BMC Pediatrics

RESEARCH Open Access

Kikuchi-Fujimoto disease concurrent with aseptic meningitis or encephalitis in children: a case-control study

Bing Liu[1†], Yi Sun[1†], Bing Hu[1], Wen-yuan Shi[2], Tian-ming Chen[1], Lin-lin Liu[1] and Gang Liu[1,3*]

Abstract

Background This study was performed to summarize the clinical and laboratory features of children with Kikuchi-Fujimoto disease concurrent with aseptic meningitis or encephalitis.

Methods A case-control study of children diagnosed with Kikuchi-Fujimoto disease at Beijing Children's Hospital from January 2015 to December 2023 was conducted to determine the characteristics of the disease when concurrent with aseptic meningitis or encephalitis.

Results Our cohort of 64 cases of Kikuchi-Fujimoto disease included 16 children with central nervous system involvement and 48 controls. Among the 16 affected children, the male: female ratio was 1.7:1.0. The age at onset ranged from 3 to 13 years, with a median age of 8 years. All 16 cases had fever, 15 had cervical lymph node tenderness, 11 had headache, and 14 showed decreased white blood cell counts in routine blood tests. Imaging of the head revealed abnormalities in 11 cases, specifically leukoencephalopathy with mostly bilateral involvement. Cervical lymph node tenderness, headache, confusion, convulsions, and elevated C-reactive protein were significantly associated with Kikuchi-Fujimoto disease concurrent with aseptic meningitis or encephalitis ($p < 0.05$). There was also a significant difference in lactate dehydrogenase levels between children with and without central nervous system involvement (575.8 ± 221.3 vs. 440.0 ± 163.1 U/L, $p = 0.014$).

Conclusions For children with Kikuchi-Fujimoto disease, careful evaluation for central nervous system involvement is warranted when cervical lymph node tenderness, elevated C-reactive protein, or elevated lactate dehydrogenase is present. In children presenting with aseptic meningitis or encephalitis, Kikuchi-Fujimoto disease should be considered as a differential diagnosis.

Clinical trial number Not applicable.

Keywords Histiocytic necrotizing lymphadenitis, Child, Meningitis and encephalitis

[†]Bing Liu and Yi Sun contributed equally to this work.

*Correspondence:
Gang Liu
liugangbch@sina.com
[1]Department of Infectious Diseases, Beijing Children's Hospital, Capital Medical University, National Center for Children's Health, National Center for Children's Infectious and Allergic Diseases Surveillance, Beijing Research Center for Respiratory Infectious Diseases, Beijing Key Laboratory of Core Technologies for the Prevention and Treatment of Emerging Infectious Diseases in Children, Key Laboratory of Major Diseases in Children, Ministry of Education, No. 56 Nan Lishi Road, Beijing 100045, China
[2]Department of Interventional Medicine, Beijing Children's Hospital, National Center for Children's Health, Capital Medical University, Beijing, China
[3]Research Unit of Critical Infection in Children, Chinese Academy of Medical Sciences, Beijing 2019RU016, China

Background

Kikuchi-Fujimoto disease (KFD), also known as histiocytic necrotizing lymphadenitis, is a self-limiting condition [1]. The cause of KFD remains unknown [2]. While earlier reports have suggested KFD predominantly affects female patients, more recent studies have shown similar or even lower rates in women than in men [3]. Common clinical signs and symptoms include lymphadenopathy, fever, rash, joint pain, weakness, and enlargement of the liver and spleen. KFD can lead to various complications, such as systemic lupus erythematosus and arthritis [4]. It may also involve the central nervous system (CNS), presenting as aseptic meningitis, encephalitis, and related conditions [5]. The clinical manifestations of KFD are diverse, and individual signs and symptoms are nonspecific. Cases involving the CNS are rarely reported, which can lead to misdiagnosis, missed diagnosis, or delayed diagnosis. Therefore, the aim of this paper is to retrospectively summarize the clinical characteristics of children admitted to Beijing Children's Hospital with KFD involving the CNS and thus provide a reference for early clinical recognition and accurate diagnosis.

Materials and methods

We conducted a case-control study of children admitted to Beijing Children's Hospital, China, from 1 January 2015 to 31 December 2023. Cases were defined as children who had KFD concurrent with aseptic meningitis or encephalitis. The inclusion criteria were an age of 29 days to 18 years, a histopathological diagnosis consistent with histiocytic necrotizing lymphadenitis based on cervical lymph node biopsy, the presence of neurological symptoms or signs, and cerebrospinal fluid (CSF) abnormalities or abnormal findings on cranial magnetic resonance imaging (MRI). Additionally, three controls were randomly selected for each case, matched by sex, age, and admission date (within 7 days of the case's admission date). Controls were defined as children who had KFD without aseptic meningitis or encephalitis.

The children's clinical information was obtained from the hospital's medical record system. General data (sex, age, and time of onset) and clinical characteristics (clinical manifestations, laboratory results, etiological findings, and medication use) were collected. Descriptive statistics were presented as mean or median for continuous variables and as frequency (percentage) for categorical variables. Continuous variables between two groups were compared using the Mann–Whitney test for nonparametric data or the independent t-test for parametric data, as appropriate. Factors associated with KFD concurrent with aseptic meningitis or encephalitis were identified through univariate analysis. A p-value of <0.05 was considered statistically significant. Statistical analyses were performed using SPSS 25.0 software (IBM Corp., Armonk, NY, USA).

Results

Of the 16 patients who met the case criteria for our study, we identified matched controls, resulting in a total of 64 study participants (16 cases and 48 controls). All 16 cases of KFD concurrent with aseptic meningitis or encephalitis were diagnosed with histiocytic necrotizing lymphadenitis by cervical lymph node biopsy. Among them, there were 10 male and 6 female, yielding a male: female ratio of 1.7:1.0. The age at onset ranged from 3 to 13 years, with a median age of 8 years. All 16 cases presented with fever; 15 had cervical lymph node tenderness, 6 had a rash, and 1 had joint pain. Regarding CNS involvement, 10 cases were diagnosed with aseptic encephalitis and 6 with aseptic meningitis. Headache occurred in 11 cases, confusion in 6, and convulsions in 5. One patient exhibited neck stiffness. The interval between lymph node enlargement and onset of CNS symptoms ranged from 2 days to 8 weeks. The duration of CNS clinical manifestations lasted from 1 to 11 weeks (Table 1). In laboratory tests, 14 of the 16 children had decreased white blood cell counts. Nine cases had an elevated C-reactive protein (CRP) concentration, and 14 cases had an increased erythrocyte sedimentation rate (ESR). Ten cases had elevated CSF cell counts, primarily monocytes. CSF glucose was decreased in 4 cases, protein was increased in 10, and intracranial pressure was elevated in 4. CSF pathogen testing—including Epstein–Barr virus, cytomegalovirus, herpes simplex virus, enterovirus, and other pathogenic nucleic acid tests—was negative. Cranial MRI results were available for all 16 patients; 11 showed abnormalities, specifically leukoencephalopathy with mostly bilateral involvement, scattered lesions, and predominant involvement of the frontoparietal white matter (Table 2). Of the 16 patients, 11 received glucocorticoid therapy and 10 received intravenous immunoglobulin therapy. Recurrence occurred in two cases, and no recurrence or sequelae were observed in the remaining cases.

In total, 48 cases of KFD without aseptic meningitis or encephalitis were diagnosed with histiocytic necrotizing lymphadenitis by cervical lymph node biopsy. Among them, there were 33 male and 15 female children, yielding a male: female ratio of 2.2:1.0. The age at onset ranged from 5 to 13 years, with a median age of 10 years. All 48 cases presented with fever; 30 had cervical lymph node tenderness, 6 had a rash, and none reported joint pain. In laboratory tests, 23 cases showed decreased white blood cell counts. Eight cases had an elevated CRP concentration, and 30 cases had an increased ESR. Seven patients received glucocorticoid therapy, and two were treated with intravenous immunoglobulin. Recurrence occurred in eight cases; no recurrence or sequelae were observed

Table 1 Clinical features of 16 children with KFD concurrent with aseptic meningitis or encephalitis

Number of patients	Gender/age(years)	Fever	Tenderness of the cervical lymph nodes	Rash	Arthrodynia	Interval*(weeks)	CNS clinical manifestations					CNS duration (weeks)	Steroids therapy	IVIG	Outcome
							Headache	Confusion	Convulsions	Neck stiffness	Kernig's signs				
1	M/7	+	+	+	–	1	+	–	–	–	–	1	+	+	C
2	F/5	+	+	+	–	2	–	–	+	–	–	2	+	–	C
3	M/6	+	+	+	–	4	–	+	–	–	–	4	+	+	R
4	M/10	+	+	+	–	0	+	–	–	–	–	2	+	–	C
5	M/11	+	+	–	–	2 days	+	–	–	–	–	1	+	–	C
6	M/9	+	+	–	–	8	+	–	+	–	–	5	+	+	C
7	M/13	+	+	+	–	0	+	–	–	–	–	4	+	–	C
8	F/6	+	–	+	+	0	+	+	–	+	–	11	–	–	C
9	M/6	+	+	–	–	3	+	–	–	–	–	6	–	–	C
10	F/13	+	+	–	–	1	+	–	–	–	–	3	–	–	C
11	F/12	+	+	–	–	3	+	+	–	–	–	4	+	+	C
12	M/12	+	+	–	–	0	+	–	+	–	–	4	–	+	C
13	M/8	+	+	–	–	1	–	+	+	–	–	7	+	+	C
14	F/8	+	+	–	–	2	+	–	–	–	–	1	–	–	R
15	F/8	+	+	–	–	4	–	+	+	–	–	1	+	+	C
16	M/3	+	+	–	–	1	–	+	–	–	–	1	+	–	C

CNS, central nervous system; IVIG, intravenous immunoglobulin; M, male; F, female; C, cure; R, relapse; *, interval between development of enlarged lymph nodes and appearance of CNS symptoms;–, absent; +, present

in the remaining cases. Table 3 presents risk factors for KFD concurrent with aseptic meningitis or encephalitis as identified by univariate analysis. Cervical lymph node tenderness, headache, confusion, convulsions, and elevated CRP were significantly associated with CNS involvement ($p < 0.05$). Additionally, there was a significant difference in lactate dehydrogenase (LDH) levels between patients with and without CNS involvement (575.8 ± 221.3 vs. 440.0 ± 163.1 U/L, $p = 0.014$).

Discussion

KFD is a benign disease of unknown etiology, most commonly seen in Asian countries and rarely reported in Western countries [6]. Its clinical manifestations are varied and lack specific symptoms or signs. CNS involvement in KFD is rare and often misdiagnosed as other neurological disorders [7]. Neurological complications such as aseptic meningitis, cerebellar ataxia, and mononeuritis multiplex occur in approximately 5% of cases [8]. In this study, children with KFD concurrent with aseptic meningitis or encephalitis ranged in age from 3 to 13 years and were predominantly male. The main clinical features included fever, cervical lymph node swelling and tenderness, and leukopenia. Headache was the most prominent symptom in cases with CNS involvement. Cervical lymph node tenderness, headache, confusion, convulsions, and elevated CRP were significantly associated with KFD concurrent with aseptic meningitis or encephalitis, and LDH levels were also significantly higher in these patients than in those without CNS involvement. Clinicians should carefully assess for neurological symptoms and promptly perform CSF analysis and cranial imaging to facilitate early diagnosis and prevent delays in treatment.

Consistent with previous research [9], CSF examination in cases of KFD concurrent with aseptic meningitis or encephalitis was largely sterile. In this study, 62.5% of cases showed increased CSF cell counts, predominantly mononuclear cells, and all CSF etiology tests were negative. Aseptic meningitis is the most commonly reported neurologic complication of KFD [10]. Head imaging changes were observed in 68.8% of cases, primarily presenting as leukoencephalopathy with mostly bilateral involvement. One report described brain MRI findings of extensive T2-weighted hyperintensity, focal diffusion restriction, and microhemorrhages within the deep gray nuclei and surrounding white matter [11]. Previous studies have also noted CNS involvement manifesting as multiple peripheral neuropathy, hemiparesis, and brachial plexus injury [12, 13], although such findings were not observed in the cases from this study, warranting further attention.

KFD concurrent with aseptic meningitis or encephalitis typically occurs 2 to 3 weeks after the onset of

Table 2 Laboratory test results and imaging findings of children with KFD concurrent with aseptic meningitis or encephalitis

Number of patients	Gender/age(years)	WBC(*10^9/L)	CRP(mg/L)	ESR(mm/h)	CSF						Head MRI abnormality
					Pressure (mmH_2O)	WBC (× 10^6/L)	Mononuclear cells(×10^6/L)	Protein (mmol/L)	Glucose (mg/L)	Chloride(mmol/L)	
1	M/7	4.17	32	117	ND	24	23	160	2.11	115	The white matter of the right frontal lobe, the left insula, the white matter of the left centrum semiovale, and the left basal ganglia demonstrated mild enhancement. There was mild enhancement observed in the left tentorium cerebelli.
2	F/5	4.41	33	55	245	4	ND	691	3.3	120	T2 FLAIR imaging revealed slightly elevated surface signals in the bilateral frontal gyri. ADC sequences demonstrated scattered areas of mild diffusion restriction within the right frontal and parietal cortical regions of the cerebral hemisphere. The bodies of the bilateral lateral ventricles appeared full.
3	M/6	1.62	<8	34	ND	13	ND	640	4.28	108.6	Fuzzy patchy T2WI and slightly elevated FLAIR signals were observed in the bilateral lenticular nucleus.
4	M/10	3.47	5	85	ND	62	59	349	3.09	125.7	T2 FLAIR imaging showed multiple flaky hyperintense signals in the bilateral fronto-parietal white matter.
5	M/11	3.78	41	25	ND	3	ND	210	4.28	130.5	T2 FLAIR shows multiple patchy hyper-signals in bilateral frontotemporo-parietal white matter.
6	M/9	11.69	<0.5	53	160	52	48	435	3.31	126.9	The bilateral ventricles were widened, and the third ventricle appeared full.
7	M/13	3.23	22	34	170	22	21	269	3.06	125.6	T2 FLAIR imaging demonstrated increased signal intensity in the right transverse sinus and linear hyperintensity in the parieto-occipital sulci.
8	F/6	2.01	<0.5	11	160	20	19	258	2.89	129.9	Multiple focal hyperintense signals were observed in the bilateral temporal lobes, the left frontal cortex, and the subcortical areas on T2 FLAIR sequences. Multiple mottled demyelination changes were noted in both frontal lobes.
9	M/6	3.99	24.36	30	190	320	77	740	2.2	124	–
10	F/13	2.11	8	42	260	82	70	1939	2.28	120	–

Table 2 (continued)

Number of patients	Gender/age(years)	WBC(*10^9/L)	CRP(mg/L)	ESR(mm/h)	CSF						Head MRI abnormality
					Pressure (mmH_2O)	WBC (× 10^6/L)	Mononuclear cells(×10^6/L)	Protein (mmol/L)	Glucose (mg/L)	Chloride(mmol/L)	
11	F/12	2.07	3	40	330	58	54	2314	2.33	124.1	T2 FLAIR sequences revealed dot-like and linear hyperintensities on the surfaces of the bilateral cerebral hemispheres. The white matter of the bilateral frontal and parietal lobes exhibited punctate signals, while the trigone and posterior corners of the bilateral ventricles showed patchy signals.
12	M/12	3.43	86	38	ND	67	66	496	2.95	111	–
13	M/8	1.58	14	1	ND	0	0	2067	3.72	128.7	Diffuse abnormal signals were detected in the bilateral cerebral hemispheres, subcortical white matter, hippocampus, medial temporal lobe, basal ganglia, and thalamus.
14	F/8	3.22	18	23	168	50	45	577	3.36	123.5	–
15	F/8	1.49	20	41	ND	8	ND	1640	5.63	138	–
16	M/3	2.78	<8	46	ND	4	ND	561	3.24	123.7	Multiple spots with slightly prolonged T2 signals were identified in the white matter of the bilateral frontal and parietal lobes as well as centrum semiovale.

MRI, magnetic resonance imaging; CSF, cerebrospinal fluid; M, male; F, female; ND, not described;–, absent; FLAIR, fluid attenuated inversion recovery; ADC, apparent diffusion coefficient; T2WI, T2-weighted image

Table 3 Patients with KFD with and without aseptic meningitis or encephalitis

	KFD With Aseptic Meningitis or Encephalitis ($n=16$)	KFD Without Aseptic Meningitis or Encephalitis ($n=48$)	*P*
Epidemiologic features			
Age, yr	8.0(6.0-11.8)	10.0(7.0–12.0)	0.470
Male sex	11(68.8)	33(68.8)	1.000
Clinical features			
Fever (≥ 39℃)	13(81.3)	39(81.3)	1.000
Cervical lymph node tenderness	15(93.8)	30(62.5)	0.032
Headache	11(68.8)	2(4.2)	0.000
Confusion	6(37.5)	0	0.000
Convulsions	5(31.3)	0	0.000
Cutaneous rash	6(37.5)	6(12.5)	0.064
Arthralgia	1(6.3)	0	0.250
Hepato-Splenomegaly	0	2(4.2)	1.000
Laboratory features			
Leukopenia	14(87.5)	23(47.9)	0.062
Increased CRP	9(56.3)	8(16.7)	0.002
Increased ESR	14(87.5)	30(62.5)	0.062
Elevated liver enzymes	4(25.0)	15(31.3)	0.874
Treatment			
Corticosteroids	11(68.8)	7(14.6)	0.000
IVIG	10(62.5)	2(4.2)	0.000

Data are presented as n (%) or median (Q1–Q3)

KFD, Kikuchi-Fujimoto disease; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; IVIG, intravenous immunoglobulin

lymphadenopathy, while meningitis as the initial symptom of KFD is rare. The course of KFD with CNS involvement usually lasts 2 to 3 weeks, although durations of 2 to 4 months have also been reported [14]. In this study, the interval between lymph node enlargement and the appearance of CNS symptoms ranged from as little as 2 days to as long as 8 weeks. The duration of CNS clinical manifestations ranged from 1 to 11 weeks.

KFD is thought to be mediated by an aberrant type I interferon response, likely driven by plasmacytoid dendritic cells and T cells [15]. Evidence of C1s deficiency in a patient with KFD further supports the role of complement abnormalities in the disease's pathogenesis [16]. Additionally, one study suggests that the encephalitis complication of KFD may be autoimmune in nature and mediated by cytotoxic T cells [17].

Because of the rarity of the disease, there are no established consensus guidelines for its treatment. Mild cases are generally believed to respond well to antipyretics and nonsteroidal anti-inflammatory drugs alone, while corticosteroids are considered necessary for extranodal manifestations such as aseptic meningitis [9]. One study reported that among 41 cases of KFD with aseptic meningitis, 24 patients received steroid therapy, with 79% achieving full recovery [14]. As shown in Table 3, among the 16 patients with KFD and CNS involvement in this study, 11 received steroid therapy, and the prognosis was favorable in most cases, with the exception of 2 instances of recurrence.

This study had two main limitations: it was retrospective in nature, and no further analysis of pathogenesis was performed.

Conclusions

For children with KFD, clinicians should be alert to the possibility of CNS involvement when lymph node tenderness, elevated CRP, or elevated LDH is present. In cases of aseptic meningitis or encephalitis in children, KFD involving the CNS should be considered as a differential diagnosis.

Abbreviations

CNS Central nervous system
CRP C-reactive protein
ESR Erythrocyte sedimentation rate
CSF Cerebrospinal fluid
KFD Kikuchi-Fujimoto disease
LDH Lactate dehydrogenase
MRI Magnetic resonance imaging

Acknowledgements

We thank Angela Morben, DVM, ELS, from Liwen Bianji (Edanz) (www.liwenbianji.cn) for editing the English text of a draft of this manuscript.

Author contributions

All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by BL and YS. The first draft of the manuscript was written by BL and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Funding
This work was supported by Capital's Funds for Health Improvement and Research (2024-1-2092) and the 2022 Beijing Major Epidemic Prevention and Control Specialty Construction Project (2-1-2-6-15).

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This study was approved by the Ethics Committee of Beijing Children's Hospital, Capital Medical University (2023-E-119-R). The requirement for informed consent to participate was waived by the Ethics Committee of Beijing Children's Hospital Affiliated to Capital Medical University because of the retrospective nature of the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 17 January 2025 / Accepted: 28 March 2025
Published online: 12 April 2025

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