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Fulminant Perianal Donovanosis Manifesting as Septic Shock in a Treatment-Naïve AIDS Patient: A Clinico-Pathological Case Report

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A B S T R A C T

Donovanosis (Granuloma Inguinale), caused by *Klebsiella granulomatis*, is a rare sexually transmitted infection that can follow a devastating course in severely immunocompromised individuals. Its clinical progression in the context of advanced acquired immunodeficiency syndrome (AIDS) is not extensively documented. We present a case of fulminant donovanosis to illustrate its potential for rapid systemic deterioration and mortality. A 20-year-old Indonesian man, with a recent diagnosis of human immunodeficiency virus (HIV) for which he was treatment-naïve, presented with a two-month history of extensive, painful perianal ulcerations. Clinical examination revealed large, coalescing, "beefy-red" ulcers in the perianal and gluteal regions. Laboratory investigations confirmed profound immunosuppression (CD4+ T-cell count: 3 cells/µL; HIV viral load: >750,000 copies/mL). The diagnosis of donovanosis was definitively established by the microscopic identification of pathognomonic intracellular Donovan bodies on a Giemsa-stained tissue smear, with findings corroborated by histopathological analysis of a skin biopsy. Despite the initiation of appropriate antibiotic and supportive therapy, the patient's condition rapidly progressed to septic shock and multi-organ dysfunction syndrome, leading to his death within six days of hospital admission. In conclusion, this case highlights the aggressive, life-threatening nature of donovanosis in the setting of advanced AIDS. The profound collapse of cell-mediated immunity likely facilitated uncontrolled bacterial replication and systemic dissemination, rendering standard antibiotic therapy ineffective. This report serves as a critical clinical reminder to maintain a high index of suspicion for donovanosis in immunocompromised patients presenting with atypical anogenital ulcers, as early diagnosis and aggressive multimodal management are paramount.

1. Introduction

Donovanosis, also known as Granuloma Inguinale, is a chronic and progressively destructive bacterial infection caused by the facultative intracellular, gram-negative bacterium *Klebsiella granulomatis* (previously classified as Calymatobacterium granulomatis).[1] While effective antibiotic treatments have led to a significant decline in its global incidence, the disease remains endemic in specific tropical and subtropical regions, including Papua New Guinea, parts of Southern Africa, India, the Caribbean, and isolated communities in Brazil and Australia. In nations like Indonesia, donovanosis is considered rare, with only sporadic cases reported, often within marginalized populations facing barriers to healthcare access.[2]

The classical clinical manifestation of donovanosis is the development of painless, slowly progressive ulcerative lesions on the genitalia or in the anogenital region.[3] The pathognomonic appearance of these ulcers is a "beefy-red," highly vascular, and friable granular base that bleeds easily on contact. Transmission is primarily understood to occur

through sexual contact, although other routes such as autoinoculation and vertical transmission have been described.[4] The insidious onset and typically painless nature of early lesions can lead to significant delays in diagnosis. This delay allows for extensive local tissue destruction, which can result in severe complications such as scarring, lymphatic obstruction leading to pseudo-elephantiasis, and an increased risk of neoplastic transformation, most commonly squamous cell carcinoma, within chronic lesions. The diagnostic gold standard is the direct microscopic identification of pathognomonic Donovan bodies—clusters of encapsulated, bipolar-staining bacteria within the cytoplasm of large mononuclear cells (macrophages)—in tissue smears or biopsy specimens.[5]

The emergence of the human immunodeficiency virus (HIV) pandemic has fundamentally altered the clinical landscape of many sexually transmitted infections (STIs).[6] Co-infection with HIV is known to modify the natural history of STIs, frequently resulting in more severe, persistent, and atypical clinical presentations, as well as an increased risk of treatment failure. The profound depletion of CD4+ T-helper cells, the hallmark of acquired immunodeficiency syndrome (AIDS), cripples the host's cell-mediated immune response. This branch of the immune system is indispensable for controlling intracellular pathogens like *K. granulomatis*.[7] Consequently, patients with advanced HIV disease and severe immunosuppression are predisposed to more aggressive and disseminated forms of donovanosis, characterized by larger, more numerous, and rapidly expanding lesions, as well as a heightened risk of extragenital dissemination to bones, joints, and visceral organs.[8]

This synergistic interaction between HIV-induced immunodeficiency and *K. granulomatis* infection poses a formidable clinical challenge, markedly increasing morbidity and mortality.[9] Despite this established biological plausibility, detailed clinico-pathological reports documenting the fulminant clinical course and fatal outcomes of donovanosis in the context of severe, untreated AIDS are scarce in contemporary literature, particularly from the Southeast Asian region.[10]

Therefore, the primary aim of this report is to provide a detailed account of a fulminant and fatal case of perianal donovanosis in a young, treatment-naïve patient with AIDS. The didactic value of this study lies in its comprehensive documentation of the rapid clinical deterioration and systemic collapse in this co-infected patient. It provides a stark, real-world example of the devastating pathophysiological synergy between *K. granulomatis* and severe HIV-induced immunodeficiency, serving as a critical reminder for clinicians to maintain a high index of suspicion for this "forgotten" STI in the modern era of global health.

2. Case Presentation

A 20-year-old, unmarried Indonesian man was referred from the internal medicine service to our dermatology and venereology clinic for the evaluation of severe, debilitating, and painful ulcerations surrounding his anus. The patient was an inpatient at Dr. Moewardi Regional General Hospital, Surakarta, initially admitted for the management of severe anemia, pneumonia, and a new diagnosis of HIV. He reported that the perianal lesions had been present for approximately two months and had progressively worsened, causing excruciating pain that severely interfered with sitting, walking, and defecation.

The patient's symptoms began insidiously two months prior with the appearance of a few small, reddish papules ("pimples") around the anal verge, which he initially attributed to poor hygiene or hemorrhoids. These lesions were accompanied by mild pruritus but were not initially painful. Over the subsequent weeks, the papules enlarged and coalesced before ulcerating. The ulcers then steadily expanded in size and depth, spreading circumferentially around the anus and extending into the gluteal cleft. The character of the lesions changed significantly about one month prior to admission, transitioning from painless to excruciatingly painful. He also reported occasional bleeding from the ulcers, particularly after defecation, and a persistent, foul-

smelling seropurulent discharge. He had not sought any medical treatment for these specific lesions before this hospitalization. His admission was ultimately prompted by a constellation of systemic symptoms, including progressive weakness, cachexia with an unintentional weight loss of approximately 10 kg over three months, intermittent fevers, a persistent non-productive cough, and several episodes of melena.

The patient had no significant past medical history of chronic illnesses such as diabetes mellitus or hypertension. He had been diagnosed with HIV three months prior at a local public health clinic after presenting with oral candidiasis. Unfortunately, he was lost to follow-up and had never initiated antiretroviral therapy (ART). He denied any history of prior STIs, genital ulcers, or urethral discharge. There was no history of intravenous drug use, tattoos, or receipt of blood transfusions.

Socially, the patient worked as a freelance artist and lived alone in a rented room. He reported smoking approximately ten cigarettes per day but denied alcohol or illicit substance use. His sexual history revealed that he identified as a man who has sex with men (MSM). He became sexually active at age 16 and reported having more than five male partners in the past year, engaging in both receptive and insertive anal intercourse as well as oral sex. He reported inconsistent condom use and was unaware of the HIV status of his sexual partners. His last reported sexual contact was approximately one year prior to this presentation.

On examination, the patient appeared chronically ill, cachectic, and was in visible, moderate distress due to pain. His vital signs on admission were: blood pressure 110/70 mmHg, heart rate 104 beats per minute, respiratory rate 22 breaths per minute, temperature 38.2°C, and oxygen saturation of 95% on room air. His body mass index (BMI) was critically low at 16.3 kg/m² (underweight). There was no palpable generalized lymphadenopathy. Oral examination revealed extensive, thick, white plaques consistent with severe oropharyngeal candidiasis.

Dermatological and venereological examination of the anogenital region revealed extensive and destructive ulcerative disease. Multiple, large ulcers had coalesced to involve the entire perianal region, extending approximately 5-6 cm superiorly into the gluteal cleft and laterally onto the buttocks (Figure 1). The ulcers displayed irregular, slightly raised (pseudo-verrucous) borders and a friable, intensely erythematous, granular base that resembled raw meat—the classic "beefy-red" appearance of donovanosis. The base of the ulcers bled profusely upon gentle manipulation with a sterile cotton swab. A significant amount of malodorous, seropurulent exudate covered the lesions, but there was no visible purulence, slough, or necrotic tissue. The surrounding skin was erythematous and edematous. The ulcers were exquisitely tender to palpation. Crucially, there was no inguinal or femoral lymphadenopathy (pseudo-buboes). Examination of the penis and scrotum was unremarkable.

A comprehensive diagnostic workup was initiated to identify the etiology of the ulcers and assess the patient's overall systemic condition. A summary of key laboratory investigations is presented in Table 1. Hematological analysis revealed severe normocytic, normochromic anemia (Hemoglobin: 4.7 g/dL), leukocytosis with a marked neutrophilic predominance (WBC: 12,500/µL; 85% neutrophils), and a normal platelet count. Inflammatory markers were significantly elevated, with an erythrocyte sedimentation rate (ESR) of 110 mm/hr. The comprehensive metabolic panel was notable for severe hypoalbuminemia (2.1 g/dL), indicative of malnutrition and chronic inflammation, along with elevated liver transaminases (AST: 55 U/L; ALT: 46 U/L) and evidence of acute kidney injury (Urea: 79 mg/dL; Creatinine: 1.6 mg/dL). The reactive anti-HIV test was confirmed. Immunological assessment revealed profound cellular immunodeficiency: the CD4+ T-cell count was critically low at 3 cells/µL (normal 500-1600 cells/µL), with an inverted CD4/CD8 ratio of 0.01. The HIV-1 viral load was exceedingly high at >750,000 copies/mL. Serological

tests for other STIs, including syphilis (VDRL and TPPA) and herpes simplex virus (Anti-HSV-1/2 IgM and IgG), were non-reactive. Hepatitis B surface antigen (HBsAg) was also non-reactive. A chest X-ray was performed to investigate the patient's persistent cough and respiratory symptoms. It revealed bilateral diffuse interstitial and reticulonodular infiltrates, a pattern non-specific but highly suspicious for an opportunistic pneumonia, potentially *Pneumocystis jirovecii* pneumonia (PJP), in a patient with this level of immunosuppression.

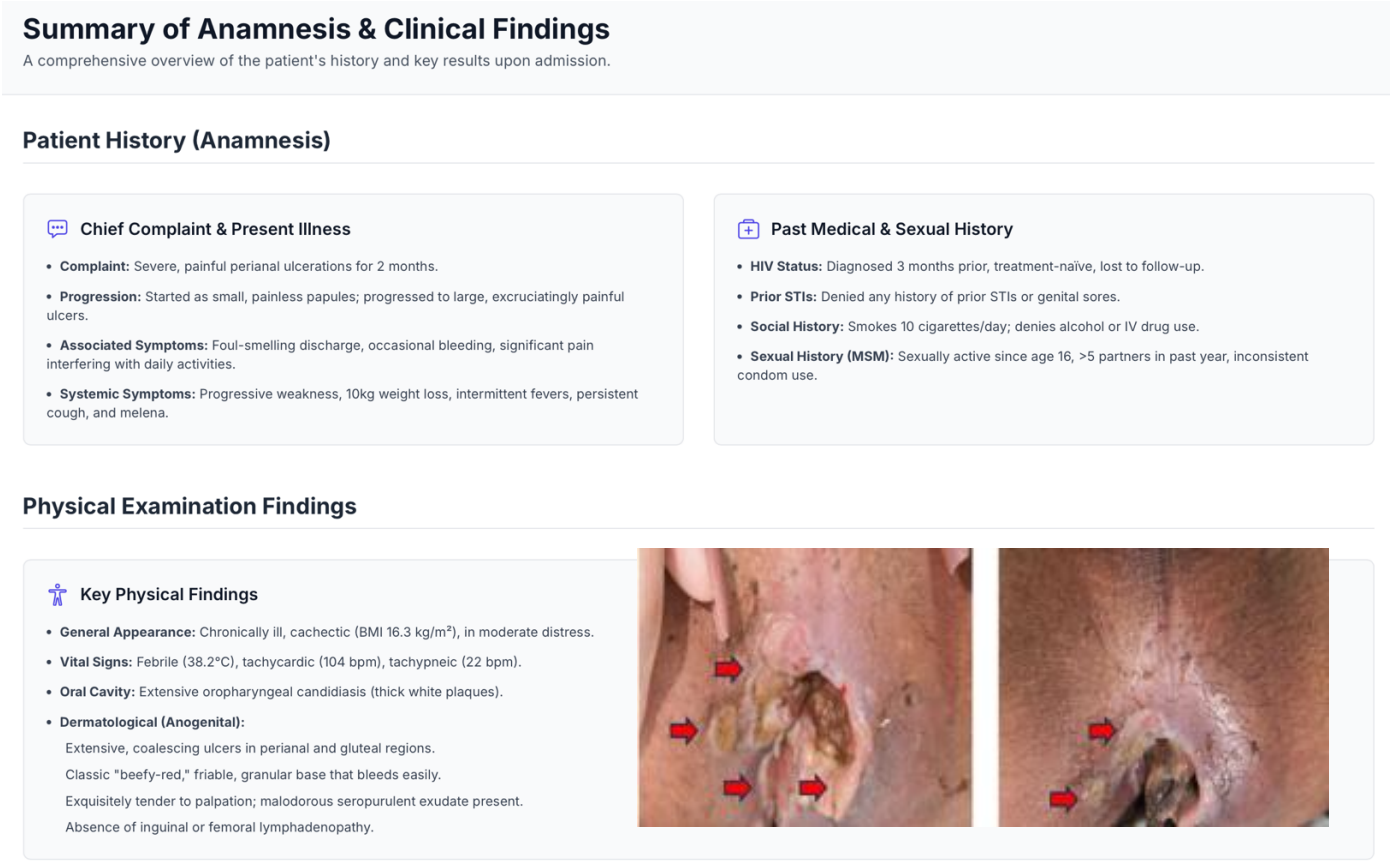

Figure 1. Summary of anamnesis and clinical findings. Clinical photograph of the extensive perianal and gluteal ulcerations. The image shows large, coalescing ulcers with intensely erythematous, granular ("beefy-red") bases and raised borders, extending from the perianal skin onto the buttocks.

To establish a definitive diagnosis for the ulcerative lesions, tissue material was collected. (1) Tissue Smears: Material was carefully collected by scraping the clean base of an ulcer. A 10% KOH mount was negative for fungal elements (Figure 2A). A Tzanck smear was negative for the multinucleated giant cells characteristic of herpesvirus infections (Figure 2B). A Gram stain showed numerous polymorphonuclear leukocytes (PMNs) and a mixed population of bacteria, with no predominant organism (Figure 2C); (2) Giemsa Stain: This was the key rapid diagnostic test. Microscopic examination of the Giemsa-stained smear under oil immersion (x1000 magnification) was pathognomonic. It revealed large mononuclear cells (macrophages) containing characteristic intracytoplasmic cysts filled with deeply staining, bipolar, "safety-pin" shaped organisms (Figure 2D). These were definitively identified as Donovan bodies,

confirming the diagnosis of donovanosis (Figure 2); (3) Histopathology: A 4-mm punch biopsy was taken from the active border of an ulcer to corroborate the smear findings and definitively rule out malignancy. Histopathological examination with Hematoxylin and Eosin (H&E) staining revealed marked pseudoepitheliomatous hyperplasia of the epidermis. The dermis showed a dense, diffuse inflammatory infiltrate composed predominantly of histiocytes and plasma cells, with scattered neutrophils and lymphocytes. Within the cytoplasm of numerous large histiocytes (macrophages), small, encapsulated, ovoid structures consistent with Donovan bodies were observed, confirming the diagnosis. There was no evidence of dysplasia or malignancy.

Table 1. Summary of key laboratory findings on admission.

Laboratory Findings

PARAMETER	PATIENT'S VALUE	REFERENCE RANGE	INTERPRETATION & SIGNIFICANCE
Complete Blood Count & Inflammatory Markers			
Hemoglobin (Hb)	**4.7 g/dL**	13.5 - 17.5 g/dL	Severe Anemia
White Blood Cell (WBC) Count	12,500 /µL	4,500 - 11,000 /µL	Leukocytosis
Neutrophils	85 %	40 - 75 %	Neutrophilia
Platelet Count	336,000 /µL	150,000 - 450,000 /µL	Normal
Erythrocyte Sedimentation Rate (ESR)	110 mm/hr	< 20 mm/hr	Markedly Elevated
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Albumin	2.1 g/dL	3.5 - 5.0 g/dL	Severe Hypoalbuminemia
Aspartate Transaminase (AST)	55 U/L	< 40 U/L	Elevated
Alanine Transaminase (ALT)	46 U/L	< 41 U/L	Elevated
Urea	79 mg/dL	10 - 50 mg/dL	Elevated (Azotemia)
Creatinine	1.6 mg/dL	0.7 - 1.3 mg/dL	Elevated (AKI)
Immunology & Serology			
Anti-HIV	Reactive	Non-reactive	Confirmed HIV Infection
CD4+ T-Cell Count	**3 cells/µL**	500 - 1600 cells/µL	Profound Immunodeficiency
CD4/CD8 Ratio	0.01	> 1.0	Severely Inverted
HIV-1 Viral Load	**>750,000 copies/mL**	Undetectable	Extremely High
VDRL / TPPA (Syphilis)	Non-reactive	Non-reactive	No evidence of Syphilis

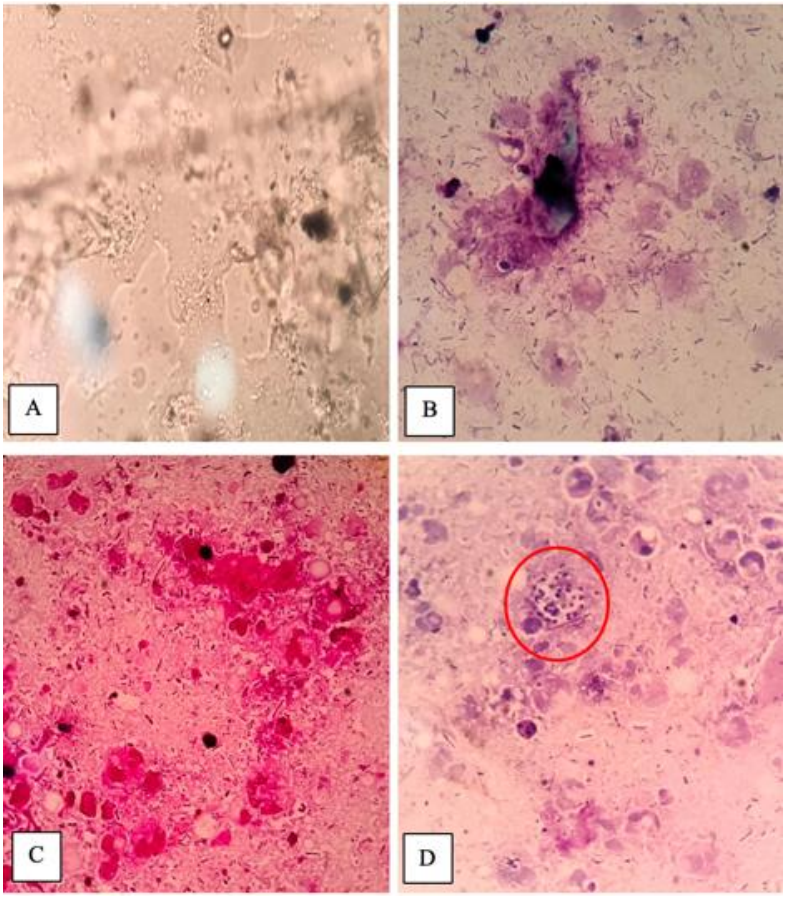

Figure 2. Microbiological and histopathological examination. (A) 10% KOH mount was negative for fungal elements (B) Tzanck smear was negative for the multinucleated giant cells characteristic of herpesvirus infections (C) Gram stain showed numerous polymorphonuclear leukocytes (PMNs) and a mixed population of bacteria, with no predominant organism (D) Photomicrograph of the Giemsa-stained smear (x1000 oil immersion). The image shows a large macrophage with its cytoplasm filled with multiple small, ovoid, bipolar-staining organisms (Donovan bodies), appearing like safety pins (red circle).

The patient was managed by a multidisciplinary team comprising internal medicine, infectious disease, and dermatology specialists. Upon the definitive diagnosis of donovanosis in the setting of advanced AIDS with multiple opportunistic infections, a comprehensive management plan was initiated: (1) Antibiotic therapy for donovanosis: Treatment was started with Azithromycin 500 mg orally once daily, a first-line therapy recommended by international guidelines; (2) Management of sepsis and pneumonia: The internal medicine team had already initiated broad-spectrum intravenous antibiotics (piperacillin-tazobactam) to cover for potential bacterial sepsis and the presumed opportunistic pneumonia; (3) Supportive care: The patient's severe anemia was managed with multiple packed red blood cell transfusions. Intravenous fluids were administered for hydration and to manage his acute kidney injury. Nutritional support was initiated; (4) Management of oropharyngeal candidiasis: Intravenous fluconazole was administered for the severe oral candidiasis; (5) Wound care: The extensive perianal ulcers were managed with gentle daily cleansing using saline solution, followed by the application of Mupirocin 2% ointment to prevent secondary bacterial infection and promote a moist healing environment; (6) Pain

management: A multimodal analgesic approach, including systemic opioids, was required to manage his severe pain; (7) Antiretroviral Therapy (ART): The infectious disease team planned to initiate ART to address the underlying immunodeficiency. However, the decision was made to defer initiation until the patient was stabilized from his acute, life-threatening infections to mitigate the risk of a paradoxical worsening due to Immune Reconstitution Inflammatory Syndrome (IRIS). Despite these aggressive, multi-pronged interventions, the patient's clinical course was one of rapid and irreversible decline. Over the subsequent 48-72 hours, his fever persisted, and he became progressively hypotensive, tachycardic, and obtunded. His renal function continued to worsen, progressing to anuria, and he developed worsening respiratory distress requiring high-flow oxygen. He fulfilled the clinical criteria for septic shock with multi-organ dysfunction syndrome (MODS). Despite aggressive resuscitation efforts in the high-dependency unit, including vasopressor support (norepinephrine infusion), the patient suffered a cardiac arrest and was pronounced dead on the sixth day of his hospital admission, only three days after the definitive diagnosis of donovanosis was made (Figure 3).

Figure 3. Hospital course, management, and outcome.

3. Discussion

This report details a catastrophic and fatal presentation of donovanosis in a young, treatment-naïve patient with advanced AIDS. The fulminant progression from a seemingly localized ulcerative disease to systemic collapse and death serves as a powerful illustration of the critical interplay between the pathogen, *Klebsiella granulomatis*, and a severely compromised host immune system.[11] The discussion will focus on the underlying pathophysiological mechanisms, the analysis of atypical clinical features, the diagnostic challenges, and the therapeutic failures that likely contributed to this devastating outcome. The fatal outcome in our patient can be directly attributed to the near-total collapse of his cell-mediated immunity, evidenced by a CD4+ T-cell count of only 3 cells/μL. *K. granulomatis* is a facultative intracellular bacterium that primarily targets and replicates within mononuclear phagocytes (macrophages and histiocytes).[12] In an immunocompetent host, the control of such pathogens is orchestrated by CD4+ T-helper cells, specifically the Th1 subset. These cells produce interferon-gamma (IFN-γ), a potent cytokine that activates macrophages, enhancing their microbicidal capacity to destroy the intracellular bacteria (Figure 4).

In our patient, the absence of CD4+ T-cells completely abrogated this crucial defense mechanism. The patient's macrophages, instead of being effective pathogen-killing effector cells, became permissive "incubators" or "Trojan horses" for uncontrolled bacterial replication. This immunological failure likely led to three critical pathophysiological consequences that drove his rapid decline: (1) Uncontrolled Local Replication and Tissue Destruction: The lack of immune containment allowed for an exponentially higher bacterial burden within the perianal lesions. This transformed what is typically an indolent, slowly progressive disease into an aggressive, necrotizing process, resulting in the unusually rapid and extensive tissue destruction observed; (2) Impaired Wound Healing: Effective tissue repair requires a coordinated immune response, angiogenesis, and collagen deposition. The patient's profound immunosuppression, compounded by severe malnutrition (hypoalbuminemia), cachexia, and anemia, completely crippled his ability to mount any effective healing response. The ulcers became non-healing, perpetually expanding portals of entry. (3) Systemic Dissemination: With no effective immune surveillance to confine the infection to the anogenital region, the hematogenous or lymphatic dissemination of infected macrophages from the primary ulcer site became highly probable. These bacteria-laden macrophages could then seed distant organs, leading to a systemic infection.[13]

A classic teaching point for donovanosis is that the ulcerative lesions are typically painless, a feature that often contributes to delayed diagnosis.[14] Our patient, however, experienced "excruciatingly painful" lesions, an atypical feature that warrants discussion. Several factors, likely acting in concert in this severely compromised host, may explain this deviation from the classic presentation: (1) Severity of Tissue Destruction: The uncontrolled bacterial replication may have led to a depth of tissue necrosis that exposed underlying somatic nerve endings, resulting in severe pain; (2) Secondary Bacterial Infection: The large, open, and exudative ulcers in the perianal region—an area with a high bacterial load—were almost certainly superinfected with other skin and gut flora. The patient's marked leukocytosis and neutrophilia support a significant secondary bacterial process.[15] This polymicrobial infection would have induced an acute, purulent inflammatory response, contributing significantly to the pain; (3) Perianal Location: The perianal skin is a highly innervated region, and any inflammatory or ulcerative process in this area is inherently more likely to be painful than lesions on the penile shaft or labia; (4) Dysregulated Inflammation: While the patient lacked an effective T-cell response, the innate immune system (neutrophils, remaining macrophages) would still have been active. In the absence of proper regulation by T-cells, this innate response may have been dysregulated, leading to an

excessive release of pro-inflammatory cytokines that contribute to hyperalgesia.[16]

While we could not confirm with a post-mortem examination, the patient's clinical trajectory from localized skin disease to death from septic shock and MODS in under 72 hours is highly suggestive of systemic bacterial seeding. The clinical evidence supporting this hypothesis is compelling: persistent high fever, progressive hypotension requiring vasopressors, acute kidney injury, elevated liver transaminases, and deteriorating mental status. This clinical syndrome is the hallmark of systemic sepsis.[17]

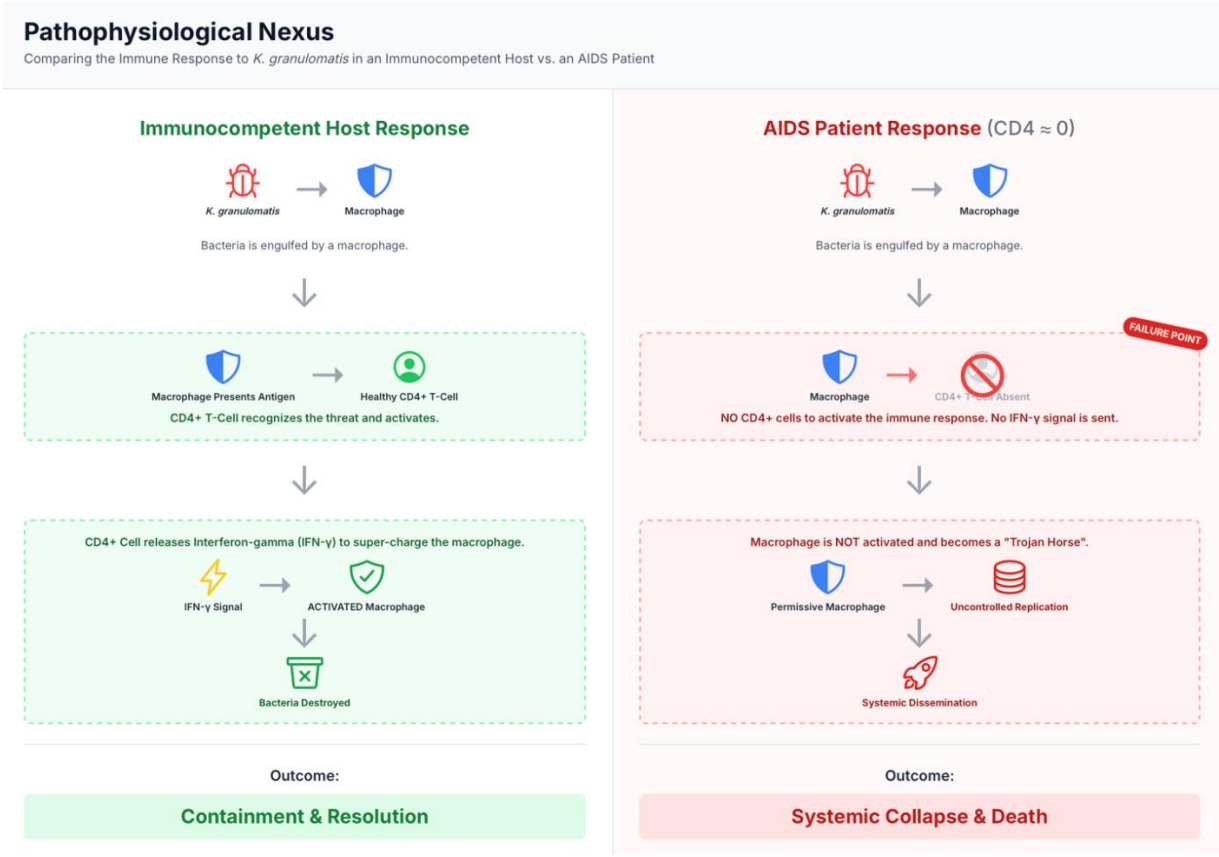

Figure 4. The pathophysiological nexus: *K. granulomatis* and profound immunodeficiency.

Fatal disseminated donovanosis involving the liver, spleen, and bones has been previously reported, almost exclusively in immunocompromised individuals. The infected macrophage, unable to kill the bacterium, likely served as the vehicle for this dissemination. The massive portal of entry provided by the extensive, non-healing perianal ulcers would have facilitated the entry of not only *K. granulomatis* but also other opportunistic pathogens from the skin and gastrointestinal tract into the bloodstream, likely contributing to the terminal septic state.[18]

Situating this case within the existing literature underscores its severity. Most reports of donovanosis in HIV-positive individuals describe more extensive or treatment-refractory disease, but not typically the hyperacute decline seen here. For instance, reports have described extragenital donovanosis of the bone in an AIDS patient with a CD4 count of 50 cells/μL who ultimately responded to prolonged therapy, and another of relapsing disease correlated with a decline in CD4 count. In contrast, our patient, with a CD4 count at the extreme nadir of 3 cells/μL, progressed to

a fatal outcome within days of diagnosis despite the initiation of standard therapy. This suggests a potential correlation between the absolute nadir of the CD4+ T-cell count and the pace of disease progression from localized to systemic. This case likely represents the most severe end of the clinical spectrum of donovanosis-HIV co-infection, where the host's immune system offers virtually zero resistance to bacterial replication and dissemination. The patient's reported last sexual contact being a year prior also raises questions about a prolonged incubation or latency period, which may be plausible in the setting of a progressively failing immune system.[19]

From a diagnostic standpoint, this case highlights the enduring utility of simple, bedside diagnostic techniques. While sophisticated molecular diagnostics for *K. granulomatis* exist, they are not widely available. The definitive diagnosis was made rapidly and cost-effectively via Giemsa staining of a tissue smear. This underscores the importance of clinicians being proficient in this classic technique, especially in resource-limited settings or when dealing with atypical presentations.[20] The decision to also perform a biopsy was prudent, as it definitively ruled out concurrent squamous cell carcinoma, a known long-term risk.

Therapeutically, this case demonstrates a tragic treatment failure. Azithromycin is the recommended first-line agent due to its excellent intracellular penetration and favorable dosing schedule. However, the success of any antibiotic therapy for intracellular pathogens relies on a host immune system capable of assisting in final bacterial clearance. In this patient, the antibiotic was initiated too late, in the face of an overwhelming bacterial load and a non-functional immune system. It is plausible that oral administration resulted in suboptimal bioavailability in a critically ill patient with potential gut malabsorption. In such severe cases, especially with suspected systemic dissemination, parenteral antibiotic regimens, including ceftriaxone or a carbapenem, might be considered, although evidence for their superiority is limited.

Finally, the case highlights the critical dilemma of ART timing in treatment-naïve AIDS patients with acute opportunistic infections. While ART is the only way to reverse the underlying immunodeficiency, its initiation during a severe infection carries a significant risk of IRIS, which could have paradoxically worsened the patient's condition. The clinical team's decision to defer ART was appropriate given the patient's instability. Unfortunately, the opportunistic infection itself proved uncontrollably lethal before the underlying immune deficiency could be addressed.

4. Conclusion

This case of fatal perianal donovanosis in a young man with advanced, untreated AIDS is a stark reminder of the devastating potential of neglected STIs in the context of severe immunodeficiency. The profound loss of cell-mediated immunity transforms donovanosis from a chronic, localized ulcerative disease into an aggressive, systemic, and rapidly fatal infection. This report emphasizes that donovanosis must be included in the differential diagnosis of any atypical, progressive, or unusually painful anogenital ulceration in patients with HIV, particularly those with low CD4 counts. Clinicians must remain vigilant, employ simple diagnostic tools like tissue smears, and recognize that even with standard antibiotic therapy, the prognosis may be grim without aggressive, multidisciplinary supportive care and an eventual attempt at immune reconstitution. This case contributes a detailed clinico-pathological perspective to the limited body of literature on the most severe end of the donovanosis-HIV co-infection spectrum and underscores the urgent, life-saving importance of early HIV diagnosis and unwavering linkage to care to prevent such catastrophic opportunistic infections.

5. References

1. Araújo MRB, Sant'Anna LO, Santos LSD. *Klebsiella granulomatis* infection in a patient with human immunodeficiency virus infection. Rev Soc Bras Med Trop. 2021; 54.

2. Vendhan DS, Vasudevan B, Gera V, Neema S. Granuloma inguinale diagnosed on the bedside. Indian J Sex Transm Dis AIDS. 2023; 44(1): 87–8.
3. Sankarananthan M. Whether reactivation of granuloma inguinale can be a possibility in a person living with HIV? Indian J Sex Transm Dis AIDS. 2025; 46(1): 78–80.
4. Ahmed N, Pillay A, Lawler M, Bobat R, Archary M. Donovanosis causing lymphadenitis, mastoiditis, and meningitis in a child. Lancet. 2015; 385(9987): 2644.
5. O'Farrell N, Hoosen A, Kingston M. 2018 UK national guideline for the management of donovanosis. Int J STD AIDS. 2018; 29(10): 946–8.
6. Ramdial PK, Sing Y, Ramburan A, Bagratee JS. Nodal donovanosis as the sentinel clue to acquired immunodeficiency syndrome. J AIDS Clin Res. 2016; 7(9).
7. Arora AK, Kumaran MS, Narang T, Saikia UN, Handa S. Donovanosis and squamous cell carcinoma: The relationship conundrum! Int J STD AIDS. 2017; 28(4): 411–4.
8. Magalhães BM, Veasey JV, Mayor SAS, Lellis RF. Donovanosis in a child victim of sexual abuse: response to doxycycline treatment. An Bras Dermatol. 2018; 93(4): 592–4.
9. Hajare SA, Mukhi JI, Rambhia KD, Singh RP. Donovanosis in central India: a series of six cases and review of literature. J Clin Diagn Res. 2019.
10. Morrone A, Scarabello A, Sperduti I, Cota C, Donà MG, Orsini D, et al. Donovanosis in migrants: a clinical case series in an Italian dermatological hospital. J Eur Acad Dermatol Venereol. 2019; 33(12): e438–40.
11. Arif T, Adil M. Donovanosis in a human immunodeficiency virus-positive female. Indian J Sex Transm Dis AIDS. 2020; 41(1): 131.
12. Muller EE, Kularatne R. The changing epidemiology of genital ulcer disease in South Africa: has donovanosis been eliminated? Sex Transm Infect. 2020; 96(8): 596–600.
13. Mawardi P, Esa Ilona S. Donovanosis and genital herpes coinfection in a woman with human immunodeficiency virus infection: a case letter. Int J Womens Dermatol. 2020; 6(2): 123–4.
14. Daniele D, Wilkerson T. Surveillance snapshot: Donovanosis among active component service members, U.S. Armed Forces, 2011-2020. MSMR. 2021; 28(12): 22.
15. Saad S, Youssef M, Idoudi S, Hadhri R, Soua Y, Toumi A, et al. Donovanosis in a Tunisian man: Atypical presentation and dermoscopic findings. Indian J Dermatol Venereol Leprol. 2021; 87(3): 393–5.
16. Sinha P, Ayub A, Madakshira MG, Praveen L, Bhattacharjee S, Alok K. Donovanosis or squamous cell carcinoma of penis: Can dermoscopy solve this enigma? Indian J Dermatol. 2022; 67(6): 807–10.
17. Sinha S, Sardana K, Agarwal A, Ahuja A. Relapsing donovanosis rhyming with unexplained decline in CD4 count in a person living with HIV/AIDS: Long-term follow up and therapeutic outcome. Int J STD AIDS. 2023; 34(6): 423–6.
18. Mangarov H, Broshtilova V, Batashki A, Velevska Y, Vatov P, Bezhanova S, et al. Donovanosis – A case report with a review of literature. Int J Clin Dermatol. 2023.
19. Kumari N, Agarwal A, Mohan A, Singh SK. A case report of donovanosis in HIV-positive female. Indian J Sex Transm Dis AIDS. 2024; 45(1): 49–51.
20. Sriram CK, Krishnaram AS. Donovanosis: Case series of a rare entity in the background of declining incidence worldwide. Indian J Sex Transm Dis AIDS. 2024; 45(2): 134–7.