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CASE REPORT

Brucellosis Presenting with Hemorrhagic Manifestations and Severe Thrombocytopenia: A Case Report

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Background: Brucellosis is a global zoonosis, commonly presenting with mild anemia and leukopenia, while severe thrombocytopenia with hemorrhagic manifestations as the initial symptom is extremely rare and easily misdiagnosed as primary hematological diseases.

Case Presentation: A 57-year-old male farmer presented with progressive fatigue, anorexia, diffuse cutaneous petechiae, oral blood blisters, hematuria, melena, and acute dyspnea. Laboratory tests showed a platelet count of 2×10^9/L. The white blood cell count was at the lower limit of normal (WBC 3.79×10^9/L), and elevated ferritin (876.9 ng/mL) was noted. Initial bone marrow aspiration suggested primary immune thrombocytopenia (ITP). The patient received intravenous immunoglobulin (IVIG) 400 mg/kg/d for 5 consecutive days and eltrombopag 50 mg/d orally for 10 days, but platelet counts remained unimproved. Subsequent blood culture identified *Brucella melitensis*, and Brucella agglutination test titer was >1:400. Targeted antimicrobial therapy (doxycycline + rifampin) combined with dexamethasone 10 mg/d intravenously for 3 days, then tapered by 2 mg every 2 days until discontinuation (total course 10 days) was initiated. Platelet counts began to rise on the 6th day of antimicrobial therapy, reached 30×10^9/L at 5 weeks, and normalized to 90×10^9/L at 10 weeks.

Conclusion: Initial misdiagnosis as ITP was due to overlapping clinical and laboratory features (severe thrombocytopenia, positive anti-platelet antibodies, and bone marrow megakaryocyte maturation arrest). Key diagnostic clues included epidemiological history of sheep contact and positive *Brucella* serology/culture. Targeted antimicrobial therapy combined with short-course corticosteroids remains the cornerstone of treatment for this rare presentation.

Keywords: brucellosis, thrombocytopenia, hemorrhagic manifestations, zoonosis, diagnosis, differential

Introduction

Brucellosis is a zoonotic disease caused by bacteria of the genus *Brucella*. This genus encompasses six primary species: *B. abortus* (bovine), *B. suis* (porcine), *B. melitensis* (ovine/caprine), *B. canis* (canine), *B. ovis*, and *B. neotomae*. Among these, *B. abortus*, *B. melitensis*, *B. suis*, and *B. canis* are pathogenic to humans, with *B. melitensis* being the most virulent and associated with the highest incidence of severe complications, including hematological abnormalities.[1–3] Human infection involves multiple systems, with typical manifestations such as fever, night sweats, arthralgia, and myalgia.[4]

Hematological involvement, though relatively common in brucellosis, is usually mild, predominantly presenting as mild anemia and leukopenia.[5] Severe thrombocytopenia leading to hemorrhagic manifestations is considerably rare, with reported incidence rates ranging from approximately 1% to 8%.[6,7] This rarity often leads to misdiagnosis as primary hematological disorders such as immune thrombocytopenic purpura (ITP).[8] Because brucellosis can present with severe thrombocytopenia, positive anti-platelet antibodies, and megakaryocyte maturation arrest on bone marrow, it shares significant diagnostic overlap with primary ITP.[9–11] Recent studies (2024–2025) have shown that brucellosis-related hematological complications are

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increasingly recognized in clinical practice.[12–14] The proposed pathogenic mechanisms of thrombocytopenia in brucellosis include immune-mediated platelet destruction, hypersplenism, bone marrow suppression, and hemophagocytosis.[15] However, the pathogenesis of severe thrombocytopenia remains incompletely understood.

This case report aims to: (1) describe a rare case of B. melitensis infection presenting with one of the most severe degrees of thrombocytopenia documented in the published literature (PLT nadir 2×10^9/L); (2) document, for the first time in a single case report, the sequential failure of four distinct ITP-directed therapies — intravenous immunoglobulin (IVIG), eltrombopag, recombinant human thrombopoietin (rhTPO), and platelet transfusion — prior to etiological diagnosis, underscoring a critical and underappreciated diagnostic pitfall; (3) provide a well-documented, quantitative longitudinal trajectory of platelet recovery following targeted combined antimicrobial and corticosteroid therapy; (4) discuss possible pathogenic mechanisms including the confounding role of concurrent vitamin B12 deficiency; and (5) through a comparative summary of previously published cases, contextualise the clinical and therapeutic features of this case within the broader literature, and emphasise the importance of brucellosis screening in patients with unexplained severe thrombocytopenia that is refractory to standard ITP therapy, particularly in endemic regions.

Case Report

Demographic and Clinical Details

A 57-year-old male farmer from a pastoral area in Shanxi Province, China, presented to the Emergency Department of the Second Hospital of Shanxi Medical University on March 1, 2024. He had a 10-year history of daily direct contact with sheep, including feeding, shearing, and assisting in lambing, without using protective equipment (eg, gloves, masks). He had no history of autoimmune diseases, liver/kidney dysfunction, hypertension, diabetes, or medication use (eg, immunosuppressants, non-steroidal anti-inflammatory drugs) prior to admission.

The patient developed unexplained fatigue and anorexia 20 days prior to admission. One week before presentation, he developed scattered petechiae over the entire body, accompanied by oral blood blisters, gross hematuria, and melena. On the morning of admission, he experienced sudden severe dyspnea, prompting emergency medical attention. The acute dyspnea was considered to be related to acute anxiety secondary to the sudden onset of gross hematuria and oral bleeding, as his SpO2 remained normal and the symptom resolved spontaneously without specific respiratory intervention.

Physical examination on admission: Temperature 36.4°C, respiratory rate 22 breaths/min, heart rate 70 beats/min, blood pressure 118/67 mmHg, oxygen saturation (SpO_2) 99%. The patient was alert but listless and pale. Scattered petechiae and ecchymoses (0.5–2 cm in diameter) were observed on the trunk and extremities. Several blood blisters (3–5 mm) with active bleeding were noted on the right buccal mucosa and tongue surface, some of which had ruptured and formed crusts. No superficial lymphadenopathy was palpated. Cardiac and pulmonary examinations were unremarkable. Abdominal examination revealed mild splenomegaly (palpable 2 cm below the left costal margin). Neurological examination showed no abnormalities.

Laboratory Investigations

Routine Blood Tests

All routine blood test results are summarized in Table 1. The key abnormalities included severe thrombocytopenia, mild anemia, elevated inflammatory markers (CRP, PCT), and significantly decreased vitamin B12 (98.81 pmol/L) which provided important clues for subsequent etiological diagnosis and treatment response evaluation. Despite clinical hemorrhagic manifestations, his hemoglobin level remained within the normal range (133 g/L), likely because the bleeding was acute and early, which was insufficient to significantly impact hemoglobin levels at the time of presentation.

Bone Marrow Aspiration and Cytology

Bone marrow aspiration was performed at the posterior superior iliac spine with 0.5 mL of bone marrow aspirate obtained. Smears were prepared using the Wright-Giemsa staining method. Bone marrow cytology showed hypercellular marrow (cellularity 80%). Megakaryocytes were normal in number, though at the higher end of the spectrum (28 per low-power field, reference range: 7–35), with maturation arrest predominantly at the granular stage (85% granular megakaryocytes, 10% immature megakaryocytes, 5% mature megakaryocytes). Platelet production was significantly reduced,

Table 1 Complete Laboratory Test Results

Item	Result	Unit	Reference Range
White Blood Cell (WBC)	3.79	$\times10^9$/L	3.5–9.5
Red Blood Cell (RBC)	3.96	$\times10^{12}$/L	4.3–5.8
Platelet (PLT)	2	$\times10^9$/L	125 - 350
Hemoglobin (Hb)	133	g/L	130 - 175
Neutrophil	1.85	$\times10^9$/L	1.8–6.3
C - Reactive Protein (CRP)	30.21	mg/L	0 - 4
Procalcitonin (PCT)	1.12	ng/mL	0 - 0.51
Reticulocyte	0.011	-	0.005–0.015
Prothrombin Time (PT)	14.6	s	13.5–15.5
Activated Partial Thromboplastin Time (APTT)	35.9	s	30 - 45
Fibrinogen	3.02	g/L	2 - 4
Folic Acid	28.48	nmol/L	13.4–56.2
Ferritin	876.9	ng/mL	23.9–336.2
Erythropoietin	14.33	mIU/mL	2.59–18.5
Vitamin B12	98.81	pmol/L	133–675

with rare platelets seen in the smear. Other hematopoietic lineages (erythroid, myeloid) showed normal maturation and morphology.

Microbiological and Serological Tests

Blood culture was performed using the BACTEC FX40 system (BD, USA) with tryptic soy broth (TSB) medium, incubated at 35°C in 5% CO_2 for 7 days. Positive growth was detected on the 5th day of culture. Identification of the pathogen was performed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS, Bruker Daltonics, Germany), confirming B. melitensis.

Brucella agglutination test was performed using the standard tube agglutination method. The titer was >1:400 (reference range: <1:100), confirming acute infection.

Imaging Investigations

Abdominal ultrasound: Probe frequency 3.5 MHz. Findings: Fatty liver; cholesterol crystal deposition in the gallbladder; splenomegaly (spleen thickness 4.8 cm, length 14.5 cm; reference range: thickness <4.0 cm, length <12.0 cm).

Chest, abdominal, and head CT: Scan layer thickness 5 mm, scan range covering the entire chest, abdomen, and brain. No abnormalities were detected (eg, pulmonary infiltration, abdominal organ lesions, intracranial hemorrhage).

Treatment and Clinical Course

The patient was admitted with a preliminary diagnosis of primary immune thrombocytopenia (ITP) complicated by severe hemorrhagic manifestations, and initial treatment was initiated as follows:

Platelet transfusion: 1 U of apheresis platelets (indication: PLT <10×10^9/L with active hemorrhage) on admission; post-transfusion PLT was 3×10^9/L (assessed 24 hours later), indicating poor response.

Intravenous immunoglobulin (IVIG): 400 mg/kg/d for 5 consecutive days (March 1–5, 2024).

Eltrombopag: 50 mg/d orally (March 2–11, 2024, 10 days total).

Recombinant human thrombopoietin (rhTPO): 15,000 U/d subcutaneously (March 1–7, 2024, 7 days total).

Hemostatic agents: Tranexamic acid 1 g intravenously twice daily for 5 days.

After 3 days of treatment, mucocutaneous/gastrointestinal bleeding stopped and dyspnea resolved (March 3, 2024), but platelets remained low (1–4×10^9/L).

On March 6, 2024 (6th hospital day), blood culture confirmed *Brucella melitensis* infection. Combined with the patient's sheep contact history, the final diagnosis was brucellosis complicated by severe thrombocytopenia. The treatment regimen was adjusted to:

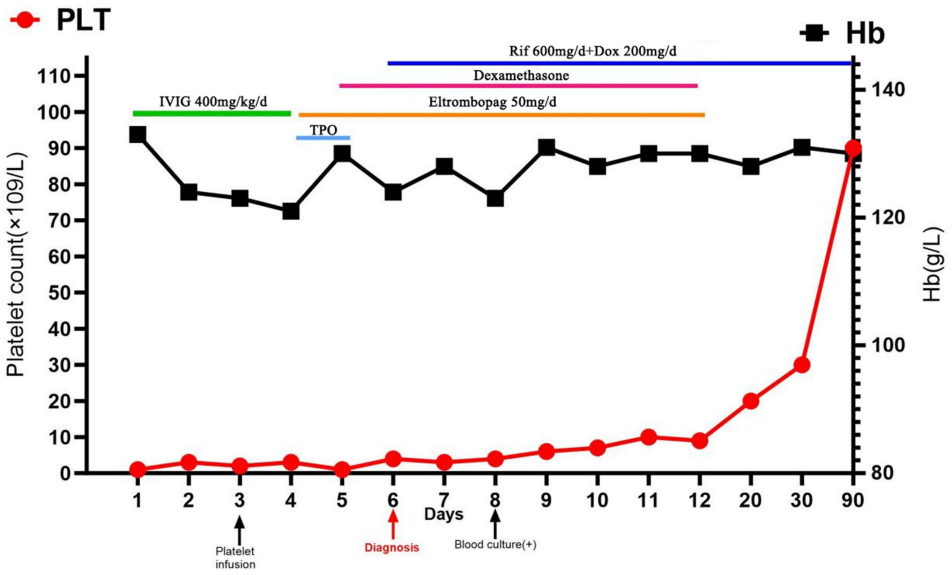

Figure 1 The changes of platelet count and hemoglobin during the treatment on admission. The patient was initially treated with intravenous immunoglobulin (IVIG), recombinant human thrombopoietin and platelet transfusion which were not effective. Then eltrombopag was initiated. On the sixth day of his admission, the patient was diagnosed with brucellosis and started on combined antibiotic and glucocorticoid therapy. The patient's platelet count gradually normalized. The horizontal axis represents the days of admission, the left vertical axis represents the platelet count, and the right vertical axis represents hemoglobin. (+) indicates a positive blood culture result.
Abbreviations: IVIG, immunoglobulin; Rif, rifampicin; Dox, doxycycline; TPO, recombinant human thrombopoietin.

Antimicrobial therapy: Oral doxycycline 100 mg twice daily + rifampin 600 mg once daily (March 6-April 30, 2024, 8 weeks total).

Corticosteroids: Intravenous dexamethasone 10 mg/d for 3 days (March 6–8, 2024), then tapered (8 mg/d for 2 days, 6 mg/d for 2 days, 4 mg/d for 2 days, 2 mg/d for 1 day; 10 days total).

Platelets began to rise on March 11, 2024 (6th day of antimicrobial therapy), reaching 10×10^9/L on March 13, 30×10^9/L on April 10 (5 weeks post-adjustment), and 90×10^9/L on May 15 (10 weeks post-adjustment). CRP (2.1 mg/L) and PCT (0.15 ng/mL) normalized by April 10. Follow-up abdominal ultrasound on May 15 showed resolved splenomegaly (thickness 3.8 cm, length 11.5 cm). Figure 1 illustrates the dynamic changes in platelet count and hemoglobin throughout the treatment course after admission. It clearly shows the ineffectiveness of initial therapies (IVIG, rhTPO, and platelet transfusion) and the subsequent gradual normalization of platelet count following the initiation of targeted antimicrobial (rifampin + doxycycline) and glucocorticoid (dexamethasone) therapy, while hemoglobin remained stable.

Follow-Up

The patient was discharged on April 30, 2024, and completed the 8-week antimicrobial course. Follow-up was conducted via outpatient visits and telephone calls at 1, 3, 6, and 12 months after discharge. Platelet counts remained within the normal range ($120–250\times10^9$/L) during follow-up, with no recurrence of bleeding or brucellosis-related symptoms. Anti-platelet antibodies normalized at 3 months (IgA 0.8 U/mL, IgG 1.2 U/mL, IgM 0.9 U/mL).

Discussion

In brucellosis, severe thrombocytopenia remains a rare presentation.[16] The precise pathogenesis of thrombocytopenia in brucellosis is not fully elucidated. Proposed mechanisms include: (i) increased platelet destruction, potentially mediated by endotoxins, exotoxins, or the production of platelet-associated antibodies; (ii) hypersplenism, leading to platelet sequestration in the spleen; (iii) hemophagocytic syndrome, involving phagocytosis of platelets by histiocytes; (iv) decreased platelet production due to bone marrow suppression; and (v) abnormal platelet adhesion to vascular surfaces or disseminated intravascular coagulation.

Some studies suggest that in cases like this, thrombocytopenia may primarily result from an immune-mediated cross-reaction, where anti-*Brucella* antibodies inadvertently target platelets, accelerating their destruction. This process can manifest with features overlapping both primary immune thrombocytopenia (ITP) and hypersplenism.[17] In the present case, the positivity for anti-platelet antibodies and the bone marrow finding of normal (but at the higher end) megakaryocyte numbers with maturation arrest—a morphological feature strongly mimicking primary ITP—along with the patient's favorable response to corticosteroids combined with anti-brucellosis therapy, support an immune-mediated component. Alternatively, other perspectives posit that thrombocytopenia in brucellosis is often multifactorial (involving bone marrow suppression, hypersplenism, etc).[18] While this multifactorial nature appears common in brucellosis-associated cytopenias, some argue that only autoimmune platelet destruction is likely to be severe enough to become life-threatening. Of note, our patient also had a decreased vitamin B12 level (98.81 pmol/L). While vitamin B12 deficiency is a well-established cause of thrombocytopenia, the rapid and sustained platelet recovery following targeted anti-brucellosis and corticosteroid therapy, without specific vitamin B12 supplementation, suggests that brucellosis was the primary driver of the severe thrombocytopenia in this instance.

Studies suggest that infection-induced immune-mediated thrombocytopenia shares pathogenic similarities with ITP.[19] While patients often respond initially to corticosteroid therapy, relapse is likely within the first two weeks if concomitant antimicrobial treatment against the underlying infection is not promptly initiated. Effective management requires addressing the primary infection; once targeted antibacterial therapy begins, platelet count recovery typically occurs within 2–3 weeks. Anti-brucellosis regimens typically employ antimicrobial agents with good intracellular penetration, such as doxycycline combined with either rifampin or an aminoglycoside, adhering to the principles of early, combined, regular, adequate, and full-course therapy. For patients presenting with hemorrhagic symptoms, a short-term adjunctive course of corticosteroids or intravenous immunoglobulin may be added.[20] In our case, the concurrent initiation of dexamethasone and antimicrobials likely provided a synergistic effect—corticosteroids rapidly halting immune-mediated platelet destruction while antimicrobials eradicated the antigenic trigger—though this dual approach makes it challenging to quantify the independent contribution of each therapy.

In the differential diagnosis of thrombocytopenia, it is essential for clinicians to consider brucellosis, particularly in endemic regions. For patients initially diagnosed with ITP who exhibit poor response to conventional therapy or present with additional symptoms—such as irregular fever, myalgia, arthralgia, hepatosplenomegaly, or lymphadenopathy—screening for brucellosis should be pursued. This is especially warranted in individuals with risk factors including occupational exposure to livestock, consumption of unpasteurized dairy products, or residence in pastoral areas.

The management of brucellosis should be individualized. In patients with severe thrombocytopenia, urgent administration of corticosteroids can rapidly increase platelet counts and help control bleeding. For those with mild to moderate thrombocytopenia and no life-threatening hemorrhage, anti-brucellosis therapy alone may be sufficient. Some guidelines suggest combining corticosteroids with antimicrobials when the platelet count falls below 10×10^9/L. In the present case, intravenous immunoglobulin was initially administered; however, prior reports indicate that in most cases of brucellosis-associated thrombocytopenia, combining corticosteroids with anti-brucellosis therapy is adequate. If corticosteroids are contraindicated or if intravenous immunoglobulin is not feasible due to economic constraints, the cornerstone of management remains a full-course, appropriate regimen of anti-brucellosis antibiotics.[16] As summarised in Table 2, the present case is consistent with the immune-mediated mechanism and favourable response to combined antimicrobial and corticosteroid therapy observed in previously published cases of brucellosis-associated severe thrombocytopenia. Notably, it is distinguished by the extreme degree of thrombocytopenia (PLT nadir 2×10^9/L), the sequential failure of four distinct ITP-directed therapies prior to correct diagnosis — a combination not previously documented in a single case report — and the detailed longitudinal platelet recovery trajectory, which collectively enhance the educational value of this report beyond prior publications.

This case has several limitations: (1) *Brucella* DNA detection and platelet antibody specificity testing were not performed to confirm cross-reactivity between anti-*Brucella* antibodies and platelet antigens; (2) cytokine profiling (eg, TNF-α, IL-6) was not conducted to further clarify the inflammatory mechanism in platelet destruction; (3) the concurrent use of dexamethasone and antimicrobials makes it difficult to definitively attribute the rapid platelet recovery to either intervention alone; (4) the presence of concurrent vitamin B12 deficiency confounds the strict characterization of "isolated" thrombocytopenia; (5) long-term follow-up beyond 12 months is needed to assess the risk of late relapse.

Table 2 Summary of Previously Published Cases of Brucellosis-Associated Severe Thrombocytopenia and Comparison with the Present Case

Author (Year) [Reference]	Age/ Sex	PLT Nadir ($\times 10^9$/L)	Brucella Species/ Diagnosis	Hemorrhagic Manifestations	Initial Misdiagnosis/Pre-Diagnosis Treatment	Definitive Treatment	Time to PLT Recovery / Outcome
Gürkan et al (2003)[10]	NR	Severe (NR)	*Brucella* spp.	Severe epistaxis, skin petechiae	Suspected ITP; no ITP-specific therapy recorded prior to diagnosis	Anti-brucellosis therapy (doxycycline + streptomycin) + short-course corticosteroids	Rapid (specific timeline NR)
Sevinc et al (2005)[9]	Female	Severe (NR)	*Brucella* spp. (blood + BM culture)	Skin petechiae; mucocutaneous bleeding	Provisional ITP; corticosteroids initiated (responded initially)	Corticosteroids + anti-brucellosis antibiotics	Weeks (NR)
Karsen et al (2012)[17] (n=10)	NR (series)	<20 (range: 2–20)	B. melitensis (56.3% culture+)	Hemorrhagic diathesis; petechiae; mucosal bleeding	Platelet transfusion only; no IVIG or corticosteroids	Doxycycline + rifampin ± platelet transfusion	All normalized by 12-month follow-up
Guzel Tunccan et al (2014)[20]	Male	Severe (NR)	*B. melitensis* (serology+)	Fever, gingival bleeding, skin petechiae	Suspected ITP (BM: ↑megakaryocytes + non-necrotic granuloma); doxycycline + rifampin (days 1–5, no response)	Doxycycline + rifampin + methylprednisolone 1 mg/kg/day (added day 6)	PLT 194×10^9/L at day 4 of corticosteroid
Al Noumani et al (2021)[15]	NR	NR (pancytopenia/ HLH)	*Brucella* spp.	Pancytopenia with hemorrhagic features; HLH syndrome	Lymphoproliferative disease workup	Anti-brucellosis therapy + IVIG (for severe thrombocytopenia)	Favorable (NR)
Ben Lahlou et al (2021)[18]	73 yr/ Male	63 (moderate; pancytopenia)	*Brucella* spp. (serology+)	Fever, chills; splenomegaly; pancytopenia	No ITP misdiagnosis; investigated for malignancy	Anti-brucellosis antibiotics (6 weeks)	Full CBC normalization at 6 weeks
Guevara et al (2023)[11]	Female	Severe (NR)	*Brucella* spp.	Petechiae, purpura, mucosal bleeding	IV corticosteroids + IVIG + platelet transfusion (4 units); partial response only	Corticosteroids + IVIG + anti-brucellosis antibiotics	PLT $>150\times10^9$/L at outpatient follow-up
Present Case (2024)	**57 yr/ Male**	**2 (extreme nadir)**	***B. melitensis*** (blood culture; titer >1:400)	**Diffuse petechiae and ecchymoses, oral blood blisters, gross hematuria, melena, dyspnea**	**IVIG + eltrombopag + rhTPO + platelet transfusion — ALL FAILED (4 distinct ITP therapies)**	**Doxycycline + rifampin + dexamethasone (tapered over 10 days)**	**PLT 10×10^9/L (day 6) → 30×10^9/L (wk 5) → 90×10^9/L (wk 10)**

Notes: The present case (bottom row, shaded) is included for direct comparison. Cases are listed in chronological order by publication year. Karsen et al (2012) report a retrospective series of 10 patients; all other entries are individual case reports. Ben Lahlou et al (2021) reported pancytopenia with a PLT of 63×10^9/L, which does not meet the strict definition of severe thrombocytopenia ($<30 \times 10^9$/L), and is included for contextual completeness. Al Noumani et al (2021) reported brucellosis-induced HLH with thrombocytopenia as a component of pancytopenia. References[9–11,15,17,18,20] correspond to the reference list of the present manuscript.

Abbreviations: PLT, platelet count; NR, not reported in the original publication; IVIG, intravenous immunoglobulin; rhTPO, recombinant human thrombopoietin; BM, bone marrow; HLH, hemophagocytic lymphohistiocytosis; ITP, immune thrombocytopenic purpura; wk, week(s); +, positive result (when used as a suffix, eg, culture+, serology+); ±, with or without; ↑, increased/elevated; →, followed by/progressed to; <, less than; >, greater than.

In conclusion, targeted antimicrobial therapy combined with short-course corticosteroids remains the cornerstone of treatment for this rare presentation.

Ethical Statement

This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of The Second Hospital of Shanxi Medical University. Written informed consent from the patients for participation and publication has been obtained.

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