ORIGINAL ARTICLE

Presentation and Management of Primary Immune Thrombocytopenia in Children at the King Abdulaziz University Hospital, Jeddah, Saudi Arabia: A Retrospective Study

Nadia M. Fida[1], MD, Enas A. Hamed[2], MD, Soad M. Jaber[1], MD, Salwa A. Alnajjar[3], MD, Dhuha Alamawi[1], MD, Fayza I. Alsiny[1], MD, and Basma Alharthy[4], MD

[1]Department of Pediatric, [3]Department of Hematology, [4]Department of Pharmacology, Faculty of Medicine, King Abdulaziz University, Jeddah, Saudi Arabia
[2]Department of Physiology, Faculty of Medicine, Assiut University, Assiut, Egypt

Correspondence
Prof. Nadia M. Fida
Department of Pediatrics
Faculty of Medicine
King Abdulaziz University
P.O. Box 80215, Jeddah 21589
Kingdom of Saudi Arabia
e.M: nadiafida2@gmail.com

Submission: 16 Aug 2020
Accepted: 16 Dec 2020

Citation
Fida NM, Hamed EA, Jaber SM, Alnajjar SA, Alamawi D, Alsiny FI, and Alharthy B. Presentation and management of primary immune thrombocytopenia in children at the King Abdulaziz University Hospital, Jeddah, Saudi Arabia: A retrospective study. JKAU Med Sci 2021; 28(1): 1-9. DOI: 10.4197/Med.28-1.2

Abstract

Immune thrombocytopenia is common bleeding disease in pediatrics caused by low antiplatelet antibodies. Its treatment is controversial. This study aims to identify patterns of newly diagnosed immune thrombocytopenia presentation and treatment for children and adolescents admitted to Pediatric Department at King Abdulaziz University Hospital over three-year period. All patients newly diagnosed with immune thrombocytopenia in pediatric clinic enrolled. Data from patients' files collected and analyzed with respect to demographics, medical history, comorbidity, presentation, bleeding incidences, laboratory investigations, and therapies. Males outnumbered females (56.8% versus 43.2%). Of 44 patients enrolled, 4 had family history of thrombocytopenia and 21 had previous viral infections. Comorbidities reported in seven patients and five were anemic. Bleeding degree was mild in 36 and moderate in eight patients. Disease manifestations were bruising and petechiae (n=30), bleeding gums (n=9), and epistaxis (n=9). Bone marrow aspirations carried out in 25 patients. Treatment modalities included observation (n=5), intravenous immunoglobulin (n=38), corticosteroid (n=11), and anti-D (n=1). In conclusion, majority of children diagnosed with primary immune thrombocytopenia in study received unnecessary intravenous immunoglobulin. Following international guidelines in management of this disease would likely reduce admission rate, treatment costs, and patient exposure to adverse therapy effects.

Keywords

Acute immune thrombocytopenia; Pediatric patients; Treatment management; International guidelines

Introduction

Immune thrombocytopenia (ITP) is a common bleeding disease in pediatrics, with a reported incidence rate of about four persons in 100,000/year[1]. It is caused by the antiplatelet antibodies, where the platelets are damaged by the reticuloendothelial system that results in reduced platelet survival[2,3]. The International Working Group Consensus categorizes ITP into three classes: 1) newly diagnosed ITP cases: those within the first three months of presentation; 2) persistent cases: those that persist from 3 to 12 months; and 3) chronic cases: those that persist for 12 months or more and that are characterized by platelet numbers below 100,000/µL[2]. Newly diagnosed and persistent cases account for about 80% of cases reported in the literature, while chronic cases make up about 20% of ITP cases in children[2,4].

Immune thrombocytopenia most frequently appears after a mild viral infection, with acute onset of purpura and bruising in an otherwise healthy child. In 70 to 90% of such cases in the literature, the disease resolved itself without treatment and with favorable prognosis within six months, and major bleeding complications were exceptional[1,4,5]. The clinical presentation of this disease is characterized by the presence of isolated thrombocytopenia, with platelet counts below 100 x 10^9/L in the absence of other clinical diseases that could cause the decline in platelets[2,6].

The management of primary ITP includes diagnostic investigations, but its treatment and follow-up management strategies are controversial[6]. Most affected children are treated as outpatients, particularly in cases with no bleeding incidences. Inpatient treatment is considered appropriate for patients with platelets below 20,000/µL and with dangerous bleeding or mucous membrane bleeding, or if the child is inaccessible or non-compliant[1,7]. Treatment options include performing splenectomy and the administering steroids of corticosteroids and blood products. These blood products derive from substances that are separated from human plasma through cold ethanol fractionation, such as polyclonal intravenous immunoglobulin (IVIG)[1,2,4] and anti-D immune globulin. IVIG contains all of the five mammalian antibodies (IgA, IgD, IgE, IgG, and IgM), with IgG as the major component. Anti-D immunoglobulin holds antibodies that target D antigen; it becomes potent in patients who bear this antigen in their blood cells, which is known as the Rh-positive blood type[1,4,7].

The aim of this retrospective research was to review the management of patients diagnosed with primary ITP in the Pediatric Department of King Abdulaziz University Hospital over a recent three-year period.

Patients and Methods

Patients

Forty-four patients with primary ITP, 25 males and 19 females, aged 1-16 years, were treated in the outpatient clinics of the Pediatric Department of King Abdulaziz University Hospital from December 2012 to January 2016, and all were included in this study. The institutional ethics committee of the Hospital approved the proposal for this research. Inclusion criteria in this study were age from 1-16 years old and diagnosed with ITP. Excluded from the study were patients with thrombocytopenia due to drugs or systemic diseases and patients with HIV infections accompanied by thrombocytopenia.

Methods

The following data from the patients' files were collected and analyzed for this study: demographic background, date of diagnosis, medical history (prior infectious illnesses), the presence of associated comorbidity, symptoms and signs of the disease at time of presentation, incidences of bleeding (with scores based on the Bolton-Maggs and Moon criteria), laboratory investigations undertaken, and prescribed therapies. The severity of bleeding using the Bolton-Maggs and Moon scale[8,13] was recorded as 1) asymptomatic: no symptoms observed; 2) mild: bruising and petechiae, sometimes with slight nose bleeding; little or no interruption to daily activity; 3) moderate: more severe skin disorders, with some mucosal lesions; bleeding per nose and menorrhagia; 4) severe: bleeding disorders (melena, epistaxis, and/or menorrhagia) that require hospitalization and/or transfusion of blood; symptoms markedly interrupt daily activity. In our hospital, during the first presentation, all patients were examined by the pediatrician or the hematologist. Patients were examined for blood cell counts, peripheral blood smear, Epstein-Barr virus, Cytomegalovirus, Parvovirus B19, *Helicobacter pylori*, hepatitis C, Human immunodeficiency virus (HIV), and autoimmune antibodies (antinuclear antibody, anti-DNA antibody, and platelet-associated antibodies). All patients with atypical symptoms and signs, such as lymphadenopathy or hepatosplenomegaly,

hypoplasia/aplasia of many cell lines, or those patients under steroid therapy who needed aspiration of bone marrow, excluded.

Statistical Analysis

IBM SPSS Statistics for Windows, Version 20 (IBM Corp., Armonk, NY USA) was used in the data processing. The statistical results are presented as mean ± standard deviation for some parameters, such as the patients' ages, platelet counts, hemoglobin level, and leukocyte counts and differential. The categorical variables between the groups were compared using the results from chi-squared tests, where a probability (*P*) level of less than 0.05 was considered significant.

Results

The number of male patients was insignificantly higher than the number of female patients, *i.e.,* 57.0% (n=25) versus 43.0% (n=19), with $P = 0.451$, and the male to female ratio is 1.32. Based on their family histories, 91.0% (n=40) reported that their families had previous bouts with thrombocytopenia. Histories of previous viral infections were found in 48.0% of patients (n=21). Comorbidity was also determined in seven patients (15.9%), where the associated conditions were mainly anemia, which was diagnosed in 11.4% of patients (n=5), followed by hyperactive airway disease in 4.5% (n=2) (see Table 1).

The degree of bleeding experienced by the patients was described as mild in 82.0% (n=36) and moderate in 18.0% patients (n=8). The symptoms and signs of the disease were mainly bruising and petechiae in 68.0% (n=30), bleeding gums (n=9, 21.0%), epistaxis (n=9, 21.0%), hematemesis and melena (n=2, 5.0%), menorrhagia (n=1, 2.0%), and hematuria (n=1, 2.0%) (see Table 2).

The patients' platelet counts ranged from 1 to 98 x 10^9/L. Their hemoglobin levels were within the range of 5.7 to 17.0 gram/dL. Their leukocytic counts were measured to be between 3.30 and 27.06 x 10^3/L. Bone marrow aspiration was performed in 57.0% (n=25) to confirm diagnosis by exclusion of other causes. Antinuclear antibodies were measured in 20 patients, among whom they were detected in 35.0% (n=7) but found absent in 65.0% (n=13). The presence of *Helicobacter pylori* was determined in three patients by salivary anti-*H. pylori* IgG immune response, and only one case (33.33%) out of the three was positive. Antiphospholipid antibodies (n=5), platelet-associated

Table 2. Frequency profiles of the clinical presentation in patients (n = 44)*

Clinical Presentation	Frequency
Severity of bleeding	
Mild	36 (82.00%)
Moderate	8 (18.00%)
Symptoms and signs	
Bruising and petechiae	30 (68.00%)
Bleeding gum	9 (21.00%)
Epistaxis	9 (21.00%)
Hematemesis and melena	2 (5.00%)
Menorrhagia	1 (2.00%)
Hematuria	1 (2.00%)

**The values are expressed as number of patients (percentage of total).*

Table 1. Demographic profile of patients with primary immune thrombocytopenia (n = 44)*

Parameters	Values	Significance
Age (years)	6.33 ± 4.39 (1.00–16.00)	-
Gender		0.451
Male	25 (57.00%)	
Female	19 (43.00%)	
Male to female ratio	1.32	
Family history with thrombocytopenia		0.0001
Yes	4 (9.00%)	
No	40 (91.00%)	
History of previous viral infection		0.763
Yes	21 (48.00%)	
No	23 (52.00%)	
Comorbidities		0.0001
None	37 (84.00%)	
Anemia	5 (11.40%)	
Hyperactive airway disease	2 (4.50%)	

**The ages of the patients are expressed as a mean ± standard deviation, and the other parameters are expressed as the number of patients (percentage of total); level of significance was calculated using a Pearson's chi-squared test.*

Table 3. Laboratory tests conducted for the patients (n = 44)*

Parameters	Values	Significance
Platelet count (10^9/L)	18.11 ± 24.16 (1.00–98.00)	-
Hemoglobin (gram/dL)	11.14 ± 2.10 (5.70–17.00)	-
Leukocyte count (10^3/L)	8.85 ± 3.79 (3.30–27.06)	-
Bone marrow aspiration		0.366
Done	25 (57.00%)	
Not done	19 (43.00%)	
Antiphospholipid antibodies		0.0001
Not done	39 (89.00%)	
Done	5 (11.00%)	
Negative†	5 (100.00%)	
Antinuclear antibodies		0.006
Not done	24 (54.00%)	
Done	20 (46.00%)	
Positive†	7 (35.00%)	
Negative†	13 (65.00%)	
Platelet-associated antibodies		0.0001
Not done	41 (93.00%)	
Done	3 (7.00%)	
Negative†	3 (100.00%)	
Hepatitis C		0.366
Not done	19 (43.00%)	
Done	25 (67.00%)	
Negative†	25 (100.00%)	
Helicobacter pylori		0.0001
Not done	41 (93.00%)	
Done	3 (7.00%)	
Positive†	1 (33.33%)	
Negative†	2 (66.67%)	

**The first three parameters are expressed as mean ± standard deviation; other parameters are expressed as number of patients (percent of total or †percent of total number taking the test); level of significance was calculated using a Pearson's chi-squared test.*

antibodies (n=3), and hepatitis C (n=25) were found to be negative in investigated cases (see Table 3).

Treatment modalities administered to the patients with primary ITP included the following: observation in the case of five patients (11.20%), IVIG treatments for 38 patients (86.00%), corticosteroid treatments for 11 patients (33.00%), and anti-D administration for one patient (2.00%) (See Table 4).

Table 4. Treatment modalities of patients with primary immune thrombocytopenia (n=44)*

Treatment Modalities	Values	Significance
Observations	5 (11.20%)	-
Corticosteroids		0.0001
Received	11 (32.00%)	
Did not receive	33 (68.00%)	
Intravenous immunoglobulin		0.0001
Received	38 (86.00%)	
Did not receive	6 (14.00%)	
Anti-D		0.0001
Received	1 (2.00%)	
Did not receive	43 (98.00%)	

**The values are the actual number of patients (percent of total); level of significance was calculated using a Pearson's chi-squared test.*

Discussion

The mean age of the children and adolescents enrolled in this research was six years. In the research of the Intercontinental Childhood Primary Immune Thrombocytopenia Study Group (ICIS), which involved 2,540 cases, the mean presentation age was five years, 70% of the patients were between the ages of 1 and 10 years old, 10% were infants between 3 and 12 months, and the remaining 20% of cases were between 10 and 16 years old[9].

In our study, the number of male patients with newly diagnosed primary ITP was insignificantly higher than that of the female patients, with a male to female ratio of 1.32:1. In this respect, it has been reported that primary ITP in children affected both males and females in the same degree, but in infancy males were affected more than females[9,10]. Kühne and Imbach reported that in affected infants the female to male ratio was 1:1.7; male predominance was less in older children, and the overall female to male ratio was 1:1.2[9].

Primary ITP in children usually comes with acute purpura or bruising**.** In this study, preceding viral infections occurred in 48.0% of patients, mostly infections of the upper respiratory tract. Ghanima *et al.*[3] reported histories of previous viral diseases in about two-thirds of pediatric cases, mostly upper respiratory tract diseases. The timing from the incidences of previous infection to the emergence of purpura varies from a few days to several weeks, with an interval of about two weeks occurring most frequently. In some children, primary ITP appears after the administration of the measles, mumps, rubella (MMR) vaccine. Measles, mumps, rubella-associated primary ITP happens rarely, i.e., in 1–3 cases/100,000 doses of vaccine[1].

In this study, four cases (9.1%) had a positive family history of thrombocytopenia. Inherited thrombocytopenia are sometimes misdiagnosed as ITP[1,5]. Inherited diseases must be suspected if thrombocytopenia was present in early life, if a positive family history for the same disorder was documented, or if the characteristic manifestations of the disease were found[1,5].

The presenting symptoms and signs in our patients were mostly bruising and petechiae, followed by bleeding gums, epistaxis, hematemesis and melena, menorrhagia, and hematuria. Manifestations of primary ITP vary widely. Many patients present either as asymptomatic or with little bruising, whereas others experience massive bleeding. It has been reported that about 60% of children with primary ITP only presented skin bleeding that included an accompanying purpuric rash, bruising, or petechial – also known as "dry" – purpura. Mucosal bleeding, or "wet purpura," has been reported in about 40% of children with primary ITP. The latter involved bleeding per nose, gingival and buccal bleeding, and, to some extent, menorrhagia, blood in urine, or GIT bleeding[4,5,7,11]. In the study by Mohammad *et al*.[12], petechiae and bruises were the most common clinical findings associated with primary ITP in children. Severe bleeding also occurred in children with platelet counts of less than 10 x 10^9/L[6]. Other than mucocutaneous bleeding, the patients looked well and had no systemic symptoms, such as weight loss, fever, or bone/joint ache. On examination, no significant hepatosplenomegaly or lymphadenopathy was found (if present, another round of diagnosis must be considered). However, small cervical adenopathy was common in young children, and a slightly palpable spleen was observed in 5% to 10% of children with primary ITP[11].

Trials have been conducted to establish scoring systems that capture the extent of bleeding, and the resulting scales can be used to standardize therapy decisions and to record platelet responses. Bolton-Maggs and Moon[13] divided the manifestations of bleeding into four categories: none, mild, moderate, and severe. Buchanan and Adix developed a scoring system that allows for semi-quantitative judgments on the severity of the bleeding, with grading on a 5-point scale that assesses bleeding on the skin, from the nose, from the mouth, and global bleeding. Global scores refer to other areas of bleeding, such as menorrhagia, internal bleeding, and gastrointestinal bleeding, depending on the results of examination and the history of new bleeding during the previous twenty-four hours[13–15]. Based on the Bolton-Maggs and Moon classification[13], 81.80% of our patients showed mild bleeding, while 18.20% showed moderate bleeding. From their histories, no severe bleeding was reported, although there were patients with platelet numbers that were less than 10 x 10^9/L.

For typical patients with childhood primary ITP, a complete blood count with a thorough clinical examination of the peripheral blood smear is needed. In this study, the mean of patients' platelet counts was 18.11 x 10^9/L, with a range of 1.00 to 98.00 x 10^9/L. In most reports, the presenting platelet count was below 30 x 10^9/L, mostly because many patients with mild disorder do not seek medical help. About 80% of patients with ITP were reported to have platelet counts below 20 x 10^9/L and 44% below 10 x 10^9/L[5,14,15,16]. In this study, the mean hemoglobin level of the patients was 11.14 g/dL, with a range from 5.70 to 17.00 g/dL. Other hematologic abnormalities were considered together with the newly diagnosed ITP, e.g., the incidence of anemia in children with significant manifestation of bleeding[5,16] or the presence of atypical lymphocytes in post-infectious patients. The exception to this is mild eosinophilia, which is a common presentation[1].

In this study, platelets associated with antibodies were tested in only three patients (7.0%), and they showed negative results. Measurement of the number of antiphospholipid antibodies was performed in five patients (11.0%) and they also showed negative results. Antinuclear antibodies were detected positive in seven patients (16.0%) and negative in 13 patients (30.0%). Antiplatelet antibodies have been detected in 60–70% of primary ITP reported in the literature[15,17], and there is no evidence to support the need for the routine examination of other autoimmune

antibodies (antiphospholipid, antinuclear, or anti-thyroid), platelet measurements (platelet volume or reticulated platelets), or serum thrombopoietin levels in the assessment of children with suspected primary ITP[16]. The test for antinuclear antibodies (ANA) can be conducted on older children with ITP or on children with chronic diseases[16]. Evidence for the utility of measuring immunoglobulins in all patients to exclude common and variable immune deficiencies is equivocal[1,15].

In this study, bone marrow aspiration was performed on 25 ITP patients (57.0%). Based on the American Society of Hematology (ASH) guidelines, bone marrow aspiration is not needed for children with typical characteristics of primary ITP or for those patients who do not respond to IVIG. Bone marrow aspiration can be performed on patients having atypical clinical or laboratory features at presentation and on those with suspected malignancy or bone marrow failure; bone pain; lymphadenopathy; hepatosplenomegaly; anemia that cannot be explained by blood loss or by abnormally high or low WBC; therapy refractory primary ITP; and on patients with new findings gathered during follow-up sessions that are inconsistent with primary ITP[5,16].

The goal of all treatment strategies for primary ITP in children is to produce a platelet count that is associated with normal hemostasis rather than a "normal" platelet number. However, treatment recommendations for childhood ITP have long been a subject of debate[19], and there is currently considerable variation in the management of treatment of children with typical primary ITP[16]. The United Kingdom has long supported a watch and wait approach, while the American Society of Hematology did not universally adopt treatment guidelines until 2011. In any case, if the platelet count is greater than 10,000/µL; with minimal bleeding signs (*i.e.* fitting the definition of "skin manifestations only," in the form of bruising and petechiae[19]), and if there is a reliable caregiver and the child has not experienced head trauma and has not taken an antiplatelet agent, such as aspirin (acetylsalicylic acid), it is appropriate to monitor the child without pharmacotherapy[2].

In this study, five cases (11.0%) were subjected to observation without therapy. Otherwise, therapeutic agents included glucocorticoids, IVIG, and anti-Rhesus (anti-D immunoglobulin)[17,18]. Eleven patients also received corticosteroid, which was used in this study to treat patients with bleeding in the oral cavity, GIT bleeding, and hematuria. Intravenous immunoglobulin (IVIG) was used to treat primary ITP when there were contraindications to the use of glucocorticoids, such as glucose intolerance, hypertension, or intestinal ulceration, or per the therapist's preference[5,16].

In this study, 38 patients (86.0%) received IVIG. IVIG was used to treat patients with oral cavity bleeding, epistaxis, menorrhagia, and GIT bleeding. Anti-D immunoglobulin, like IVIG, is a pooled blood product licensed for the treatment of patients with primary ITP and with patients who possess a D positive blood group, are non-splenectomies, and have normal hemoglobin levels[2,11,18]. In this study, one patient (2.0%) with menorrhagia received IV anti-D immunoglobulins with a dose of 75 µg/kg, administered once during a 20-minute infusion.

In this study, platelet transfusion was administered to 20 patients (46.0%) subsequent to diagnosis. Transfusion is generally contraindicated except in the case of patients with life-threatening hemorrhages or patients requiring surgical treatment[15].

Reporting on a recent study conducted in Philadelphia, USA, Güngör *et al.*[19] state that significant behavioral changes were observed in connection with the use of medications and the rate of cases under observation without treatment increased. This supports the view that children with primary ITP should not be hospitalized unless they have significant bleeding, and treatment, where provided, should be based on platelet count and severity of symptoms. Platelet transfusion should be given only in cases with significant hemorrhage. There is absence of evidence that its use delayed or mitigated symptoms and is pending further research on the effect of IVIG on manifestation of primary ITP in children

Limitations of the Study

Limitations include the small number of the cases considered in the study. Also, the fact that this was a retrospective study resulted in several cases having to be excluded due to inadequate data.

Conclusions

In comparing the treatment modalities employed in the study to previous research and international guidelines, the study found that IVIG appears to have been dispensed unnecessarily in a number of cases of

children newly diagnosed with acute ITP. Thus, the study supports following international guidelines in terms of preferring a "watch and wait" approach to primary ITP in children in the majority of cases. This finding may help in determining what patients should be admitted for hospitalization, and this is important because where admission and/or treatment is not deemed necessary, costs are reduced, inpatient space can be assigned to more acute patients, and fewer patients are exposed to potentially unjustifiable adverse effects of therapy[20].

Acknowledgement

The authors would like to acknowledge Dr. Salwa Hindawi, Department of Hematology, for reviewing the manuscript, and Ms. Rola Almalki for her assistance with data collection.

Conflict of Interest

The authors declared that there is no conflict of interest that is related to this study and this article.

Disclosure

The authors did not receive any type of commercial support either in the form of compensation or financial support for this case report. The authors have no financial interest in any of the products, devices, or drugs mentioned in this article.

Ethical Approval

The study was approved by the Ethics Committee of the KAUH in Jeddah, Kingdom of Saudi Arabia, also known as the Institutional Review Board of Hospitals.

References

[1] Labarque V, Van Geet C. Clinical practice: immune thrombocytopenia in paediatrics. Eur J Pediatr 2014; 173(2): 163-72.

[2] Bredlau AL, Semple JW, Segel GB. Management of immune thrombocytopenic purpura in children: potential role of novel agents. Paediatr Drugs 2011; 13(4): 213-223. .

[3] Ghanima W, Godeau B, Cines DB, Bussel JB. How I treat immune thrombocytopenia: the choice between splenectomy or a medical therapy as a second-line treatment. Blood 2012; 120(5): 960-969.

[4] McCrae K. Immune thrombocytopenia: no longer 'idiopathic'. Cleve Clin J Med 2011; 78(6): 358-373.

[5] Provan D, Stasi R, Newland AC, Blanchette VS, Bolton-Maggs P, Bussel JB, Chong BH, Cines DB, Gernsheimer TB, Godeau B, Grainger J, Greer I, Hunt BJ, Imbach PA, Lyons G, McMillan R, Rodeghiero F, Sanz MA, Tarantino M, Watson S, Young J, Kuter DJ. International consensus report on the investigation and management of primary immune thrombocytopenia. Blood 2010; 115(2): 168-186.

[6] Kühne T, Imbach P. Management of children and adolescents with primary immune thrombocytopenia: controversies and solutions. Vox Sang 2013; 104(1): 55-66.

[7] Grainger JD, Rees JL, Reeves M, Bolton-Maggs PH. Changing trends in the UK management of childhood ITP. Arch Dis Child 2012; 97(1): 8-11.

[8] Neunert CE, Buchanan GR, Imbach P, Bolton-Maggs PH, Bennett CM, Neufeld E, Vesely SK, Adix L, Blanchette VS, Kühne T; Intercontinental Cooperative ITP Study Group Registry II Participants. Bleeding manifestations and management of children with persistent and chronic immune thrombocytopenia: data from the Intercontinental Cooperative ITP Study Group (ICIS). Blood 2013; 121(22): 4457-4462.

[9] Kühne T, Buchanan GR, Zimmerman S, Michaels LA, Kohan R, Berchtold W, Imbach P; Intercontinental Childhood ITP Study Group; Intercontinental Childhood ITP Study Group. A prospective comparative study of 2540 infants and children with newly diagnosed idiopathic thrombocytopenic purpura (ITP) from the Intercontinental Childhood ITP Study Group. J Pediatr. 2003; 143(5): 605-608.

[10] Choi PY, Gordon JE, Harvey M, Chong BH. Presentation and outcome of idiopathic thrombocytopenic purpura in a single Australian centre. Intern Med J 2012; 42(7): 841-845.

[11] Blanchette V, Bolton-Maggs P. Childhood immune thrombocytopenic purpura: diagnosis and management. Hematol Oncol Clin North Am 2010; 24(1): 249-273. .

[12] Mohammad J, Rahim F, Nayyar J. Frequency of clinical features of idiopathic thrombocytopenic purpura. J Med Sci 2011; 19(3): 126–128.

[13] Bolton-Maggs PH, Moon I. Assessment of UK practice for management of acute childhood idiopathic thrombocytopenic purpura against published guidelines. Lancet 1997; 350(9078): 620-623.

[14] Roganović J. Idiopathic thrombocytopenic purpura in children. Acta Med Acad 2009; 38(1): 21–34.

[15] Roganović J. Immune thrombocytopenia in children. Rad 524 Med Sci 2015; 42: 59–72.

[16] Neunert C, Lim W, Crowther M, Cohen A, Solberg L Jr, Crowther MA; American Society of Hematology. The American Society of Hematology 2011 evidence-based practice guideline for immune thrombocytopenia. Blood 2011; 117(16): 4190-4207.

[17] Kistangari G, McCrae KR. Immune thrombocytopenia. Hematol Oncol Clin North Am 2013; 27(3): 495-520.

[18] Faki Osman ME. Childhood immune thrombocytopenia:

Clinical presentation and management. Sudan J Paediatr 2012; 12(1): 27-39.

[19] Güngör T, Arman Bilir Ö, Koşan Çulha V, Güngör A, Kara A, Azık FM, Yaralı HN. Retrospective evaluation of children with immune thrombocytopenic purpura and factors contributing to chronicity. Pediatr Neonatol 2019; 60(4): 411-416.

[20] Schultz CL, Mitra N, Schapira MM, Lambert MP. Influence of the American Society of Hematology guidelines on the management of newly diagnosed childhood immune thrombocytopenia. JAMA Pediatr 2014; 168(10): e142214.

الاستعلان السريرى و معالجة حالات نقص الصفيحات المناعية الأولية عند الأطفال في مستشفى جامعة الملك عبد العزيز ، جدة ، المملكة العربية السعودية: دراسة مرجعية

نادية فدا[١]، إيناس حامد[٢]، سعاد جابر[١]، سلوى النجار[٣]، ضحى العماوي[١]، فايزة الصيني[١]، بسمة الحارثي[٤]

[١]قسم طب الأطفال، [٢]قسم أمراض الدم، [٣]قسم الصيدلة، [٤]كلية الطب، جامعة الملك عبد العزيز، جدة - المملكة العربية السعودية
[٣]قسم علم وظائف الأعضاء، كلية الطب، جامعة أسيوط، أسيوط، مصر

المستخلص. مرض نقص الصفيحات المناعي شائع في الأطفال بسبب الأجسام المضادة للصفيحات. تشخيص وعلاج نقص الصفيحات المناعي مثير للجدل. هدفت هذه الدراسة إلى التعرف على نمط التعامل مع حالات نقص الصفيحات المناعي المشخصة حديثًا فى الأطفال المترددين على قسم طب الأطفال، جامعة الملك عبد العزيز، جدة ، السعودية على مدار السنوات الثلاث الماضية. تم جمع وتحليل البيانات من ملفات المرضى مثل: خلفيتهم الديموغرافية، وتاريخ تشخيصهم ، وتاريخهم الطبي، ووجود الأمراض المصاحبة، وأعراض المرض، ودرجات النزيف، والاختبارات المعملية، والعلاج. كانت نسبة الذكور (٥٦،٨٠٪) أعلى من الإناث (٤٣،٢٠٪). تم العثور على تاريخ عائلي إيجابي لنقص الصفيحات في ٤ مرضى وتاريخ الإصابة السابقة بالفيروس في ٢١ مريض، أمراض مصاحبة في ٧ مرضى ووجد ٥ مرضى مصابين بالانيميا. كانت درجة النزف قليله في ٨١،٨٠٪ ومعتدلة في ١٨،٢٠٪. وكانت الأعراض والعلامات بشكل رئيسي كدمات ونزيف تحت الجلد (٦٨،٢٠٪) ثم نزيف اللثة (٢٠،٥٠٪) ورعاف (٢٠،٥٠٪). فحص نخاع العظم في ٥٦،٨٠٪. كانت طريقة علاج نقص الصفيحات المناعي هي الملاحظة فى (١١،٢٠٪)، الغلوبولين المناعي الوريدي (٨٦،٤٠٪)، كورتيكوستيرويد (٣٢،٣٠٪) ، مضاد الدى (٢،٣٠٪). تم استنتاج إن معظم الأطفال الذين تم تشخيصهم بنقص الصفيحات المناعي الأساسي يتلقون الأجسام المضادة عن طريق الوريد وقد لايحتاجوه. اتباع الإرشادات الدولية من شأنه أن يقلل من معدل نسبة تنويم المرضى والتكلفة والتعرض للآثار الضارة المحتملة غير المبررة لعلاج نقص الصفيحات المناعي الأساسي.