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Original Article

Hematological Complications in Familial Mediterranean Fever: A Case Report and Literature Review

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Abstract

Familial Mediterranean Fever (FMF) is an inherited autoinflammatory disease caused by mutations in the MEFV gene. These patients typically present with lymphocytosis and thrombocytosis during periods of inflammation; however, some patients may manifest leukopenia along with other symptoms. Demographic data, medical history, laboratory data, and genetic findings of the cases were collected by reviewing clinical records of the patient. Whole-genome sequencing test revealed a mutation in MEFV gene. A systematic searched was conducted in four databases: PubMed, Web of Science, Scopus, and PerQuest, using keywords related to blood abnormalities in FMF disease. A mutation in the MEFV gene was confirmed in a 29-year-old patient with FMF. He experienced periodic and regular decreases in the number of neutrophils, lymphocytes, and platelets during periods of inflammation. Our literature review revealed neutropenia (17.6%), lymphopenia (8.8%), thrombocytopenia (11.8%), leukopenia (61.8%), and anemia (20.6%) are the frequent most common hematologic complications. Genetic analysis in 28 patients revealed M694V as the most prevalent mutation (57.1%), followed by E148Q (21.4%), M680I (10.7%), and others. Reporting this case and others highlights that hematological manifestations in FMF can be observed periodic and simultaneous decreases in neutrophils, lymphocytes, and platelet counts can in patients with FMF.

Keywords: Autoinflammatory Disease; Familial Mediterranean Fever; Hematological Manifestations; MEFV

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Introduction

Familial Mediterranean Fever (FMF) is an autoinflammatory disease with autosomal recessive inheritance. A gain-of-function mutation in the Mediterranean fever (MEFV) gene encoding pyrin, a protein involved in immune regulation, is responsible for the manifestation of FMF. FMF is identified by self-limiting episodes of fever and polyserositis, often accompanied by severe long-term complications such as renal amyloi-

dosis. Some patients with FMF may also exhibit hematological abnormalities, including leukopenia and neutropenia (1). Autophagy and mutations in genes involved in apoptosis processes are probably associated with the development of leukopenia in FMF (2). We report a patient with FMF who experienced recurrent, synchronized reductions in neutrophil, lymphocyte, and platelet counts. This is an interesting case indicating that FMF patients can undergo these simultaneous dips at regular intervals, every two to three months. This case underscores the importance of systematic hematological monitoring in patients with FMF.

Patients and Methods

Demographic data, medical history, laboratory data, and genetic findings of the cases were collected by reviewing clinical records of the patient. All data were collected after obtaining written informed consent from the patient. The current study was approved by the Ethics Committee of the Tehran University of Medical Sciences (IR.NIMAD.REC.1395.024). Hence, we conducted a comprehensive search to identify FMF cases presenting with hematological abnormalities. Our search was performed in PubMed, Web of Science, Scopus, and ProQuest using the following keywords: "Familial Mediterranean Fever," "FMF," "hematological complications," "neutropenia," "thrombocytopenia," "lymphopenia," "leukopenia," and "anemia." At first, the articles were screened based on the title and abstract to remove all irrelevant studies. After reviewing the full-text manuscripts, the selected articles were assessed based on specific criteria: articles in the English language, human subjects, and at least one patient diagnosed with FMF, along with detailed descriptions of clinical and immunological features.

Results

Initially, a total of 620 articles were identified through a systematic search across the databases PubMed, Web of Science, Scopus, and ProQuest. After removing 115 duplicate articles, 505 titles and abstracts were screened. As a result, 62 articles were selected for full-text review and further assessment. Following this, based on the full-text evaluation and application of inclusion criteria, 16 articles were ultimately chosen for final analysis. Additionally, one study was included based on a manual search. In total, 17 studies related to hematological complications in FMF patients were identified.

Case Presentation

The patient is a 29-year-old male and the seventh child born to healthy, non-consanguineous parents. At the age of 2 years, he experienced multiple seizures and non-periodic fevers without reductions in neutrophil, lymphocyte, or platelet counts. These fevers led to hospitalizations, during which he was treated with antibiotics and corticosteroids (betamethasone). The episodes of non-periodic fever ameliorated with age. The patient's family history revealed that the first child had died at three months of age due to unknown causes. However, no evidence of periodic fever or reduced blood cell counts was reported among the patient's close relatives.

In March 2021, during the COVID-19 pandemic in Iran, the patient, who was 26 years old, presented with symptoms of fever, chills, severe fatigue, body pain, weakness, anorexia, and proteinuria, along with reduced lymphocyte, neutrophil, and platelet counts. On the first day of hospitalization, a polymerase chain reaction (PCR) sample was taken, and he was hospitalized with a suspected diagnosis of COVID-19. He received intravenous fluid resuscitation, antibiotics (imipenem), and cetirizine tablets (10 mg per day). After three days of hospitalization, the PCR test result was negative, and the patient was discharged from the hospital due to the improvement of symptoms. Approximately three months later, the patient was hospitalized again with the same previous symptoms. He was treated with intravenous fluid resuscitation and antibiotics (ciprofloxacin, meropenem, and amphotericin B). Given the reduction in absolute neutrophil count to 361 cells per mm3, he received a single dose of granulocyte colony-stimulating factor (G-CSF) (300 mg). Bone marrow aspiration and biopsy, as well as bone marrow smear tests, showed reduced bone marrow cellularity (15% cellularity). Immunological and laboratory findings are summarized in **Table1**.

Table 1. Laboratory and immunologic data at the age of 26, following the COVID-19 pandemic in Iran.

Laboratory Test	Values	Reference values [a]
Complete blood count		
WBC/mm3	0.88↓	4000–11,000
Red blood cells (10^6/µL)	4.44↓	4.7-6.0
Hemoglobin (gr/dl)	12.3↓	13.5-18
Hematocrit (%)	35.4↓	42-52
Neutrophils (%)	41.1	20–70
Absolute neutrophil count(mm3)	361	1500-8000
Lymphocytes (%)	45.3	45–75
Absolute lymphocyte count	398	1000-4800
Platelets (10^3/µL)	55↓	140-440
ESR 1h	23↑	0-10
Serum immunoglobulins		
IgM (mg/dL)	65	40-230
IgG (mg/dL)	1143	700-1600
IgA (mg/dL)	301	70-400
IgE (IU/ml)	299.2	<280
Infections panel		
HIV1,2	Negative	Negative
HBS Antigens	Negative	Negative
Anti HCV	Negative	Negative
Coagulation test		
PT patient(sec)	14.8	13.5
PTControl (sec)	13.5	12
INR(Index)	1	0.9-1.0
PTT(sec)	25	25-38
Liver enzymes		
AST(IU/L)	38↑	0-37
ALT(IU/L)	15	0-41
ALK(IU/L)	125	80-360
LDH(U/L)	662↑	1-480
Thyroid markers		
T3(ng/dl)	83	80-200
T4(micg/dl)	7.45	4.5-11.7
TSH(mlu/l)	10.4↑	0.3-4.2
Immunology panel		
RF(quantitative)	1	UP to 20
C3(mg/dl)	139	90-180
C4(mg/dl)	47	10-40
CH50(%)	94	>=90%
Lupus anticoagulant screening(sec)	46↑	28.8-44.4
FANA(Titer)	Negative	UP to 1/40
Anti DNA(ds)(IgG)(IU/ml)	10	Negative<25
Wright(Titer)	Negative	Negative

Table 1. Continued

Direct coombs(Qual))	Negative	Negative
2ME Wright(Titer)	Negative	Negative
Kidney function tests		
Blood Urea(mg/dl)	23↑	9-20
Creatinine(mg/dl)	1.2	0.7-1.4
Sodium (meq/L)	140	136-145
Potassium (meq/L)	4.3	3.7-5.5
Biochemistry test		
FBS(mg/dl)	88	70-115
TIBC(micg/dl)	258	230-440
Ferrittin(micgr/l)	185	18.2-341.2
Serum iron(micg/dl)	50↓	60-180
Inorganic P (mg/dl)	2.97	2.6-4.5
Serum CA (mg/dl)	8.24	8.6-10.3
Total Bilirubin(mg/dl)	0.48	0.1-1.2
Direct Bilirubin(mg/dl)	0.12	0-0.2
Vitamin D(ng/ml)	34.5	30-100
Urine Analysis		
Random urine protein(mg/dl)	40↑	UP to 14

Abbreviations: WBC, white blood cell count; ESR, erythrocyte sedimentation rate; AST, aspartate transaminase; ALT, alanine transaminase; LDH, lactate dehydrogenase; TSH, thyroid-stimulating hormone; RF, rheumatoid factors; FANA, fluorescent antinuclear antibody; FBS, fasting blood sugar; TIBC, total iron binding capacity

According to the patient's laboratory information, decreased cellularity was observed in the bone marrow aspiration results, confirmed by flow cytometry. The CBC showed significant leukopenia and thrombocytopenia, while red blood cell (RBC) levels remained normal. Flow cytometry revealed normal CD55 and CD59 markers, ruling out paroxysmal nocturnal hemoglobinuria. Additionally, the peripheral blood smear test results were normal. These findings confirmed bicytopenia and were attributed to impaired bone marrow function. Based on this evidence, a diagnosis of aplastic anemia was proposed for the patient.

Over approximately four years, periodic episodes of fevers, fatigue, anorexia, headache, and muscle cramps, and proteinuria, along with reductions in white blood cell (WBC) and platelet counts, recurred every two to three months. The duration of these episodes was approximately 10 days, although some episodes lasted less or more than 10 days. The hematological changes during disease episodes and the intervals between episodes (from 2020 to 2024) are depicted in **Figure 1**. Over this four-year period, the disease initially manifested with bone pain and headache, accompanied by a progressive decrease in WBC counts. This pattern enabled the patient to anticipate the onset of subsequent episodes. The recurrent nature of these symptoms resulted in frequent hospitalizations and treatments with various antibiotics, including imipenem, meropenem, ciprofloxacin, and vancomycin, as well as G-CSF injections administered during periods of neutropenia. Additionally, the patient received prophylactic medications such as vitamins, antibiotics, antiviral drugs, and danazol to help manage and prevent future episodes.

Finally, in September 2024, whole exome sequencing revealed a mutation in the MEFV gene (P.Val726Ala). The MEFV gene, located on chromosome 16, encodes a protein called pyrin, which is crucial for regulating inflammation and the immune response. The heterozygous mutation c.2177T>C (p.Val726Ala) in exon 10 of the MEFV gene is pathogenic for FMF and follows an autosomal dominant inheritance pattern, meaning that the presence of just one mutated copy of the gene from either parent is sufficient to cause

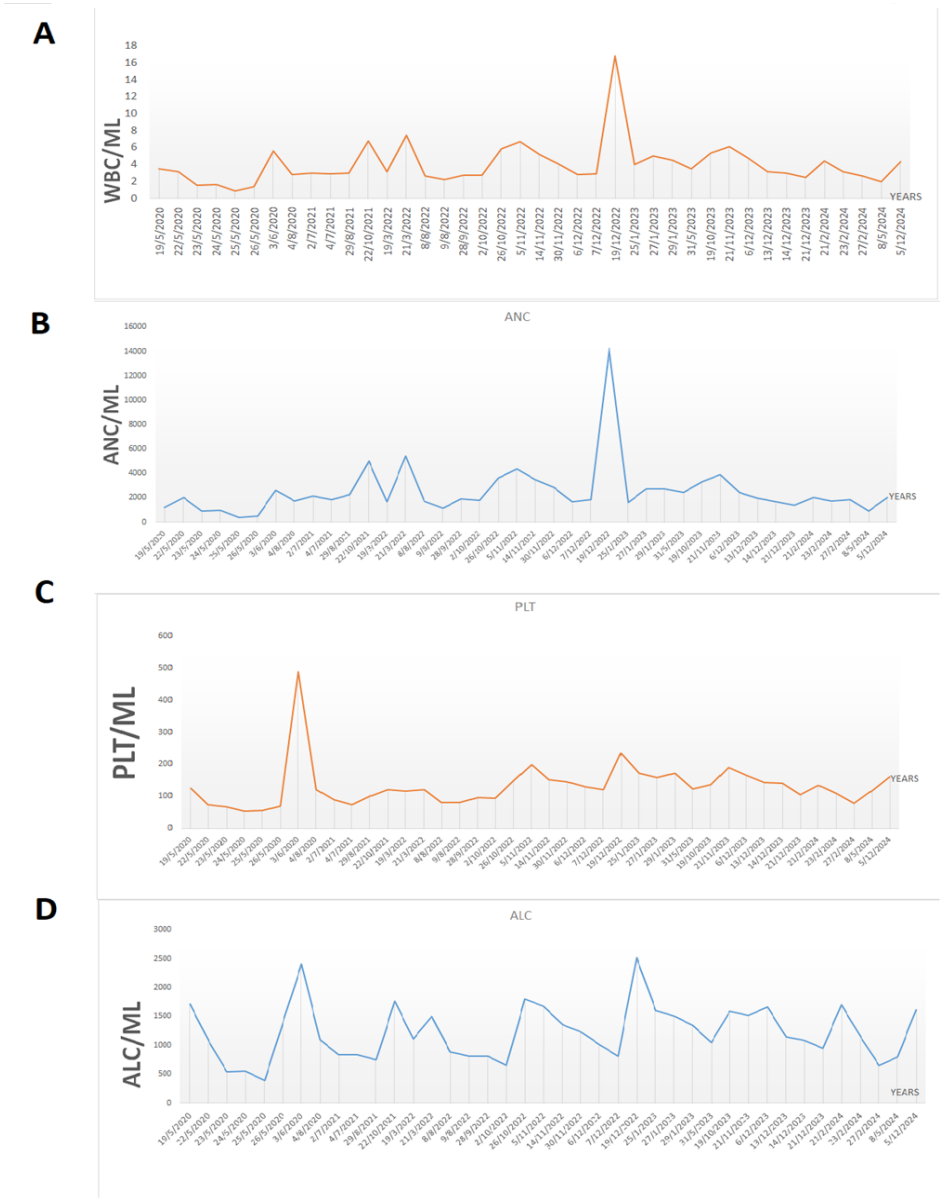

Figure 1. Hematological changes from 2020 to 2024 during attack and attack-free periods of familial Mediterranean fever A. Changes in white blood cell count during attack and attack-free periods of familial Mediterranean fever B. Changes in absolute neutrophil count during attack and attack-free periods of familial Mediterranean fever C. Changes in absolute lymphocyte count during attack and attack-free periods of familial Mediterranean fever D. Changes in platelet count during attack and attack-free periods of familial Mediterranean fever. It is also noteworthy that the patient may have received G-CSF injections during these periods, which could have influenced the hematological parameters. Abbreviations: ANC: Absolut neutrophil count; PLC: Platelet count; ALC: Absolut lymphocyte count.

the disorder. After diagnosing FMF with the MEFV gene, the patient was treated daily with 1 gram of colchicine, leading to a decline in fluctuations in WBC counts. Additionally, the patient has not required hospitalization or G-CSF injections. The periodic fevers, fatigue, anorexia, headache, and muscle cramps, and proteinuria, along with recurrent reductions in WBC and platelet counts every two to three months for approximately four years, have been well controlled with colchicine treatment.

Literature Review

Hematologic data for ANC were unavailable in 28 out of 34 patients (82.4%) **(Table 2)**. Of the six patients with available absolute neutrophil count (ANC) data, two (33.3%) had ANC below 1500/mm^3, while four (66.7%) had ANC above this

Table2. Hematological changes in 34 cases diagnosed with FMF

Case	Age (y)/gender	Ethnicity	WBC (mm3)	ANC (mm3)	Neutrophil%	ALC (mm3)	lymphocyte%	PLT (mm3)	FMF mutation	Allele status	Hematologic complications	Diagnose disease	Clinical manifestations	Years	Authors/Reference
1	5/F	Mixed Ethnicity	NA	NA	NA	NA	NA	NA	Met694val	heterozygous	Mild thrombocytopenia	FMF	Fever-leg pain- Feeling cold-Mild abdominal pain	2019	R. Uruthirakumar et al (12)
2	49/F	Japanese	NA	NA	NA	NA	NA	NA	R410H, E84K, E148Q	Uncertain	thrombocytopenia	FMF	Fever- Arthritis- Erythema	2018	D. Kishida (13)
3	13/F	Turkish	3510	1400	NA	1700	NA	200,000	M680I/ M694V	Heterozygous	Leukopenia-Neutropenia	FMF	Abdominal pain-ankle pain, swelling, and redness (hyperemia)	2018	I. Beyitler et al (1)
4	3..5/M	NA	NA	NA	NA	NA	NA	108,000	M680I	Heterozygous	Thrombocytopenia-Anemia	FMF-HIDS	Hepatosplenomegaly - cervical lymphadenopathy - fever- urticarial like rash,-abdominal pain-diarrhe- cervical lymphadenopathy - chest pain	2017	M. Çakan etal (14)
5	34/F	Turkish	4180	2320	NA	NA	NA	NA	M694V/ R202Q	Heterozygous	Leukopenia	FMF	Abdominal pain	2014	D. Aslan et al (15)
6	6/M	Turkish	4260	1580	NA	NA	NA	NA	M694V/N and R202Q/ R202Q	Heterozygous-Homozygous	Leukopenia	FMF	Abdominal pain	2014	D. Aslan et al (15)
7	10/F	Turkish	3700	2100	NA	NA	NA	NA	M694V/ M694V	Homozygous	Leukopenia	FMF	Fever -abdominal pain-Arthralgia	2014	D. Aslan et al (15)
8	44/F	Turkish	3700	2500	NA	NA	NA	NA	M694V/ N	Heterozygous	Leukopenia	FMF	Abdominal pain	2014	D. Aslan et al (15)
9	15/M	Turkish	2700	900	NA	NA	NA	NA	V726A/N	Heterozygous	Leukopenia-Neutropenia	FMF	Abdominal pain	2014	D. Aslan et al (15)
10	14/F	Turkish	3600	2300	NA	NA	NA	NA	M694V/ M694V	Homozygous	Leukopenia	FMF	Abdominal pain	2014	D. Aslan et al (15)
11	7/M	Turkish	3880	1690	NA	NA	NA	NA	R761H/ N	Heterozygous	Leukopenia	FMF	Fever -abdominal pain	2014	D. Aslan et al (15)
12	12/F	Turkish	4600	2200	NA	NA	NA	NA	M694V/ M694V	Homozygous	Leukopenia	FMF	Abdominal pain-Arthralgia	2014	D. Aslan et al (15)
13	11/F	Turkish	4530	2040	NA	NA	NA	NA	M694V/ M680I (G/A)	Heterozygous	Leukopenia	FMF	Abdominal pain	2014	D. Aslan et al (15)
14	14/M	Turkish	4730	2500	NA	NA	NA	NA	M694V/ M680I (G/A)	Heterozygous	Leukopenia	FMF	Abdominal pain-skin rash on buttocks and legs	2014	D. Aslan et al (15)
15	16/F	Turkish	4780	2420	NA	NA	NA	NA	M680I (G/C)/N	Heterozygous	Leukopenia	FMF	Abdominal pain-Arthralgia	2014	D. Aslan et al (15)
16	9/M	Turkish	3100	100	NA	NA	NA	NA	M694V/ M694V	Homozygous	Leukopenia-Neutropenia	FMF	Arthralgia	2014	D. Aslan et al (15)
17	13/M	Turkish	2600	1200	NA	NA	NA	NA	E148Q/ N	Heterozygous	Leukopenia	FMF	Fever -abdominal pain	2014	D. Aslan et al (15)
18	7/F	Turkish	827	389	NA	NA	NA	NA	E148Q/ N	Heterozygous	Leukopenia-Neutropenia	FMF	Abdominal pain	2014	D. Aslan et al (15)
19	3/F	Turkish	4030	2430	NA	NA	NA	NA	M694V/ M694V	Homozygous	Leukopenia	FMF	Fever	2014	D. Aslan et al (15)
20	15/M	Turkish	2.500 to 3.000	NA	NA	NA	NA	NA	V726A	Heterozygous	Leukopenia-Microcytic hypochromic anemia	FMF-Alpha thalassemia	Abdominal pain -Fever-Chill-Nausea- Fatigue	2013	D. Aslan (16)
21	20/F	NA	NA	NA	NA	NA	NA	NA	NA	NA	Leucopenia-Pancytopenia-Thrombocytopenia	FMF	Recurrent fever-Shaking chills-Profuse sweating-Bilateral diffuse arthralgia - Focal myositis-Hypocholesterolemia-Hepatosplenomegaly	2013	F. Adragna etal (17)
22	8/M	Korean	NA	NA	NA	NA	NA	NA	Pro369Ser, Arg408Gln	Uncertain	Anemia	FMF	Incomplete abdominal attack-Fever	2012	K. Y. Koo etal (18)
23	14/M	Turkish	2700	1120	41.40%	1100	40.70%	Adequate platelets	M694V/E148Q	Heterozygous	Leukopenia-Neutropenia	FMF	Abdominal pain-Periodical fever-Arthralgia	2012	O. Sakallioglu et al (8)

Table 2. Continued

24	32/F	Japanese	3900	NA	NA	NA	NA	NA	G304R/G304R (G/A)	Homozygous	Leukopenia	FMF	Abdominal pain and lumbago	2011	Y. Tone et al (19)
25	61/(17)M	Japanese	3200	NA	NA	NA	NA	NA	G304R/G304R (G/A)	Homozygous	Leukopenia	FMF	Periodic episodes of fever - Abdominal pain	2011	Y. Tone et al (19)
26	24/F	North African Jewish	3400	NA	NA	NA	NA	NA	Clinical diagnosis	NA	Leucopenia	FMF-SLE	Abdominal pain -Fever-Monoarthritic	2008	M. Lidar etal (20)
27	22/F	North African Jewish	NA	NA	NA	NA	NA	NA	M694V / E148Q	Uncertain	Microcytic anemia	FMF-SLE	Diffuse abdominal pain-Fever-Weight loss - Arthralgias -Peripheral lymphadenopathy	2008	M. Lidar etal (20)
28	38/M	North African Jewish	3700	NA	NA	NA	NA	NA	M694V	Heterozygous	Leucopenia-Mild anemia	FMF-SLE	Fever- Recurrent episodes of ankle monoarthritic - Erysipeloid erythema - Severe myalgia	2008	M. Lidar etal (20)
29	57/M	Caucasian	2000 to 4000	NA	NA	NA	NA	100 to 140	E148Q	Heterozygous	Modest leucopenia-Mild anemia (12)-Mild thrombocytopenia	FMF-SLE	Malar rash and photosensitivity-	2008	B. E. Schreiber etal (21)
30	14/M	Egyptian	500 to 1000	NA	NA	NA	NA	NA	NA	NA	Lymphopenia-Pancytopenia	FMF	Chronic diarrhea- Failure to thrive-Hepatosplenomegaly-Thyroid dysfunction-Proteinuria	2008	J. Woodard etal (22)
31	12/F	Moroccan	300–6848 (min-max)	1700 (median)†-(Range 300/mm3 to 6,848/mm3)	NA	2225	NA	NA	M694V/M694V	Homozygous	Leukopenia-cyclic neutropenia -Mild Lymphopenia-Elevated monocyte and platelet counts	FMF	Febrile diarrhea-Abdominal pain-Gingivostomatitis-Skin infections (furunculosis and cellulitis)-Upper airway infections-Severe dental caries-Proteinuria and renal failure-AA amyloidosis-Polyclonal hypergammaglobulinemia-Edema of the limbs	2008	K. GaniouTidjani et al (23)
32	19/M	NA	2500	NA	NA	NA	NA	469,000	NA	NA	Leukopenia-Mild Lymphopenia-Severe Anemia-Febrile neutropenia	FMF	Secondary amyloidosis - Nephrotic syndrome-abdominal complaints	2008	E. Koca et al (24)
33	36/M	Sephardic Jew	NA	NA	NA	NA	NA	NA	NA	NA	Anemia	FMF-Vasculitis	Abdominal pain - Fever severe- muscular pain-joint pain	2006	A. Balbir-Gurman (25)
34	17/F	Turkish	4000	NA	NA	NA	NA	NA	Clinical diagnosis	NA	Leukopenia-Neutropenia	Cyclic neutropenia associated with FMF	Recurrent infections-Renal AA-type amyloidosis	2000	Metin et al (26)

Abbreviations: WBC, white blood cell count; FMF, familial Mediterranean fever; ANC, absolute neutrophil count; NA: not available; ALC, Absolute lymphocyte count; PLT, Platelet count; SLE, Systemic Lupus Erythematosus; HIDS, Hyperimmunoglobulinemia D syndrome

threshold. Based on hematologic side effects, six patients (17.6%) exhibited neutropenia. Data on absolute lymphocyte count (ALC) were absent in 32 patients (94.1%). Regarding hematologic adverse effects, three patients (8.8%) showed mild lymphopenia. Platelet count data were unavailable for 30 patients (88.2%). Hematologic side effects included thrombocytopenia in four patients (11.8%), while an increase in platelet count was documented in one patient (2.9%). Regarding hematologic adverse effects, twenty-one patients (61.8%) exhibited leukopenia, and seven patients (20.6%) had anemia. Conversely, an increase in platelet count was observed in one patient (2.9%).

Genetic profiles were determined in twenty-eight cases. The most common mutation identified was M694V, present in sixteen patients (57.1%). Other mutations included G304R (two patients, 7.1%), E148Q (six patients, 21.4%), M680I (three patients, 10.7%), R202Q (two patients, 7.1%), V726A (two patients, 7.1%), R761H (one patient, 3.6%), as well as R410H/E84K and Pro369Ser/Arg408Gln, each in one patient (3.6%). In two cases (7.1%), the diagnosis was based solely on clinical features.

Clinically, abdominal pain was reported in twenty-six of thirty-four patients (76.5%), with fever documented in the same proportion. Arthralgia and arthritis were observed in nine patients (26.5%). Skin manifestations were noted in five patients (14.7%), amyloidosis in three (8.8%), and proteinuria in two (5.9%). Other symptoms presented less frequently.

Discussion

We herein report the first case with a history of seizures and intermittent childhood fevers, who developed recurrent episodes of fever, fatigue, and cytopenias in adulthood. Genetic testing identified a heterozygous P.Val726Ala mutation in the MEFV gene, which is pathogenic for FMF and follows an autosomal dominant inheritance pattern. Reporting this case, along with previously reported FMF cases with hematological complications, helps broaden our understanding of this rare and complex IEI.

Hematological findings in this patient, particularly leukopenia and thrombocytopenia during disease flares, are noteworthy within the context of our literature search results. While studies have demonstrated that FMF attacks are typically accompanied by elevated inflammatory markers, leukocytosis, and thrombocytosis (3), our literature review indicated that leukopenia and thrombocytopenia can also occur in a significant subset of FMF patients (21 cases of leukopenia and 4 of thrombocytopenia among 34 patients). This suggests that hematological manifestations in FMF can be diverse and are not limited to increased blood cell counts.

Multiple mechanisms may contribute to the development of leukopenia and thrombocytopenia in FMF patients. Elevated apoptosis in neutrophils and monocytes during inflammatory episodes may help modulate the immune response and prevent chronic inflammation (4), yet can also lead to transient reductions in blood cell levels. Additionally, the mTOR metabolic pathway plays an important role in regulating pyrin production and autophagy in the leukocytes of FMF patients (4). Autophagy activation during attacks might facilitate the clearance of damaged inflammatory cells. Copper deficiency (5) and bone marrow suppression effects of colchicine (6, 7) have also been proposed as potential causes of leukopenia and anemia in FMF. Interestingly, in one case, colchicine therapy initiation was associated with a gradual increase in WBC and neutrophil counts (8), indicating that colchicine's effects on hematopoiesis may be complex and patient-dependent (5).

In our case, treatment with colchicine following the FMF diagnosis was effective in reducing WBC fluctuations and controlling episodic symptoms, thereby eliminating the need for hospitalization and G-CSF injections. This therapeutic response aligns with colchicine's primary role in FMF management, especially through inflammation suppression and neutrophil migration inhibition (9, 10). Although colchicine can cause hematological side effects, including bone marrow suppression and leukopenia (11), the benefits of disease control and hematological improvement outweighed potential adverse effects in our patient.

Hematological manifestations in FMF are diverse and are not solely characterized by increased blood cell counts. When evaluating such findings, clinicians should consider factors like disease severity, attack phase, treatments administered,

and other concurrent infections. Future studies should aim to elucidate the underlying mechanisms of leukopenia and thrombocytopenia in FMF. Early diagnosis and effective treatment—particularly with colchicine—remain essential for disease control and the prevention of long-term complications.

Conclusion

We reported a unique case of FMF and its associated hematological changes. The findings suggest that hematological manifestations in FMF patients are not limited to elevated blood cell counts; instead, they can also include periodic and simultaneous decreases in neutrophils, lymphocytes, and platelets. This highlights the diverse and complex nature of hematological involvements in FMF patients and underscore the importance of comprehensive, multidisciplinary evaluation for effective management and treatment of the condition.

Conflicts of Interest

The authors have no conflict of interest related to this publication. The authors have no financial or non-financial interests to disclose.

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