Kikuchi-Fujimoto Disease in a Young Woman: A Rare Cause of Cervical Lymphadenopathy

Abstract

Background: Kikuchi-Fujimoto disease (KFD) is an infrequently encountered, self-resolving lymph node pathology predominantly seen among young females. In regions with high tuberculosis burden, its clinical manifestations frequently overlap with tuberculous lymphadenitis, creating diagnostic uncertainty and potentially resulting in unwarranted anti-tubercular pharmacotherapy.

Case Presentation: We describe a 24-year-old female who presented with right cervical lymph node enlargement accompanied by high-grade pyrexia, generalized weakness, unintentional weight reduction, and diminished appetite. Laboratory evaluation demonstrated leucopenia (WBC 3700/µL) alongside raised ESR (40 mm/hr). QuantiFERON-TB Gold assay yielded negative results. Excisional lymph node biopsy was performed, and AFB staining and GeneXpert MTB/RIF assay on the biopsy specimen were negative. Histopathological examination revealed hallmark features of KFD, including necrotic zones containing karyorrhectic debris enveloped by histiocytic and plasmacytoid cellular infiltrates. Serological markers for autoimmune disease were unremarkable. The patient received conservative management and achieved complete clinical and sonographic resolution over a 6-month observation period.

Conclusion: Clinicians practicing in tuberculosis-endemic settings should maintain a high index of suspicion for KFD when evaluating cervical lymphadenopathy in young women. Histopathological verification through excisional biopsy remains indispensable for establishing an accurate diagnosis and preventing unnecessary anti-tubercular pharmacotherapy.

Keywords: Kikuchi-Fujimoto disease; Histiocytic necrotizing lymphadenitis; Cervical lymphadenopathy; Tuberculosis; Excisional biopsy

INTRODUCTION

Kikuchi-Fujimoto disease (KFD), alternatively termed histiocytic necrotizing lymphadenitis, represents an infrequently diagnosed, benign lymphoproliferative condition with a marked predilection for young females[1]. This entity was first delineated independently by two Japanese pathologists, Kikuchi and Fujimoto, in the year 1972[3]. Patients typically manifest with painful cervical or regional lymph node enlargement, frequently accompanied by febrile episodes[4]. In cases with more pronounced disease severity, additional findings such as splenic enlargement, unintentional weight reduction, leucopenia, and raised erythrocyte sedimentation rate (ESR) may be encountered[2]. The exact etiopathogenesis of KFD continues to elude researchers; nonetheless, prevailing theories implicate viral infectious triggers and aberrant

autoimmune responses as contributory mechanisms[5]. Confirmatory diagnosis necessitates excisional biopsy of the affected lymph node, which reveals distinctive histomorphological patterns[4].

Herein, we report the case of a 24-year-old Indian woman whose clinical presentation of KFD closely mimicked tuberculous lymphadenitis.

CASE PRESENTATION

A 24-year-old female with no pre-existing medical conditions presented to our department with a two-week history of right-sided cervical pain and swelling, high-grade intermittent pyrexia, diffuse abdominal discomfort, and generalized fatigue. She additionally reported progressive weight loss and appetite reduction over the preceding two months. Hemodynamic parameters were within normal limits. Physical examination revealed a tender, soft, approximately 2 cm right cervical lymph node demonstrating matting. Respiratory auscultation was unremarkable with bilateral equal air entry. Abdominal ultrasonography demonstrated splenomegaly.

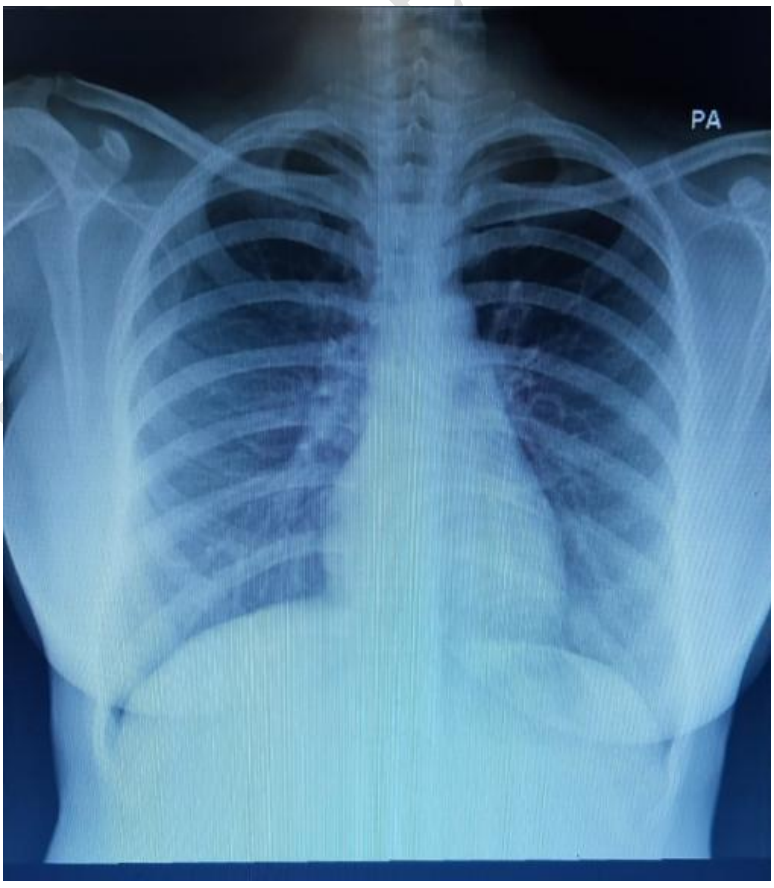

Figure 1: Chest X-ray (PA view) showing normal lung fields with no evidence of mediastinal widening, hilar lymphadenopathy, or parenchymal infiltrates.

Hematological evaluation revealed leucopenia with a total WBC count of 3700/µL. Viral serological markers were non-reactive. The ESR was elevated at 40 mm/hr, while CRP and procalcitonin remained within reference ranges. Chest radiography demonstrated no abnormalities (Figure 1). Ultrasound of the neck showed an enlarged right-sided cervical lymph node. Considering the high tuberculosis prevalence in the Indian subcontinent, mycobacterial lymphadenitis was the primary differential diagnosis given the constellation of constitutional symptoms and cervical lymphadenopathy. QuantiFERON-TB Gold assay was performed and returned a negative result.

An excisional biopsy of the right cervical lymph node was subsequently undertaken. Acid-fast bacilli (AFB) staining and GeneXpert MTB/RIF assay both yielded negative results. Histopathological evaluation (HPE) revealed focal necrotic areas containing copious karyorrhectic nuclear debris, surrounded by abundant histiocytic infiltrates, transformed lymphocytes, and plasmacytoid dendritic cells – findings consistent with Kikuchi-Fujimoto Disease (Figure 2, 3). Notably, follicular hyperplasia was absent. Autoimmune workup including rheumatoid factor, antinuclear antibodies (ANA), and anti-double stranded DNA antibodies were all non-reactive.

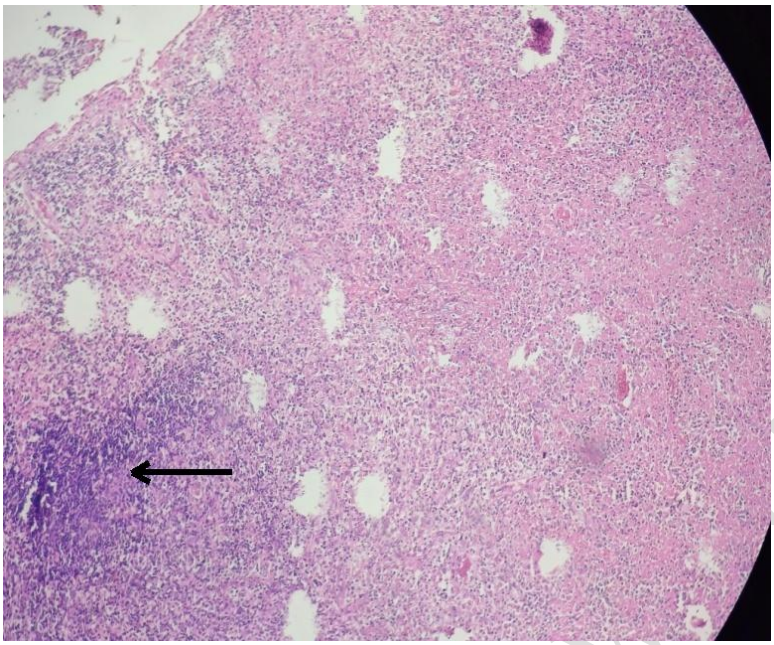

Figure 2: HPE showing histiocytes and necrosis (karyorrhectic debris – black arrow). Low power magnification. Hematoxylin and Eosin stain.

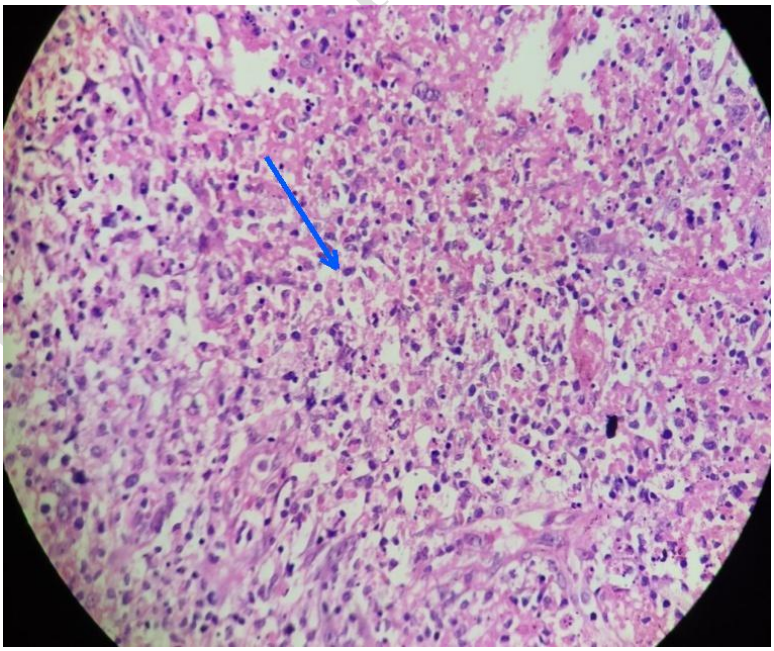

Figure 3: HPE showing histiocytes and lymphocytes (blue arrow). High power magnification. Hematoxylin and Eosin stain.

Conservative management was instituted comprising Tablet Naproxen 250 mg administered twice daily for five days along with supportive care. The patient was monitored at bimonthly intervals over a six-month duration. Progressive clinical improvement was documented with gradual regression of the cervical lymphadenopathy, and follow-up ultrasonography confirmed complete resolution of the previously enlarged lymph nodes.

DISCUSSION

KFD constitutes an uncommon lymph node pathology typified by its benign, self-resolving natural history[3,4]. The demographic profile of our patient – a 24-year-old woman with cervical lymphadenopathy – is consistent with the observations of Hoan et al. (2021), who documented that KFD preferentially affects females within the 20–35 year age bracket[5]. The clinical presentation in our case, characterized by persistent high-grade pyrexia and cervical lymph node enlargement, mirrors the findings of Ye et al. (2025) who described an analogous presentation in a young adult[4]. Furthermore, the female sex and Asian ethnicity of our patient corroborate the epidemiological data presented by Mahajan et al. (2023), who established an approximate female-to-male preponderance of 4:1 with higher incidence among Asian populations[2].

The laboratory derangements observed in our patient – leucopenia (WBC 3700/µL), raised ESR (40 mm/hr), and splenic enlargement – are concordant with the findings of Mahajan et al. (2023), who identified these as frequently encountered hematological aberrations in KFD[2]. Subahi et al. (2026) documented comparable leucopenia and systemic manifestations in their KFD patient who exhibited generalized lymphadenopathy, further supporting our observations[6]. Notably, in contrast to the widespread lymph node involvement described by Subahi et al., our patient demonstrated isolated cervical lymphadenopathy – a pattern identified by Deb et al. (2024) as the predominant mode of presentation in their multi-case analysis[3].

The diagnostic dilemma encountered in our case resonates with existing literature. Motwani et al. (2025) underscored that KFD presents considerable diagnostic hurdles owing to its phenotypic overlap with mycobacterial infection, lymphoproliferative malignancies, and connective tissue disorders[10]. Our initial clinical suspicion of tuberculous lymphadenitis, driven by the endemic context, parallels the diagnostic trajectory reported by Motwani et al. in their case[10]. The systematic exclusion of tuberculosis through AFB staining, GeneXpert, and QuantiFERON-TB Gold in our patient is consistent with the diagnostic approach advocated by Masab et al. (2023), who stressed the imperative of comprehensively ruling out infectious etiologies prior to confirming KFD[7].

With respect to etiopathogenesis, both Mahajan et al. (2023) and Subahi et al. (2026) have postulated the involvement of various viral pathogens – including EBV, HHV-6, CMV, and parvovirus B19 – as potential immunological triggers for KFD2,6. Although specific EBV DNA testing was not performed in our patient, the non-reactive viral serology does not contradict these reports, as Hutchinson and Wang (2010) elucidated that viral agents may serve as initial immunological triggers without remaining serologically detectable at the time of clinical manifestation9. This observation lends credence to the prevailing hypothesis that KFD reflects a dysregulated T-lymphocyte mediated immune cascade rather than an ongoing viral replicative process.

The histomorphological profile in our case – comprising necrotic zones replete with karyorrhectic debris, enveloped by histiocytic infiltrates, activated lymphocytes, and plasmacytoid cells, conspicuously devoid of neutrophilic or granulomatous components –

demonstrates complete concordance with the pathognomonic histological triad delineated by Puri et al. (2017)[1] and corroborated by Hutchinson and Wang (2010)[9]. Puri et al. particularly emphasized that neutrophilic absence serves as a critical differentiating criterion from suppurative lymphadenitis – a feature unequivocally demonstrated in our specimen[1]. Moreover, the conspicuous absence of granulomatous formations in our biopsy material effectively excluded tuberculous etiology at the histological level, aligning with the diagnostic framework proposed by Bosch and Guilabert (2006)[8].

Although the non-reactive autoimmune panel (ANA, anti-dsDNA, RF) in our patient provides reassurance, Bosch and Guilabert (2006) cautioned that KFD may temporally precede, coexist with, or develop subsequent to systemic lupus erythematosus[8], thereby warranting continued clinical vigilance. The favorable therapeutic outcome in our patient – complete resolution following conservative NSAID-based management over six months – corroborates the self-limiting disease trajectory documented by Deb et al. (2024), who reported spontaneous resolution within one to four months in the majority of cases[3]. This outcome also parallels the experience of Hoan et al. (2021), who observed analogous spontaneous remission with supportive therapeutic measures[5]. The absence of corticosteroid requirement in our case is consistent with the therapeutic stratification proposed by Subahi et al. (2026), who recommended reserving glucocorticoid therapy for severe or treatment-refractory presentations[6].

Our report carries certain limitations. Immunohistochemical characterization utilizing CD68 and MPO markers was not performed, which could have provided additional diagnostic confirmation as advocated by Masab et al. (2023)[7]. Furthermore, while the six-month

surveillance period adequately documented disease resolution, it may prove insufficient for detecting delayed recurrences, given that Deb et al. (2024) documented recurrence rates approximating 3–4% in published literature[3].

CONCLUSION

In tuberculosis-endemic nations such as India, the clinical triad of cervical lymphadenopathy, elevated ESR, and constitutional symptoms warrants a comprehensive diagnostic evaluation rather than empirical initiation of anti-tubercular pharmacotherapy. Histopathological assessment of an excised lymph node remains the cornerstone for establishing a definitive diagnosis. Clinicians must maintain awareness of alternative etiologies, particularly KFD and autoimmune lymphadenopathy including systemic lupus erythematosus, which may present with overlapping clinical features. Prolonged clinical surveillance is recommended to monitor for potential disease recurrence or evolution into autoimmune pathology.

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