

Coxiella burnetii Aortitis Leading to Post-EVAR Vascular Graft Infection Complicated by Aorto-Colic Fistula: A Case Report with Literature Review

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Case Report

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Abstract

Background: Persistent localized infection caused by *Coxiella burnetii* can lead to fatal vascular graft infection (VGI). Q fever graft infection secondary to endovascular aortic repair (EVAR) is extremely rare and presents with a highly insidious clinical course. Due to the inherent difficulty of obtaining positive results from conventional microbial cultures, such infections are highly susceptible to clinical misdiagnosis or therapeutic delays; once complicated by an aortoenteric fistula or aneurysm rupture, patient mortality is catastrophically high.

Case Presentation: A 73-year-old male patient, who had undergone EVAR for an abdominal aortic aneurysm (AAA) over 3 years prior, was admitted with a 12-day history of recurrent fever accompanied by persistently elevated inflammatory markers. An 18F-FDG PET-CT scan demonstrated irregular soft-tissue thickening and pathological air pockets around the aortic stent-graft, accompanied by intense glucose hypermetabolism, features clearly indicative of VGI. Emergency laparotomy revealed dense inflammatory adhesions between the aneurysmal wall and the sigmoid colon, confirming the formation of an aortocolic fistula. This finding constituted a major clinical criterion according to the Management of Aortic Graft Infection Collaboration (MAGIC) guidelines. The patient subsequently underwent partial aneurysmectomy, complete explantation of the infected stent-graft, and in situ reconstruction using a rifampicin-bonded synthetic graft. Concurrently, to block intestinal contamination, primary closure of the colonic defect and a sigmoid colostomy (Hartmann's procedure) were performed. Postoperatively, an indirect immunofluorescence assay (IFA) revealed both phase I and phase II IgG titers of 1:512, confirming *C. burnetii* infection. The patient has been initiated on a long-term targeted regimen of doxycycline combined with hydroxychloroquine.

Conclusions: *C. burnetii* should be considered as a differential diagnosis in cases of unexplained, culture-negative VGI following abdominal aortic repair. When clinical manifestations align with the MAGIC diagnostic criteria and 18F-FDG PET-CT confirms a hypermetabolic perigraft lesion, a diagnosis of persistent Q fever should be strongly considered, even if the serological antibody titers fall below the traditional diagnostic threshold of 1:800. Successful management hinges on early recognition followed by thorough surgical debridement of the infectious source and vascular reconstruction. Postoperatively, a long-term (18 to 24 months) dual-antibiotic regimen of doxycycline and hydroxychloroquine is mandatory to completely eradicate the intracellular pathogen by manipulating the intralysosomal microenvironment of host macrophages.

Introduction

Q fever is a globally distributed zoonosis caused by *Coxiella burnetii*, an obligate intracellular Gram-negative coccobacillus. Humans are primarily infected via the inhalation of airborne aerosols contaminated by the placenta, amniotic fluid, or feces of infected livestock, such as sheep, goats, and cattle. Due to the remarkable environmental stability of its small cell variant (SCV), which allows long-distance dissemination via air currents, many patients lack a definitive history of direct animal contact.

Although acute Q fever typically manifests as a self-limiting influenza-like illness or atypical pneumonia, approximately 4% to 5% of patients experience significant immune evasion, transitioning into a persistent localized infection [1]. This persistent stage exhibits a pronounced tropism for vascular and cardiac tissues; notably, vascular graft infection (VGI) accounts for 7% to 8% of these persistent localized cases, predominantly affecting patients with a history of aneurysms or those who have undergone endovascular aortic repair (EVAR) [2]. While *C. burnetii* VGI secondary to EVAR is exceedingly rare, its prognosis remains dismal [3]. Pathophysiologically, the bacterium replicates within the acidic phagolysosomes of host macrophages and induces localized endotolerance by upregulating immunomodulatory factors such as interleukin-10 (IL-10). This mechanism promotes the formation of recalcitrant biofilms and destructive chronic granulomas surrounding the vascular prosthesis [4]. Because conventional microbial cultures are characteristically negative, diagnosing Q fever-associated VGI is exceptionally challenging [5]. In the absence of timely intervention, the infection relentlessly erodes adjacent retroperitoneal organs, triggering catastrophic hemorrhage from an aortoenteric fistula or fulminant sepsis, carrying a 3-year mortality rate of up to 25% [6]. Herein, we describe a patient presenting with *C. burnetii* VGI and a subsequent aortocolic fistula over 3 years post-EVAR, and elucidate the diagnostic framework and the pathophysiology-driven multidisciplinary salvage strategy deployed for successful eradication.

Case presentation

A 73-year-old male was admitted in mid-January 2026 with a 12-day history of recurrent fevers. His past medical history was notable for an infrarenal abdominal aortic aneurysm (AAA) treated with EVAR, right internal iliac artery embolization, and left common iliac artery stenting 3 years prior (October 2022), from which he initially achieved an uneventful recovery (Figs. 1A–1C). Epidemiological investigation revealed that the patient resided in a peri-urban area near small-scale livestock facilities. Although he denied direct animal contact, a potential environmental exposure via airborne aerosols—which can disseminate *Coxiella burnetii* over distances of up to 18 km—could not be excluded [7]. The patient had no history of diabetes, malignancy, or other predisposing immunosuppressive conditions. Prior to admission, the patient received a 1-week course of empirical broad-spectrum antibiotic therapy (piperacillin/tazobactam 4.5 g q8h and imipenem 1 g q8h) at a local hospital, which failed to alleviate his symptoms. Upon admission, physical examination revealed a body temperature of 38.1°C, a heart rate of 101 beats/minute, and blood pressure of 163/98 mmHg. Abdominal palpation revealed a pulsatile mass in the periumbilical region with localized tenderness. Peripheral pulses, including the bilateral dorsalis pedis arteries, were palpable, and no lower extremity edema was noted. His home medications included atorvastatin (20 mg QD) and rivaroxaban (10 mg QD).

Clinical Findings and Diagnostic Work-up

Admission laboratory tests revealed a white blood cell count of $9.1 \times 10^9/\text{L}$, with a significantly elevated neutrophil ratio of 90.1%, indicating severe systemic bacterial inflammation. C-reactive protein (CRP) was markedly increased at 132.4 mg/L. Given the suspicion of infective endocarditis (IE), transthoracic

echocardiography was performed, which revealed no valvular vegetation or severe regurgitation, effectively ruling out dual cardiovascular infection. Furthermore, three sets of aerobic, anaerobic, and fungal blood cultures were negative. This clinical triad of "culture-negative status, failure of broad-spectrum antibiotics, and high inflammatory state with localized tenderness" strongly prompted the suspicion of occult intracellular pathogen infection. To identify the infectious source, 18F-FDG PET-CT was performed, demonstrating irregular, significant soft-tissue thickening with heterogeneous density around the abdominal aortic and iliac metal stent graft. Pathological air pockets were observed within the graft and surrounding tissues. Notably, the lesion exhibited intense, non-homogeneous FDG hypermetabolism, accompanied by retroperitoneal structural disarray and extensive inflammatory exudation (Fig. 2A, 2B) [8]. While contrast-enhanced CT failed to capture direct evidence of an aorto-enteric fistula, these typical imaging features strongly indicated a Vascular Graft Infection (VGI). According to the Management of Aortic Graft Infection Collaboration (MAGIC) criteria, the patient met multiple minor diagnostic criteria, including unexplained persistent fever ($\geq 38^{\circ}\text{C}$), graft tenderness, elevated inflammatory markers, and PET-CT findings suggestive of perigraft inflammation and pathological air presence [9]. Diagnostic precision was achieved through serum mNGS and indirect immunofluorescence assays (IFA). mNGS identified *Enterococcus faecium* (7.05% relative abundance) and *Klebsiella aerogenes* (0.67% relative abundance), consistent with intraoperative tissue biopsy results, suggesting a gut-derived polymicrobial infection. IFA results were negative for *Bartonella henselae* and *Bartonella quintana* IgM; however, *B. henselae* IgG was positive. Given the absence of feline contact history or characteristic lymphadenopathy, this was interpreted as past exposure rather than active infection. Critically, IFA revealed *Coxiella burnetii* phase I and II IgG titers of 1:512. Although traditional diagnostic criteria for chronic Q fever often suggest a threshold of $\geq 1:800$, recent studies indicate that lower titers (1:200 to 1:512) are sufficient for diagnosis in patients with confirmed localized infections via PET-CT or surgery [10]. This phenomenon likely reflects the sequestration of *C. burnetii* within dense biofilm or fibrotic tissue, limiting systemic antigenic exposure. Finally, the intraoperative confirmation of an aorto-enteric fistula met the MAGIC "major clinical criterion," completing the diagnostic evidence chain for Q fever-related VGI [11].

Treatment and Implementation

Given the presence of a VGI with a mature biofilm eroding into adjacent structures, conservative antimicrobial therapy alone was deemed futile. The vascular surgery team proceeded with an emergency exploratory laparotomy. Intraoperative exploration revealed dense, hostile inflammatory adhesions between the lower pole of the aneurysm sac and the sigmoid colon, accompanied by localized purulent exudate (Fig. 3A). The surgical procedure was executed as follows: the infrarenal abdominal aorta was first isolated in a healthy proximal zone, ensuring meticulous protection of the adjacent duodenum, left renal vein, and bilateral renal arteries. Proximal cross-clamping was applied, and the bilateral common and internal iliac arteries were looped and controlled. The anterior wall of the infected aneurysm sac was incised, and the encapsulated EVAR stent-graft was completely explanted (Fig. 3B). Following radical debridement of necrotic retroperitoneal tissue and meticulous hemostasis of the sac wall, in situ

reconstruction was performed using a rifampicin-bonded bifurcated synthetic graft [12]. The proximal end-to-end anastomosis was performed at the infrarenal aorta; the left limb was anastomosed end-to-end to the left internal iliac artery and end-to-side to the left common iliac artery, while the right limb was anastomosed end-to-end to the right common iliac artery. Upon graft declamping, robust pulsatile flow was restored to both lower extremities (Fig. 3C). During adhesiolysis, a perforation was found on the posterior wall of the sigmoid colon, confirming an aortocolic fistula [13]. To prevent continuous colorectal soilage and secondary contamination of the newly implanted graft, a collaborative procedure was performed with the general surgery team: following primary closure of the colonic defect, a temporary sigmoid colostomy (Hartmann's procedure) was performed. Finally, a pedicled autologous omental flap was harvested and wrapped around the synthetic vascular reconstruction to serve as a vascularized biological barrier [14]. Postoperatively, empirical broad-spectrum coverage with intravenous meropenem (1 g q8h) and vancomycin (1 g q12h) was initiated to cover gut-derived pathogens. Following IFA confirmation of persistent *C. burnetii* infection on postoperative day 12, the regimen was targeted. Based on international consensus for persistent Q fever, the patient was transitioned to oral doxycycline (100 mg BID) combined with hydroxychloroquine (200 mg BID) [15]. Simultaneously, intravenous cefoperazone/sulbactam (3 g q8h) and vancomycin were continued for 6 weeks to treat secondary polymicrobial graft contamination (Fig. 4).

Follow-up and Outcomes

At the 3-month outpatient follow-up, the patient exhibited excellent tolerance to the dual-antibiotic regimen, with no reported gastrointestinal distress or QT interval prolongation. Given the potential for dose-dependent ocular toxicity associated with long-term hydroxychloroquine use, baseline and interval ophthalmological assessments were strictly performed; the patient reported no visual disturbances. Currently, the patient remains afebrile with normalized inflammatory markers. Abdominal high-resolution computed tomography angiography (CTA) revealed excellent patency of the rifampicin-bonded synthetic graft, with no evidence of contrast extravasation, pseudoaneurysm formation, or recurrent localized inflammation (Fig. 1D). Considering the high recurrence risk of Q fever VGI, maintenance therapy with the doxycycline and hydroxychloroquine regimen is mandated for at least 18 to 24 months [16]. A rigorous follow-up protocol was established: clinical and inflammatory marker assessments every 3 months, and serological IFA monitoring every 6 months. Concurrently, ophthalmological screening is conducted every 6 months to monitor for hydroxychloroquine-induced ocular toxicity. Treatment cessation will be considered only upon the attainment of clinical remission, imaging normalization, and strict serological stabilization—specifically, a sustained decrease in phase I IgG titers to below 1:200 [17].

Discussion

This case provides a compelling exemplar of *Coxiella burnetii*-induced vascular graft infection (VGI), highlighting the complexity of its immune evasion mechanisms, diagnostic challenges, and the necessity of a multidisciplinary management strategy. Pathophysiologically, *C. burnetii* displays a distinct tropism

for damaged vascular endothelium and synthetic prosthetic material. Upon aerosolized entry, the bacterium utilizes its Type IVB secretion system (T4BSS) to deploy effector proteins (e.g., CaeA, AnkG) into the host cytoplasm to inhibit apoptosis [18]. Concurrently, *C. burnetii* manipulates host cytokine networks to induce IL-10 secretion, promoting macrophage polarization toward an M2 anti-inflammatory phenotype. This downregulates TNF- α expression and inhibits phagosome-lysosome fusion [4]. This engineered immune-tolerant microenvironment enables the pathogen to replicate within protective biofilms and destructive chronic granulomas, relentlessly eroding the aortic wall and predisposing the site to aneurysmal structural collapse and fistula formation. The subsequent breach of the bowel-aorta barrier results in polymicrobial contamination, explaining the 25% 3-year mortality rate observed in conservatively managed cases [6]. While *C. burnetii* is a primary pathogen in culture-negative infective endocarditis (IE), our case presented as an isolated VGI without cardiac involvement. This deviation may be ascribed to the lower antibody titers (Phase I IgG 1:512), indicating an early or localized stage of infection rather than the classic chronic IE (Phase I IgG $\geq$ 1:800) [19]. Furthermore, the synthetic graft provided a highly susceptible abiotic surface for biofilm formation under turbulent flow, potentially capturing the pathogen before systemic cardiac seeding occurred. The pathological gas visualized on PET-CT was likely a result of the synergistic interplay between microbial metabolism and physical disruption of the bowel-aorta barrier. Specifically, *Klebsiella aerogenes* provided direct gas production via metabolic fermentation, facilitated by the microenvironment established by the *Enterococcus faecium* biofilm. While *C. burnetii* itself is not gas-producing, its chronic granulomatous inflammation predisposed the aortic wall to fistulization, acting as a physical conduit for intestinal gas and polymicrobial inoculation. The failure of plasma mNGS to detect *C. burnetii* fragments in this case is highly noteworthy. This negative result is heavily consistent with the biological characteristics of persistent localized Q fever, where the pathogen remains intracellularly sequestered within dense retroperitoneal granulomatous stroma and mature prosthetic biofilms, resulting in transient or negligible systemic bacteremia [20]. This underscores that serological IFA remains the indispensable gold standard. However, we advocate for a prudent interpretation of serological thresholds. While traditional guidelines cite a Phase I IgG titer of $\geq$ 1:800 for chronic Q fever, our case—supported by PET-CT findings and the MAGIC diagnostic criteria—demonstrates that lower titers (1:512) are clinically diagnostic when antigen shedding is limited by dense fibrosis or biofilm [10]. Finally, this case reinforces the pharmacodynamic rationale for the doxycycline-hydroxychloroquine combination. Hydroxychloroquine, as a lysosomotropic weak base, raises the intralysosomal pH, thereby enhancing the bactericidal potency of doxycycline against intracellular *C. burnetii* [15]. In conclusion, radical surgical debridement of the biofilm-laden prosthesis combined with long-term targeted modulation of the intracellular microenvironment is the cornerstone of successful management for this recalcitrant infection.

Conclusions

In clinical practice, *Coxiella burnetii* infection should be prioritized in the differential diagnosis for patients presenting with unexplained fever and culture-negative vascular graft infection (VGI) following abdominal aortic repair. This report demonstrates that clinicians should not rely exclusively on rigid

serological thresholds (e.g., $\geq 1:800$). When clinical suspicion is bolstered by PET-CT evidence of hypermetabolic foci and corroborated by MAGIC criteria, a diagnosis of persistent Q fever remains tenable even with sub-threshold antibody titers. Successful management of such recalcitrant cases hinges on a multidisciplinary approach: prompt radical surgical debridement, anatomical vascular reconstruction, and a prolonged (18–24 months) dual-targeted antimicrobial regimen of doxycycline and hydroxychloroquine. Continued surveillance is essential to assess long-term outcomes and recurrence risk.

Declarations

Ethics approval and consent to participate. This study was reviewed and approved by the Ethics Committee of Hangzhou First People's Hospital (Approval No. 2026ZN271-1). Informed consent to participate was obtained from the patient prior to the study.

Consent for publication. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials. All data generated or analysed during this study are included in this published article and its supplementary information files.

Competing interests. The authors declare that they have no competing interests.

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Authors' contributions. S.Z. wrote the main manuscript text and prepared the figures. X.P. analyzed the pharmacological data. N.Z., C.Z., and P.C. collected the clinical data and contributed to the literature review. X.F. and X.M. supervised the patient management, designed the report, revised the manuscript. All authors reviewed and approved the final manuscript.

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Figures

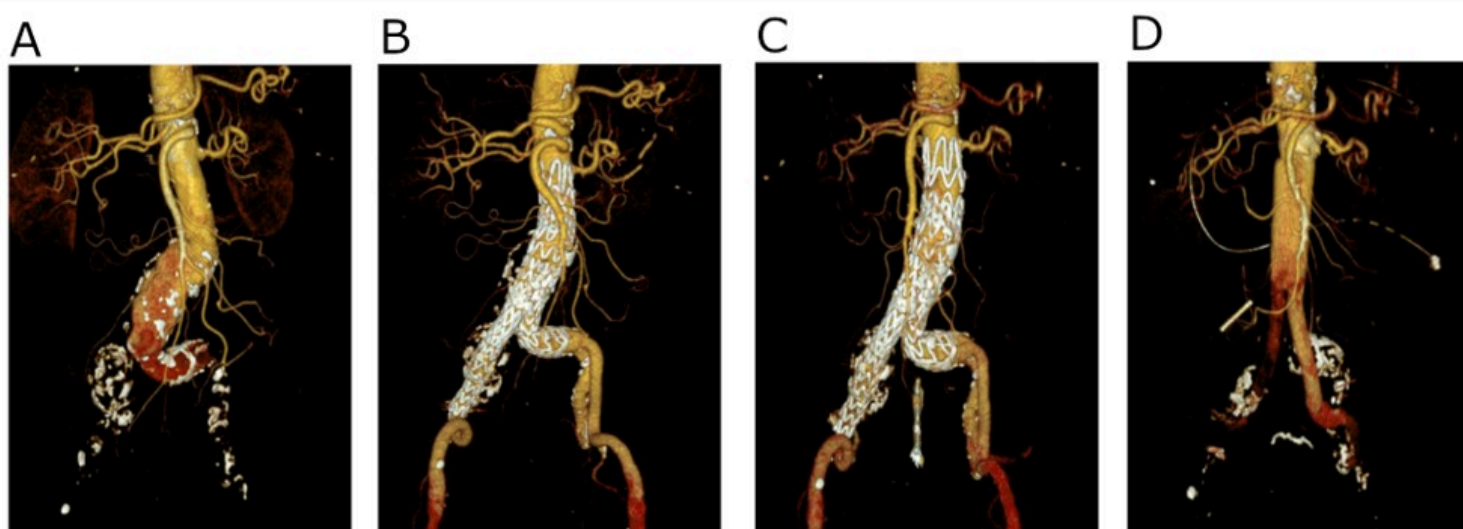


Figure 1

Chronological computed tomography angiography (CTA) series from initial presentation to post-revision follow-up.

(A) Preoperative CTA (October 2022) demonstrating the primary infrarenal AAA. (B) Immediate postoperative CTA following EVAR, confirming appropriate deployment of the aortic stent-graft, successful right iliac artery embolization, and left iliac artery stenting. (C) Mid-term follow-up CTA at 6 months post-EVAR, depicting stable stent-graft configuration, well-preserved distal perfusion, and absence of early endoleaks or peri-graft complications. (D) Short-term follow-up CTA at 3 months after the open radical revision, demonstrating excellent patency of the rifampicin-bonded Y-shaped synthetic vascular graft with intact distal anatomical blood flow and no evidence of recurrent infection, contrast extravasation, or pseudoaneurysm formation.

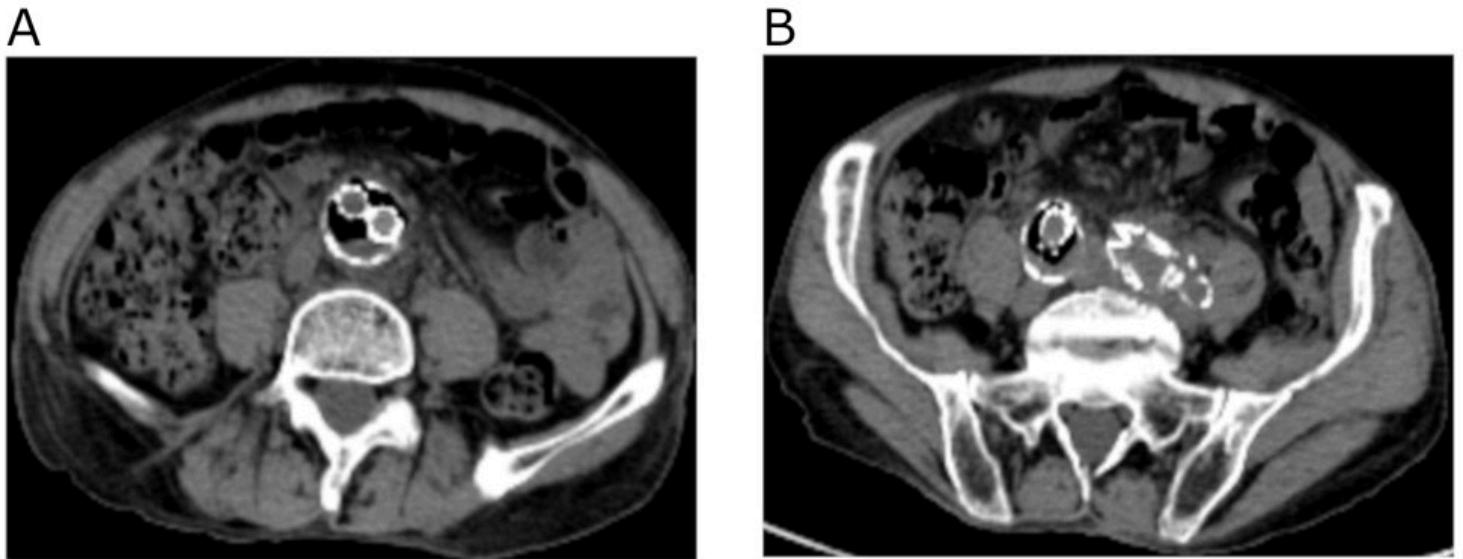


Figure 2

Preoperative 18F-FDG PET-CT imaging of vascular graft infection (VGI).

(A) Irregular, prominent soft tissue thickening and dense patchy shadows around the abdominal aortic stent-graft, accompanied by multiple foci of pathological gas accumulation within and perigraft. (B) Perigraft gas accumulation and marked inflammatory exudation adjacent to the right iliac artery stent-graft.

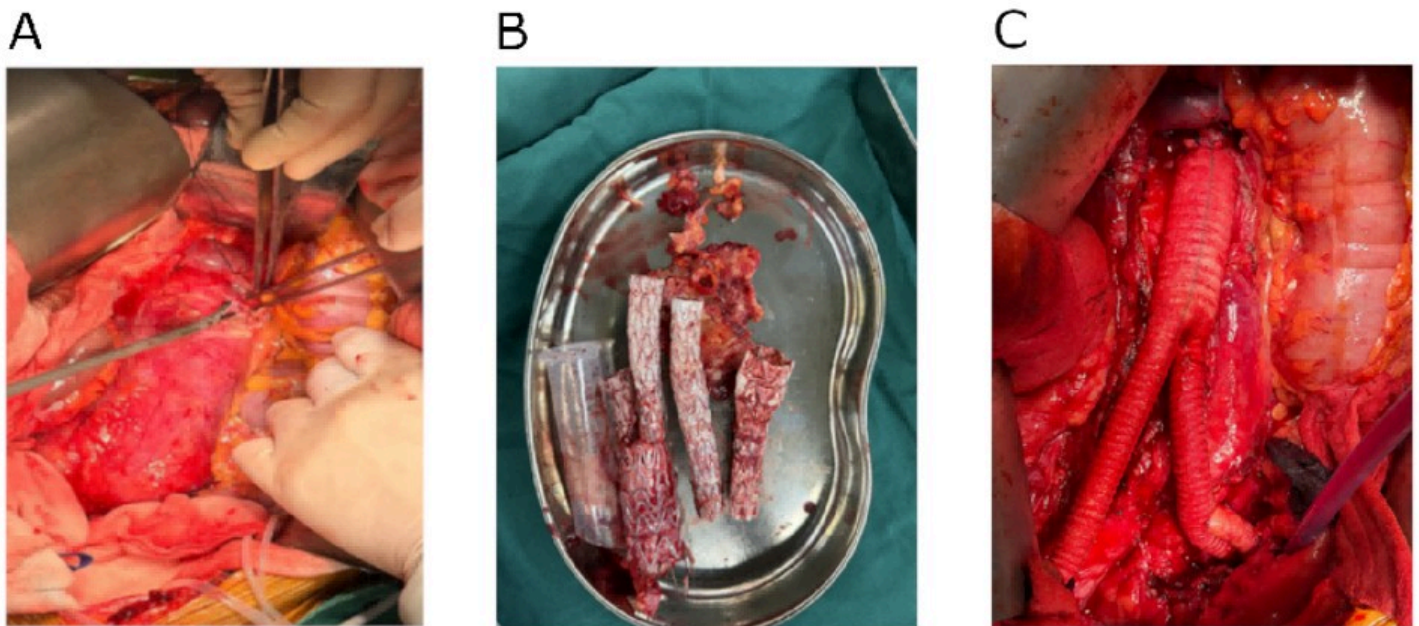


Figure 3

Intraoperative findings during emergency exploratory laparotomy and vascular reconstruction.

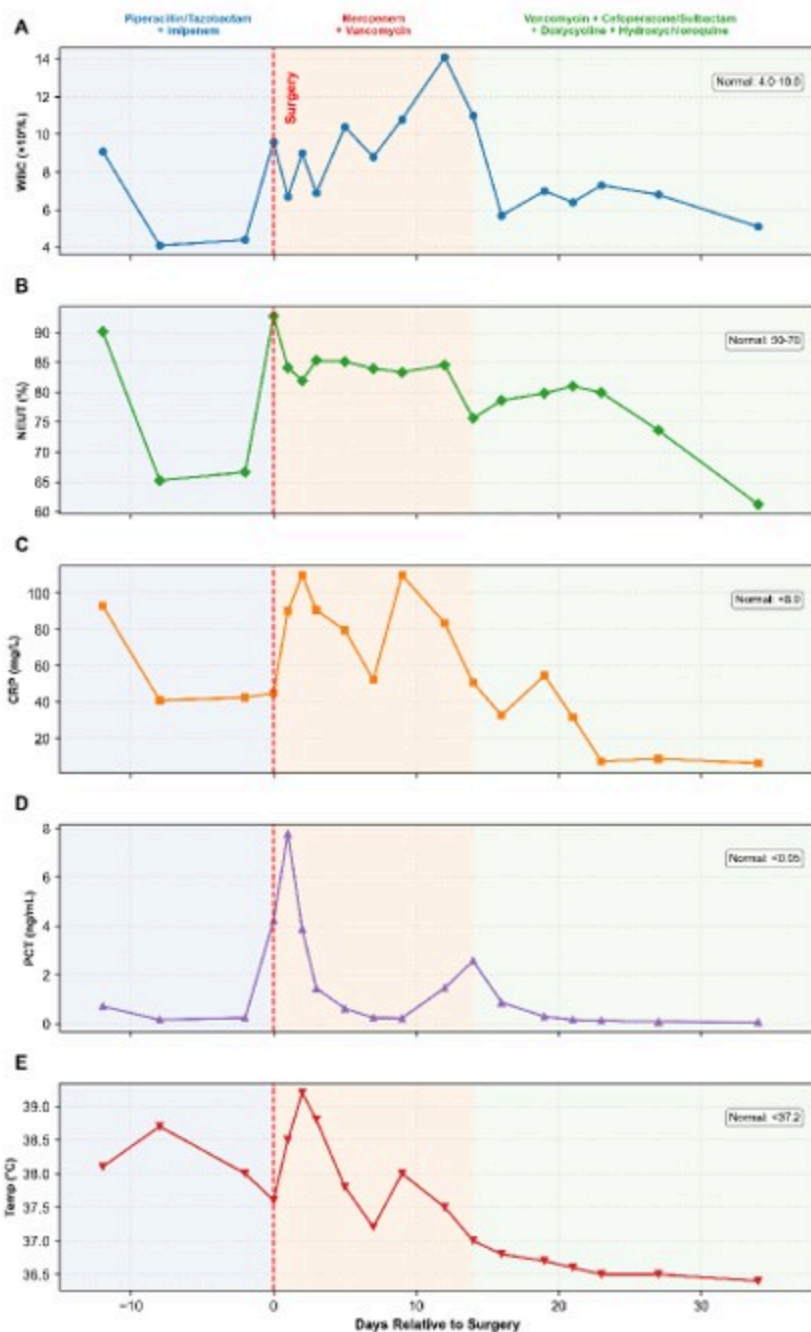


Figure 4

Perioperative clinical course and therapeutic evolution profile aligned to the timeline of surgical intervention (Day 0).

Longitudinal monitoring demonstrates the dynamic changes in: (A) White blood cell (WBC) ; (B) Neutrophil percentage (NEUT, %); (C) C-reactive protein(CRP) ; (D)Procalcitonin(PCT) and (E) Body temperature(°C) The timeline color blocks delineate the sequential antimicrobial strategies: empirical broad-spectrum therapy (piperacillin/tazobactam + imipenem) prior to surgery (blue zone); post-laparotomy intensive regimens (meropenem + vancomycin) (orange zone); and final long-term targeted

combination therapy (vancomycin + cefoperazone/sulbactam + doxycycline + hydroxychloroquine) following the serological confirmation of *Coxiella burnetii* (green zone). Dashed horizontal boxes indicate the upper/lower limits of physiological reference intervals for each laboratory parameter.

Supplementary Files

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