ORIGINAL ARTICLE

# Kikuchi-Fujimoto Disease: An Experience from a Tertiary Care Center in South India

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## Abstract

**Introduction:** Kikuchi-Fujimoto disease (KFD), also known as histiocytic necrotizing lymphadenitis is a benign, usually self-limiting condition, that usually affects females under the age of 30 years. The etiology remains unknown.

**Methods:** Analysis of all cases of KFD diagnosed at our center from January 2019 to March 2021 was carried out. A total of seven cases with KFD were included in the study. The clinical and laboratory parameters, associated comorbidities, and treatment outcomes were studied and analyzed in detail.

**Results:** The mean age of presentation was 11 years with a female predominance. Fever and lymphadenopathy were noted in the majority; while weight loss and fatigue were noted in 5/7. One patient had a recurrence of KFD, whereas two sisters presented with familial KFD. Most patients had leukopenia and raised ESR at presentation. ANA was positive in 5/7 (71%) of patients, of which two had systemic lupus erythematous at presentation. Steroids were used in all except one patient.

**Conclusion:** We hereby report our experience in the diagnosis and management of KFD and re-emphasize that KFD must be considered as a possibility in febrile children with lymphadenopathy.


**Keywords:** Familial Kikuchi disease, Histiocytic necrotizing lymphadenitis, Kikuchi-Fujimoto disease, Lupus erythematosus, Recurrent Kikuchi disease, Systemic.

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## Introduction

Kikuchi-Fujimoto disease (KFD) also known as histiocytic necrotizing lymphadenitis was first described by Kikuchi and Fujimoto from Japan in 1972.[1,2] KFD is more common among Asian patients, possibly linked to some haplotypes.[3]

Kikuchi disease is known to be a benign, usually self-limiting condition, that usually affects females under the age of 30 years. The etiology is however unknown, although viral or autoimmune pathogenesis has been suggested.[4] The classic presentation includes fever and tender cervical lymphadenopathy. KFD most commonly involves the posterior cervical lymph node group and most often the involvement is unilateral. The other less common manifestations include fatigue, weight loss, axillary and mesenteric lymphadenopathy, splenomegaly, cutaneous rash, parotid gland enlargement, myalgia, arthralgia and aseptic meningitis, interstitial lung disease, and bone marrow hemophagocytosis. Elevated erythrocyte sedimentation rate (ESR), mild neutropenia, and lymphocytosis is seen in most patients.[5–8]

This study reports a case series of seven patients diagnosed with KFD at our center. The clinical and laboratory parameters, associated comorbidities, and treatment outcomes have been studied and analyzed in detail.

### Patients and Methods

This is a retrospective, observational study of cases diagnosed with KFD in the Pediatric Immunology & Rheumatology Unit at Aster CMI Hospital, Bengaluru, India. Our hospital serves as a tertiary care center in the Southern city of Bengaluru in India. During the period, January 2019 to March 2021, a total of seven children were diagnosed with KFD based on clinical and histopathological findings. The inclusion criteria were all patients diagnosed with KFD and confirmed on lymph node biopsy. Lymph node biopsy samples were subjected to routine Gram stain, Ziehl-Neelsen (ZN) stain, and bacterial and fungal cultures. The study included three male and four female patients in the age-group of 10–18 years. The age, gender, year of diagnosis, symptoms, localization of lymphadenopathy, findings on physical examination, laboratory findings, comorbid diseases, and treatment choices were recorded. This work has been approved by the Institute Ethics committee Board.

### Case 1 (P1)

A 13-year-old boy presented to our institute in December 2020 with the second episode of KFD. He had the first episode at the age of 12 when he developed fever and right cervical lymphadenopathy and fine-needle aspiration cytology (FNAC) of the node showed

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necrotizing lymphadenitis suggestive of KFD. He had leukopenia and antinuclear antibody (ANA) by IF was found to be positive (+1). He responded to NSAIDs during the first episode.

Now, he presented with left-sided neck swelling for 2 months and fever for 10 days. He had been treated with intravenous antimicrobials with no response.

On examination, he had tender left-sided lower cervical and supraclavicular lymphadenopathy and aphthous ulcers on the buccal mucosa. Investigations showed hemoglobin of 138 gm/L, a total leukocyte count of 2.85 x $10^9$/L ($N_{45}$ $L_{44}$ $M_0$), and a platelet count of 200 x $10^9$/L. ESR (40 mm/hr) was elevated whereas C-reactive protein (6 mg/L) was normal. Tests for tropical infections like dengue, malaria, tuberculosis, and typhoid were negative. ANA was positive (2+) and C3, C4 was normal. He underwent a lymph node biopsy that showed paracortical necrosis, karyorrhectic debris surrounded by crescentic histiocytes, and the absence of neutrophils. A diagnosis of recurrent KFD was made and he was started on naproxen. Despite 3 weeks of regular therapy, he continued to remain febrile and had persistent leukopenia [TC 1.78 x $10^9$/L ($N_{49}$ $L_{41}$ $M_6$)]. In view of the ongoing disease activity, he was started on oral steroids (1 mg/kg/day). He responded promptly, became afebrile by 1 week, and counts normalized. Steroids were stopped by 6 weeks. On follow-up, he continues to remain well for the last 6 months.

## Case 2 (P2)

A 14-year-old girl presented with fever for 2½ months associated with easy fatigability and swelling on the right side of the neck. She also complained of decreased appetite and weight loss (2 kg) over the past 2½ months.

On examination, she had tender right posterior cervical lymphadenopathy. Her Hb was 120 gm/L, total counts were 3.70 x $10^9$/L and platelet counts were 240 x $10^9$/L. ESR was raised (25 mm/hr) and CRP was normal. Tests for all tropical infections were negative. ANA test was positive (2+). However, ANA profile was negative. Complement levels C3 (100) and C4 (25) were normal. USG neck showed matting of left cervical lymph nodes following which a biopsy was done which reported necrotizing histiocytic lymphadenitis suggestive of KFD. She was started on oral steroids to which she responded promptly. Steroids were tapered over 4 weeks and stopped. On follow-up she remains well and has had no recurrence of symptoms for the past 1.5 years.

## Case 3 (P3)

An 18-year-old-girl presented with fever for a month associated with weight loss (1 kg). She had no other systemic symptoms. On examination, her left submandibular node and right cervical lymph node were enlarged and tender. A year back her younger sister (case 2) was diagnosed with KFD and was treated for the same. On investigation, she had a hemoglobin of 111 gm/L, total counts were 2.7 x $10^9$/L with a platelet count of 160 x $10^9$/L. CRP was normal. Test for EBV infection was negative. Considering lymphoma as a clinical possibility, the treating physician performed a CT scan chest and abdomen that showed enlarged cervical, mesenteric, supraclavicular, axillary, and retroperitoneal lymph nodes. Lymph node biopsy of right cervical lymph node reported changes of KFD. ANA test performed was negative. She was started on naproxen therapy to which she responded temporarily followed by recurrence of fever spikes 2 weeks later. Her counts continued to remain low (3.54 x $10^9$/L) and her ESR was high (47 mm/hr). Due to ongoing disease activity, she was started on oral steroids at (1 mg/kg/day) to which she responded promptly. Her neck swellings reduced in size and she continues to remain afebrile on follow-up.

## Case 4 (P4)

A 17-year-old girl presented with fever on and off for 3 months. She also complained of swelling on the left side of her neck 3 months back which had subsided after a course of antibiotics. A month later she developed swelling on the right side of her neck which was tender and associated with high-grade fever. She complained of poor appetite and had lost 9 kg weight over a period of 3 months. She also had a history of hair fall and oral ulcers.

On examination, she had pallor and bilateral tender postauricular lymphadenopathy. Her hemoglobin was 70 g/L, total counts were 3.08 x $10^9$/L ($N_{73}$ $L_{22}$) with a platelet count of 207 x $10^9$/L. Her ESR (120 mm/hr) and CRP (76 gm/L) were raised, however tests for all tropical infections were negative. With a high suspicion for systemic lupus erythematosus (SLE), she was investigated further. ANA by Immunofluorescence was positive (3+, homogeneous pattern). ANA profile was positive for multiple antibodies (RNP/Sm, anti-Sm antibody, anti-RO-52, anti-SS-A antibody, anti-histone antibody, and anti-ribosomal-P-protein antibody), and her complement levels (C3, C4) were low. Lymph node biopsy confirmed the diagnosis of KFD. She was diagnosed to have SLE with KFD and was given an intravenous pulse dose of methylprednisolone and started on hydroxychloroquine. She became afebrile and lymphadenopathy decreased in size. On follow-up, her leukopenia improved and she has been managed on steroids, HCQS, and methotrexate.

## Case 5 (P5)

A 14-year-old boy presented with high-grade fever for 14 days, fatigue, and loss of appetite without any localizing features (cough/diarrhea/ear pain/burning micturition/arthritis/jaundice). He had no response to a course of antibiotics and hence was referred to our center.

On examination, he had no lymphadenopathy or organomegaly. He had a hemoglobin of 128 gm/L, total counts of 4.60 x $10^9$/L ($N_{52}$ $L_{41}$), and platelets counts of 255 x $10^9$/L. ESR (45 mm/hr) was high but his CRP (6.7 gm/L) was normal. He was extensively investigated for tropical infections like dengue, malaria, leptospira, brucella, tuberculosis, and typhoid but all tests were reported negative. He continued to spike temperatures on the first three days of admission and developed progressive leukopenia (1.60 x $10^9$/L) and mild thrombocytopenia (1.36 x $10^9$/L). Since no clear focus was identified a PET-CT scan was performed which showed enlarged left axillary lymph nodes with significant uptake. Lymphoma being high on cards, a lymph node biopsy was performed. Biopsy showed features suggestive of KFD and malignancy was excluded. On further evaluation, the ANA test was reported positive (2+) and the ANA profile was positive for RNP/Sm (3+). Complement levels were normal. He was started on naproxen therapy following which fever spikes reduced but he continued to have a daily low-grade fever and leukopenia and thrombocytopenia persisted and three days later, oral steroids (1 mg/kg/day) were added, following which he became afebrile, ESR, and CRP improved and cytopenia showed resolving trend. Steroids were tapered over a period of 2 months and on follow-up, he continues to remain well.

## Case 6 (P6)

A 6-year-old girl was referred to our center in view of prolonged fever on and off for 6 months and periorbital puffiness for the last 1 week and abdominal distension for a day.

On examination, she was noted to have periorbital edema, generalized lymphadenopathy, and splenomegaly. Initial investigations revealed anemia (81 gm/L), total counts

**Table 1:** Characteristics of patients with Kikuchi-Fujimoto disease

| *Baseline characteristics* | *N (n = 7)* |
|---|---|
| Age | 14 (10–18 yr) |
| Sex | |
| Male | 3 (42) |
| Female | 4 (57.1) |
| Clinical features | |
| Fever (≥39.0) | 7 (100) |
| Fatigue | 4 (57) |
| Weight loss | 5 (71) |
| Cervical lymphadenopathy | 3 (57) |
| Axillary lymphadenopathy | 2 (28) |
| Lymph nodes (>2 localizations) | 2 (14) |
| Tender lymphadenopathy | 5 (71) |
| Splenomegaly | 1 (14) |
| Oral ulcers | 2 (28) |
| Laboratory features | |
| Raised ESR | 5 |
| Leukopenia | 5 |
| Thrombocytopenia | 1 |
| ANA | 5 |
| Anti-dsDNA | 2 |
| Other antibodies | 3 |
| Treatment | |
| NSAIDs | 5 |
| Corticosteroids | 6 |
| SLE with KFD | 2 |
| Recurrence of KFD | 1 |
| Familial KFD | 1 |

(7.50 x $10^9$/L) and platelets (156 x $10^9$/L) were normal. ESR (108 mm/hr) was high. Direct Coombs test was positive and peripheral smear was reported to have schistocytes. Tests for all tropical infections were negative. Blood and urine cultures were sterile. ANA test reported positive (4+, homogeneous pattern) and ANA profile was positive for anti-smith and anti-dsDNA antibodies. Serum complement levels C3 and C4 were markedly reduced. During the course of the hospital stay, the child developed proteinuria, and a renal biopsy was done to report class 3 lupus nephritis. Therefore, a diagnosis of SLE with lupus nephritis and auto-immune hemolytic anemia was made. As the child had generalized lymphadenopathy, a lymph node biopsy of the right cervical lymph node was performed which revealed necrotizing histiocytic lymphadenitis suggestive of KFD. She was initiated on injection methylprednisolone pulse dose (30 mg/kg/day x 5 doses) to which she responded promptly. Fever subsided and edema along with lymphadenopathy and splenomegaly resolved by the next 7 days. On further evaluation, she had a positive lupus anticoagulant and was started on aspirin (5 mg/kg/day). She was treated with steroids, mycophenolate mofetil, hydroxychloroquine, and remains well for the past 4 years.

### Case 7 (P7)

A 12-year-old boy presented with a high-grade fever for 14 days. A month back he also had joint pains and was treated elsewhere with a short course of analgesics and steroids. Later he developed a hyperpigmented rash over the face and limbs. His symptoms resolved with intravenous antibiotics however he continued to spike temperatures. He had also lost weight (1 kg) over the last 1 month.

On examination, he had axillary lymphadenopathy (right>left) 2 cms in size which was non-tender. His hemoglobin was 116 gm/L, total counts were 4.1* $10^9$/L, and platelet counts were 150 x $10^9$/L. Workup for pyrexia of unknown origin (PUO) was negative. On serial CBC his counts increased from 4 x $10^9$/L to 12 x $10^9$/L. A lymph node biopsy of the right axillary lymph node was performed which showed changes in KFD. ANA test done was negative. He was started on naproxen therapy following which he became afebrile and remains well on follow-up.

## Results

A total of seven patients with KFD were included in the study. The main characteristics of these patients are shown in Table 1. The mean age at diagnosis was 14 years (range 10–18 years). Of the seven patients enrolled in the study, three were males and four were females. While all patients had a fever, clinical lymphadenopathy was noted in 6/7. Two of them were diagnosed to have SLE at presentation. Recurrence of disease was seen in one patient. We had one case of familial Kikuchi involving two sisters, diagnosed a year apart from each other.

Most patients ($n$ = 5) were treated with Non-steroidal anti-inflammatory drugs (NSAIDs), and almost all patients required the addition of steroids for the resolution of symptoms. Only one responded to NSAIDs.

## Discussion

Kikuchi-Fujimoto disease is a rare, self-limiting, idiopathic disease, affecting adolescents, and young adults who usually present with fever and cervical lymphadenopathy.[9,10] It is fairly common among the Asian population.[11] This case series includes seven patients with KFD diagnosed at our center.

The mean age of presentation in our study was 14 years, with a male to female ratio of 1:1.33. A case series from Northern India reported six patients with a mean age of 10.8 years and a male to female ratio of 2:1.[12] Kucukardali et al. in their analysis of a large cohort of KFD reported that 70% of the patients were younger than 30 years with the majority being females.[13]

All children in our cohort presented with fever whereas, easy fatigability, weight loss, and tender cervical lymphadenopathy were the other common presentations. These findings were consistent with other studies which reported fever and cervical lymphadenopathy as the commonest presentations.[10,13,14]

Weight loss was reported by a significant number of patients (71%) in our cohort. Though reported previously, the exact incidence of weight loss in patients with KFD has not been well defined. Dumas et al. reported that 47% of patients had weight loss as one of the presenting symptoms.[15]

Axillary lymphadenopathy (28%) was the next common site of lymph node involvement in our patients (P1 and P6). A Korean study of 95 cases of KFD reported axillary lymph node involvement in 12.5% of cases.[16] Generalized lymphadenopathy and splenomegaly were reported to be relatively uncommon.[17] In the current series, one patient (P6) had splenomegaly; however, it must be noted that she had SLE along with KFD.

Leukopenia was a consistent finding in most of our patients (71%). This is in consistency with the previous reports on KFD. Leukopenia was reported in 50–87% of patients with KFD.[16,18] Leukopenia could be considered as a marker of response as most patients who failed to respond to NSAID, had persistent leukopenia which improved with steroid therapy.

ANA positivity was seen in five out of seven patients (71%) in our cohort of which two patients fulfilled the criteria for SLE. However, three patients were ANA positive but had no features suggestive of SLE. Kucukardali et al. reported 18 KFD patients who had a positive ANA test of which eight patients (44%) had ANA positivity even in the absence of features suggestive of SLE.[13]

The Association of SLE with KFD is well known. SLE can either present before the onset of KFD (30%), simultaneously (47%), or after developing KFD (23%).[19,20] In our study, both patients with SLE had KFD at presentation. One of these patients, (P6) had generalized lymphadenopathy and lupus nephritis at the time of presentation. Patients with SLE can present with generalized lymphadenopathy,[21] however absence of neutrophils and absence of hematoxylin bodies is consistent with a diagnosis of KFD in SLE.[22]

Recurrent KFD though uncommon has been reported in many studies. Cheng et al. described a total of 14.6% (14 out of 95) patients to have a recurrence of KFD at a mean interval of 2 years. Five of these patients developed autoimmune diseases like SLE, Graves disease, and mixed connective tissue disease.[23] In our study we had one patient (P1) with recurrence of KFD at an interval of 1 year. ANA test has been positive on both occasions, though he has no clinical features to suggest SLE. He has been under close follow-up.

Our cohort included two sisters (P2, P3) who developed KFD one year apart. Amir et al. reported two sisters who presented with KFD but the time lag between their presentations was 10 years. They also reported that they had a common HLA phenotype though not associated with SLE or KFD.[24] Kikuchi et al. reported A11 and DR12 phenotypes were found to have a significantly higher association with KFD.[25] However, HLA typing was not performed in our patients. Previously reported familial cases also have been found to be positive for certain viral agents like human T-lymphotropic virus type-1 (HTLV-1).[26] EBV infection was excluded in P3.

While KFD is a self-limiting disease, patients with the underlying autoimmune disease must be treated aggressively. Both patients with SLE were treated with steroids for their primary disease and KFD improved after therapy. NSAIDs have been recommended as the first-line therapy in patients with KFD. NSAIDs failed to produce a sustained response in four patients (non-SLE KFD) and needed steroids. Only one patient responded promptly to NSAIDs. Park et al. reported 16 patients with KFD of which six patients received steroid therapy, four patients recovered during inappropriate use of antibiotics, whereas six patients underwent a lymph node excision biopsy and recovered without treatment.[17] The outcome is usually favorable, but rare cases with fatal outcomes have been published. Therefore, most authors have recommended long-term monitoring of KFD patients. Our observation suggests steroids may be a better choice than NSAIDs in the treatment of KFD as they significantly reduce the duration of illness and help reduce the duration of school absenteeism in these patients. We however realize that the number of patients reported in this series is small to provide any definitive recommendation.

The strengths of our study are the robust clinical and laboratory data that was collected, standardized treatment across patients, and the availability of a pediatric rheumatologist at our center. However, we do acknowledge the limitations of our study. Being a retrospective study involving a small number of patients from a single center are some of the limitations.

## Conclusion

We hereby present seven cases with Kikuchi-Fujimoto disease. Their clinical features, laboratory parameters, and response to NSAIDs and steroids have been discussed in detail.

### Author's Contribution

RS-Initial draft of manuscript and review of literature.
SB-Review of literature, final draft of the manuscript.
TK- Assessment of histopathology specimens.
VM- Assessment of histopathology specimens.

**Ethics committee approval**: obtained
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