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БОЛЕЗНЬ КИКУЧИ-ФУДЖИМОТО НА ФОНЕ ПЕРВИЧНОГО ИММУНОДЕФИЦИТА И РАССЕЯННОГО СКЛЕРОЗА: КЛИНИЧЕСКИЙ СЛУЧАЙ

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Kikuchi-Fujimoto's Disease on The Background of Primary Immunodeficiency and Multiple Sclerosis: A Clinical Case

Резюме

Впервые описано сочетание болезни Кикучи-Фуджимото с общей вариабельной иммунной недостаточностью и рассеянным склерозом. Наличие у пациентки первичного иммунодефицита, сопровождающегося частыми и тяжелыми обострениями хронических заболеваний, привело к затяжному диагностическому поиску. Случай подчеркивает важность своевременного морфологического исследования при стойкой лимфаденопатии, даже у пациентов с известными иммунными нарушениями.

Ключевые слова: Болезнь Кикучи-Фуджимото, гистиоцитарный некротизирующий лимфаденит, первичный иммунодефицит, общая вариабельная иммунная недостаточность, рассеянный склероз

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

Источники финансирования

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Соответствие принципам этики

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Abstract

For the first time, a combination of Kikuchi-Fujimoto's disease with general variable immune deficiency and multiple sclerosis has been described. The patient's primary immunodeficiency, accompanied by frequent and severe exacerbations of chronic diseases, led to a protracted diagnostic search. The case highlights the importance of timely morphological examination in persistent lymphadenopathy, even in patients with known immune disorders.

Key words: Kikuchi-Fujimoto's disease, histiocytic necrotizing lymphadenitis, primary immunodeficiency, general variable immune deficiency; multiple sclerosis

Conflict of interests

The authors declare no conflict of interests

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Conformity with the principles of ethics

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KFD — Kikuchi-Fujimoto disease, LN — lymph nodes, SLE — systemic lupus erythematosus, PID GVID — primary immunodeficiency: general variable immunodeficiency, MRI — magnetic resonance imaging, MS — multiple sclerosis, aHSCT — autologous haematopoietic stem cell transplantation, Ig — immunoglobulin

Introduction

Kikuchi-Fujimoto disease (KFD) is a benign self-limiting inflammatory disease characterised by histiocytic necrotising lymphadenitis. The condition affects primarily cervical lymph nodes (LNs); however, the pathologic process can also involve axillary and inguinal LNs. Due to the similarity of the clinical and anatomico-pathological presentation with lymphoproliferative disorders, the primary diagnoses is often wrong; therefore, it is important to raise awareness of this disorder among clinicians and pathologists [1, 2].

Clinically, KFD manifests as painful lymphadenopathy (the main criterion for the diagnosis, which is observed in all reported cases), fever and marked weakness. The clinical presentation can include diarrhea, weight loss, dyspepsia, arthralgia, skin rashes, and enlarged liver and spleen. In individual cases, the disease is aggravated by aseptic meningitis or optic neuritis. Skin manifestations can vary from simple papules to necrosis of individual keratinocytes, and are reported in 30 % of cases. Histologic examination of affected skin of KFD patients shows lymphohistiocytic infiltration of the derma, nuclear disruption, mucin deposits, panniculitis, and disrupted dermoepidermal junction. Clinical blood analysis comes back normal; however, anemia, leukopenia, leukocytosis, lymphocytosis, and slightly elevated erythrocyte sedimentation rate can be reported [3–5].

The cause of the disease remains unclear. There is no connection with the race; and the condition usually manifests in individuals of 30 to 40 years of age. It is assumed that the disease is triggered by Epstein-Barr virus, human herpes virus 6 and 8, parvovirus B19, cytomegalovirus, *Yersinia enterocolitica*, *Toxoplasma gondii*, and *Brucella*. An infectious or non-infectious trigger causes an immune reaction mediated by T-cells and histiocytes. The viral origin is supported by elevated levels of alpha-interferon and its stimulating agents, as

well as tubuloreticular structures in the cytoplasm of stimulated lymphocytes, histiocytes, and vascular endothelium. A favourable therapeutic response to tominocycline, ciprofloxacin and ofloxacin in several clinical cases proposes the possible pathogenic role of other non-viral microorganisms, which are sensitive to antimicrobial agents. There is a theory about an autoimmune mechanism, since there are numerous patients with autoimmune pathologies before, during, and after the diagnosis of KFD is confirmed. Systemic lupus erythematosus (SLE) is most common, assuming the existence of common pathogenic links. The histological pattern of lymphadenitis in SLE patients can resemble that of KFD; however, the key distinguishing characteristic in SLE is the presence of hematoxylin bodies. Moreover, KFD is a self-limiting condition and is not a chronic pathology. KFD can be associated with other autoimmune conditions, e.g. primary Sjögren's syndrome, Hashimoto's thyroiditis, antiphospholipid syndrome, and hypersensitivity vasculitis [1–4, 6].

KFD is diagnosed on the basis of histological and immunohistochemical examinations of a removed LN.

There are three stages of the disease:

1. Early (proliferative) stage: the paracortical layer of histiocytes and plasmacytoid dendritic cells is widened; the infiltrate incorporates lymphocytes and karyorrhectic nuclear debris.
2. Intermediate (necrotic) stage: areas of necrosis start appearing in the paracortex.
3. Late (xanthomatous) stage: foamy histiocytes prevail, irrespective of the presence or absence of necrosis [3, 6].

The complex histological pattern, which depends on the stage of the disease, can make the diagnosis even more challenging.

KFD regresses spontaneously within several months and is associated with a favourable prognosis. Patients

should be followed up for several years after recovery, since 5 % of cases relapse or show signs of an autoimmune pathology. There is no KFD-specific therapy, since the evidence base is limited to the data from observational studies. In severe cases, symptomatic treatment with non-steroidal anti-inflammatory drugs (NSAIDs) with glucocorticosteroids is used [3, 6, 7].

A PubMed analysis as of 20.10.2025 using “Kikuchi-Fujimoto disease” search returned 397 publications from 1996 to 2025, including 267 clinical cases; the rest are literature reviews. It confirms that this pathology is quite rare.

Clinical Case Report

Patient A., 42 years of age, has primary immunodeficiency: general variable immunodeficiency (PID GVID), and receives life-long therapy with human immunoglobulin once a month. PID is caused by heterozygous mutation in exon 6 chr4:102567013 c.285G>T, p.Lys95Asn (NM_003998.4) of gene *NFKB1*. Immunodeficiency resulted in frequent episodes of chronic tonsillitis; acute respiratory viral infections were associated with lingering cough.

The family history shows rheumatoid arthritis in her mother and confirmed alopecia localis in her younger daughter.

When she was 20 years of age, the patient complained of facial numbness, headaches, and double vision. NSAIDs had a short positive effect. Three years later, magnetic resonance imaging (MRI) showed areas of demyelination in the brain. The patient was later diagnosed with multiple sclerosis (MS). She was treated with interferon-beta-1b and vascular therapy, and also attended exercise therapy sessions. Six years later, MS progression was confirmed: she had lurching walk; brain and cervical spine contrast-enhanced MRI showed multiple areas of demyelination of up to 11 mm in periventricular and supraventricular regions and cerebellum. There was also a 9x4 mm area at the C3-4 level in the spine. In order to manage the aggressive progression of MS, autologous haematopoietic stem cell transplantation (aHSCT) was performed with BEAM modified immunosuppression therapy. The therapy was associated with grade IV neutropenia (max. 0.2 ths/ μ L), grade IV thrombocytopenia (max. 18 ths/ μ L), grade 2 hepatotoxicity (ALT 198 U/L, AST 62 U/L, total bilirubin 30.8 μ mol/L).

Long after the transplantation, the patient had lingering catarrhal diseases, which required regular antibacterial therapy. She had frequent episodes of chronic bronchitis, tonsillitis, sinusitis, otitis, and maxillitis.

A follow-up MRI performed three years after the aHSCT showed supraventricular areas of inactive demye-

linating disease. Blood immunoglobulin lab results demonstrated lower immunoglobulin G (IgG) (291 mg/dL; reference range: 900–1800 mg/dL); IgM values were normal (226 mg/dL; reference range: 80–250 mg/dL); IgA levels dropped to 5 mg/dL (reference range: 100–350 mg/dL). An extended lymphocyte immunophenotyping with T-cell subpopulation identification returned the following results: CD switch 0.9–2.7 %, CD4 1190, CD8 810, CD19 430, NK 300, CD21low 5,3, CD transient 1.5–1.2. A comprehensive immunoassay aimed at the restoration of the humoral and cellular components of the immune system; facilitation of the decision whether a replacement immunoglobulin therapy was required; assessment the risks of infectious complications of PID GVID; and MS therapy correction. These assays were performed for the first time, since the initial levels of blood immunoglobulins and lymphocyte subpopulations were unknown.

From February 2023 to November 2024, the patient had to interrupt her therapy with human immunoglobulin. During this period, she had monthly penicillin antimicrobials for frequent respiratory diseases. In November 2024, immunoglobulin therapy was resumed.

Due to regular catarrhal diseases and episodes of chronic conditions, the patient was repeatedly admitted to the allergology and immunology ward. During one of the admissions in April 2024, polymerase chain reactions for cytomegalovirus, Epstein-Barr virus, and human herpes virus 6 returned negative results. Helminthic involvement was excluded when negative anti-parasite antibodies results were received. Immunoblotting for a wide range of antinuclear antibodies was performed, which allowed excluding SLE, Sjögren's syndrome, systemic scleroderma, and other diffuse connective tissue disorders. Besides, C3-complement component levels dropped to 0.84 g/L with normal C4-complement component values (0.178 g/L), which is a sign of the classical complement pathway activation and can evidence the presence of an immunoinflammatory process of any other nature. A sputum sample was tested for *M. tuberculosis*, the result was negative. Peripheral LN ultrasound showed peripheral lymphadenopathy.

In April 2025, when the patient was 42 years of age, she noticed sudden marked enlargement of cervical LNs on the left; fever of up to 38.5 degrees Celcius in the evening, difficulty in swallowing, and sore throat with prodromes. Her GP prescribed levofloxacin; the therapy was ineffective. Several days later, her LNs got enlarged on the right side of the neck, these were painful to the touch; and diarrhea appeared. The patient started amoxicillin with clavulanic acid, with no effect.

Three months later, the patient started feeling very poorly; she developed extreme weakness, haze, excessive

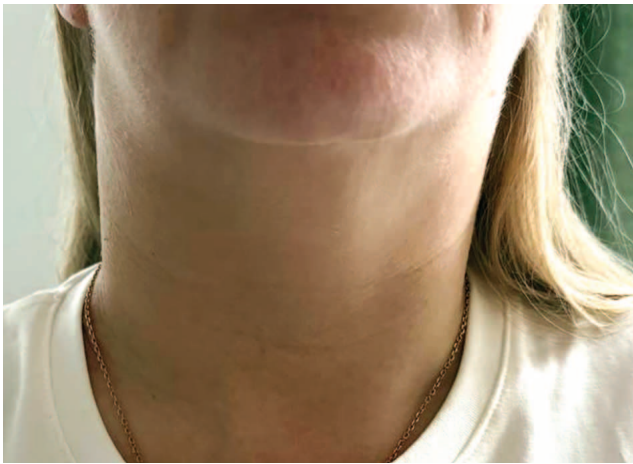


Figure 1. Enlarged lymph nodes of the neck

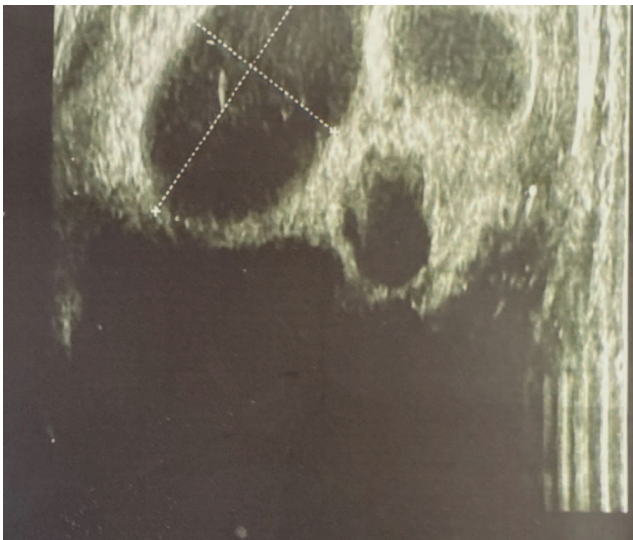


Figure 2. Ultrasound examination of the lymph nodes of the neck

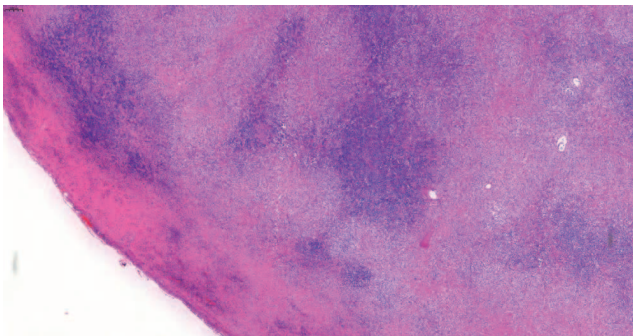


Figure 3a. Hematoxylin and eosin staining ×400. Expansion of the paracortical zone with the formation of foci of necrosis. The cellular infiltrate includes a mixed cell population, including histiocytes with a virtually complete absence of neutrophils and eosinophils.

sweat, dry mouth, polydipsia, and loss of appetite, where she lost 10 kg of her weight. Over time, the LNs enlarged even more and the patient started feeling as if her neck skin was stretched. Severe headaches appeared, with NSAIDs therapy being of little to no use.

By September, the elevated body temperature was observed immediately after awakening, with the highest values reaching 38.7°C with NSAIDs therapy. Since the symptoms progressed and the patient’s condition was resistant to the antibiotic treatment, the patient was admitted to the allergology and immunology ward. Stool test results: *Escherichia coli* 10⁵ CFU/mL, *Klebsiella oxytoca* 10⁴ CFU/mL, *Candida albicans* 10⁵ CFU/mL. Tests for acute enteric infection pathogens (*Shigella spp*, *Salmonella*, *Campylobacter spp*, *Adenovirus F*, *Enterovirus*, *Rotavirus grA*, *Norovirus G2*, *Astrovirus*) came back negative. Blood draw results showed an inflammatory response; however, imaging examinations did not find the focus of inflammation.

Since a malignancy was suspected, the patient was consulted by a cancer specialist. On palpation, left and right cervical LNs, left axillary LNs were enlarged up to 3 cm, moderately painful and soft (Fig. 1). During the consultation, another LN ultrasound was performed, which showed the following results: cervical LNs: multiple on both sides, not tending to fuse, impaired corticomedullary differentiation, ovoid, with even contours, without hypervascularisation, up to 26x21mm on the right and up to 24x18 mm on the left. Axillary LNs: individual on both sides, not tending to fuse, preserved corticomedullary differentiation, ovoid, with uneven contours, without hypervascularisation, up to 25x14 mm on the right and up to 21x15 mm on the left. Inguinal LNs: individual on both sides, not tending to fuse, preserved corticomedullary differentiation, ovoid, with even contours, without hypervascularisation, up to 16x15 mm on the right and up to 14x16 mm to the left (Fig. 2).

As part of the differential diagnosis of the systemic blood disease, an excision biopsy and histological examination of a cervical LN was performed, which showed morphological signs of necrotising lymphadenitis. The material was also sent for immunohistochemistry; the changes correspond to necrotising lymphadenitis in Kikuchi-Fujimoto disease (Fig. 3a, 3b, 3c, 3d).

The clinical presentation and immunohistochemistry results allowed diagnosis Kikuchi-Fujimoto disease.

It was decided to initiate the weekly symptomatic treatment with dexamethasone 12 mg, ascorbic acid 10 mL, cytoflavin 10 mL, Reamberin 500 mL, and omeprazole 40 mg. Since the clinical response to the therapy was poor, daily IV injections of prednisolone 40 mg were introduced. After therapy correction, the symptoms of the disease improved.

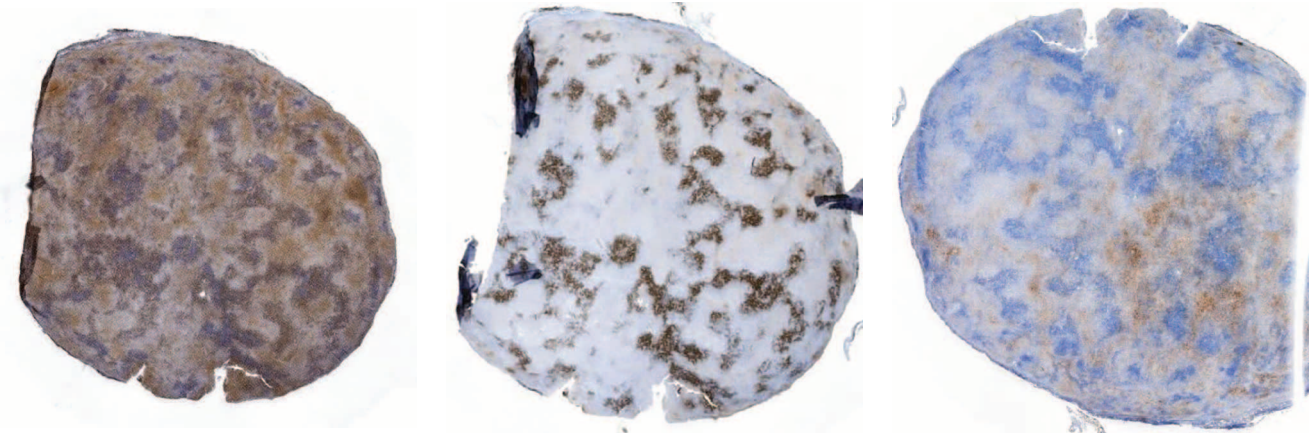


Figure 3b. Enzyme immunoassay, diffuse positive reaction with anti-bodies to CD3 (magnification ×100)

Figure 3c. ELISA reveals a positive reaction in the form of isolated foci with antibodies to CD20 (magnification ×100). Extensive areas of necrosis and infiltrate are unstained

Figure 3d. Enzyme immunoassay, focal positive reaction with antibodies to CD10 (magnification ×100), localized mainly in the germinal centers of lymphoid follicles

Table 1. Dynamics of laboratory parameters

Mark	09.2024	09.2025	11.2025
Total IgG (900-1800 mg/dL)	370 mg/dL	963 mg/dL	928 mg/dL
Total IgM (80-250 mg/dL)	330 mg/dL	262,8 mg/dL	281 mg/dL
Total IgA (100-350 mg/dL)	20 mg/dL	0,001 mg/dL	1 mg/dL
Total IgE (up to 100 IU/ml)	<1 IU/ml	-	<1,00 IU/ml
Hb (120–140 g/l)	143 g/l	118 g/l	115 g/l
PLT (179.00-403.00 10 ⁹ /l)	255 x 10 ⁹ /l	137 x 10 ⁹ /l	132 x 10 ⁹ /l
Leukocytes (4.50-11.00 10 ⁹ /l)	7,8 x 10 ⁹ /l	1,94 x 10 ⁹ /l	9,72 x 10 ⁹ /l
Neutrophils (2.10-8.89 10 ⁹ /l)	3,49 x 10 ⁹ /l	0,86 x 10 ⁹ /l	9,07 x 10 ⁹ /l
Eosinophils (0.01-0.40 10 ⁹ /l)	0,64 x 10 ⁹ /l	0,0 x 10 ⁹ /l	0,0 x 10 ⁹ /l
Basophils (0.00-0.07 10 ⁹ /l)	-	0,0 x 10 ⁹ /l	0,0 x 10 ⁹ /l
Lymphocytes (1.26-3.35 10 ⁹ /l)	3 x 10 ⁹ /l	0,93 x 10 ⁹ /l	0,49 x 10 ⁹ /l
Monocytes (0.25-0.84 10 ⁹ /l)	0,62 x 10 ⁹ /l	0,15 x 10 ⁹ /l	0,16 x 10 ⁹ /l
ESR (0.00-30.00 mm/hour)	21 mm/hour	11,0 mm/hour	23 mm/hour
CRP (up to 5 mg/l)	-	7,77 mg/l	0,19 mg/l

Note: Ig — immunoglobulins, Hb — hemoglobin, PLT — platelets, ESR — erythrocyte sedimentation rate, CRP — C-reactive protein

Discussion

This clinical observation is a case of KFD in a patient with multiple comorbidities. The main diagnostic challenge in this case was the history of PID and MS. From an early age, the patient has had frequent infections and chronic lymphadenopathy, and even being an adult, they were treated as manifestations of PID or its complications. In turn, it resulted in a long-lasting diagnostic search. This case demonstrates the importance of cancer alertness in persistent lymphadenopathy resistant to empiric antimicrobial therapy.

The analysed publications do not report any patients with KFD and PID GVID or MS, evidencing the unique

nature of this clinical case. Vaidya H. et al. described a case of a 35-year-old patient complaining of severe abdominal pain, vomitting, general weakness, episodes of fever, and joint pain both in legs and arms. Lab results demonstrated marked anaemia, and abdominal ultrasound showed enlarged liver and delated portal and splenic veins. The patient also complained of impaired vision acuity. An ophthalmic examination revealed multiple cotton wool-shaped macules on the retina of both eyes, haemorrhage in the left eye along the lower arcade, areas of paracentral acute moderate macular retinopathy to the right, while the left eye had a thickened outer plexiform layer [8].

The case of a 20-year-old patient described by Gurung S. et al. reported standard clinical manifestations of lymphadenopathy, fever, and asthenia. Lab results were within the normal range [9].

There are also numerous cases of disease manifestations following SARS-CoV-2 vaccination. For instance, Ikeda K. et al. described a case of a 20-year-old patient with progressing complains after vaccination. Lab test results demonstrated leukopaenia, inflammatory syndrome, and elevated lactate dehydrogenase levels. G, A and M immunoglobulins, C3 and C4 complement concentrations, and antinuclear antibody titer were normal. SARS-CoV-2 PCR test was negative. Imaging examinations revealed enlarged liver and spleen [10].

Identification of the precise trigger of KFD is rather challenging; therefore, the causes of the disease remain unknown. The theory of the association between KFD and autoimmune diseases is common; however, in the present case the specific markers of SLE and other diffuse connective tissue disorders were negative; a history of MS and a family history of autoimmune pathologies evidence potential general susceptibility to immune dysregulation. This is in line with the theory of excessive or inadequate immune response to various triggers (viral, bacterial) in susceptible individuals.

An analysis of the reported cases and comparison with the described clinical case assume that the classical presentation of KFD (lymphadenopathy, fever, and asthenia) remains the same. However, the patient in this case study had a more lingering and more severe disease, caused by a long-lasting diagnostic search and PID.

Conclusions

KFD is a very rare disease, which should not be negated in the differential diagnosis of lymphadenopathy and idiopathic fever. It can mimic lymphoma or systemic autoimmune diseases, requiring mandatory morphological confirmation. Diagnosis of KFD can be particularly challenging in patients with concomitant immunodeficiencies. This case study underlines the importance of a multidisciplinary assessment of the clinical presentation and prompt biopsy in atypical or refractory lymphadenopathy.

PID GVID and MS in the patient make possible an assumption of the link between transcription factor protein dysregulation, autoimmune susceptibility, and KFD development.

Despite the challenging diagnosing path, the KFD prognosis remains favourable. The disease is self-limiting and responds well to symptomatic therapy, including glucocorticosteroids. This case demonstrates a favourable disease outcome even in a patient with severe KFD and a severe comorbidity.

Therefore, higher KFD awareness among health-care professionals and inclusion of the disease into the differential diagnosis in patients with fever and lymphadenopathy allow timely diagnosis, prevent unnecessary tests and examinations, and ensure adequate treatment.

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Aranovich E.V.: concept development, text preparation and editing;

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