

Evaluation of children with immune thrombocytopenia

¹Ceren TÜRKÖZKAN İBİŞ

²Sema YILDIRIM

³Aylin CANBOLAT AYHAN

⁴Hüsnü Fahri OVALI

¹Department of Pediatrics, Kartal Dr. Lutfi Kırdar City Hospital, Istanbul, Turkey

²Department of Pediatrics, Goztepe Prof. Dr. Süleyman Yalçın City Hospital, Istanbul, Turkey

³Division of Hematology and Oncology, Department of Pediatrics, Istanbul Medeniyet University, Istanbul, Turkey

⁴Division of Neonatology, Department of Pediatrics, Istanbul Medeniyet University, Istanbul, Turkey

ORCID ID

CTİ : 0000-0003-2563-2429

SY : 0000-0001-7311-519X

ACA : 0000-0001-6173-2350

HFO : 0000-0002-9717-313X



ABSTRACT

Objective: Immune thrombocytopenia (ITP) is an autoimmune disease. We aimed to examine the demographic, clinical, and laboratory features of ITP patients, identify the etiological factors, evaluate and compare treatment options, and analyze the effects of these parameters on the course of the disease.

Material and Methods: This retrospective study was conducted by reviewing the medical records of all patients under 18 years of age diagnosed with ITP at the Pediatric Hematology Department of a tertiary healthcare institution between January 2015 and December 2022. A total of 154 patients were divided into three groups according to age, initial thrombocyte count, and response to treatment. All groups were compared in terms of the course of the disease.

Results: The mean age at diagnosis was 5±4.1 years. The most common findings at referral were ecchymosis (n=84; 54.5%) and petechiae (n=67; 43.5%). Sixty-two patients (40.3%) had an infectious disease prior to being diagnosed with ITP. The presence of triggering factors was significantly higher in the acute ITP group (p=0.031). Chronic disease was significantly more common in children aged 10 to 18 years (p<0.001). A 'wait and watch' strategy was followed for 40 patients, and these patients tended to develop a persistent (40%) or chronic (52.9%) course of disease (p<0.001). The rate of chronicity (70.6%) was higher in patients who received corticosteroids (p<0.001). The remission time was shorter among patients who received only IVIG therapy (p<0.001). Seventeen (11%) of the patients required second-line therapy.

Conclusion: ITP is a benign disease with a very low mortality rate and tends to resolve spontaneously.

Keywords: Age, children, immune thrombocytopenia prognosis, treatment.

This article was presented orally at the 5th Congress of the Pediatric Association of the Balkan in November 2023, in İstanbul, Turkey.

Cite this article as: Türközkcan İbiş C, Yıldırım S, Canbolat Ayhan A, Ovalı HF. Evaluation of children with immune thrombocytopenia. Zeynep Kamil Med J 2025;56(2):90–97.

Received: November 15, 2024

Revised: January 23, 2025

Accepted: January 31, 2025

Online: May 23, 2025

Correspondence: Sema YILDIRIM, MD. Göztepe Prof. Dr. Süleyman Yalçın Şehir Hastanesi, Çocuk Sağlığı ve Hastalıkları Kliniği, İstanbul, Türkiye.

Tel: +90 505 474 13 03 **e-mail:** yldrsm@gmail.com

Zeynep Kamil Medical Journal published by Kare Publishing. Zeynep Kamil Tıp Dergisi, Kare Yayıncılık tarafından basılmıştır.

OPEN ACCESS This is an open access article under the CC BY-NC license (<http://creativecommons.org/licenses/by-nc/4.0/>).



INTRODUCTION

Immune thrombocytopenia (ITP) is an autoimmune disorder in which antibody-sensitized thrombocytes are destroyed in the reticuloendothelial system. Thrombocyte count is below $100 \times 10^9/L$.

[1] It is the most common cause of thrombocytopenia among the pediatric population.[2] ITP is mostly seen in children aged two to five years. Its incidence is reported as 2–5/100,000.[3] Etiologically, it is classified as primary and secondary ITP. Primary ITP occurs idiopathically, while secondary ITP is caused by infections, medications, or vaccination. Another classification is based on the duration of the disease: acute disease resolves within three months; persistent disease lasts between three–twelve months; and chronic disease lasts longer than one year.[4]

In the pediatric population, ITP is generally a self-limited and benign disorder.[5] Symptoms are generally mild, and it is often asymptomatic. The most common symptoms are petechiae, purpura, ecchymosis, and mild mucosal bleeding such as epistaxis or gingival bleeding. Mortal bleeding is quite rare, and intracranial hemorrhage incidence is under 1%. If thrombocyte count is below $10 \times 10^9/L$, a medical history of anti-thrombocyte or anticoagulant agent intake or the presence of any physical trauma increases the risk of major bleeding.[6]

Treatment is symptomatic. The aim of treatment is to increase thrombocyte count to a relatively safe zone. It is recommended to 'wait and watch' if the thrombocyte count is $>20 \times 10^9/L$ or if the patient is asymptomatic or has mild bleeding symptoms.[5] Intravenous immunoglobulins (IVIG) and corticosteroids are the first-line treatment for patients with an increased risk of bleeding or for symptomatic ones. Thrombopoietin receptor agonists (TPO-RA), Rituximab, and immunosuppressant agents are used for chronic or refractory disease. Splenectomy is another option for treatment.[3]

In this study, we aim to examine the demographic, clinical, and laboratory features of patients with ITP, determine the etiological factors, examine and compare treatment options, and analyze the effects of these parameters on the progress of the disease.

MATERIAL AND METHOD

We retrospectively reviewed the medical records of all patients under 18 years old, diagnosed with ITP at the Pediatric Hematology Clinic in a tertiary healthcare institution between January 2015–December 2022. A total of 154 patients were included in this study. Demographic features, medical history of the patient and the family, clinical features, laboratory findings, treatment modalities, response to treatment, and the duration of the disease were recorded.

The patients were divided into three groups according to age (0–2, 2–10, and 10–18 years) and thrombocyte count ($<20 \times 10^9/L$, $20–50 \times 10^9/L$, $>50 \times 10^9/L$). The bleeding symptoms were classified as minor (mucosal bleeding, petechiae, purpura, etc., with no need for blood transfusion) and major (intracranial hemorrhage or other life-threatening bleeding requiring blood transfusion).

According to the response to treatment, the patients were divided into three groups: if thrombocyte count was $>100 \times 10^9/L$ with no bleeding symptoms after treatment, it was considered as complete response; if it was $>30 \times 10^9/L$ with at least a two-fold

increase from baseline and no bleeding symptoms, it was considered as responsive; if it was $<30 \times 10^9/L$ or there was less than a two-fold increase from baseline or bleeding was present, it was considered as non-responsive disease.

According to the course of the disease, patients were grouped as acute, persistent, and chronic (as described above). Patients aged under one month or over 18 years and those who discontinued follow-up visits before 12 months were excluded from the study.

This study was approved by the local ethics committee (No. 2022/0721, dated 17.12.2022) before the study began and was conducted in accordance with the principles set forth in the Helsinki Declaration.

Statistical Analysis

Statistical analysis was performed using IBM SPSS 23.0 (IBM Corp., Armonk, NY) package program. Number (n) and percentage (%) were used for descriptive categorical variables. Mean±standard deviation (SD) was used for continuous variables when the normal distribution assumption was met, and median values were used when it was not. The normality assumption was checked using the Kolmogorov–Smirnov test.

Fisher's Exact Test, Pearson chi-square test, and Yates and Bonferroni corrections were used to analyze the relationships between categorical variables. The Kruskal–Wallis H test and post hoc Bonferroni correction were used for non-parametric comparisons of continuous variables among more than two independent groups.

Univariate and multivariate logistic regression analyses were performed to determine the independent risk factors associated with dependent variables, and variables with $p < 0.05$ in univariate analyses were included in the multivariate model. Obtained results are presented with odds ratio (OR) and 95% confidence intervals. $p < 0.05$ values were considered statistically significant.

RESULTS

Of the 154 ITP patients, 79 (51.3%) were girls. The mean age was 5 ± 4.1 years, and the median age was 4.3 years (1 month–17.8 years). Forty-eight (31.1%) of the patients were under two years of age, 82 (56.4%) were between two and ten years, and 14 (12.3%) were over ten years of age.

Admissions to the hospital occurred mostly during winter ($n=54$; 35.1%), particularly in January ($n=24$; 15.6%).

Etiologically, 62 (40.3%) patients had an infectious disease prior to ITP. Upper respiratory tract infections were the most common ($n=38$; 57.5%) among them.

Thirty (19.5%) of the patients were asymptomatic. The most common symptoms were ecchymosis ($n=84$; 54.5%) and petechiae ($n=67$; 43.5%). The demographic and clinical data are shown in Table 1.

The mean thrombocyte count was $18.5 \pm 19.7 \times 10^9/L$ (range: $1–87 \times 10^9/L$), and the mean MPV was 10.4 ± 1.9 fL (range: 6–15.7). Viral serology or PCR tests were performed for 116 patients. The most detected agents were parvovirus ($n=6$; 5.2%), COVID-19 ($n=5$; 7.5%), and EBV ($n=4$; 3.4%). Laboratory data are shown in Table 2.

Table 1: Demographic and clinical characteristics of the children with ITP

Variable	n (%)
Gender	
Female	79 (51.3)
Male	75 (48.7)
Age	
0–2 years	48 (31.1)
2–10 years	82 (56.4)
10–18 years	14 (12.3)
Season	
Winter	54 (35.1)
Spring	30 (19.5)
Summer	39 (25.3)
Fall	31 (20.1)
Presence of autoimmune disease	4 (2.6)
Family history of ITP	4 (2.6)
Positive viral tests	27 (28.4)
Positive autoimmune markers	17 (23.1)
Vaccination in previous 2 week	6 (3.9)
Infections in previous 2 weeks	62 (40.3)
Type of infection	
Acute upper tract infection	38 (57.5)
Viremia	9 (13.6)
Acute gastroenteritis	8 (12.1)
Acute lower tract infections	4 (6)
Urinary tract infections	2 (3)
Otitis media	2 (3)
Tonsillitis	1 (1.5)
Acute appendicitis	1 (1.5)
Clinical findings	
No symptoms	30 (19.5)
Ecchymosis	84 (54.5)
Petechia	67 (43.5)
Wet purpura	13 (8.4)
Purpura	5 (3.2)
Gingival bleeding	3 (1.9)
Hematochezia	2 (1.3)
ITP: Immune thrombocytopenia.	

A 'wait and watch' strategy was followed for 40 patients, with a mean duration of 12.3 ± 4.1 days (range: 8–51). Twenty-one (13.6%) of them did not receive any medical treatment but were observed throughout the entire course. Thrombocyte counts of the remaining 19 patients did not increase as expected; thus, 8 (5.1%) received

Table 2: Laboratory finding of the children with ITP

Variable	Median (Min–Max)	Mean \pm SD
Thrombocyte count ($\times 10^3/L$)	11 (1–87)	18.578 \pm 19.708
MPV (fL)	10.7 (6–15.7)	10.4 \pm 1.9
PDW (fL)	16.3 (12.7–24.3)	16.7 \pm 2
Hemoglobin (g/dL)	12 (7.9–16.7)	11.9 \pm 1.3
White blood cells ($\times 10^3/L$)	9.1 (2.230–24.8)	9.893 \pm 3.754
Eosinophils (mm^3)	200 (0–910)	252 \pm 214
CRP (mg/dl)	0.2 (0–62.9)	4.7 \pm 10.9
Sedimentation rate (mm/h)	14 (2–72)	19.8 \pm 18.7
Ferritin (ml/ng)	38 (1.4–852)	74 \pm 120
SD: Standard deviation; ITP: Immune thrombocytopenia; MPV: Mean platelet volume; PDW: Platelet distribution width; CRP: C reactive protein.		

IVIg therapy, 1 (0.6%) received corticosteroids, and 10 (6.5%) received both corticosteroids and IVIg therapy.

Seventy-three (47.4%) patients received solely IVIg therapy as initial treatment. Three (1.9%) received only corticosteroids. Thirty (19.4%) patients received both IVIg and corticosteroids. One patient (0.6%) received IVIg, corticosteroids, and anti-D Ig altogether. Five (3.2%) patients were regarded as steroid-resistant. Seventeen (11%) patients received second-line therapy, and eltrombopag was the most frequently used agent (94%). One patient was resistant to all medical agents and therefore underwent splenectomy.

No complications developed in 143 (95.3%) patients. Five (3.3%) had hematuria, 1 (0.6%) had hematochezia, 1 (0.6%) had intracranial hemorrhage, and 1 (0.6%) had intraocular hemorrhage during the disease course.

As for the prognosis, 89 (60.1%) patients had acute, 25 (16.9%) had persistent, and 23 (34.0%) had chronic ITP. The mean age of acute ITP patients was 3.4 ± 2.8 years, and that of chronic patients was 7.5 ± 4.5 years. Among age groups, acute disease was significantly higher in patients under two years of age, and chronic disease was significantly higher in patients aged 10–18 years ($p < 0.001$) (Table 3).

The presence of trigger factors such as infections, medications, and vaccinations was significantly higher in the acute ITP group ($p = 0.031$). Additionally, the rate of symptomatic patients was higher in the acute ITP group compared to the persistent group ($p = 0.007$). While petechiae were more commonly seen among acute patients than chronic ones ($p = 0.030$), ecchymosis was more frequent in the chronic group than in the persistent group ($p = 0.029$) (Table 3).

A platelet count of $< 20 \times 10^9/L$ at the time of diagnosis was observed more frequently in patients diagnosed with acute ITP compared to those with persistent ITP ($p = 0.037$). Laboratory data of the patients according to prognosis are shown in Table 4.

We examined the duration of the disease according to the initial treatment. Patients managed with the 'wait and watch' approach tended to develop a persistent (40.0%) or chronic (52.9%) course ($p < 0.001$). The rate of chronicity in those who received corticosteroids

Table 3: Demographic and clinical data of the patients according to prognosis

	Acute n (%)	Persistent n (%)	Chronic n (%)	p
Age				<0.001
<2 years	40 (44.9) ^a	5 (20) ^{a,b}	3 (8.8) ^b	
2–10 years	46 (51.7) ^a	16 (64) ^a	20 (58.8) ^a	
10–18 years	3 (3.4) ^a	4 (16) ^{a,b}	11 (32.4) ^b	
Gender				0.319
Female	42 (47.2)	14 (56)	21 (61.8)	
Male	47 (52.8)	11 (44)	13 (38.2)	
Season				0.013
Summer	25 (28.1)	2 (8)	11 (32.4)	
Fall	19 (21.3)	3 (12)	7 (20.6)	
Winter	34 (38.2) ^{a,b}	13 (52) ^b	6 (17.6) ^a	
Spring	11 (12.4)	7 (28)	10 (29.4)	
Trigger factors	46 (51.7)	9 (36)	9 (26.5)	0.031
Clinical findings				
No symptoms	11 (12.4) ^a	10 (40) ^b	7 (20.6) ^{a,b}	0.007
Symptomatic	78 (87.6) ^a	15 (60) ^b	27 (79.4) ^{a,b}	
Ecchymosis	52 (58.4) ^{a,b}	8 (32) ^b	22 (64.7) ^a	0.029
Epistaxis	4 (4.5)	3 (12)	1 (2.9)	0.302
Petechia	46 (51.7) ^a	9 (36) ^{a,b}	9 (26.5) ^b	0.030
Purpura	3 (3.4)	0 (0)	1 (2.9)	0.999
Wet purpura	10 (11.2)	0 (0)	2 (5.9)	0.162
Gingival bleeding	1 (1.1)	1 (4)	0 (0)	0.362
Hematochezia	2 (2.2)	0 (0)	0 (0)	0.999

a, b: Indicate statistically significant differences between the groups.

was higher (70.6%) compared to the acute (14.6%) and persistent (32.0%) groups ($p<0.001$). IVIG therapy at referral did not result in a statistically significant difference among groups ($p=0.101$).

The mean duration of disease was 3 ± 5 months in the observation group, 3 ± 7 months in the IVIG group, 33 ± 35 months in the corticosteroid group, and 16 ± 18 months in those who received both IVIG and corticosteroids ($p<0.001$). The remission time was shorter among those who received only IVIG therapy compared to those who received only corticosteroids ($p=0.002$) and those who received a combination of IVIG and corticosteroids ($p<0.001$). In addition, among patients who did not receive any therapy, the remission time was shorter compared to those who received corticosteroids ($p=0.030$) and those who received both IVIG and corticosteroids ($p=0.005$).

We examined treatment responses for the initial therapies. Eleven (13.9%) of 79 patients who received only IVIG therapy were non-responsive, 27 (34.2%) were completely responsive, and 41 (51.9%) were responsive. One (25.0%) of four patients who received only corticosteroid therapy was non-responsive, 1 (25.0%) was completely responsive, and 2 (50.0%) were responsive. Among 48 patients who received both IVIG and corticosteroids, 10 (20.8%) were

completely responsive, 25 (52.1%) were responsive, and 13 (27.1%) were non-responsive. According to treatment responses, there was no statistically significant difference among IVIG, corticosteroids, and combination therapies ($p=0.295$) (Table 5).

Older age, absence of triggering factors, and corticosteroid treatment were associated with chronic disease. The risk factors for chronicity are shown in Table 6.

DISCUSSION

The mean age at referral for ITP is reported as between one and six years.^[7] In a Croatian study, the mean age was reported as 5.7 ± 4.4 years, and in an Argentinian study, it was 4.6 ± 3.8 years.^[8,9] Similarly, in our study, the mean age was 5 ± 4.1 years. Many studies conducted in different centers around the world have shown that the incidence of chronicity increases if the patient is older than ten years at referral.^[7–10] The ASH 2019 guideline states that the spontaneous remission rate under one year of age is 74%, while it is 62% above ten years and 20–45% in the adult population.^[3] In our study, the mean age of patients with acute ITP was 3.4 ± 2.8 years, and for chronic ITP, it was

Table 4: Laboratory data of the patients according to prognosis

	Acute n (%)	Persistent n (%)	Chronic n (%)	p
Thrombocyte count				0.037
<20X10 ⁹ /L	69 (77.5) ^a	13 (52) ^b	20 (58.8) ^{a,b}	
20–50X10 ⁹ /L	15 (16.9) ^a	8 (32) ^a	8 (23.5) ^a	
>50X10 ⁹ /L	5 (5.6) ^a	4 (16) ^a	6 (17.6) ^a	
MPV				0.075
<11 FL	71 (79.8)	16 (64)	30 (88.2)	
>11 FL	18 (20.2)	9 (36)	4 (11.8)	
Hemoglobin				0.192
<12.5 G/DL	66 (74.2)	22 (88)	29 (85.3)	
<12.5 G/DL	23 (25.8)	3 (12)	5 (14.7)	
PDW				0.308
25–65%	40 (44.9)	12 (48)	20 (58.8)	
<25%	12 (13.5)	4 (16)	7 (20.6)	
>65%	37 (41.6)	9 (36)	7 (20.6)	
White blood cells				0.287
4–11 X10 ³	77 (86.5)	23 (92)	33 (97.1)	
>11X10 ³	9 (10.1)	2 (8)	0 (0)	
<4X10 ³	3 (3.4)	0 (0)	1 (2.9)	
Eosinophils				0.959
<500/MCL	74 (83.1)	21 (84)	29 (85.3)	
>500/MCL	15 (16.9)	4 (16)	5 (14.7)	
CRP				0.858
0–5 mg/dl	68 (79.1)	14 (77.8)	25 (83.3)	
>5 mg/dl	18 (20.9)	4 (22.2)	5 (16.7)	
Sedimentation rate				0.730
0-15 mm/h	17 (65.4)	7 (70)	7 (53.8)	
>15 mm/h	9 (34.6)	3 (30)	6 (46.2)	
Ferritin				0.956
20–200 ng/ml	32 (91.4)	12 (85.7)	16 (88.9)	
>200 ng/ml	2 (5.7)	1 (7.1)	1 (5.6)	
<20 ng/ml	1 (2.9)	1 (7.1)	1 (5.6)	

a, b: Indicate statistically significant differences between the groups; MPV: Mean platelet volume; PDW: Platelet distribution width; CRP: C reactive protein.

7.5±4.5 years. Patients diagnosed under two years of age tended to have an acute course, whereas patients aged ten years or older were more likely to develop chronic disease, consistent with the literature.

It is known that infections, vaccination, and some drugs can initiate ITP.^[1] The ASH 2019 guideline emphasizes especially the MMR vaccine and includes many related recommendations.^[3] A previous study conducted in Türkiye showed a significant association between preceding viral infections and disease onset.^[10] Bruin et al.^[11] reported that in the absence of triggering factors, the risk of

chronicity increases three- to six-fold. In our study, 44.2% of patients had at least one triggering factor, the most common of which was upper respiratory tract infection. The presence of at least one trigger was significantly higher in the acute group than in the chronic group. Furthermore, having at least one triggering factor was statistically associated with a decreased risk of chronicity.

The most common presentation of ITP is petechiae and ecchymosis.^[12] Neunert et al.^[13] reported that the most common bleeding symptoms are cutaneous (i.e., petechiae, purpura,

Table 5: Treatment response to initial therapy

Treatment	Treatment response to initial therapy			p
	Non-responsive (n=28) n (%)	Complete response (n=42) n (%)	Responsive (n=83) n (%)	
IVIG	11 (13.9)	27 (34.2)	41 (51.9)	0.295
Corticosteroids	1 (25.0)	1 (25.0)	2 (50.0)	
IVIG+corticosteroids	13 (27.1)	10 (20.8)	25 (52.1)	

IVIG: Intravenous immunoglobulin.

Table 6: Risk factors for chronicity

Chronicity variable	Univariate		Multivariate	
	OR (%95 CI)	p	OR (%95 CI)	p
Age (year)	1.214 (1.100–1.339)	<0.001	1.074 (0.946–1.219)	0.269
Triggering factor	0.386 (0.166–0.900)	0.027	2.473 (0.822–7.439)	0.107
Symptom	0.871 (0.335–2.267)	0.777		
Winter season	0.305 (0.117–0.796)	0.015	2.380 (0.737–7.693)	0.147
Ecchymosis	1.650 (0.746–3.649)	0.216		
Petechia	0.386 (0.166–0.900)	0.027	1.395 (0.434–4.488)	0.577
Viral tests	0.154 (0.019–1.217)	0.076		
Thrombocyte count (<20x10 ⁹ /L)	0.557 (0.252–1.236)	0.150		
Observation	4.982 (2.187–11.349)	<0.001	12.544 (3.005–52.357)	0.001
Steroid	10.629 (4.424–25.538)	<0.001	0.093 (0.006–1.339)	0.081
High dose MP	6.714 (2.909–15.495)	<0.001	0.406 (0.025–6.548)	0.525

CI: Confidence interval; OR: Odds ratio; MP: Methylprednisolone.

ecchymosis), followed by mucosal bleeding (epistaxis). In our study, 19.5% of patients were asymptomatic. Among symptomatic patients, petechiae, ecchymosis, and wet purpura were the most frequently observed findings. Symptomatic presentation was more common in the acute group. In the chronic group, ecchymosis was the predominant symptom, which was statistically significant. Conversely, the presence of petechiae as the initial finding was significantly associated with acute disease. In conclusion, petechiae were correlated with an acute disease course.

A meta-analysis including 12 studies demonstrated that higher initial thrombocyte counts are associated with increased risk of chronicity.^[14] Several studies report that when thrombocyte count is $\geq 20 \times 10^9/L$ at diagnosis, the risk of developing chronic disease is higher.^[9,10,15] In accordance with these findings, we observed that a thrombocyte count $< 20 \times 10^9/L$ at referral was associated with an acute prognosis.

ITP is a self-limiting and benign disease. 'Watch and wait' is a commonly preferred strategy instead of immediate medical therapies. It is reported that early medical treatment does not prevent

complications or chronicity.^[3] Güngör et al.^[10] followed the 'wait and watch' strategy in 11.8% of their study population and found that the incidence of chronicity was higher compared to the medically treated population. Roganović et al.^[6] applied the same strategy to 77% of their study group and found no significant difference in recovery time compared to the medically treated population. In our study, 13.2% of patients did not receive any medical therapy, and this had no significant effect on chronicity.

It is well known that none of the first-line therapies for ITP have an advantage over the others. However, the use of corticosteroids is recommended as a first-line treatment due to their accessibility and ease of use.^[3] Many studies have shown a rapid increase in thrombocyte counts and a high rate of complete response with IVIG therapy. However, there is no evidence that any treatment choice affects remission time or long-term prognosis.^[10,16,17] In accordance with the literature, our study also showed that IVIG therapy did not prevent chronicity. Among 73 patients who received only IVIG therapy, the incidence of acute disease was significantly higher. Similarly, remission time was significantly shorter in this group.

Corticosteroids are recommended for short-term use because of their potential adverse effects.^[3] It has been shown that there is no difference in prognosis between long-term and short-term corticosteroid administration. High-dose methylprednisolone therapy provides a more rapid platelet response compared to low-dose corticosteroids for 5–7 days; however, no difference in long-term outcomes has been demonstrated between these regimens.^[3,18–21] In our study, we observed a higher rate of chronicity and a longer remission time among patients treated with corticosteroids. No significant differences were found regarding treatment response.

Intracranial hemorrhage and other life-threatening hemorrhages are the most severe complications of ITP. In a review article by Neunert et al.,^[22] the incidence of intracranial hemorrhage was reported to be less than 1%. In the same article, the rate of major bleeding events requiring hospitalization or transfusion was 2.5%. Although bleeding events are often independent of platelet counts, the risk of bleeding increases when the platelet count is $<30 \times 10^9/L$. In a prospective study, no intracranial hemorrhage was observed over a six-year follow-up period.^[13] In our study, the incidence of intracranial hemorrhage was 0.6%. This single case was resistant to all first- and second-line treatments, and the patient's platelet count remained $<1 \times 10^9/L$ for a prolonged period.

The major limitation of our study is its retrospective design. Since our study is single-centered and covers a limited time frame, the findings should not be generalized. More reliable data on factors influencing the etiology of ITP, risk factors for chronicity, and treatment outcomes should be obtained through multicenter, long-term, prospectively designed studies.

CONCLUSION

Since ITP is a benign disease with a very low mortality rate and tends to resolve spontaneously, less invasive approaches should be preferred over aggressive treatment practices. Detailed laboratory evaluation will generally be sufficient to elucidate the underlying etiology.

Statement

Ethics Committee Approval: The Istanbul Medeniyet University Göztepe Training and Research Hospital Clinical Research Ethics Committee granted approval for this study (date: 17.12.2022, number: 2022/0721).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

Use of AI for Writing Assistance: Not declared.

Author Contributions: Concept – SY; Design – SY, CTİ; Supervision – SY; Materials – SY, CTİ, ACA; Data collection and/or processing – SY, CTİ, ACA; Analysis and/or interpretation – CTİ, SY; Literature search – CTİ, SY; Writing – CTİ, SY; Critical review – SY, ACA, HFO.

Peer-review: Externally peer-reviewed.

REFERENCES

1. Nugent DJ. Immune thrombocytopenic purpura of childhood. *Hematology Am Soc Hematol Educ Program* 2006;97–103.
2. Singh G, Bansal D, Wright NAM. Immune thrombocytopenia in children: Consensus and controversies. *Indian J Pediatr* 2020;87:150–7.
3. Neunert C, Terrell DR, Arnold DM, Buchanan G, Cines DB, Cooper N, et al. American Society of Hematology 2019 guidelines for immune thrombocytopenia. *Blood Adv* 2019;3:3829–66. Erratum in: *Blood Adv* 2020;4:252.
4. Rodeghiero F, Stasi R, Gernsheimer T, Michel M, Provan D, Arnold DM, et al. Standardization of terminology, definitions and outcome criteria in immune thrombocytopenic purpura of adults and children: Report from an international working group. *Blood* 2009;113:2386–93.
5. Blanchette V, Bolton-Maggs P. Childhood immune thrombocytopenic purpura: Diagnosis and management. *Pediatr Clin North Am* 2008;55:393–420.
6. Evim MS, Baytan B, Güneş AM. Childhood immune thrombocytopenia: Long-term follow-up data evaluated by the criteria of the international working group on immune thrombocytopenic purpura. *Turk J Haematol* 2014;31:32–9.
7. Kühne T. Diagnosis and management of immune thrombocytopenia in childhood. *Hamostaseologie* 2017;37:36–44.
8. Roganovic J, Letica-Crepulja M. Idiopathic thrombocytopenic purpura: A 15-year natural history study at the Children's Hospital Rijeka, Croatia. *Pediatr Blood Cancer* 2006;47:662–4.
9. Donato H, Picón A, Martinez M, Rapetti MC, Rosso A, Gomez S, et al. Demographic data, natural history, and prognostic factors of idiopathic thrombocytopenic purpura in children: A multicentered study from Argentina. *Pediatr Blood Cancer* 2009;52:491–6.
10. Güngör T, Arman Bilir Ö, Koşan Çulha V, Güngör A, Kara A, Azık FM, et al. Retrospective evaluation of children with immune thrombocytopenic purpura and factors contributing to chronicity. *Pediatr Neonatol* 2019;60:411–6.
11. Bruin M, Bierings M, Uiterwaal C, Révész T, Bode L, Wiesman ME, et al. Platelet count, previous infection and FCGR2B genotype predict development of chronic disease in newly diagnosed idiopathic thrombocytopenia in childhood: Results of a prospective study. *Br J Haematol* 2004;127:561–7.
12. Koçak U, Aral YZ, Kaya Z, Oztürk G, Gürsel T. Evaluation of clinical characteristics, diagnosis and management in childhood immune thrombocytopenic purpura: A single center's experience. *Turk J Pediatr* 2007;49:250–5.
13. Neunert CE, Buchanan GR, Imbach P, Bolton-Maggs PH, Bennett CM, Neufeld E, et al. Bleeding manifestations and management of children with persistent and chronic immune thrombocytopenia: Data from the Intercontinental Cooperative ITP Study Group (ICIS). *Blood* 2013;121:4457–62.
14. Heitink-Pollé KM, Nijsten J, Boonacker CW, de Haas M, Bruin MC. Clinical and laboratory predictors of chronic immune thrombocytopenia in children: A systematic review and meta-analysis. *Blood* 2014;124:3295–307.
15. Kühne T, Berchtold W, Michaels LA, Wu R, Donato H, Espina B, et al. Newly diagnosed immune thrombocytopenia in children and adults: A comparative prospective observational registry of the Intercontinental Cooperative Immune Thrombocytopenia Study Group. *Haematologica* 2011;96:1831–7.

16. Erduran E, Aslan Y, Gedik Y, Orhan F. A randomized and comparative study of intravenous immunoglobulin and mega dose methylprednisolone treatments in children with acute idiopathic thrombocytopenic purpura. *Turk J Pediatr* 2003;45:295–300.
17. Shirahata A, Fujisawa K, Ishii E, Ohta S, Sako M, Takahashi Y, et al. A nationwide survey of newly diagnosed childhood idiopathic thrombocytopenic purpura in Japan. *J Pediatr Hematol Oncol* 2009;31:27–32.
18. Provan D, Arnold DM, Bussell JB, Chong BH, Cooper N, Gernsheimer T, et al. Updated international consensus report on the investigation and management of primary immune thrombocytopenia. *Blood Adv* 2019;3:3780–817.
19. Monteagudo E, Astigarraga I, Cervera Á, Dasí MA, Sastre A, Berrueto R, et al. Protocol for the study and treatment of primary immune thrombocytopenia: ITP-2018. *An Pediatr (Engl Ed)* 2019;91:127.
20. De Mattia D, Del Principe D, Del Vecchio GC, Jankovic M, Arrighini A, Giordano P, et al. Acute childhood idiopathic thrombocytopenic purpura: AIEOP consensus guidelines for diagnosis and treatment. *Haematologica* 2000;85:420–4.
21. British Committee for Standards in Haematology General Haematology Task Force. Guidelines for the investigation and management of idiopathic thrombocytopenic purpura in adults, children and in pregnancy. *Br J Haematol* 2003;120:574–96.
22. Neunert C, Noroozi N, Norman G, Buchanan GR, Goy J, Nazi I, et al. Severe bleeding events in adults and children with primary immune thrombocytopenia: A systematic review. *J Thromb Haemost* 2015;13:457–64.