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Evaluation of Clinical Characteristics of Patients with PFAPA Syndrome According to Febrile Seizure History

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Abstract

Although the clinical phenotype of periodic fever, aphthous stomatitis, pharyngitis, and cervical adenitis (PFAPA) syndrome is well defined, its association with febrile seizures (FS) remains unclear. This study aimed to evaluate potential risk factors associated with FS development by comparing PFAPA patients with and without FS. The study included 169 children who were diagnosed with PFAPA by a pediatric rheumatologist according to EUROFEVER/PRINTO criteria, and followed up in our tertiary care center for at least six months. According to the FS assessment, individuals were assigned to one of two groups. Demographic, clinical, and laboratory characteristics were analyzed comparatively between the groups. The prevalence of FS in children with PFAPA was 8.9%, which was higher compared to the general pediatric population. The age at diagnosis was significantly lower in patients with FS, compared to those without FS [32 (13-110) months vs. 53 (12-116) months, p=0.044]. There were no significant variations between the two groups regarding demographic, clinical, or laboratory parameters. Although the basic clinical phenotype of PFAPA appears largely independent of the presence of FS, the younger age at diagnosis in patients with a history of FS suggests that seizures may contribute to earlier clinical recognition of the syndrome. Our findings emphasize the importance of considering neurological outcomes in the management of PFAPA.

Keywords: Aphthous, febrile seizures, fever, pharyngitis, stomatitis

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Introduction

Periodic fever, aphthous stomatitis, pharyngitis, and cervical lymphadenitis (PFAPA) is the most common autoinflammatory disorder of childhood, usually beginning before the age of five; lasting 3-7 days; and it is characterized by recurrent episodes of fever every 2-8 weeks. During fever-free periods, children are completely asymptomatic and exhibit normal growth and development[1]. Although clinically benign, frequent attacks and uncertainties in the diagnostic process can reduce children's well-being and cause stress for parents[2,3]. Therefore, there is increasing interest in treatment options aimed at reducing the frequency of attacks[4].

Highlights

- The prevalence of fever syndrome prevalence in periodic fever, aphthous stomatitis, pharyngitis, and adenitis (PFAPA) children is higher than in the general population.
- PFAPA patients with fever syndrome were diagnosed at a younger age.
- Neurological outcomes should be considered in PFAPA management.

Although the main clinical features of PFAPA are well characterized, the association with febrile seizures (FS) remains unclear[1]. Recurrent episodes of high fever, especially in children between 6 months and 6 years of age, are a well-known risk factor for the development of FS[5]. In this context, the possibility of FS in patients with PFAPA should not be dismissed[6]. It has been suggested that periodic fever syndromes such as PFAPA and Familial Mediterranean fever may be associated with a higher rate of FS compared to healthy children[5,6].

Furthermore, genetic predisposition, family history of recurrent fever, and duration of attacks were found to be associated with FS in children with periodic fever syndromes[5,7]. Although the frequent episodes of high fever in PFAPA may lower the seizure threshold, the increasing frequency of reported children experiencing FS, associated with PFAPA in recent years, suggests that the clinical spectrum of the syndrome may be broader than previously thought[5,6]. In addition, the fact that cases of aseptic encephalitis with seizures during a PFAPA attack have been reported reinforces the need for neurological follow-up[8].

This study aimed to assess the clinical and demographic characteristics of PFAPA patients with a history of FS, compare them to PFAPA patients without such a history, and identify potential risk factors linked to the development of FS.

Materials and Methods

Study Cohort and Data Collection

The study included children in whom PFAPA was identified by a pediatric rheumatologist based on the EUROFEVER/PRINTO criteria, and who had been followed at our tertiary care center for a minimum of six months[9].

Demographic, clinical, genetic [Mediterranean Fever (MEFV) gene variation]; laboratory, and history of FS data were obtained retrospectively from the patient medical records. The "attack period" laboratory data include complete blood count values, C-reactive protein, and erythrocyte sedimentation rate, recorded during the initial attack after the patients were admitted to our department. Conversely, the "non-attack period" laboratory data consist of the same markers, measured two weeks after the initial assessment, when the children were entirely asymptomatic.

This study was approved by the İstanbul Medipol University Non-Interventional Clinical Research Ethics Committee (approval number: 725, date: 18.07.2024) and conducted in accordance with the ethical principles of the Declaration of Helsinki.

Statistical Analysis

The statistical evaluation of the data was carried out using SPSS software (version 26.0, Chicago, IL, USA). For categorical variables, frequencies and percentages were reported, and comparisons across groups were examined with the chi-square test. Continuous variables were expressed according to their distribution characteristics as mean ± standard deviation when normally distributed and as median values with ranges when the distribution was skewed. Distributional assumptions were checked with the Kolmogorov-Smirnov test. Depending on normality, either the Student's t-test or the Mann-Whitney U test was applied. Logistic regression analysis was employed to determine independent predictors of FS. Initially, univariate binary logistic regression was performed for variables with a p-value <0.2 in the comparison between patients with and without FS. Then, these variables and clinically significant confounding variables were included in the multivariate model. Family history of periodic fever and tonsillectomy were considered as confounding factors. Odds ratio along with its 95% confidence interval was estimated for all variables. The significance level used to determine statistical significance was set at $p<0.05$.

Results

Demographic, Clinical and Laboratory Findings of the Cohort

The median age at disease onset among the 169 patients diagnosed with PFAPA was 30 (2-92) months; the median age at diagnosis was 52 (12-116) months; and 65.7% (n=111) were male. The median duration of attacks was 4 (1-10) days, and the median time between attacks was 20 (7-90) days. The median of the highest temperature value in the attack was 40 (38-41) °C. The median number of monthly attacks was 1 (0-4).

A history of preterm delivery was observed in 16% (n=27) of the patients, while a history of small for gestational age (SGA) was noted in 6.5% (n=11). Neonatal intensive care unit (NICU) hospitalization occurred in 24.9% (n=42) of the subjects, and FS were present in 8.9% (n=15). The median duration of breastfeeding was 21 (0-54) months. Consanguineous marriages were reported in 14.6% (n=25) of the patients. The family history of

FS was 4.1%, (n=7), family history of periodic fever was 63.9%, (n=108), and family history of tonsillectomy was 47.3% (n=80). Additionally, the presence of the *MEFV* variation was found in 31.4% (n=53) of the participants.

The clinical findings during the attacks included sore throat (n=166, 98.2%), lymphadenitis (n=127, 75.1%), abdominal pain (n=101, 59.8%), aphthous stomatitis (n=88, 52.1%), fatigue (n=86, 50.9%), myalgia (n=79, 46.7%), arthralgia (n=67, 39.6%), vomiting (n=51, 30.2%), headache (n=44, 26%), diarrhea (n=33, 19.5%), rash (n=11, 6.5%), constipation (n=8, 4.7%), chest pain (n=8, 4.7%), conjunctivitis (n=6, 3.6%), periorbital edema (n=3, 1.8%), cough (n=3, 1.8%), rhinorrhea (n=2, 1.2%), blood in the stool (n=1, 0.6%), arthritis (n=1, 0.6%), and ecchymosis (n=1, 0.6%).

Comparison of PFAPA Patients with and Without Febrile Seizures

The age at diagnosis was significantly lower in patients with FS compared to those without FS [median 32 (range 13-110) months vs. median 53 (range 12-116) months, p=0.044]. No statistically significant differences were observed in other demographic, clinical, or laboratory characteristics between the two groups (**Table 1**).

Risk Factors for Febrile Seizures

Univariate and multivariate logistic regression analyses revealed no statistically significant risk factor for FS (p>0.05) (**Table 2**).

Discussion

In this study, clinical, demographic, and laboratory characteristics associated with the development of FS in patients with PFAPA were evaluated. These parameters were evaluated in patients with and without a history of FS. The prevalence of FS in patients with PFAPA was 8.9%. The age at diagnosis was significantly lower in patients with a history of FS, whereas the groups did not differ with respect to other clinical and laboratory findings.

The relationship between periodic fever syndromes and FS has received increasing attention in the literature[5,6,10]. While existing studies have generally evaluated limited parameters, such as fever or genetic predisposition[5,6], our study addresses this relationship within a much broader clinical and demographic spectrum and presents one of the most comprehensive patient series in the literature. While the prevalence of FS in the general pediatric population varies between 2-5%,[11,12] it has been reported to be between 8.6% and 18.2% in PFAPA patients[5,6]. In our study, the prevalence of FS was found to be 8.9%, which is higher than that in healthy children, but consistent with the available data in the literature.

Episodes of fever exceeding 39 to 40 °C, which are frequent in PFAPA, are one of the strongest environmental triggers for FS in childhood, increasing neuronal excitability and lowering the seizure threshold[6]. In our study, the groups did not differ significantly with respect to the highest fever values during the attack, and high fever was not associated with the development of FS. This may be explained by the different pathophysiological characteristics of the fever response in PFAPA compared to other febrile states. It has been reported that PFAPA fevers are shorter and more self-limiting than fevers caused by infections, and thermal exposure may not last long enough to exceed the seizure threshold[5,6]. It has also been suggested that the systemic inflammatory response seen in PFAPA stimulates anti-inflammatory mechanisms along with proinflammatory cytokine release and that this balance may modulate the thermal stress response in the brain[13-16].

The development of FS is associated with various genetic, congenital, environmental, and immunologic risk factors, including male sex, family history of FS or epilepsy, prematurity, low birth weight, history of hospitalization in the NICU, short-term or no breastfeeding, iron deficiency anemia, genetic mutations such as *PRRT2* and *SCN1A*, history of seizures with low fever, and rapid rise in fever[17-21]. It has been previously demonstrated that the age at first attack and age at diagnosis are significantly lower in PFAPA patients with a history of FS, compared to those without. While male sex, a family history of periodic fever and tonsillectomy, and MEFV variation are more prevalent among PFAPA patients with FS history, these differences do not reach statistical significance[5]. However, the relationship between FS and prenatal history and breastfeeding duration in patients with PFAPA has not been studied to date[5,6]. In our study, no statistically significant difference was found in terms of age at first episode, male sex, preterm birth, SGA, NICU hospitalization, duration of breastfeeding, family history of FS, periodic fever and tonsillectomy, and MEFV variation. However, the age at diagnosis was found to be significantly lower in patients with a history of FS, and this finding is consistent with existing data in the literature[5]. The proinflammatory cytokine response, particularly interleukin-1β, is more prominent in patients with FS at an early age, lowering the seizure threshold accompanying fever and accelerating inflammatory processes[22,23]. This hyperreactive immune response is one of the main pathophysiological mechanisms of PFAPA syndrome[9]. It is therefore likely that symptoms of PFAPA will appear at an earlier age in these children. Furthermore, careful monitoring of febrile episodes in those with FS may allow for increased clinical awareness and earlier recognition of PFAPA.

Study Limitations

The most important limitation of our study was the limited sample size of patients presenting a history of FS, which may have restricted the demonstration of stronger statistical associations. However, our study included one of the largest PFAPA cohorts reported in the literature, and systematically compared the demographic and clinical profiles of patients, stratified by history of FS. In addition, the detailed evaluation of variables such as perinatal history, breastfeeding duration, and family history provides new perspectives in the literature on early life factors that may affect the development of FS in PFAPA syndrome.

Table 1.
Comparison of PFAPA patients with and without febrile seizures

	PFAPA patients with febrile seizures (n=15)	PFAPA patients without febrile seizures (n=154)	p
Male gender (n, %)	12 (80%)	99 (64.3%)	0.348
Age at first attack (month) [Median (Min-Max)]	12 (6-53)	30 (2-92)	0.087
Age at diagnosis (month) [Median (Min-Max)]	32 (13-110)	53 (12-116)	0.044
Time from first attack to diagnosis (month) [Median (Min-Max)]	14 (6-98)	17 (2-88)	0.767
Duration of attack (day) [Median (Min-Max)]	4 (3-10)	4 (1-10)	0.810
Time between attacks (day) [Median (Min-Max)]	20 (10-60)	20 (7-90)	0.988
Number of monthly attacks [Median (Min-Max)]	1.5 (0-2.5)	1 (0-4)	0.966
Highest fever value in the attack (°C) [Median (Min-Max)]	40 (38.7-41)	40 (38-41)	0.917
Gestational age (n, %)			
Preterm (n, %)	4 (26.7%)	23 (14.9%)	0.265
Term (n, %)	11 (73.3%)	131 (85.1%)	0.265
Birth weight (n, %)			
SGA (n, %)	2 (13.3%)	9 (5.8%)	0.424
AGA (n, %)	13 (86.7%)	135 (87.7%)	0.424
LGA (n, %)	0 (0%)	10 (6.5%)	0.424
NICU hospitalization (n, %)	4 (26.7%)	38 (24.7%)	1.0
Breastfeeding duration (month) [Median (Min-Max)]	22 (0.25-30)	20.5 (0-54)	0.543
Vaccination status (n, %)	15 (100%)	139 (90.3%)	0.366
Allergy history (n, %)	9 (60%)	70 (45.5%)	0.420
Family history			
Febrile seizures (n, %)	2 (13.3%)	5 (3.2%)	0.119
Periodic fever (n, %)	10 (66.7%)	98 (63.6%)	1.0
Tonsillectomy (n, %)	6 (40%)	74 (48.1%)	0.745
Consanguineous marriage (n, %)	2 (13.3%)	23 (14.9%)	1.0
MEFV mutation (n, %)	5 (45.5%)	48 (51.1%)	0.973
Exome 10 variation (n, %)	3 (27.3%)	27 (29%)	1.0
M694V variation (n, %)	2 (18.2%)	16 (17%)	1.0
Clinical findings during the attack (n, %)			
Sore throat	14 (93.3%)	152 (98.7%)	0.245
Lymphadenitis	10 (66.7%)	117 (76%)	0.531
Abdominal pain	11 (73.3%)	90 (58.4%)	0.397
Fatigue	6 (40%)	80 (51.9%)	0.540
Aphthous stomatitis	8 (53.3%)	80 (51.9%)	1.0
Myalgia	5 (33.3%)	74 (48.1%)	0.412
Arthralgia	5 (33.3%)	62 (40.3%)	0.805
Vomiting	2 (13.3%)	49 (31.8%)	0.236
Headache	4 (26.7%)	40 (26%)	1.0
Diarrhea	2 (13.3%)	31 (20.1%)	0.738
Rash	2 (13.3%)	9 (5.8%)	0.253
Constipation	2 (13.3%)	6 (3.9%)	0.151
Chest Pain	0 (0%)	8 (5.2%)	1.0
Conjunctivitis	2 (13.3%)	4 (2.6%)	0.090
Periorbital edema	0 (0%)	3 (1.9%)	1.0
Cough	0 (0%)	3 (1.9%)	1.0
Rhinorrhea	0 (0%)	2 (1.3%)	1.0
Blood in stool	0 (0%)	1 (0.6%)	1.0
Arthritis	0 (0%)	1 (0.6%)	1.0
Ecchymosis	0 (0%)	1 (0.6%)	1.0
Laboratory findings in the attack period			
Neutrophil count ($\times10^9$/L) [Median (Min-Max)]	8.8 (3-15.4)	9 (1.2-27.1)	0.607
Lymphocyte count ($\times10^9$/L) [Median (Min-Max)]	3.4 (0.9-7.7)	2.8 (1-9.8)	0.552
Platelet count ($\times10^9$/L) [Median (Min-Max)]	298 (171-428)	301 (183-603)	0.716
CRP (mg/L) [Median (Min-Max)]	43.9 (8.5-152.4)	56.1 (7.9-338)	0.265
ESR (mm/h) [Median (Min-Max)]	21 (15-51)	25.5 (4-89)	0.836

AGA: Appropriate for gestational age, CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate, LGA: Large for gestational age, MEFV: Mediterranean Fever gene, NICU: Neonatal intensive care unit, PFAPA: Periodic fever, aphthous stomatitis, pharyngitis, and adenitis, SGA: Small for gestational age, Min: Minimum, Max: Maximum

Table 2.
Risk factors for febrile seizures

	Univariate analysis			Multivariate analysis		
	OR	95% CI	p	OR	95% CI	p
Age at first attack (month)	0.973	0.943-1.004	0.083	0.984	0.945-1.025	0.443
Age at diagnosis (month)	0.978	0.954-1.003	0.078	0.990	0.958-1.023	0.541
Family history of febrile seizures	0.218	0.038-1.237	0.085	0.314	0.048-2.033	0.224
Family history of periodic fever	1.143	0.372-3.512	0.816	1.282	0.365-4.500	0.698
Family history of tonsillectomy	0.721	0.245-2.123	0.552	0.630	0.190-2.091	0.450

CI: Confidence interval, OR: Odds ratio

Conclusion

The frequency of FS was higher in children with PFAPA than in the general pediatric population. Although the basic clinical phenotype of PFAPA appears largely independent of the presence of FS, patients with a history of FS are diagnosed at an earlier age, suggesting that these individuals may have been recognized clinically earlier. Our findings emphasize the need to consider neurological outcomes in the management of PFAPA and suggest that future prospective research is required to better clarify the association between autoinflammatory mechanisms and susceptibility to FS.

Ethics

Ethics Committee Approval: This study was approved by the İstanbul Medipol University Non-Interventional Clinical Research Ethics Committee (approval number: 725, date: 18.07.2024) and conducted in accordance with the ethical principles of the Declaration of Helsinki.

Informed Consent: Because the study was designed retrospectively no written informed consent form was obtained from the patients.

Footnotes

Author Contributions: Küçük E: Concept, Design, Data Collection or Processing, Analysis or Interpretation, Literature Search, Writing; Yabancı Erten ES: Data Collection or Processing, Literature Search; Özdemir UF: Data Collection or Processing, Literature Search; Özen Balcı M: Data Collection or Processing, Literature Search; Dursun HK: Data Collection or Processing, Literature Search; Dizman EN: Data Collection or Processing, Literature Search; Aydın Z: Data Collection or Processing, Literature Search; Koru L: Data Collection or Processing, Literature Search; Kaya F:, Öztürk K: Concept, Design, Analysis or Interpretation, Literature Search, Haslak F: Concept, Design, Analysis or Interpretation, Literature Search.

Conflict of Interest: The authors declare no conflicts of interest.

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