process may be confined to a segment of the aorta without affecting blood flow through the major branches. However, this feature may lead to diagnostic confusion with lumbar spine pathology, potentially resulting in delayed diagnosis.

Regarding etiological diagnosis, this case illustrates a systematic approach to differential diagnosis. Persistent postoperative fever remained a key clue throughout the diagnostic process. Common causes that needed sequential exclusion included:[8,9] 1) Infectious etiologies: Mycobacterium tuberculosis is an important pathogen in infected aneurysms.[10] Although the patient had systemic symptoms such as night sweats and fatigue, specific tests for tuberculosis

(interferon-gamma release assay and imaging) were negative, effectively ruling out this etiology. 2) Neoplastic etiologies:[11,12] Given the patient's age and the nature of the aneurysm, malignancies such as primary angiosarcoma or metastatic carcinoma required consideration. Comprehensive tumor marker testing and imaging assessment revealed no supportive evidence. 3) Non-infectious inflammatory diseases:[13] Large-vessel vasculitis and other rheumatic immune diseases can cause aneurysmal lesions. In this case, no abnormal rheumatologic antibodies or associated inflammatory markers were found, arguing against such diagnoses. 4) Hereditary connective tissue disorders: Conditions such as Marfan syndrome are common causes of aortic aneurysms in young patients.[14,15] This was highly unlikely given the absence of a relevant family history and the lack of typical phenotypic features.

In this context, it is necessary to consider other Gram-negative bacteria that cause similar vascular injury by intracellular mechanisms. Coxiella burnetii (the causative agent of Q fever) is a classic example: in an epidemic region in the Netherlands, 16.9% of 770 patients with abdominal aortic/iliac disease were seropositive, and 30.8% of those had chronic Q fever; patients with chronic infection had significantly higher rates of acute aneurysm-related complications (rupture, symptomatic aneurysm, aorto-duodenal fistula) compared to seronegative patients (30.0% vs. 9.0%, respectively, P=0.013).[16] Furthermore, pre-existing aortic aneurysm or vascular graft was an independent risk factor for developing chronic Q fever (odds ratios 25.9 and 26.8, respectively).[17] These findings align with the biological characteristics of brucellosis: pre-existing aortic pathology provides a niche for intracellular bacteria, leading to rapid vessel wall destruction. Therefore, in patients with unexplained fever and aortic aneurysm, clinicians should consider a possible diagnosis of infected aneurysm.[18] Obtaining microbiological evidence is critical to guide therapy. In this case, initial blood cultures were negative, possibly due to transient bacterial suppression by perioperative empirical antibiotics. Successful isolation of *Brucella melitensis* was achieved through standardized repeat blood culture sampling at different time points and from different sites (upper and lower extremities). This facilitated the transition from empirical to targeted therapy. This process demonstrates that in suspected endovascular infection, obtaining multiple blood culture sets from different sites is essential to maximize pathogen detection yield.

Nevertheless, the exact mechanism by which *Brucella* infection leads to aortic rupture in this setting remains unclear. Based on available clinical and microbiological data, two non-mutually exclusive possibilities may explain this association: ① Infected aneurysm – *Brucella* may directly invade the aortic wall, inducing local inflammation, medial necrosis, and saccular aneurysm formation, culminating in rupture.② Bacterial aortitis – The patient may have had pre-existing degenerative aortic disease, and *Brucella* bacteremia led to secondary infection of the aortic wall (so-called "bacterial aortitis"), causing local destruction and rupture without true aneurysm formation.

Preoperative CTA did not reveal typical features of a mycotic aneurysm (eg., saccular shape, periaortic soft tissue). Therefore, we cannot definitively distinguish between these two mechanisms. We propose that *Brucella*-associated aortic rupture may result from either infected aneurysm or aortitis with bacteremia, and clinicians should consider both possibilities when encountering similar cases.

In this emergency setting, given the patient's severe hemodynamic instability and the absence of obvious purulent fluid collections or mycotic aneurysm morphology on imaging, EVAR was selected over open debridement with in-situ or extra-anatomic bypass. However, the use of stent grafts in infected fields remains controversial, as it may serve as a nidus for persistent or recurrent infection. In this case, rapid detection of *Brucella* and initiation of targeted dual antibiotic therapy likely contributed to the successful outcome without graft infection. Nonetheless, long-term surveillance is required.

We observed only a modest elevation in plasma procalcitonin (PCT) levels in this patient, consistent with previous findings that brucellosis typically does not cause marked PCT elevation (unlike Gram-negative bacterial sepsis). This pattern may help distinguish brucellosis from other bacterial infections when clinical suspicion exists.

From an infectious disease perspective, this case offers several important insights: Vascular complications of brucellosis may initially present as critical conditions such as aneurysm rupture, while typical features such as undulant fever or clear exposure history in an endemic area may be absent, greatly increasing diagnostic difficulty[19,20.] The pathogenesis involves hematogenous dissemination of *Brucella*, bacterial adhesion to damaged vascular endothelium, and intracellular survival and replication within macrophages. This triggers local granulomatous inflammation and structural destruction of the vessel wall, ultimately leading to aneurysm formation and possible rupture.[5,21] Therefore,

in patients presenting with unexplained fever accompanied by back pain and abnormal inflammatory markers (eg., elevated neutrophil percentage, elevated PCT, decreased lymphocyte or eosinophil percentage), particularly those with potential epidemiological risk factors, clinicians should maintain a high degree of suspicion for *Brucella* aortitis. Once suspected, emergency thoracic and abdominal aortic CTA is the imaging modality of choice to assess aortic morphology and confirm the presence of aneurysm or rupture.

The management team must adhere to the principle of multidisciplinary collaboration, which is based on two core elements: first, immediate vascular surgical or endovascular intervention to resolve the rupture crisis and control hemorrhage; second, prompt initiation of a prolonged, combination antibiotic regimen with good intracellular penetration (eg., doxycycline plus rifampicin) based on microbiological findings. This dual strategy is essential to eradicate intracellular bacteria, control infection, prevent relapse, and ultimately improve prognosis.

Short-term doxycycline (6 weeks to 3 months) in patients with abdominal aortic aneurysm reduces neutrophil and cytotoxic T-cell infiltration within the aortic wall and lowers levels of pro-inflammatory chemokines such as IL-6 and IL-8. This selective anti-inflammatory effect helps stabilize the infected aortic wall and reduces the risk of early rupture.[22] The favorable short-term prognosis in this case, achieved with emergency stent grafting followed by combination doxycycline, may be explained by this mechanism.

However, short-term benefits cannot be extrapolated to long-term therapy. In an already compromised aorta, long-term doxycycline or potent anti-inflammatory treatment may be detrimental.[23] Transcriptomic analysis has revealed significant metabolic reprogramming in human abdominal aortic aneurysms and in the angiotensin II (AngII) mouse model (but not the elastase model): downregulation of oxidative phosphorylation and upregulation of glycolysis, suggesting mitochondrial dysfunction in aortic smooth muscle cells.[24] Treatment with the glycolysis inhibitor PFK15 in the AngII model significantly reduced aneurysm formation (25% in treated group vs. 71% in controls).[24] Mitochondria, which are of bacterial origin, are susceptible to tetracyclines; these agents inhibit mitochondrial protein synthesis.[25] In a metabolically depleted aneurysmal aorta,[26] long-term doxycycline may further suppress mitochondrial bioenergetic metabolism, accelerating cell loss and promoting matrix degradation.[27] Therefore, in the present case, short-term (eg., 6 weeks) doxycycline was necessary and relatively safe, but long-term or repeated use may impose additional iatrogenic stress on a vulnerable aortic wall.

Mechanistic studies further confirm that doxycycline directly impairs mitochondrial function in cardiac and aortic smooth muscle cells (SMCs). In cultured human aortic SMCs, doxycycline induces mitochondrial protein imbalance, reduces mitochondrial membrane potential and oxygen consumption, and downregulates contractile proteins such as α-smooth muscle actin and calponin, effectively promoting SMC dedifferentiation.[28] Studies in cardiomyocytes show that doxycycline shifts cellular metabolism from oxidative phosphorylation to glycolysis, increases mitochondrial fragmentation, and impairs contractile reserve, an effect that is amplified in metabolically vulnerable states such as diabetes.[29]

Based on the above evidence, the following practical recommendations are proposed for similar cases: (1) Timely surgical control combined with targeted long-course antibiotics remains the standard of care; (2) For unexplained fever with aortic pathology, *Brucella*, *Coxiella*, and other intracellular pathogens should be considered; (3) Doxycycline is a double-edged sword – short courses are necessary, but ultra-long courses should be used with caution in patients with pre-existing aneurysms; (4) Adjunctive therapies that support mitochondrial health (eg., metformin) warrant further investigation; (5) Long-term surveillance is required after EVAR for infected aneurysms. In summary, this case of aortic aneurysm rupture due to *Brucella* highlights the intersection of infectious disease and vascular pathology and underscores that even necessary therapies may have unintended adverse effects on a metabolically compromised aortic wall, necessitating a nuanced, individualized approach to achieve optimal outcomes.

Conclusion

Brucella is an obligate intracellular pathogen. In this case, *Brucella melitensis* infection was temporally associated with abdominal aortic rupture and retroperitoneal hematoma. The exact pathological relationship remains to be clarified, but the rupture may be explained by either an infectious aneurysm or aortitis secondary to bacteremia—or a combination of both.

The primary diagnostic challenge lies in the frequently atypical systemic manifestations and the difficulty in isolating the pathogen. Definitive etiological evaluation requires standardized, repeated blood cultures integrated with comprehensive imaging. The cornerstone of successful management is multidisciplinary collaboration, combining urgent

vascular intervention (EVAR or open surgery) with timely, long-term combination antibiotic therapy guided by microbiological findings, to eradicate the infection and prevent recurrence.

Clinicians should be aware that brucellosis can be associated with life-threatening aortic rupture, even in the absence of classic risk factors or typical imaging features of an infectious aneurysm.

We acknowledge an important limitation of this case report: the absence of histopathological or microbiological examination of the aortic wall itself. Aneurysm wall biopsy or culture would have definitively distinguished between a primary infectious aneurysm (ie., Brucella-induced saccular aneurysm with wall degeneration) and *Brucella* aortitis leading to focal wall destruction without a pre-existing aneurysm, or secondary infection of a degenerative aneurysm. The preoperative CTA did not show typical features of an infectious aneurysm. Therefore, we emphasize that while a temporal association exists, causality cannot be firmly established. The causal relationship should be interpreted with caution, though the temporal association, positive blood cultures, and rapid response to anti-*Brucella* therapy strongly support a causative role of *Brucella*.

Reporting Standards

This case report was prepared in accordance with the CARE guidelines.[30]

Data Sharing Statement

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

Ethical Approval

Written informed consent was obtained from the patient for the publication of all the images and data included in this article. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). Ethical review and approval were not required to publish the case details in accordance with the institutional requirements.

Consent to Publish

The patient was admitted to the ICU in an unawakened state immediately after surgery; therefore, initial informed consent for publication was obtained from his family. After the patient regained consciousness, he also provided his own written informed consent. The final consent to publish is based on the patient's own authorization.

Author Contributions

Tenghao Shao: Conceptualization, Investigation, Data curation, Writing – original draft, Writing – review & editing.

Ping Sheng: Conceptualization, Methodology, Writing – original draft, Writing – review & editing, Supervision.

Ning Chen: Conceptualization, Data curation, Writing – review & editing.

Qinlong Sun: Data curation, Writing – review & editing.

All authors made a significant contribution to the work reported, whether 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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