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Septicemic Shock Secondary to Staphylococcal Osteomyelitis in a Ten (10)-Month-Old Thomson's Gazelle (*Eudorcas thomsonii*): A Case Report

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ABSTRACT
Septicemic shock, occurring as a complication of staphylococcal osteomyelitis, caused the fatal outcome in a 10-month-old male Thomson's gazelle (Eudorcas thomsonii) described in this report. The gazelle was presented with severe, non–weight-bearing lameness of the right forelimb following a recent translocation. Clinical examination revealed a deep, necrotizing, foul-smelling wound with an exposed, fractured ulna. Despite initial antiseptic wound care and supportive therapy, the animal's condition deteriorated rapidly. Hematology indicated severe systemic inflammation with marked leukocytosis, and a wound culture confirmed pure growth of *Staphylococcus aureus*. The gazelle succumbed to the infection approximately 20 hours after presentation. Necropsy findings documented chronic-active necrotizing osteomyelitis of the right radius and ulna, complicated by fibrinous polyserositis and multi-organ congestion, consistent with septicemic shock. This case highlights the vulnerability of translocated wildlife to opportunistic infections and underscores the challenges associated with managing advanced infections in such compromised patients. The report emphasizes the need for aggressive and timely veterinary intervention, appropriate antimicrobial therapy, and enhanced biosecurity protocols for captive wildlife.

Keywords:
Eudorcas thomsonii,
Staphylococcus aureus,
Osteomyelitis,
Septicemic shock,
Wildlife translocation

INTRODUCTION

Wildlife translocation, encompassing conservation reintroductions, trade, and private collection, has become a widespread practice globally (Thelwall & Kousha, 2017). While intended to serve conservation goals, these movements introduce substantial risks to animal health, welfare, and the dynamics of disease transmission (Thelwall & Kousha, 2017; O'Brien et al., 2018). Translocation can expose animals to unfamiliar pathogens in their new environment or introduce novel diseases into recipient ecosystems, potentially threatening immunologically naive native species (O'Brien et al., 2018). Furthermore, the stress associated with capture, transport, and adaptation to captivity can cause immunosuppression, increasing susceptibility to opportunistic infections (Morisse et al., 2017). This is a particular concern for species like Thomson's gazelles (*Eudorcas thomsonii*), a small East African antelope that faces increased risk due to potential stress and unfamiliarity with captive environments when managed in facilities, especially outside their native range (Morgan & Tromborg, 2007).

Clinical reports involving Thomson's gazelles, particularly those in captive settings, are infrequent in the veterinary literature (Niyazov et al., 2016). A retrospective study on mortality across multiple captive gazelle species identified trauma as the leading cause of death, followed by bronchopneumonia and maternal neglect (Niyazov et al., 2016). The same study noted that neonatal death was prevalent in many gazelle groups, including Thomson's gazelles, with primary causes identified as low birth weight and failure of passive transfer (Niyazov et al., 2016). This leaves young animals particularly vulnerable to bacterial infections (Gimza & Cassat, 2021).

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Given their inherent flighty nature and the risks associated with confined spaces, gazelles are highly susceptible to traumatic injuries (Mason et al., 2010). This is particularly problematic for young animals whose long bones are not fully developed, as they are at higher risk for fractures and subsequent complications (Morgan & Tromborg, 2007).

Osteomyelitis, an infection of the bone, is a serious complication of traumatic injuries, particularly open fractures where the bone is exposed to environmental bacteria (Niyazov et al., 2016; Cassat & Smeltzer, 2020). The most common bacterial pathogens associated with osteomyelitis in both humans and animals are commensal staphylococci, including *Staphylococcus aureus* (Noy & Nitzan, 2022; Cassat & Smeltzer, 2020). *S. aureus* is an opportunistic pathogen capable of producing various toxins and virulence factors that facilitate infection and evade host immune responses (Tong et al., 2015). Key virulence factors associated with osteomyelitis-causing strains include surface proteins, such as MSCRAMMs, that promote adhesion to host tissues like collagen (Foster et al., 1998), as well as enzymes that allow for dissemination (Tong et al., 2015). In wild ungulates, virulent and drug-resistant *S. aureus* has been isolated from healthy individuals, highlighting the bacteria's potential to cause serious disease when predisposing factors like trauma are present (Jamali, 2017; O'Brien et al., 2018).

If left untreated or if the infection is particularly aggressive, osteomyelitis can progress to a systemic infection, leading to septicemia and, ultimately, life-threatening septicemic shock (Seilie & Wardenburg, 2017). Septic shock is a severe syndrome characterized by fever, hypotension (dangerously low blood pressure), and multi-organ dysfunction, often triggered by bacterial toxins that induce an overwhelming immune response (Seilie & Wardenburg, 2017). The pathway from localized staphylococcal infection to fatal systemic shock is well-documented in domestic animals and humans (Niyazov et al., 2016; Cassat & Smeltzer, 2020).

Despite the known disease risks associated with translocation and the prevalence of staphylococcal osteomyelitis in domestic species, cases of this specific disease in wild gazelles are rarely reported in the veterinary literature (Niyazov et al., 2016). This lack of documented cases is particularly acute in regions like West Africa, where captive wildlife veterinary practices face significant challenges, including limited diagnostic infrastructure and a scarcity of specialized knowledge (Morisse et al., 2017; Morgan & Tromborg, 2007). This report documents a fatal instance of septicemic shock stemming from staphylococcal osteomyelitis in a young, captive Thomson's gazelle. The report provides valuable insights into the potential health risks for this species in managed settings and underscores the vulnerability of translocated wildlife to common, yet serious, bacterial diseases (O'Brien et al., 2018; Thelwall & Kousha, 2017).

MATERIALS AND METHODS

Case Presentation

Patient Signalment and History

On July 10, 2025, a 10-month-old, sexually intact male Thomson's gazelle (*Eudorcas thomsonii*) weighing approximately 15 kg was presented to the Large Animal Unit of the Veterinary Teaching Hospital, Usmanu Danfodiyo University, Sokoto. The gazelle's primary presenting complaint was a noticeable difficulty walking, according to its owner.

The animal was part of a group of four gazelles purchased and transported from Maiduguri, Borno State, to Sokoto. The owner reported a prior death in the group during the translocation. A wound on the right forelimb was first observed four days prior to presentation, shortly after the gazelles arrived at their destination.

Upon initial clinical examination, the gazelle was bright, alert, and responsive (BAR). However, it exhibited severe, non-weight-bearing lameness of the right forelimb. A physical examination revealed a deep, necrotizing, foul-smelling wound on the proximal radioulnar region. The wound was discharging a large amount of thick, purulent material and had exposed a fractured section of the ulna.

Significant bilateral prescapular lymphadenopathy was noted, with the right prescapular lymph node being more severely enlarged and firmer on palpation than the left. Vital signs were recorded as a temperature of 38.6 °C, a heart rate of 90 beats per minute, and a respiratory rate of 13 breaths per minute. While a definitive reference range for Thomson's gazelles is scarce, a comparison with domestic ruminants and other gazelle species suggests the temperature was within a low-normal range, and the respiratory rate was slightly decreased. The gazelle's overall physical condition was poor, with a body condition score (BCS) of 2/5. The mucous membranes were pink, and the capillary refill time (CRT) was less than two seconds, indicating adequate peripheral perfusion at the time of examination.

This initial assessment strongly suggested a localized, infected fracture complicated by regional lymphadenopathy, with potential systemic implications given the duration and nature of the infection.

Case Management

Initial Therapeutic Interventions and Diagnostics

Upon presentation, the wound was thoroughly lavaged with antiseptic solutions, including chlorhexidine, alcohol, and povidone-iodine. A systemic antimicrobial was also administered intravenously. The clinical team

scheduled radiography and a surgical review to further assess the injury and planned for a more extended course of antimicrobial therapy.

Hematology results indicated a marked leukocytosis (14.88 × 10^3/µL) with concurrent neutrophilia, monocytosis, and lymphopenia, and microcytic hypochromic erythrocytes. A wound swab was submitted for microbiological culture, which revealed a pure growth of *Staphylococcus aureus*. These findings collectively confirmed a diagnosis of severe staphylococcal osteomyelitis of the right ulna with systemic complications.

Despite the initiated interventions, the animal's condition deteriorated rapidly. It succumbed to septicemic shock approximately 20 hours after its presentation on July 11, 2025.

Post-mortem Examination and Findings

Gross Pathology

A complete necropsy was conducted at the Department of Veterinary Pathology. External Examination: A full-thickness, ulcerative wound, measuring approximately 0.5 × 1.5 cm, was identified on the right antebrachium. The wound margins were necrotic and dark, and a foul-smelling, purulent discharge was present. The fracture was evident through the exposed underlying bone. The surrounding subcutaneous tissues were markedly edematous and discolored (Fig 1.9, Fig 1.10).

Systemic Examination

Lymphatic System: Both prescapular lymph nodes were conspicuously enlarged, with the right node exhibiting more severe, palpable enlargement (Fig 1.4).

Musculoskeletal System: Deep dissection of the wound site confirmed a diagnosis of chronic-active necrotizing osteomyelitis affecting the right radius and ulna. A sequestrum, or fragment of necrotic bone, was present, surrounded by putrid exudate (Fig 1.9, 1.10).

Respiratory System: The lungs were bilaterally congested and heavy, indicative of severe circulatory disturbance. Frothy, pink-tinged fluid was observed within the trachea and major bronchi, suggesting pulmonary edema (Fig 1.6).

Cardiovascular System: The pericardium was diffusely opaque and thickened due to the presence of a fibrinous exudate, consistent with fibrinous pericarditis, a common finding in systemic sepsis (Fig 1.5).

Abdominal Viscera: The liver capsule showed signs of inflammation consistent with perihepatitis (Fig 1.11). The spleen was contracted (Fig 1.7), a finding often associated with septic shock and intravascular volume depletion. The renal cortices showed multifocal petechial hemorrhages, suggesting disseminated intravascular coagulation (DIC) or micro-embolism.

Histopathology

Microscopic examination of tissue sections revealed the following:

Lung: Histopathology of the lung tissue confirmed severe pulmonary congestion and a mild, diffuse interstitial pneumonitis (Fig 2.1, 2.2, 2.3, 2.4).

Bone (Radius): Sections of the right radius showed extensive osteolysis (bone destruction), marked infiltration by a mixed population of leukocytes, and abundant necrotic debris (Fig 2.5, 2.6, 2.7, 2.8). The presence of abundant fibrovascular connective tissue and sequestrum formation was consistent with chronic-active necrotizing osteomyelitis.

RESULTS AND DISCUSSION

Pathological Diagnosis

The post-mortem findings support a final diagnosis of chronic-active necrotizing staphylococcal osteomyelitis of the right radius and ulna, complicated by systemic fibrinous inflammation, pulmonary congestion, and mild pneumonitis. The constellation of pathological findings, including systemic organ dysfunction, is pathognomonic for septicemic shock, which was determined to be the ultimate cause of death.

The Pathology of Staphylococcal Osteomyelitis and Septicemia

This case exemplifies the rapid and fatal progression of *Staphylococcus aureus* osteomyelitis in a young, captive wild ungulate following a traumatic injury. The clinicopathological presentation in this gazelle aligns with, yet intensifies, findings reported in other wild ungulates and young animals. While cases of localized osteomyelitis have been documented in various captive species, the rapid hematogenous dissemination leading to fatal polyserositis and shock, as seen here, is less commonly detailed in the literature for gazelles. Notably, the severe systemic inflammation (marked leukocytosis, fibrinous pericarditis, perihepatitis) mirrors the pathological cascade described in fatal staphylococcal sepsis in neonatal foals and wildlife trauma cases. However, the finding of a well-formed sequestrum alongside acute septicemic shock is distinctive, indicating a smoldering chronic infection that abruptly transitioned to an acute systemic crisis, a progression underscoring the heightened vulnerability of juvenile animals under stress. The predisposing open fracture, as observed in this case, provided an entry point for bacterial colonization, an event mediated by *S. aureus*'s expression of microbial surface components recognizing adhesive matrix molecules (MSCRAMMs) (Tong et al., 2015). These surface proteins, including those that bind collagen (e.g., adhesin Cna), fibrinogen (e.g., clumping factor A), and fibronectin (e.g., FnBPA and FnBPB), allow the bacteria

to adhere robustly to the bone matrix and surrounding host tissues (Foster et al., 1998; Arrecubieta et al., 2022). The established local infection triggered a severe inflammatory response, characterized by marked neutrophilic infiltration and subsequent osteolysis, as evidenced by the histopathological findings. The compromised vascularity within the infected and necrotic bone created an ideal microenvironment for the formation of a sequestrum, a hallmark of chronic osteomyelitis (Cassat & Smeltzer, 2020). This pathological sequela is consistent with reports of osteomyelitis in other hoof-stock species, such as deer and antelope, where trauma precedes chronic bone infection. This avascular necrotic bone, along with the formation of protective bacterial biofilms, severely impeded the penetration and efficacy of antimicrobial agents, contributing to treatment failure (Gimza & Cassat, 2021; Cassat & Smeltzer, 2020). Biofilms are particularly detrimental in bone infections, as they confer a high degree of antimicrobial resistance, often exceeding planktonic bacteria by up to 1000-fold (Ren et al., 2022).

The progression from localized infection to systemic disease was likely facilitated by several key *S. aureus* virulence factors and reflects a more fulminant course than often reported in adult animals. Secreted exotoxins, including various hemolysins and leukocidins, contributed to tissue damage and systemic inflammation (Seilie & Wardenburg, 2017). These toxins can cause widespread organ dysfunction and, through their cytolytic effects on immune cells and other host cells, enable bacterial dissemination (Tong et al., 2015). This septicemic process ultimately led to the observed fibrinous polyserositis, pulmonary congestion, and multi-organ petechiation, a pathological cascade culminating in fatal septicemic shock (Tong et al., 2015; Seilie & Wardenburg, 2017). The resulting pathological picture, fibrinous polyserositis, pulmonary edema, and a contracted spleen, is a classic, albeit severe, manifestation of septicemic shock that has been described in septicemic cases across numerous domestic and wild mammal species. The hematological findings of marked leukocytosis, neutrophilia, and lymphopenia were indicative of a severe systemic inflammatory response (Cassat & Smeltzer, 2020), while the microcytic hypochromic anemia suggested a more chronic inflammatory process was already underway (Noy & Nitzan, 2022). The contracted spleen observed on necropsy is a common finding in acute shock, reflecting splenic smooth muscle contraction in response to systemic crisis (D'Ettorre et al., 2021).

This case underscores the critical importance of proactive veterinary care in captive wildlife, particularly concerning traumatic injuries. When compared to managed cases in domestic species, the rapid demise of this gazelle highlights the diagnostic challenge posed by the marked stoicism of wildlife, which can mask signs until crisis. Early, aggressive, and appropriately targeted antimicrobial therapy, coupled with prompt surgical debridement for effective source control, are essential for managing osteomyelitis and preventing potentially lethal systemic complications (Cassat & Smeltzer, 2020). The failure of initial supportive measures demonstrates the difficulty of managing advanced infections like osteomyelitis, especially when complicated by sequestra and biofilm formation in a compromised host. Overcoming these challenges requires not only vigilance but also continued research into new therapeutic strategies. This includes exploring alternative or adjunctive treatments, such as natural compounds with demonstrated anti-staphylococcal activity in computational models (Sani et al., 2025). This case provides a comparative record for the veterinary community, illustrating that the progression and pathology of staphylococcal sepsis in vulnerable wildlife can be more rapid and severe than in their domestic counterparts, necessitating heightened vigilance.

CONCLUSION

This case achieved a detailed documentation of fatal septicemic shock secondary to disseminated *Staphylococcus aureus* osteomyelitis in a Thomson's gazelle. Objectives included characterizing the rapid disease progression from an open fracture to systemic collapse and correlating the subtle clinical presentation with definitive post-mortem findings, including severe osteomyelitis and fibrinous polyserositis. The case underscores the severe impact of concurrent stress and trauma in young, non-domestic animals, where advanced infection can develop with masked clinical signs until a terminal stage.

FIGURES
GROSS IMAGES

Fig 1.1: Gazelle post - wound

Fig 1.2: Gazelle on PM Table.

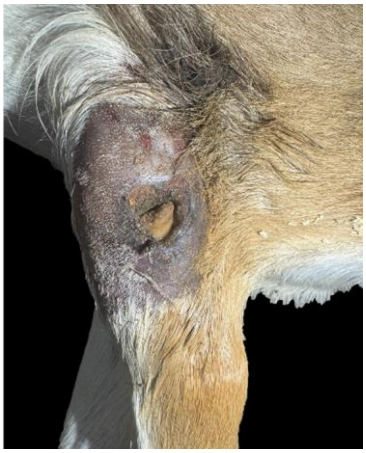

Fig 1.3: Deep wound –

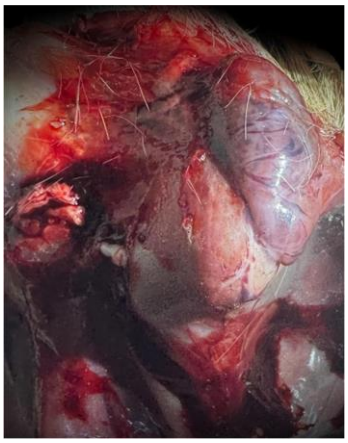

Fig 1.4: Right Prescapular Lymphadenitis

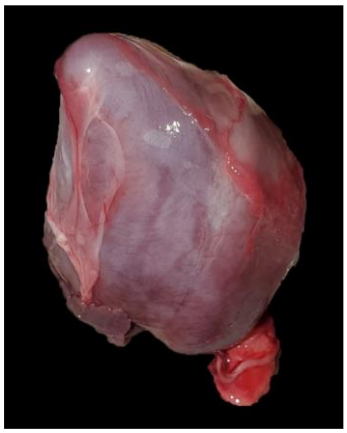

Fig 1.5: The fibrinous pericarditis

Fig 1.6: Congested Lungs & Frothy Fluid in the Trachea

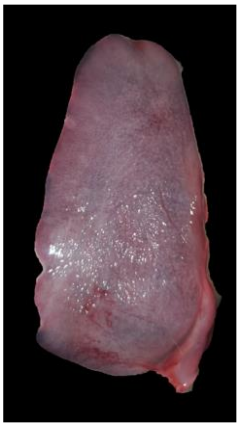

Fig 1.7: Pale and Contracted Spleen

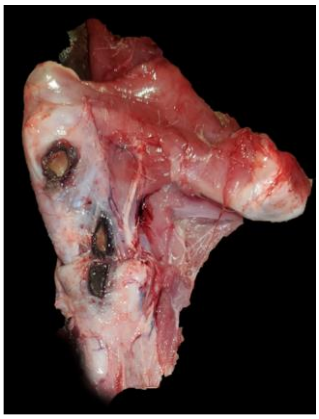

Fig 1.8: Subcutaneous Tracking Wound

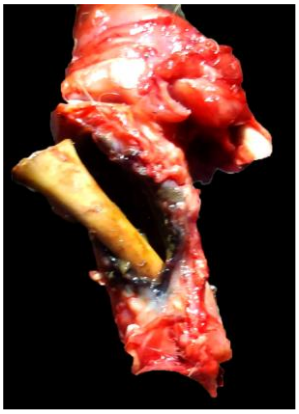

Fig 1.9: Distal Fragment Loosely Attached

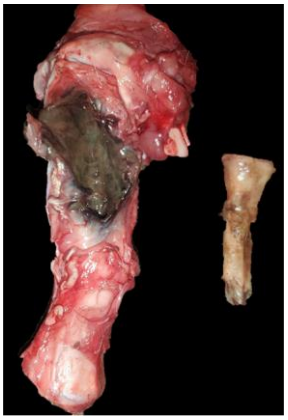

Fig 1.10: Purulent, Greenish necrotic material at fracture site

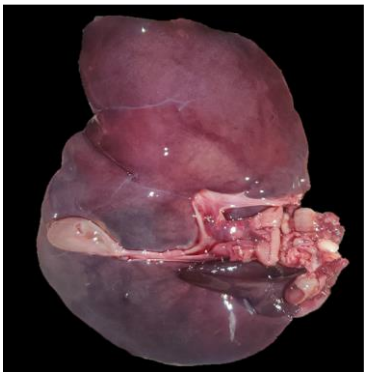

Fig 1.11: Whitish Capsule Lining

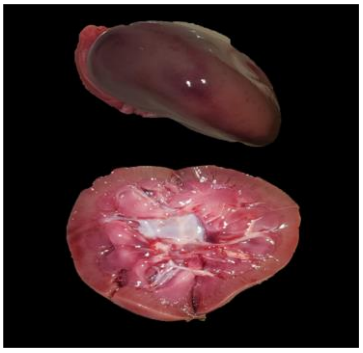

Fig 1.12: Multifocal Cortical Hemorrhages in the Kidney

HISTOLOGY SLIDES

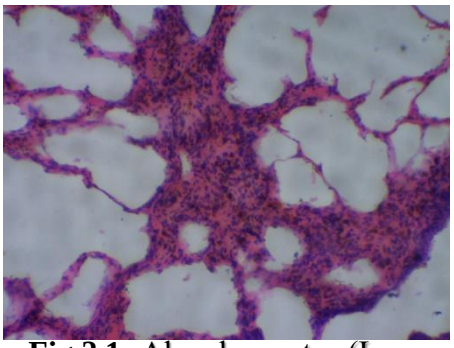

Fig 2.1: Alveolar septae (Lungs x40)

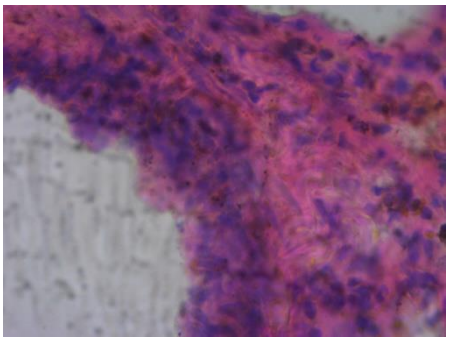

Fig 2.2: Inflammatory infiltrates (Lungs x100)

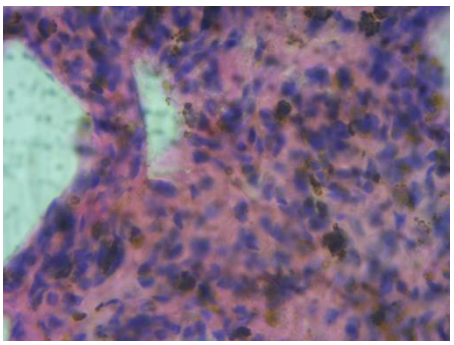

Fig 2.3: Inflammatory infiltrates (Lungs x400)

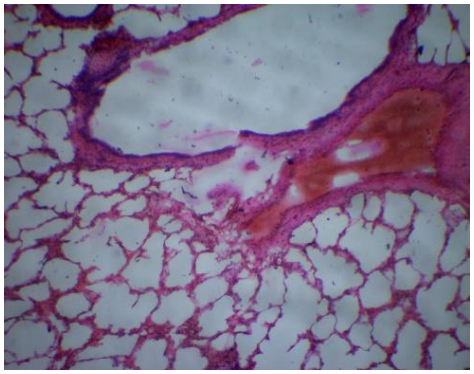

Fig 2.4: Vascular congestion (Lungs x40)

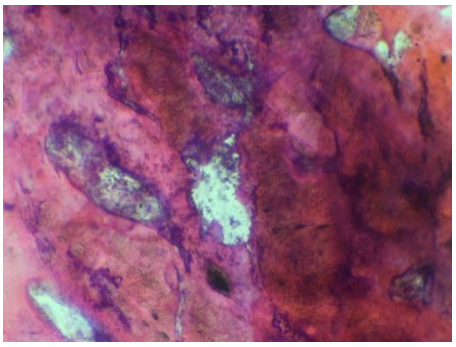

Fig 2.5: Dense fibrous and fibrovascular CT (Bone x100)

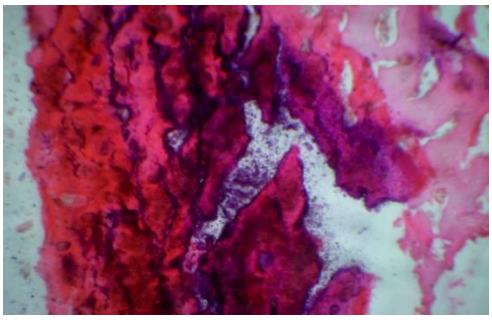

Fig 2.6: Scalloped bone (Bone x40)

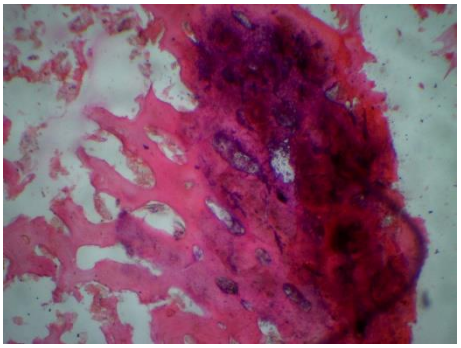

Fig 2.7: Ongoing osteolytic process (Bone x40)

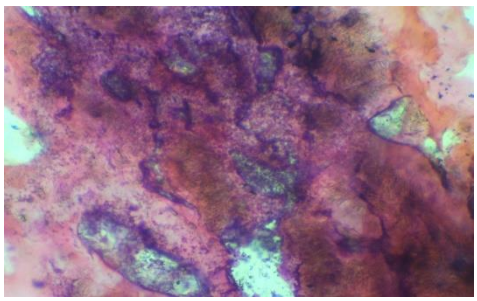

Fig 2.8: Necrotic debris and inflammatory cells (Bone x100)

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