SAS Journal of Surgery
Abbreviated Key Title: SAS J Surg
ISSN 2454-5104
Journal homepage: https://www.saspublishers.com

OPEN ACCESS

Maxillo-facial Surgery

Nodular Sclerosis Hodgkin Lymphoma Presenting as Widespread Necrotic Lymphadenopathy: A Case Emphasizing Diagnostic Pitfalls

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DOI: https://doi.org/10.36347/sasjs.2025.v11i10.007 | **Received:** 13.08.2025 | **Accepted:** 06.10.2025 | **Published:** 07.10.2025

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Abstract **Case Report**

Hodgkin lymphoma (HL) most commonly presents with painless, rubbery lymphadenopathy, whereas necrotic nodes are rare and frequently misattributed to infectious or metastatic etiologies. We report the case of a 44-year-old man with widespread necrotic lymphadenopathy involving cervical, abdominal, pelvic, and inguinal regions. Histopathology confirmed nodular sclerosis HL (NSHL) with CD30+/CD15+ Reed–Sternberg cells. The patient achieved complete remission following six cycles of ABVD chemotherapy. This case underscores the diagnostic pitfalls of necrotic lymphadenopathy, particularly in tuberculosis-endemic regions, and highlights the importance of early excisional biopsy and immunohistochemistry for accurate diagnosis and timely treatment.

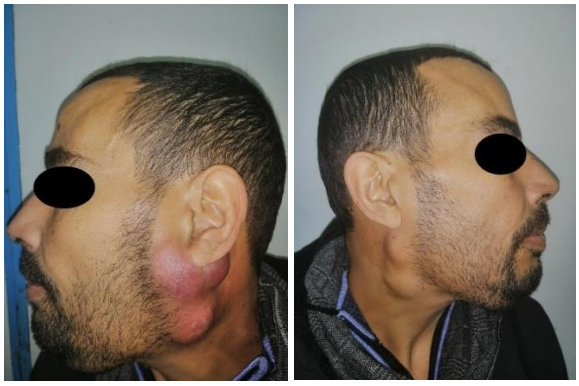
Keywords: Hodgkin lymphoma, necrotic lymphadenopathy, immunohistochemistry, ABVD, diagnostic delay.

INTRODUCTION

Nodular sclerosis Hodgkin lymphoma (NSHL) accounts for approximately 70% of HL cases, typically manifesting as localized cervical or mediastinal lymphadenopathy in young adults [1,3]. Globally, HL represents about 10% of all lymphomas, with an incidence of 2–3 cases per 100,000 annually. Necrotic lymph nodes are distinctly uncommon in HL (<10% of cases) and are more frequently associated with tuberculosis, metastatic carcinoma, or non-Hodgkin lymphoma [2,5]. Such atypical presentations may lead to diagnostic delays and inappropriate empiric therapies, particularly in tuberculosis-endemic regions [4]. We describe a rare case of NSHL with extensive necrotic lymphadenopathy, emphasizing the diagnostic challenges and the multidisciplinary approach required.

CASE REPORT

A 44-year-old man with no significant past medical history presented with a one-year history of progressive, painless swelling in the neck and submental region. He denied fever, night sweats, or weight loss. Physical examination revealed firm, non-tender lymph nodes in the bilateral cervical (submandibular and sublobular), submental, and inguinal regions. No hepatosplenomegaly was detected. [Figures 1, 2]



Figures 1 and 2: Preoperative lateral views showing cervical lymphadenopathy

Laboratory tests showed mild anemia (Hb 11.2 g/dL), elevated ESR (65 mm/h), and normal LDH. HIV and EBV serologies were negative.

Cervical ultrasound demonstrated hypoechoic lymph nodes with central necrosis. Contrast-enhanced CT confirmed multiple necrotic lymphadenopathies in cervical [Figure 3] abdominal, pelvic, and bilateral inguinal regions.

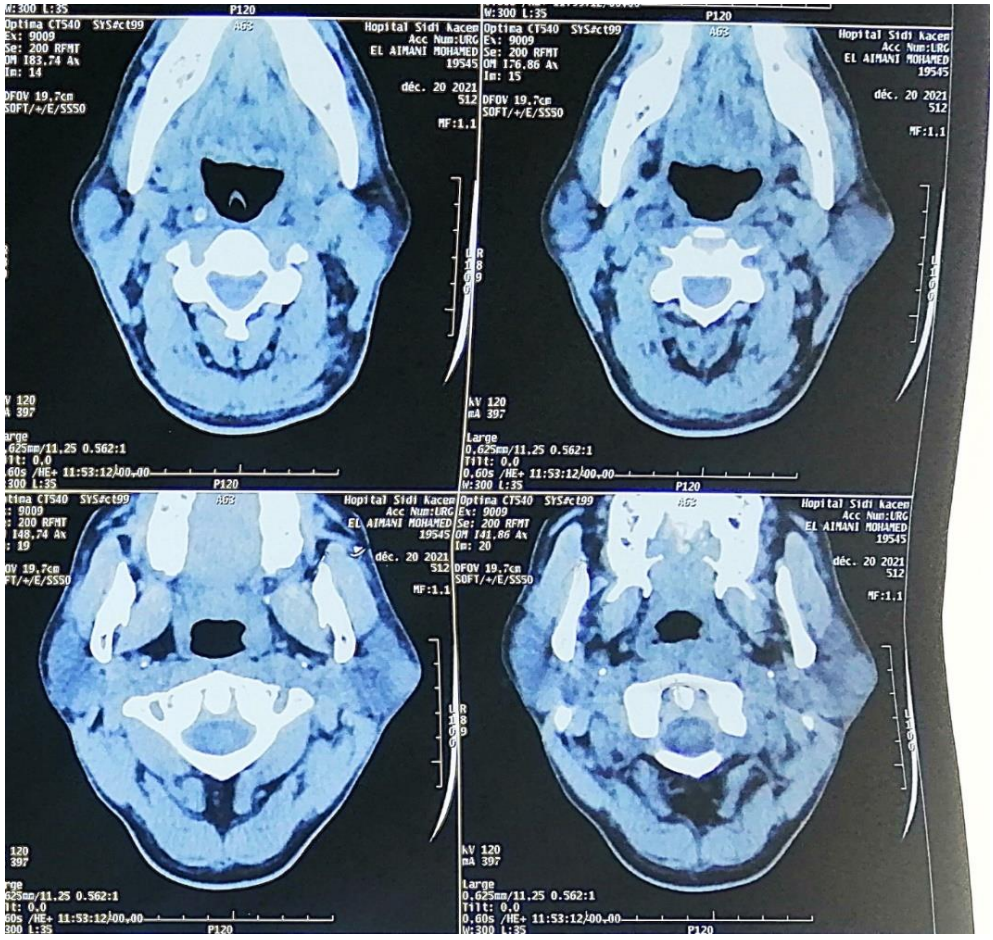


Figure 3: CT scan demonstrating necrotic cervical lymphadenopathy

Nasoendoscopy and chest X-ray were unremarkable. PET-CT revealed hypermetabolic necrotic nodes consistent with Ann Arbor stage IIIB disease.

Excisional biopsy of a submental lymph node showed fibrous bands dividing the lymphoid tissue into nodules, with scattered Reed–Sternberg cells. Immunohistochemistry demonstrated CD30+/CD15+ positivity, weak PAX5, and negative CD45/ALK. EBV in situ hybridization was negative.

The patient received six cycles of ABVD chemotherapy (doxorubicin 25 mg/m², bleomycin 10,000 IU/m², vinblastine 6 mg/m², dacarbazine 375 mg/m²) with granulocyte colony-stimulating factor support. Interim PET-CT after four cycles demonstrated a Deauville score of 2, consistent with complete metabolic response. At one-year follow-up, clinical examination and CT scan showed no residual disease, and the patient remained asymptomatic.

DISCUSSION

Necrotic lymphadenopathy is an exceptional presentation of Hodgkin lymphoma (HL), historically associated with granulomatous infections or metastatic malignancies [1]. Our case adds to the limited literature describing HL with extensive nodal necrosis, particularly in submental and sublobular regions, a pattern reported in fewer than 2% of cases [2]. While HL typically manifests as non-tender, rubbery lymphadenopathy in young adults [3], the presence of necrosis in this patient initially diverted the diagnostic workup toward tuberculosis, echoing findings from Sharma *et al.,*[4], who reported a 6-month delay in HL diagnosis due to empiric antitubercular therapy. This underscores a critical clinical pitfall: necrotic nodes in HL often lack systemic symptoms (e.g., fever, weight loss), mimicking indolent infections [5].

The anatomical distribution in our case further challenges classical paradigms. Submental lymph node involvement, observed here and in only three prior cases

[6], contrasts with the predilection of nodular sclerosis HL for cervical and mediastinal regions [7]. Müller *et al.,*[8] hypothesized that atypical nodal sites in HL may reflect aberrant cytokine signaling or microenvironmental interactions, though this remains speculative. Importantly, the coexistence of abdominal and inguinal involvement aligns with advanced-stage disease (Ann Arbor IIIB), a finding correlated with necrosis in retrospective analyses [9]. For instance, Gallamini *et al.,*[10] demonstrated that necrotic nodes on PET-CT predict bulky disease (OR 3.2, p=0.01), though our patient's rapid response to ABVD chemotherapy (complete metabolic response by cycle 4) contradicts their association of necrosis with chemoresistance. This discrepancy may reflect heterogeneity in HL biology or the efficacy of modern supportive care (e.g., G-CSF), which mitigates treatment delays [11].

Immunohistochemically, the CD30+/CD15+/EBV- profile in our case is typical of nodular sclerosis HL [12], yet the absence of B-cell markers (CD20-/PAX5-) helped exclude aggressive B-cell lymphomas, a distinction emphasized in the 2022 WHO classification [13]. Notably, the fibrous bands characteristic of nodular sclerosis were interspersed with necrotic foci, a histologic overlap with granulomatous inflammation that complicates frozen-section interpretation [14]. This reinforces the necessity of excisional (rather than core-needle) biopsies in such cases, as advocated by National Comprehensive Cancer Network guidelines [15].

Prognostically, while necrosis has been linked to inferior outcomes in some cohorts [16], our patient's sustained remission at one year aligns with larger studies showing 5-year survival rates exceeding 85% for advanced-stage HL treated with ABVD [17]. However, long-term surveillance remains crucial, as Gupta *et al.,*[18] documented a 20% relapse rate in HL patients with necrotic nodes, often within the first 24 months.

CONCLUSION

Necrotic lymphadenopathy in HL is a diagnostic challenge that requires early biopsy and immunohistochemical confirmation. Even in atypical presentations, standard chemotherapy achieves excellent outcomes. Clinicians must consider HL in the differential diagnosis of necrotic nodes to avoid delays in treatment.

ACKNOWLEDGEMENTS

The authors declare that there are no conflicts of interest related to this study. Additionally, this work did not receive any financial support from external funding sources.

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