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# RESEARCH ARTICLE

## CASE REPORT: KIKUCHI-FUJIMOTO DISEASE PRESENTING WITH PERSISTENT FEVER AND GENERALIZED LYMPHADENOPATHY IN AN ADOLESCENT GIRL

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## ABSTRACT

**Introduction:** Kikuchi-Fujimoto disease, or histiocytic necrotising lymphadenitis, has an indeterminate aetiology, with potential autoimmune or viral precipitating factors. The diagnosis necessitates a substantial level of clinical suspicion owing to its generic manifestation and the imperative to rule out viral and neoplastic aetiologies. We present a case of a 16-year-old female with persistent fever, cervical and axillary lymphadenopathy, and urticarial eruptions, ultimately diagnosed with Kikuchi-Fujimoto disease following excisional lymph node biopsy. **Case Presentation**: A 16-year-old female presented to the outpatient department with a 1.5-month history of high-grade fever, cephalalgia, myalgia, and anorexia. A painful mass was identified in her left axilla, accompanied by cervical swellings. Facial and thoracic rashes manifested one week prior to presentation. Following ineffective empirical treatment at a different hospital, she was admitted for additional evaluation. Palpation indicated painful left axillary lymphadenopathy and enlarged cervical lymph nodes at levels II and IV. Papular and urticarial eruptions affected the chest and face. Soreness in the upper belly was detected during the systemic examination; however, cardiovascular, respiratory, and neurological assessments yielded normal results. The complete blood count, liver function tests, kidney function tests, and chest X-ray results were all normal. Culture of sterile urine and blood samples. Elevated ESR (40 mm/hr) and CRP (80.8 mg/L). CECT of the abdomen revealed hepatomegaly, moderate ascites, and mesenteric lymphadenopathy, while CECT of the chest showed necrotic axillary lymphadenitis. The ANA and Mantoux tests yielded negative results. Serology results indicate negative HIV, HBsAg, and HCV. General anaesthesia was employed for the excisional biopsy of the left axillary lymph node. Histopathological findings of necrotising lymphadenitis indicated Kikuchi disease, corroborated by immunohistochemistry. The initial therapy comprised intravenous antibiotics and paracetamol. She defervesced and was discharged after stabilisation. Naproxen was used for fever during the follow-up. We initiated and gradually reduced oral steroids (Prednisolone 30 mg). She has shown clinical improvement and has remained asymptomatic for one month. **Conclusion:** In adolescents exhibiting protracted fever, lymphadenopathy, and rash, Kikuchi-Fujimoto disease must be contemplated following the exclusion of more prevalent causes. Excisional lymph node biopsy is essential for diagnosis, and prompt commencement of anti-inflammatory treatment can lead to full recovery.



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# INTRODUCTION

Fever and subacute necrotizing regional lymphadenopathy are the hallmarks of Kikuchi–Fujimoto disease (KFD), also known as histiocytic necrotizing lymphadenitis. (1) It was first documented in 1972 by Japanese pathologists Kikuchi and Fujimoto (2,3). Although there are cases in juveniles, it mainly affects women aged 20 to 35, with a mixed gender distribution (4). Although the exact cause of Kikuchi-Fujimoto disease is unknown, it is believed to be brought on by an inflammatory cascade that is triggered by an infectious agent, an autoimmune condition, or physical trauma. In the end, this cycle results in the release of inflammatory cytokines and T-cell-mediated death. (5) This extreme inflammatory reaction can have negative effects, such as the development of cancer or systemic lupus erythematosus.

An excisional biopsy of an enlarged lymph node is necessary to diagnose Kikuchi-Fujimoto disease, and immunohistochemistry is crucial for ruling out other differential diagnoses. (6) To confirm the diagnosis of Kikuchi-Fujimoto disease and differentiate it from other conditions, such as lymphoma, immunohistochemistry is crucial. Myeloperoxidase, lysozyme, CD68, CD163, and CD4 all show positive staining in the typical immunohistochemical profile (7, 8). Since Kikuchi-Fujimoto disease frequently follows a self-limiting trajectory with spontaneous remission occurring within 1 to 4 months or 1 to 6 months, there is no proven treatment. A key component of treatment is supportive management, which includes using analgesics and antipyretics to reduce symptoms. After infectious causes have been ruled out, patients with severe sickness may require a prolonged corticosteroid taper. Other therapies that have shown promise in the past include intravenous immunoglobulin, minocycline, or

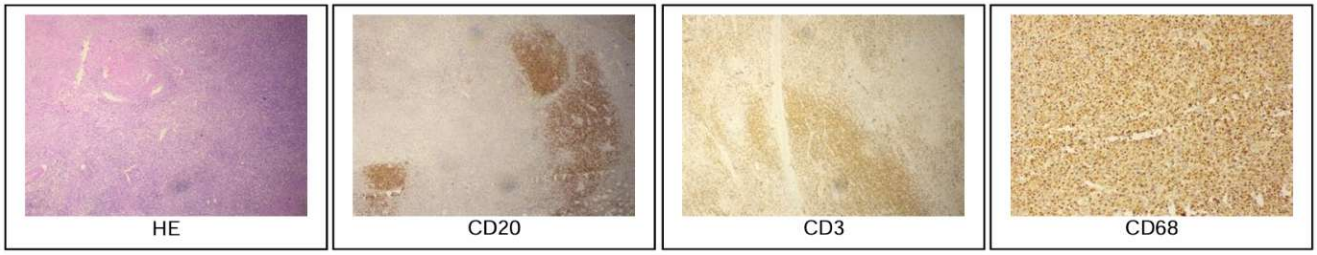


**Figure 1.Histopathology markers**

hydroxychloroquine (9, 10). We report a case of a 16-year-old girl presenting with prolonged fever, cervical and axillary lymphadenopathy, and urticarial rashes, ultimately diagnosed as KFD following excisional lymph node biopsy.

**CASE REPORT**

A 16-year-old girl, weighing 62 kg, came to the outpatient department with complaints of high-grade fever that came and went for 1.5 months. She also had headaches, body aches all over, and a loss of appetite. She also felt pain and swelling in the left axilla and felt swellings in the neck while she was sick. One week before the presentation, she got rashes on her face and chest that were red and raised, and they looked like hives. She had received empirical medical treatment at a different healthcare facility, including antibiotics and antipyretics, but her symptoms did not get better. She was then admitted for more tests. The patient looked sick and had a fever, with a temperature of 102.8°F. Her vital signs were stable: her oxygen saturation was 97% on room air, her pulse rate was 110 beats per minute (regular), and her blood pressure was 120/70 mmHg. The general physical exam showed that the cervical lymph nodes at levels II and IV were enlarged, firm, and slightly tender. There was also a swollen, painful lymph node in the left axilla. A skin exam showed papular and urticarial rashes on the chest and face. The abdominal exam showed that the upper abdomen was sore, but there was no organomegaly when palpated. The cardiovascular, respiratory, and neurological exams were normal.

**Investigations:** Baseline laboratory tests, such as a complete blood count, liver function tests, kidney function tests, and a chest X-ray, were all normal. There were no bacteria in the blood or urine cultures. The erythrocyte sedimentation rate (ESR) was 40 mm/hr and the C-reactive protein (CRP) was 80.8 mg/L, which are both signs of inflammation. Contrast-enhanced computed tomography (CECT) of the abdomen showed that the liver was enlarged, there was a little bit of fluid in the abdomen, and there were swollen lymph nodes in the mesentery. CECT of the chest showed necrotic left axillary lymphadenitis. Autoimmune screening, which included testing for antinuclear antibodies (ANA), came back negative. The Mantoux test for tuberculosis came back negative. Serological assays for HIV 1 and 2, hepatitis B surface antigen (HBsAg), and hepatitis C virus (HCV) yielded negative results. Due to ongoing fever and lymphadenopathy with inconclusive non-invasive tests, an excisional biopsy of the left axillary lymph node was conducted under general anesthesia. Histopathological examination demonstrated the disruption of nodal architecture, marked by extensive coagulative necrosis, substantial karyorrhectic debris, histiocytes, crescent-shaped nuclei, and the absence of neutrophils or granulomas. The Ziehl–Neelsen test for acid-fast bacilli yielded a negative result. These findings suggested necrotizing lymphadenitis. Immunohistochemistry revealed CD68-positive histiocytes, numerous CD8-positive T lymphocytes, an intact follicular dendritic cell network accentuated by CD21, and an elevated Ki-67 proliferation index in proximity to necrotic regions. The B-cell markers (CD20, PAX-5) displayed reactive patterns, and the findings were consistent with Kikuchi–Fujimoto disease.Differential Diagnosis: The differential diagnoses included tubercular lymphadenitis, lymphoma, systemic lupus erythematosus, and viral infections. Negative microbiological studies, autoimmune markers, the absence of Reed–Sternberg cells, the lack of granulomas, and unique histopathological characteristics enabled the exclusion of these conditions. Management and Outcome: The initial treatment administered was antibiotics and antipyretics (paracetamol) by intravenous infusion. She had ceased to exhibit a fever and was sufficiently stable to be discharged from the hospital. Upon her return for a follow-up, she continued to experience intermittent fever, prompting the addition of naproxen. The symptoms persisted, prompting us to administer prednisolone at a dosage of 30 mg daily, which was gradually reduced over the subsequent weeks. The patient exhibited significant clinical improvement, with full remission of fever, lymphadenopathy, and cutaneous rash. At the one-month follow-up, she remained asymptomatic, with no symptom recurrence.

# DISCUSSION

One uncommon cause of lymphadenopathy is KFD, also known as histiocytic necrotizing lymphadenitis. Patients in Asia are more likely to have its symptoms (11). Because KFD is prone to being mistaken with other lymphadenopathy etiologies, its exact incidence cannot be determined. 330 cases of KFD since 1991 were reported in a 2007 review by Kucukardali et al. (12). According to a 2014 research by Feder et al. (13), there had been 10 instances reported in the US. Although the etiology and pathophysiology of KFD are yet unknown, pathogenic organisms and an autoimmune reaction are thought to be the two main causes (14). KFD can begin suddenly or subacutely and go away on its own in a matter of weeks. The lymph nodes are between 0.5 and 4.0 cm in size. Patients with KFD frequently have painful lymphadenopathy and fever. For one to seven weeks, up to 90% of people experience fevers with temperatures between 38.6°C and 40.5°C. 82% of KFD patients had painful lymph nodes. Signs include weakness, headache, nausea, vomiting, weight loss, nocturnal sweating, and upper respiratory symptoms (15,16). Histological results show preserved nodal architecture, varying degrees of coagulative necrosis with substantial karyorrhectic debris in the paracortical regions, and neither neutrophils nor eosinophils (17). Necrotic areas are usually surrounded by plasma-cytoid monocytes and crescent-shaped histiocytes (18). Histiocytes from patients with chronic renal disease express CD68, myeloperoxidase, and CD4. The main sign of our patient's illness was a prolonged, high-grade fever that lasted for 1.5 months and was accompanied by cutaneous, constitutional, and painful lymphadenopathy symptoms. The two symptoms of KFD that are most frequently described are fever and cervical lymphadenopathy. But involvement of the axillary and mesenteric lymph nodes, as in this instance, is less commonly reported and may lead to other diagnoses, such as metastatic illness, lymphoma, or tuberculosis. About 10–30% of cases have a skin rash, which complicates the clinical presentation and may indicate autoimmune illnesses, including systemic lupus erythematosus (SLE). The specificity of laboratory data for Kikuchi disease is often lacking. Our patient had a normal complete blood count, despite the common observation of leukopenia and slight anemia, demonstrating that good hematological parameters do not rule out the diagnosis. This instance demonstrates that high inflammatory markers, such as CRP and ESR, are typical and indicate an ongoing inflammatory process. Imaging tests revealed mesenteric lymphadenopathy, hepatomegaly, mild ascites, and necrotic axillary lymphadenitis. Although they are uncommon, severe or widespread cases of the disease have been observed to exhibit them. The low specificity of imaging in detecting

KFD is highlighted by the fact that these radiological features often resemble cancers or viral causes. Given that Kikuchi illness typically recovers spontaneously, supportive care constitutes the primary therapeutic approach. Nonsteroidal anti-inflammatory drugs adequately alleviate symptoms for most people. Due to a continuous fever and systemic involvement, our patient necessitated systemic corticosteroid therapy, which swiftly and permanently resolved their illness. This corroborates previous research indicating that corticosteroids are effective when NSAIDs are insufficient or in cases of severe, chronic, or extranodal disease. (19)

# CONCLUSION

This case highlights the necessity of evaluating Kikuchi–Fujimoto disease in adolescents exhibiting persistent fever and lymphadenopathy. To avoid misdiagnosis and unnecessary aggressive treatment, it is important to do an early excisional lymph node biopsy and get histopathological confirmation. Prompt commencement of corticosteroids in chronic cases results in superior clinical outcomes.

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