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Case Report

A case of Kikuchi–Fujimoto Disease in a SLE patient; potential importance of OR8U8 gene polymorphisms

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Abstract

Background: A Kikuchi–Fujimoto Disease (KFD) is a benign, self-limiting lymphadenopathy condition which mostly diagnosed by a pathologist due to a lymph node biopsy. In this study, we tried to report a case of KFD in a 38-year-old Iranian woman. Furthermore, an evaluation of some genetic polymorphisms in the patient's peripheral blood was performed.

Case presentation: The patient was a 38-year-old female with a background of systemic lupus erythematosus, hypertension, hypothyroidism, and type 2 diabetes. The patient was under antibiotic treatment which was without any results. So she was referred to the rheumatology department for more evaluation. The patient went through various tests, Furthermore, the lymph node biopsy confirmed KFD. We could not find the EBV genome in the patient's serum. The polymorphisms evaluation revealed wild-type alleles in all rs78460947 in OR8U8, rs34068039 in RIOX1, rs2799077 in ZSCAN26, and rs2273346 in MASP-2 but some other polymorphisms in OR8U8 gene. Patient treatment was performed by the lymph node excision during sampling and anti-inflammation.

Conclusion: KFD is a rare disease in Iranian patients. No specific Polymorphisms could be found in this study in KFD except in the OR8U8 gene. Further evaluation of these polymorphisms provides a diagnostic or prognostic tool for KFD by more comprehensive studies.

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1. Introduction

Regardless of the worldwide distribution of Kikuchi-Fujimoto disease (KFD), this condition seems to be extremely rare. Young women in Asia are most likely affected cases. The nature of the disease is benign and self-limiting but differentiation of KFD from other diseases that cause lymphadenopathy (e.g., leukemia, lymphoma, and infectious diseases) is the most challenging part (1, 2).

The fact is, the etiology of KFD remains unclear. Some theories suggest that it is caused by viral infections, that trigger a self-limiting autoimmune process in subjects with a genetic predisposition to an autoimmune response (3). Numerous agents have been proposed for KFD triggering, including Epstein-Barr virus (EBV), human herpesvirus 6 (HHV-6), human herpesvirus 8 (HHV-8), human immunodeficiency virus (HIV), parvovirus B19, paramyxoviruses, parainfluenza virus,

Yersinia enterocolitica, and Toxoplasma (2, 4). Furthermore, recent studies suggested some polymorphisms in osteoclast activity, cancer, and olfactory are associated with KFD (1).

The onset of diseases mostly is acute or subacute, with painful cervical nodes lymphadenopathy, and fever in previously young or healthy individuals. Extranodal manifestations are generally rare in KFD but in most cases can affect the skin (16–40% of patients) with nonspecific lesions, and sometimes similar to non-Hodgkin lymphoma (NHL), reactive lymphadenopathy or systemic lupus erythematosus (SLE) (5).

The exact diagnosis could be provided based on the pathologic examination of the lymph node biopsy. Histologic changes suggest an immune response of T cells and histiocytes to an infectious agent. Follicular hyperplasia, expanded paracortex, areas of necrosis with abundant karyorrhectic and nuclear debris with a large accumulation of histiocytes, plasmacytoid dendritic cells, and immunoblasts at the edge of necrosis are considered pathological features. Among these histocytes, there are some activated T cells and plasma cells. A clue to the KFD diagnosis is the absence of Neutrophils and eosinophils. Three distinct histological patterns for KFD are proposed including proliferative, necrotizing, and xanthomatous. The proliferative pattern is defined by the expanded paracortex with sheets of histiocytes and plasmacytoid dendritic cells, small lymphocytes, and karyorrhectic nuclear debris. The necrotic phase is characterized by necrosis while the xanthomatous phase is presented by the absence of necrosis and the predominance of foamy histiocytes (2, 6). The differential diagnosis of KFD includes infectious lymphadenitis of different etiologies, autoimmune lymphadenopathy (SLE lymphadenopathy), and lymphoma (2, 6).

Furthermore, by considering the nature of KFD disease some candidate genes seems to be involved in disease pathogenesis or development. In this regard some genes were more interesting due to more prevalence of polymorphism in KFD patients (1). In this regard, we could mention OR8U8 (Olfactory Receptor Family 8 Subfamily U Member 8, Gene ID: 504189), RIOX1 (Ribosomal Oxygenase 1, Gene ID: 79697), ZSCAN26 (Zinc Finger And SCAN Domain Containing 26, Gene ID: 7741), and also MASP-2 (mannose-binding protein-associated serine protease 2, Gene ID: 10747). The OR8U8 is a primary gene in neuronal responses to odors. There is a cluster of genes associated with odor which are known as OR (Olfactory Receptor) genes which is known as one of the large gene families in human genomes. This gene polymorphisms seems to be associated with obesity (7). Another important gene is ZSCAN26 is transcription activator factor and DNA binding element with high expression in lymphoid tissue (8). The ZSCAN26 also known as the butyrophilin subfamily 3 member A2 gene (BTN3A2) and reported to be associated with risk for gastric cancer development (9). Furthermore, RIOX1 is a negative factor for histone lysine demethylation and regulation of transcription (10). This gene seems to be associated with acute myeloid leukemia (AML) (10), osteoporosis (11) and DNA repair (12). In addition, MASP-2 is considered a critical element in complement system activation (13). This role makes this gene as critical factor in immune responses, especially in viral infections (14, 15).

There are some reports of KFD from Iran in recent years (16-19). In the current study, we tried to report a case of KFD in a patient with an SLE background and review reported cases from Iran. Furthermore, we evaluated some polymorphisms in OR8U8, RIOX1, ZSCAN26, and MASP-2.

2. Case presentation

A 38-year-old female with a history of hypothyroidism and SLE, presented with fever, arthralgia, and skin erythema, with cervical and axillary lymphadenopathy. The main clinical presentation that happened in 2017 was fever, chills, arthralgia, myalgia, diffuse erythematous skin lesions, and night sweats that started 2 weeks before admission. A lupus flair-up was reported in the last 2 years. Lupus flair-up was controlled by standard multi-drug treatments including Methylprednisolone, Mycophenolic acid, Hydroxychloroquine, and Azathioprine.

At the primary point for the current admission, the SLE flair-up or infectious lymphadenopathy was considered for the patient. However, due to more investigations lymph node biopsy was performed **(Figure 1).** Multiple axillary and cervical lymphadenopathy were observed. short-axis diameter (SAD) of lymph nodes represented a maximum of 7mm lymph nodes in the cervical era and 8.5mm and 13mm in the left and right axillary era. A summary of the patient's laboratory and clinical profile is provided in **Table 1**. The lymph node biopsy represented necrotizing lymphadenopathy and KFD confirmed by an expert pathologist.

Furthermore, the patient's evaluation for EBV infection

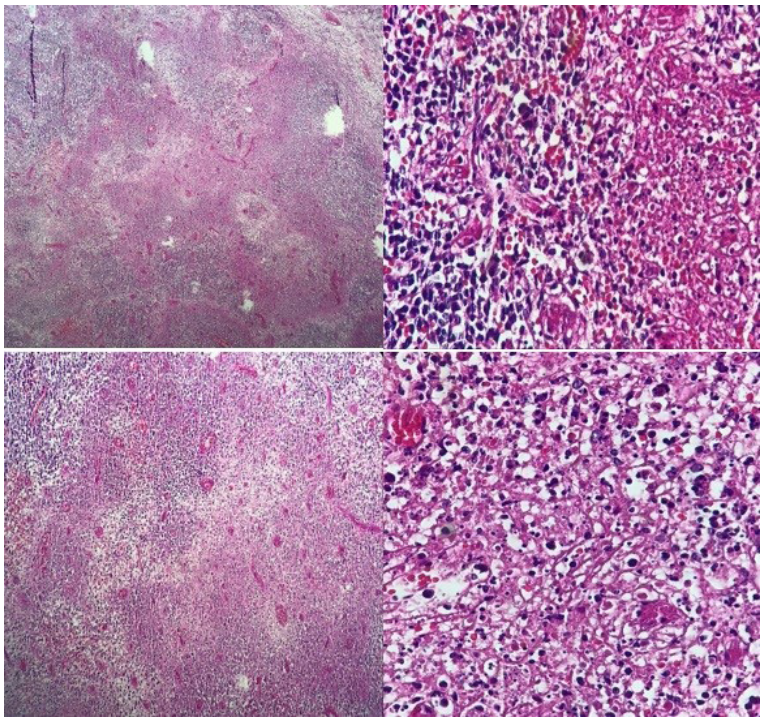

Figure 1. Microscopic examination of axillary lymph node show focal paracortical area of necrosis with abundant karyorrhectic nuclear debris without any intact neutrophil. Necrotic area surrounded by mononuclear cell. In addition, nuclear debris are extracellular and phagocytosed by histiocytes are represented

Table 1. A summary of patient laboratory and clinical profile at the time of diagnosis for KFD

Clinical presentations		
Fever, arthralgia, and skin erythema, with cervical and axillary lymphadenopathy		
Laboratory panel	*Laboratory parameter*	*Value*
Inflammatorymarkers	ESR	91 mm/hr
	CRP	Negative
CBC	WBC	7.9×10^9/L
	Hb	11 g/dL
	Plt	281×10^9/L
Rheumatologic	C3	0.67 g/L
	C4	1.96 g/L
	P-ANCA	Negative
	C-ANCA	Negative
	Anti SSA	Positive
	Anti SSB	Negative
	Anti ds-DNA	222.5 IU/ml
	ANA	Positive
	RF	Positive
	Blood culture	Negative
	Stool Exam	Negative

CBC: complete blood count, Anti SSA: Autoantibodies directed against Ro/SSA autoantigens, Anti SSB: Autoantibodies directed against La/SSB autoantigens, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, ANA: Antinuclear Antibody, RF: rheumatoid factor.

in the serum sample by PCR (polymerase chain reaction) did not show any viral genome presence. Genome polymorphisms evaluation represented that there were no polymorphisms in rs78460947 in OR8U8, rs34068039 in RIOX1, rs2799077 in ZSCAN26, rs2273346 in MASP-2. The polymorphism names, genes and primer that used for the PCR reaction are listed in **Table 2**.

A 5mL of peripheral blood was collected from the patient in EDTA (Ethylenediaminetetraacetic acid) tube. The whole blood was used for DNA extraction by AddPrep Genomic DNA Extraction Kit (Add Bio, South Korea). The extracted DNA was used for PCR. The PCR reactions were performed in a total of 25 µL volume which it was include 1µg from template DNA, 12 µL of 2X supper master mix (Yekta Tajhiz, Tehran, Iran), 1µL from each forward and reverse primers, and round out to 25 µL by water. The temperature program included 95°C for 10 min, 40 cycles at 95°C for 20 seconds, 30 seconds for annealing based on **Table 2**, 72°C for 40 seconds, and a final extension of 72°C for 10 minutes. PCR products were visualized on 1% agarose gel electrophoresis. The electrophoresis result is provided in **Figure 2**. The results for Sanger sequencing are summarized in **Figure 3.** Furthermore, some other polymorphisms were seen in OR8U8 and ZSCAN26, which are listed in **Table 3**.

3. Discussion

Kikuchi–Fujimoto Disease (KFD) is a benign lymphadenopathy that is self-limited and often happens in the course of other autoimmune conditions such as SLE. The KFD clinically confirms by lymph node biopsy (2); in our case a 38-year-old female with a background of SLE that had other symptoms like fever, arthralgia, and skin erythema, with cervical and axillary lymphadenopathy which lymph node biopsy represented necrotizing lymphadenopathy or KFD. Furthermore, laboratory parameters revealed Hb 11 g/dL, elevated ESR (91 mm/hr) and normal WBC.

As previous studies represented leukopenia and elevated inflammatory markers such as ESR or CRP seen in 50% of KFD cases (21), while anemia was rarely reported in some cases (22). As a general view and based on previous reports (2), it could be concluded that none of these parameters are diagnostic markers in KFD. In recent years, there are some cases reports of KFD in Iran. There are 6 case reports from 2018 to 2021 (17, 19, 23-25).

A list of reported cases of KFD in Iran during recent years is provided in **Table 4**. Only one study reports relapse during 1 year. This might be due to the limited time of follow-up of patients in other studies or could be a the nature of the disease. The majority of patients were female and aged less than 50 years. The curse of the disease and clinical presentation seems to be similar between our current case and previous studies (as listed in **Table 4**). The number of evaluated or reported cases in Iran seems to be increased in recent years (more than 6 reports since 2018). This might be due to more interest in reporting this kind of case or could be due to the increase in the prevalence of KFD in Iran, which needs further comprehensive studies. Furthermore, this should be considered that the evaluation of some genes or polymorphisms in one or two cases of KFD is not conclusive for considering these genes or alteration as an important element and only could make this genes as target for future evaluation in larger studies.

None of these reported cases from Iran did not represent any clue about SLE as a background disease. However, there are multiple cases of KFD presentation in SLE all around the world (5, 26). Confirming KFD in SLE or patients without any clinical background condition is based on biopsy. There is a critical matter of differentiating the diagnosis of KFD from other malignancy conditions (2, 5). In the current study, we tried to evaluate some gene polymorphisms in our case. The evaluated genes are selected based on the previous study (1). Anuntakarun et al. (1), proposed fourteen polymorphisms as possible markers for KFD. Some of these genes with more potential for polymorphisms were evaluated in our study. The evaluated genes are selected based on previous study by Anuntakaram and colleagues (1). These genes include OR8U8, RIOX1, ZSCAN26, and also MASP-2 due to its association with SLE were included either (27). Our findings represent some polymorphisms in ZSCAN26 and OR8U8, while the evaluated sequences from other genes did not show any alteration. The OR8U8 is an important element in odor sensation (28). The OR8U8 association with infectious disease especially RSV (Respiratory syncytial virus) infection is demonstrated (29). This finding seems to be a clue for further studies about the importance of Olfactory Receptor genes in respiratory infections. A clear association between OR8U8 and KFD is not available. Furthermore, our current study results seem to be similar to Anuntakarun et al.

Table 2. The polymorphism names, genes and primer which it was used for PCR reaction

Primer name	Gene	Sense	Sequence	Aneling temperature	Product size	Ref
KFD polymorphisms	rs78460947 in OR8U8	F	5′-CCATCCTCACCTTCTGCCTC -3′	59	264	This study
		R	5′-CTGCCAGCATGTGAGAGCTA -3′			
	rs34068039 in RIOX1	F	5′-CGCAATACCTGGGGTGACTT -3′	60	705	
		R	5′-CAGTGGCAGCGGAGAATCA -3′			
	rs2799077 in ZSCAN26	F	5′-ACCTTTCAATCCAAACGTATGTCC-3′	59	580	
		R	5′-AGGGAAGATTCTCTGCACCC -3′			
	rs2273346 in MASP-2	F	5′-GGGGGCTCAAGTTCCAAGT -3′	57	478	
		R	5′-GGAAAGGCAGCACACCTCTTC -3′			
EBV	EBNA3C	F	5′- AGAAGGGGAGCGTGTGTTGT-3′	55	153 EBV1	(20)
		R	5′- GGCTCGTTTTTGACGTCGGC -3′			

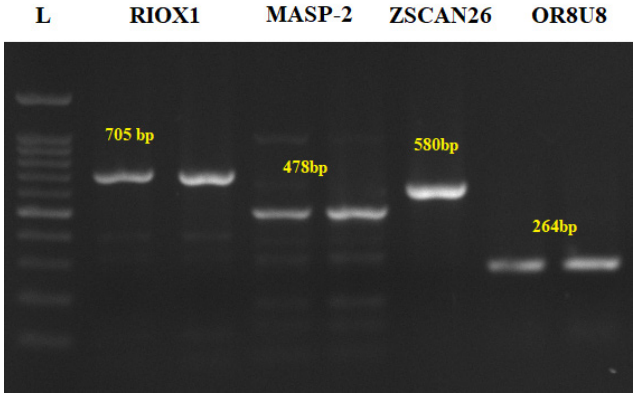

Figure 2. The electrophoresis result is provided for evaluated genes (100 to 1000bp ladder).

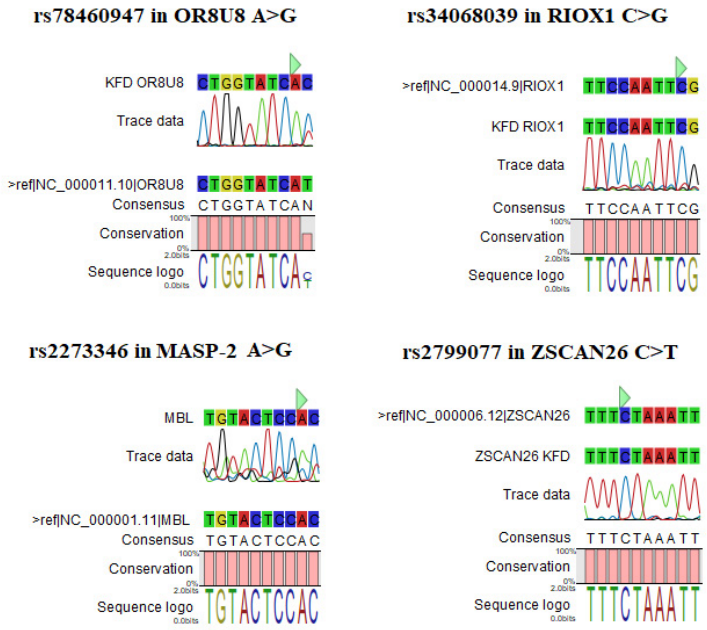

Figure 3. Polymorphism evaluation in the KFD patient.

Table 3. Some other polymorphisms were seen in OR8U8 and ZSCAN26 that are listed

Evaluated gene	Polymorphism	Substitution
ZSCAN26	rs200126967	C>A*
OR8U8	rs10896309	T>C
	rs10896310	G>A*
	rs1852796694	C>T
	rs753240579	C>G
	rs78729591	G>A
	rs202193035	A>G*
	rs201727202	G>A
	rs200433243	G>C

*Represents mutations in heterozygote form

Table 4. A list of reported cases of KFD in Iran during recent years (2009 to 2021)

Author	Year	age	gender	Clinical presentation	Background condition	Flair up	ref
Asadi	2009	17	Female	fever and cervical lymphadenopathy, leukopenia	Non	Non	(30)
Hafezi-Ahmadi	2011	30	Male	cervical lymphadenopathy malaise, fever, night sweats and weight loss	Non	Non	(31)
Afrasiabian	2014	38	Female	lymphadenopathy, fever, and generalized arthropathy	renal stone and dysuria	Non	(32)
Taghvaei	2015	32	Female	Fever, night sweats, severe weight loss and anorexia, Lymphadenopathy	Non	Non	(18)
Masoumi	2018	29	Female	low grade fever, fatigue, malaise, night sweats, gradual loss of appetite, and 5 kilograms, weight loss	Non	Non	(19)
Emami	2018	33	Male	lymphadenopathy	Non	Non	(24)
Baziboroun	2019	26	Female	fever and painful lymphadenopathy fever, nausea and fatigue	Non	Non	(25)
Ferdosian	2020	13	Male	prolonged fever occasional abdominal pain, and also reduced appetite and weight loss	Non	Non	(23)
Servatyari	2020	56	Female	Fever, malaise, loss of weight, loss of appetite, and she had noticed swelling of neck glands	Non	1 year	(16)
Kaveh Jaseb	2021	16	Female	lymphadenopathy frequent fevers, night sweats, myalgia, and weight loss	Non	Non	(17)

(1), regarding the possible importance of OR8U8 polymorphisms in KFD. The fact is none of the mentioned genes did not show a clear association with any immune, autoimmune or infectious condition. Also, a glimpse into possible functions for OR8U8 (Olfactory function), RIOX1 (expression regulation), ZSCAN26 (gene expression regulation), and MASP-2 (complement system) is provided (1).

Ethics approval and consent to participate

All the study protocols approved based on Declaration of Helsinki. Written informed consent was obtained from all subjects.

Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Conflict of interest

There is no conflict of interest.

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Not applicable.

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