Original Article

Henoch-Schönlein purpura in children: A cross sectional study

Fatih Karaaslan¹, Betul Gemici Karaaslan¹, Huseyin Dag¹, Soner Sazak¹, Emine Turkkan¹, Bilal Yılmaz¹, Gulsen Kose Midillioglu²

From Department of Pediatrics, ¹University of Health Sciences, Istanbul Okmeydani Training and Research Hospital, ²Şişli Hamidiye Etfal Training and Research Hospital, Istanbul, Turkey

Correspondence to: Dr. Huseyin Dag, Okmeydanı Egitim Ve Araştırma Hastanesi, Pediatri Klinigi Darülaceze cad. No: 27 Şişli/Istanbul, Istanbul, Turkey. E-mail: huseyindag2003@gmail.com

Received - 11 February 2019

Initial Review - 02 March 2019

Accepted - 14 March 2019

ABSTRACT

Background: Henoch-Schönlein purpura (HSP) is a leukocytoclastic vasculitis with small vessel involvement and mainly affects the skin as well as joints, the gastrointestinal system (GIS), kidneys, and, more rarely, other organs. **Objective:** The objective of this study was to evaluate the sociodemographic characteristics, and clinical and laboratory findings of patients diagnosed with HSP. **Materials and Methods:** This was a retrospective study done to find out the sociodemographic data, clinical, laboratory findings, and treatment information of patients diagnosed with HSP and was admitted to the Pediatric Clinic of a tertiary care hospital between January 1, 2008, and August 31, 2013. The data were obtained from the hospital's data processing system. HSP cases were validated according to EULAR/PRINTO/PRES criteria. Mean standard deviation, median, lowest and highest, frequency, and ratio values were used in the descriptive statistics of the data. **Results:** The study included 85 patients between the ages of 2 and 16 years, wherein 49 patients (57.6%) were male and 36 (42.4%) were female. The mean age was 9.9±3.3 years and 53 patients (62.4%) were under 10 years of age. The most common precipitating factor was upper respiratory tract infections. Purpura was the only symptom observed in all the patients and joint involvement was the second most common symptom (60%). GIS involvement with mild nephropathy. The most frequently observed laboratory findings were increased C-reactive protein (47%) and leukocytosis (31%). **Conclusion:** HSP is commonly seen in children and leads to life-threatening complications in a minority of patients. Whole patients with GIS and renal involvement should be examined and monitored to assess the severity of the disease and any complications.

Key words: Children, Complications, Henoch-Schönlein, Purpura

Heberden, in 1801, and its relationship with arthritis was highlighted by Schönlein, in 1837 [1]. HSP is a leukocytoclastic vasculitis with small vessel involvement; it mainly affects the skin as well as joints, the gastrointestinal system (GIS), kidneys, and, more rarely, other organs. Its etiology is not fully known [2] and it is the most widespread reason of non-thrombocytopenic purpura in children. It is a disease that is generally seen in children between the ages of 5 and 15 years. The estimated incidence of the disease in children <17 is 13–20/100.000 [3].

Although the etiology of HSP is unknown, it has been reported that various factors such as infections, vaccines, drugs, foods, and insect bites trigger the disease [4]. The fact that two-thirds of the patients had a history of upper respiratory tract infection (URTI) and the disease was more commonly seen in winter and spring, when URTIs are very frequent, could be incidental, but it is more likely that HSP is linked to infectious diseases. The most important microorganisms are streptococci; in addition, many viral and other bacterial agents have also been reported to precipitating the disease [3]. IgA plays an important role in the pathogenesis of HSP. Studies have shown renal mesenchymal IgA accumulation, increased serum IgA, and the presence of IgA-containing immune complexes in patients with HSP [5].

The most common and distinct sign of the disease is a skin rash in the form of non-thrombocytopenic palpable purpura that appears, especially on lower extremities. Non-thrombocytopenic purpura, arthritis, and abdominal pain in the early phase of the disease constitute the classic triad of symptoms of HSP and facilitate a clinical diagnosis [5]. The skin rash can be accompanied by soft tissue swelling, especially on the scalp, on the tibial aspect of the lower extremities, and on the back of the hands. Less frequently, localized edema on the eyelids, lips, ears, and perineum can be seen especially in younger children [6]. This edema is not associated with proteinuria, but it generally stems from angioedema and does not leave an indented mark when pressed [7]. Joint involvement in HSP manifests as arthralgia or arthritis. Knee, elbow, wrist, and ankle involvement are frequently seen. There is no intra-articular hemorrhage or effusion, nor permanent deformity [8].

GIS involvement generally manifests as abdominal pain. If abdominal pain occurs before the rash, it can be confused with acute abdomen. Acute abdomen can be accompanied by vomiting and blood in stools [8,9]. In GIS involvement, serious life-threatening complications other than bleeding include intussusception and perforation. The diagnosis should always be considered in the case of very severe and long-term pain [8].

Long-term prognosis of HSP is determined by the severity of kidney involvement. The most common sign of kidney involvement is hematuria, which generally develops within the first 4 weeks. Proteinuria can be seen in various degrees of severity, ranging from mild proteinuria to severe proteinuria, as in nephrotic syndrome. Hypertension can generally be seen in the early stages. Kidney function tests are usually normal; however, some patients may develop severe kidney involvement and progressive glomerulonephritis [10].

Neurologic involvement is rarely seen in HSP. Central nervous system damage occurs either due to the direct effect of vasculitis or the indirect effect of systemic inflammation. Severe neurologic involvement is rare in HSP, whereas mild effects have been reported more frequently. In one study, 244 cases were retrospectively screened and it was found that 17 (6.9%) patients experienced convulsions and confusion. Recent studies have reported a much lower frequency of such symptoms [11].

HSP is the most common vasculitis of childhood and can cause serious complications. The aim of this study was to investigate the epidemiologic, clinical, and laboratory characteristics of patients with HSP who were admitted to our clinic and to review the treatment approaches for these patients.

MATERIALS AND METHODS

In this retrospective cross-sectional study, the sociodemographic data, clinical, laboratory findings, and treatment information of the patients hospitalized between January 1, 2008, and August 31, 2013, with HSP in the pediatric clinic of a hospital, were recorded and evaluated.

HSP cases were validated according to EULAR/PRINTO/ PRES criteria [12]. Sociodemographic and epidemiologic characteristics (age, sex, and season), history (previous infections, insect bites, and trauma), complaints at admission, clinical progress, laboratory findings (full blood count, C-reactive protein [CRP], biochemistry, and complete urinalysis) of the patients, treatment approaches, and complications were retrospectively evaluated. Leukocyte count >13,000/mm³ was considered as leukocytosis; hemoglobin count <11 g/dl as anemia; platelet count >450,000/mm³ as thrombocytosis; and CRP >5 was considered to be high.

SPSS 22.0 software was used for the statistical analysis. Mean standard deviation, median, lowest and highest, frequency, and ratio values were used in the descriptive statistics of the data. Fisher's exact test was used to analyze the qualitative independent variables. Statistical significance was construed as p<0.05.

A total of 85 patients with HSP were included in the study, of which, 49 patients (57.6%) were male and 36 (42.4%) were female, with male:female ratio of 1:36. The ages of the patients were between 2 and 16 years, and the mean age was 9.9 ± 3.3 years. 53 patients (62.4%) were under 10 and 32 patients (37.6%) were over 10. Considering the distribution of patients by the season of admission, 32 patients (37.6%) were presented in winter, 22 (25.9%) in autumn, 19 (22.4%) in spring, and 12 (14.1%) in summer. Of the total study patients, 23 (27%) had a history of a potentially triggering factor before the onset of HSP. As a triggering factor, 20 patients (23.5%) had a history of URTI (rhinitis, otitis, tonsillitis, and pharyngitis) and three patients had a history of insect bites.

It was found that all patients had non-thrombocytopenic purpura ranging from petechiae to ecchymosis, and these lesions were localized most frequently on the lower extremities and hips, and less frequently on the upper extremities. Joint involvement was observed in 51 patients (60%). Joint involvement manifested as arthritis in 33 (38.8%) patients and arthralgia in 18 (21.2%) patients.

Gastrointestinal involvement was found in 50 patients (58.8%) and all these 50 patients had abdominal pain and 36 (42.3%) had gastrointestinal bleeding. The bleeding manifested as fecal occult blood in 33 patients (38.8%) and as gross bleeding in three patients. Abdominal ultrasonography (USG) revealed intussusceptions in one patient with severe abdominal pain. It was found that abdominal pain was an early symptom in 15 patients (17.6%) followed by purpuric rash. Renal involvement was detected in 9 patients (10.6%). These patients had mild nephropathy along with microscopic hematuria. None of the patients had proteinuria or severe nephropathy.

The main laboratory findings in the acute phase of the disease were as follows: Mean leukocyte count was 11.5 ± 3.4 ($1000\times/\mu$ l) and 26 patients (30.6%) had leukocytosis. The mean hemoglobin count was 13.3 ± 1.1 g/dl and 10 patients (11.7%) were anemic. The mean platelet count was 364.2 ± 117.9 ($1000\times/\mu$ l) and 19 patients (22.4%) had thrombocytosis. CRP was positive (>8 mg/dl) in 40 patients (47%). The mean urea value was 24.3 ± 8.5 mg/dL, and the mean creatinine value was 0.5 ± 0.2 mg/dL. Creatinine values were within normal limits in all patients, whereas urea was higher than normal in four patients. The CRP-positive group had a significantly higher rate of arthritis as compared to the CRP-negative group (p<0.05). There was no significant difference between CRP positivity and other clinical findings.

The rate of renal involvement with hematuria was significantly higher in the group with leukocytosis in comparison to the group without leukocytosis [Table 1].

It was observed that nonsteroidal anti-inflammatory drugs (ibuprofen, 30 mg/kg/day) were administered to 29 patients (34.1%), corticosteroids were administered to 38 patients (44.7%), and 18 patients (21.2%) were followed up without any treatment. Among the patients that received treatment, 44 (65.6%) had only GIS involvement, 2 (2.9%) had only renal involvement, and 7 (10.4%)

Table 1: Relationsh	p of the clinical	findings to leukocytosis

Findings	Yes/no	Leukocytosis	No leukocytosis	
		n (%)	n (%)	р
Abdominal pain	Yes	18 (69.20)	32 (54.20)	0.196
	No	8 (30.80)	27 (45.80)	
Arthritis	Yes	11 (42.30)	22 (37.30)	0.602
	No	15 (57.70)	37 (62.70)	
Arthralgia	Yes	4 (15.40)	14 (23.70)	0.386
	No	22 (84.60)	45 (76.30)	
Hematuria	Yes	6 (23.10)	3 (5.10)	0.013*
	No	20 (76.90)	56 (94.90)	
Fecal occult blood	Yes	12 (46.20)	21 (35.60)	0.357
	No	14 (53.80)	38 (64.40)	
GIS hemorrhage	Yes	1 (3.80)	2 (3.40)	1.000
	No	25 (96.20)	57 (96.60)	
GIS involvement	Yes	17 (65.40)	33 (55.90)	0.415
	No	9 (34.60)	26 (44.10)	
Joint	Yes	15 (57.70)	36 (61.00)	0.773
involvement	No	11 (42.30)	23 (39.00)	
Renal involvement	Yes	6 (23.10)	3 (5.10)	0.013*
	No	20 (76.90)	56 (94.90)	

*p<0.05. GIS: Gastrointestinal system

had both GIS and renal involvement. It was found that one patient developed intussusception as a complication and the other patients did not have any complications during the hospitalization period.

DISCUSSION

The findings of our study, i.e. age of the patients and the maleto-female ratio (1.36:1) were similar to the data reported in other studies [13,14]. Although the disease is seen throughout the year, it is known that the disease is more frequent in winter as seen in our study; furthermore, it is followed by autumn. There are studies reporting that HSP is associated with respiratory tract infections, and the fact that the disease is more frequently seen in cold weather, especially in autumn and winter, implies that infections could be the triggering factor in the pathogenesis of this disease. However, this may be a coincidence on account of the increased incidence of URTI in children during winter [15].

Clinical findings from the patients revealed that all patients had a rash in the form of purpura. Lesions were localized generally on the lower extremities and rarely on the upper extremities and torso. Sometimes, skin findings are not present as the first symptom and can appear after arthritis or GIS symptoms. The occurrence of skin findings following joint and GIS involvement ranges between 25 and 50% [15,16]. Two different studies reported that joint involvement was seen in 15% and 25% of patients, respectively, and abdominal pain in 12% and 20% of patients, respectively, as initial symptoms [17,18]. Among our patients, 17.6% had abdominal pain and 18.8% had joint pain as initial symptoms and the results were similar to those in literature.

Arthritis is the second most common clinical presentation following rash in HSP patients. The rate of joint involvement ranges between 62 and 82%; the most common joint involvement was seen in the ankles and knees. Effusions are usually serous and not hemorrhagic and do not develop residual deformity or joint damage [15,16,19]. In our study, 60% of the patients had joint involvement. The most common joint involvement was seen in the ankles and knees, which is consistent with literature.

Studies reported a GIS participation rate of 38-75% in HSP. In one study, it was obtained that the rate of GIS involvement was 62.3% and incident of fecal occult blood was 37.7% [3]. In our study, 58.8% of the patients had GIS involvement. All patients had abdominal pain, 38.8% of the patients had fecal occult blood and 3.5% had fecal fresh red blood, all consistent with the data reported in literature. The most common surgical complication of HSP is intussusception. Radiologic, USG, and endoscopic assessment of patients presenting to the clinic with GI symptoms significantly reduced the number of unnecessary laparotomies. Studies that carried out gastrointestinal USG examinations in children with HSP, who also had abdominal pain, revealed increased duodenum wall thickness and echogenicity in 82%, increased duodenum diameter in 64%, and presence of gallbladder hydrops in 36% of these children. Some studies showed intestinal wall thickening, segmental dilatation of the intestine, and intestinal hypomotility in patients with HSP who were evaluated using abdominal USG [20,21].

In another study conducted by Connolly *et al.* [22], repeated USG of 12 patients with HSP showed decreased bowel wall thickening, a reduced amount of peritoneal fluid, return of peristalsis, and improved loose invaginations, and the improvement in sonographic findings were consistent with the clinical improvement. Therefore, it was reported that the severity and extent of gastrointestinal involvement could be monitored in HSP with repeat USG. In our study, an abdominal USG examination of a patient with abdominal pain showed requiring surgery.

The rate of renal involvement in HSP was reported to have a range between 10 and 60%, and the first signs of renal pathology are generally seen within 2 weeks after purpuric lesions are observed [23]. HSP nephritis generally manifests as mild proteinuria and/or microscopic hematuria that resolves spontaneously. However, renal involvement can be the primary cause of chronic kidney disease (CKD) in HSP. Therefore, kidney functions in HSP patients should be closely monitored, and treatment provided in the case of severe involvement [24]. A study by Sano *et al.* found that at the age of > 4 years, presence of severe gastrointestinal involvement, and purpura tending to last more than 1 month, was reported as risk factors for renal disease, and the most significant factor determining prognosis was reported the severity of renal symptoms at disease onset [25]. In our study, it was found that 10.6% of patients had renal involvement with microscopic hematuria, and renal symptoms appeared along with the rash or within the first few days. None of the patients had proteinuria and their blood pressure was within normal limits throughout hospitalization. In a study by Hamdan and Barqauri, the effect of age distribution on systemic involvement was investigated in 68 patients with HSP, and it was found that 19%

of the patients younger than 5 years and 67% of the patients older than 10 years had developed nephritis. Hence, it was highlighted that the development of nephritis was age dependent and the incidence of nephritis was higher in older children [26]. Similarly, our study has also demonstrated that 89% of the patients with renal involvement were older than 7 years. Consequently, urinalysis results and blood pressure should be monitored more carefully in the presence of renal involvement, especially in older children.

Laboratory tests are generally supplementary rather than diagnostic in HSP. In our study, the mean values of hemoglobin, leukocyte, platelet, urea, and creatinine parameters were within normal limits. Urea levels were higher than normal in four patients, but a relationship between elevated urea level and HSP could not be established in these patients as they did not have renal involvement. In all, 30.6% of the patients had leukocytosis, 22.4% had thrombocytosis, and 47% had CRP positivity. The detected high levels of acute phase reactants such as CRP, platelets, and leukocytes were consistent with other studies [19,27] and show that inflammation plays a role in the pathogenesis of HSP. The absence of other diseases and symptoms that may cause arthritis, URTI, or leukocytosis in patients with hematuria means that the risk of kidney involvement may increase in patients with leukocytosis. Even so, this field requires further prospective studies.

HSP is generally a self-limiting disease that does not require any special treatment other than support. Joint pain can usually be managed using analgesics such as ibuprofen. In patients with active GIS bleeding, nonsteroidal anti-inflammatory treatment for joint symptoms is undesirable. Hypertension is managed with suitable antihypertensive drugs. If there is an infection, it is treated with suitable antibiotics depending on the infectious agent and severity of infection. Surgery may be required for serious GIS complications. Severe skin symptoms, abdominal pain, and protein loss respond dramatically to oral corticosteroid treatment within a few days [16,27]. Corticosteroid treatment can lead to remission of arthritis and abdominal pain; however, it does not shorten the duration of the disease or prevent relapse. Starting steroid treatment in a patient with abdominal pain but no bleeding is considered undesirable as it may mask more severe complications. GIS symptoms exhibit remission without treatment in cases other than those requiring surgery. Starting treatment in the initial phase does not prevent the symptoms from developing. A few studies asserted that starting steroid treatment in the initial phase in patients (older than 7, with severe GIS involvement, and long-term purpura) who are suspected to have a high risk of developing kidney involvement could prevent renal damage [17,28].

Treatment protocols for Henoch-Schönlein nephritis are based on small series studies and a common consensus has not been reached on any of these protocols. Recent findings show that the combination of high-dose intravenous pulse methylprednisolone with azathioprine or cyclophosphamide could be beneficial in patients with severe nephritis [27]. There are also attempts to use plasmapheresis, Factor XIII concentrate, and urokinase in patients with severe nephritis. It is also reported that Dapsone treatment could contribute to the remission of HSP symptoms [17,28]. Recent studies showed that administering mycophenolate mofetil to patients who presented with nephrotic syndrome led to a significant remission in proteinuria after 1 month and full remission in 3 months, and slowed down the progression to CKD [29]. In our study, nonsteroidal anti-inflammatory drugs (ibuprofen, 30 mg/kg/day) were administered to 34.1% of the patients, steroids (methylprednisolone 2 mg/kg/day) were administered to 44.7% of the patients, and 21.2% of the patients were followed up without any treatment. It was found that joint symptoms in patients who only had joint involvement had clinically resolved without any residual damage after administration of nonsteroidal anti-inflammatory treatment for 1-2 weeks on average, during the hospitalization period. Steroid treatment was found to be needed in patients with severe gastrointestinal involvement (severe abdominal pain and GIS hemorrhage) and renal involvement for 2-3 weeks on average. All patients who received steroid treatment due to severe GIS involvement exhibited clinical improvement in the acute phase, except for one patient. It was found that one patient with severe GIS symptoms developed intussusception as a complication and was operated on, whereas the other patients did not have any complications during the hospitalization period.

CONCLUSION

HSP is more commonly seen in children and leads to lifethreatening complications in a minority of patients. All patients with GIS symptoms should be examined using imaging methods and monitored to assess the severity of the disease, and any GIS complications should be observed and followed up. The risk of developing chronic glomerular disease is higher in older children (especially older than 7 years of age), patients who have more severe and long-term skin symptoms, leukocytosis, early renal findings, and serious abdominal symptoms. Therefore, these patients should be closely monitored.

REFERENCES

- Schijnlein H. Allgemeine und Specielle Pathologie und Therapie. 3rd. ed., Vol. 2. Wurzburg: Herisau; 1837.
- Yang YH, Yu HH, Chiang BL. The diagnosis and classification of henochschönlein purpura: An updated review. Autoimmun Rev 2014;13:355-8.
- Özer S, Kasap T, Yılmaz R, Sönmezgöz E, Ünüvar S. Henoch-Schönlein purpura in Children: Retrospective evaluation of 53 cases. J Contemp Med 2015;5:111-5.
- Gürgöze MK, Gündüzalp M. Çocuklarda Henoch-Schönlein Purpurası: 50 Olgunun Retrospektif Değerlendirilmesi. Fırat Tıp Dergisi 2010;15:27-30.
- Ersen A, Aydınoz S, Karademir F, Süleymanoğlu S, Meral C, Ozkaya H, et al. Henoch-Schonlein purpura of childhood: retrospective analysis of 42 cases. Dirim. 2009;84:35-41.
- Kasapçopur Ö, Arısoy N. Henoch-Schönlein purpurası. Türk Pediatri Arşivi 2002; 37:122-129.
- Özçakar Ö, Fitöz S, Yalçınkaya F. Henoch Schönlein purpura. Klinik Gelişim dergisi 2006;19:52-5.
- Miller ML, Pachman LM. Vasculitis syndromes, Henoch Schönlein purpura. IN: Behrmann RE, Kliegman RM, Jenson HB, editors. Nelson Textbook of Pediatrics. 17th ed. Philadelphia, PA: WB Saunders Company; 2004. p. 826-8.
- Altay D, Gürgöze MK, Deveci U, Saraç M. Can Henoch-Schönlein purpura be mixed with acute appendicitis?. Fırat Üniv Sağlık Bilimleri Tıp Derg 2016, 30(3):131-134
- 10. Trnka P. Henoch-schönlein purpura in children. J Paediatr Child Health

2013;49:995-1003.

- 11. Berube MD, Blais N, Lanthier S. Neurologic manifestations of henochschönlein purpura. Handb Clin Neurol 2014;120:1101-11.
- 12. Ozen S, Ruperto N, Dillon MJ, Bagga A, Barron K, Davin JC, *et al.* EULAR/ PRES endorsed consensus criteria for the classification of childhood vasculitides. Ann Rheum Dis 2006;65:936-41.
- 13. Eryılmaz D, Ece A. Evaluation of follow-up results in children with henochschönlein purpura. J Clin Exp Invest 2016;7:269-77.
- 14. Chen JY, Mao JH. Henoch-schönlein purpura nephritis in children: Incidence, pathogenesis and management. World J Pediatr 2015;11:29-34.
- 15. Watts RA, Scott DG. Epidemiology of the vasculitides. Semin Respir Crit Care Med 2004;25:455-64.
- 16. Trapani S, Micheli A, Grisolia F, Resti M, Chiappini E, Falcini F, *et al.* Henoch schonlein purpura in childhood: Epidemiological and clinical analysis of 150 cases over a 5-year period and review of literature. Semin Arthritis Rheum 2005;35:143-53.
- 17. Saulsbury FT. Henoch-schönlein purpura inchildren report of 100 patients and review of the literature. Med 1999;78:395-409.
- 18. Robson WL, Leung AK. Henoch-schönlen purpura. Adv Pediatr 1994;41:163-94.
- 19. Çakır M, Orhan F, Mungan I, Sönmez M, Aslan Y. Henoch-schönlein purpura in North-Eastern Turkey. Ann Trop Pediatr 2006;26:59-65.
- Kagimoto S. Duodenal findings on ultrasound in children with schonleinhenoch purpura and gastrointestinal symptoms. J Pediatr Gastr Nutr 1993;16:178-82.
- Özdemir H, Işık S, Buyan N, Radiol EJ. Sonographic demonstration of intestinal involvement in henoch-schonlein syndrome. Eur J Radiol 1995;20:32-4.
- 22. Conolly B, O'Halpin D. Sonographic evaluation of the abdomen in HSP. Clin radiol 1994;49:320-3.

- Bilge I. Çocukluk çaği vaskülitleri ve böbrek. Turkiye Klinikleri J Pediatr Sci. 2008;4(1):94-105.
- 24. Pohl M. Henoch-schönlein purpura nephritis. Pediatr Nephrol 2015;30:245-52.
- Sano H, Izumida M, Shimizu H, Ogawa Y. Risk factors on renal involvement and significant proteinuria in henoch schönlein purpura. Eur J Pediatr 2002;161:196-201.
- Hamdan JM, Barqauri MA. Henoch-schonlein purpura in children. Influence of age on the incidence of nephritis and arthritis. Saudi Med J 2008;29:549-52.
- Nong B, Huang Y, Chuang C, Liu C, Hsieh K. Fifteen-year experience of children with henoch-schönlein purpura in southern Taiwan, 1991-2005. J Microbiol Immunol Infect 2007;40:371-6.
- Bagga A, Dillon MJ. Leukocytoclastic vasculitis. In: Cassidy JT, Petty RE editors. Textbook of Pediatric Rheumatology. Philadelphia, PA: WB Saunders Company; 2001. p. 569-79.
- Hackl A, Becker JU, Körner LM, Ehren R, Habbig S, Nüsken E, *et al.* Mycophenolate mofetil following glucocorticoid treatment in henochschönlein purpuranephritis: The role of early initiation and therapeutic drug monitoring. Pediatr Nephrol 2018;33:619-29.

Funding: None; Conflict of Interest: None Stated.

How to cite this article: Karaaslan F, Karaaslan BG, Dag H, Sazak S, Turkkan E, Yılmaz B, Midillioglu GK. Henoch-Schönlein purpura in children: A cross sectional study. Indian J Child Health. 2019; 6(3):99-103.

Doi: 10.32677/IJCH.2019.v06.i03.001