

# Fifteen-year experience of children with Henoch-Schönlein purpura in southern Taiwan, 1991-2005

Bao-Ren Nong, Yung-Feng Huang, Chih-Ming Chuang, Chia-Chia Liu, Kai-Sheng Hsieh

*Department of Pediatrics, Veterans General Hospital-Kaohsiung, Kaohsiung, Taiwan*

Received: September 2, 2005 Revised: July 20, 2006 Accepted: September 15, 2006

**Background and Purpose:** Although Henoch-Schönlein purpura (HSP) is the most common cause of systemic vasculitis in children, long-term and large-scale Taiwanese studies on HSP are rare. We reviewed the records of 107 Taiwanese pediatric patients diagnosed with HSP at our institution between 1991 and 2005.

**Methods:** The first clinical manifestations, laboratory findings, and outcome evaluations of the patients were analyzed. Data were grouped according to the presence of fever and upper respiratory tract infection (URI) as a presenting symptom and also by gender. Chi-squared test was used for statistical analysis.

**Results:** The children had a mean age of  $6.2 \pm 2.5$  years (range, 2 to 13 years), with a male-to-female ratio of 1.0:0.7. Main clinical symptoms included skin rashes (95.3%), gastrointestinal (GI) symptoms (72.0%), joint involvement (46.7%), and kidney involvement (28.0%). The most common first manifestations were skin rashes (56.1%), GI symptoms (35.5%), and joint involvement (12.1%). There was no significant association between first manifestations and fever presence or gender. However, the non-URI patients had a significantly higher incidence of GI problems than the URI group ( $p=0.01$ ). Fever as a symptom was not associated with elevation of C-reactive protein ( $p=0.45$ ). Immunoglobulin A levels were within the normal range. No chronic renal failure or end-stage renal disease was detected, and overall the prognosis of patients was good.

**Conclusions:** The categories used did not predict the expression of HSP, with the exception of an association between absence of URI and GI manifestations. Overall, HSP showed a good prognosis.

**Key words:** Purpura, Schoenlein-Henoch; Retrospective studies; Risk factors

## Introduction

Henoch-Schönlein purpura (HSP) characterized by skin rashes, gastrointestinal (GI) symptoms, joint involvement, and kidney involvement is the most common cause of systemic vasculitis in children all over the world [1-7]. The annual incidence of HSP is reported to be 13.5 per 100,000 children [8,9]. It may develop at any age, from early infancy to the geriatric period, but is most common in early childhood [8]. Although many reports have dealt with this disease, long-term and large-scale Taiwanese studies of HSP are rare, especially in southern Taiwan. We reviewed 107 patients with HSP from the Veterans General Hospital at Kaohsiung, a

teaching hospital in Taiwan, over a 15-year period from 1991 to 2005.

We analyzed the first clinical manifestations, laboratory findings, and outcome evaluations of these patients. Patients were classified according to their symptoms as those with fever (fever group) or without fever (non-fever group), those with upper respiratory tract infection (URI) [URI group] or without URI (non-URI group), and based on their gender. The aim was to evaluate whether these criteria were of predictive value for the disease symptomatology.

## Methods

According to the 1990 American College of Rheumatology criteria for the classification of HSP, patients suffering from "at least 2 of the 4 criteria" were diagnosed as having HSP.

*Corresponding author: Dr. Yung-Feng Huang, MD, Department of Pediatrics, Veterans General Hospital-Kaohsiung, No. 386 Ta-Chung First Road, Kaohsiung 813, Taiwan.  
E-mail: brnong@yahoo.com.tw*

The 4 criteria are: (1) palpable purpura; (2) age <20 years at disease onset; (3) bowel angina; and (4) wall granulocytes on biopsy [1,2].

Children who were diagnosed with HSP and consistently visiting the pediatrics department at the Veterans General Hospital at Kaohsiung from November 1991 to December 2005 were studied.

Skin rash was defined as urticarial, maculopapular lesions [10,11], petechiae, or purpura. The rash was red or brown in color and did not blanch with pressure [8] or angioma [12]. GI symptoms were defined as abdominal pain, vomiting, melena, or hematemesis. Joint involvement referred to pain, swelling, limitation in movement, redness, or warmth of the joint.

The URI group comprised patients with antecedent URI events; the rest were placed in the non-URI group. The fever group consisted of patients who developed fever within 3 days of disease onset; the rest were in the non-fever group.

Complete blood counts, differential counts, and urine routines were checked. Hematuria was defined as more than 10 red blood cells per high-power field read in the centrifuged urine. Isolated hematuria was defined as hematuria without renal insufficiency or other urinary abnormalities, such as red cell, other casts, or proteinuria. Proteinuria was defined as a positive dip stick test or urine protein excretion >4 mg/m<sup>2</sup>/h [3]. Standard definitions were applied to acute nephritis syndrome, nephrotic syndrome, and renal impairment [4,5]. Kidney involvement referred to isolated hematuria, isolated proteinuria, hematuria with proteinuria, nephritis, or nephrotic syndrome.

Patients who suffered from nephrotic syndrome, a rapid progressive deterioration in glomerular filtration rate, nephritic syndrome with macroscopic hematuria, hypertension, or renal insufficiency that persisted longer than 2 to 4 weeks were advised to have kidney biopsies. The classification of Henoch-Schönlein nephritis (HSN) was based on the "modified Heaton's classification" (Table 1) [13].

C-reactive protein, immunoglobulin G, immunoglobulin A (IgA), and immunoglobulin M (IgM) were analyzed by nephelometry and were used to define the abnormal ranges in Chinese children of a particular age (mean + standard deviation) [6,14]. Antistreptolysin O titer was measured by the passive hemagglutination test and was interpreted as elevated if it was >300 Todd units. Antinuclear antibody test was done by the indirect fluorescent immunoassay method. The chi-squared test was employed for statistical analysis.

## Results

A total of 107 patients were involved in the study, with 63 males and 44 females. The male-to-female ratio was 1.0:0.7. Their ages ranged from 2 to 13 years, with a mean of  $6.2 \pm 2.5$ . Peak incidence was seen in winter (37/107; 34.6%), followed by spring (25/107; 23.4%), autumn (23/107; 21.5%), and summer (22/107; 20.6%).

Main clinical features were skin rashes (102/107; 95.3%), GI symptoms (77/107; 72.0%), joint involvement (50/107; 46.7%), and kidney involvement (30/107; 28.0%). Skin rashes were often palpable. Some patients predominately presented with petechial lesions, while others had purpuric lesions or a mixture of the two. The skin lesions were distributed mainly over the buttocks and legs. GI involvement manifested itself most commonly in the form of abdominal pain, a symptom present in all victims, and tenderness was detectable in all patients by direct palpation over the abdomen. Of all the subjects, 6 patients suffered from vomiting combined with abdominal pain, and 5 had melena after abdominal pain. Knees and ankles were the most common sites of joint involvement. Pain, swelling, and movement limitation of the involved joints were more frequent than redness and warmth. The proportion of patients who had kidney involvement along with the clinical features of skin rashes, GI symptoms, and joint involvement was 100.0% (30/30), 66.7% (20/30), and 53.3% (16/30), respectively.

**Table 1.** Classification of Henoch-Schönlein nephritis based on the "modified Heaton's classification" [13]

Class I	No crescents, minimal glomerular alteration
Class II	No crescents
	A) Pure mesangial proliferation
	B) Focal-segmental endocapillary proliferation
	C) Diffuse endocapillary proliferation
Class III	Crescents in some glomeruli (<50%)
	A) Focal endocapillary proliferation
	B) Diffuse endocapillary proliferation
Class IV	Crescents in many glomeruli (50% to 75%)
	A) Focal endocapillary proliferation
	B) Diffuse endocapillary proliferation
Class V	Crescents in most glomeruli (>75%)
	A) Focal endocapillary proliferation
	B) Diffuse endocapillary proliferation
Class VI	Pseudomembranoproliferative glomerulonephritis

**Table 2.** Comparison of the main clinical features of skin rash, gastrointestinal (GI) symptoms, and arthritis between patients with/without hematuria or proteinuria

	No hematuria or proteinuria (n = 77)	Hematuria or proteinuria (n = 30)	<i>p</i>
	No. (%)	No. (%)	
Skin rash	73 (95)	30 (100)	0.21
GI symptoms	57 (74)	20 (67)	0.45
Arthritis	34 (44)	16 (53)	0.40

However, no significant difference was found between the patients with and without kidney involvement with respect to these 3 main clinical features (Table 2).

Skin rashes were the most common first manifestation, accounting for 56.1% (60/107) of all cases, followed by GI symptoms (38/107; 35.5%) and joint involvement (13/107; 12.1%). Of the 38 cases with GI symptoms, 36 had abdominal pain alone while 2 had abdominal pain combined with vomiting. On analysis, no significant differences were found between the fever and non-fever group patients, male and female patients, and patients with or without kidney involvement with respect to the proportion of the 3 first manifestations. However, the non-URI group showed a significantly higher percentage of GI symptoms than the URI group ( $p=0.01$ ) [Table 3].

The URI and fever groups did not show significantly higher incidences of kidney involvement in comparison with the patients of the non-URI ( $p=0.235$ ) or non-fever ( $p=0.072$ ) groups, respectively. Also, no remarkable difference was found between males and females in terms of the percentage of patients with hematuria, proteinuria, hematuria combined with proteinuria, and kidney involvement (Table 4).

The most common major manifestation of kidney involvement was isolated hematuria (20/30), followed

by isolated proteinuria (2/30) and hematuria combined with proteinuria (8/30). Of these, 3 patients had nephrotic syndrome, 1 had nephrotic syndrome combined with hypertension, and 1 had acute renal failure combined with hypertension. Seven patients received kidney biopsies, and results revealed that 2 patients were class III A HSN, 1 class III B, 2 class II B (one of them suffered from acute renal failure with hypertension), 1 class II C (nephrotic syndrome with hypertension), and 1 had IgM nephropathy. All patients were responsive to medication, and no chronic renal failure or end-stage renal disease was found, except in one of the class III A HSN girls who had cerebral vasculitis. Sequela of seizure disorder was found in this patient.

Without exception, all antistreptolysin O titers and antinuclear antibody tests were negative. Serum IgA levels were all within normal range with respect to patient age [14]. C-reactive protein serum levels also indicated no significant difference between patients in the non-fever and fever groups.

Ten patients received skin biopsies and all pathological findings were compatible with the typical HSP skin manifestation — leucocytoclastic vasculitis that affects small vessels (arterioles and venules) with neutrophil infiltration in and around dermal vessels.

**Table 3.** Skin rash, abdominal pain, and arthritis as first presentations by gender, and according to the presence/absence of fever, upper respiratory tract infection (URI), hematuria or proteinuria

Variable	Skin rash	Gastrointestinal symptoms	Arthritis
	No. (%)	No. (%)	No. (%)
Fever group (n = 77)	45 (58)	29 (38)	7 (9)
Non-fever group (n = 30)	15 (50)	9 (30)	6 (20)
<i>p</i>	0.43	0.45	0.12
URI group (n = 64)	33 (52)	29 (45)	6 (9)
Non-URI group (n = 43)	27 (63)	9 (21)	7 (16)
<i>p</i>	0.25	0.01	0.28
Male (n = 63)	33 (52)	25 (40)	7 (11)
Female (n = 44)	26 (59)	13 (30)	6 (14)
<i>p</i>	0.60	0.28	0.69
No hematuria or proteinuria (n = 78)	41 (53)	30 (38)	11 (14)
Hematuria or proteinuria (n = 29)	19 (66)	8 (28)	2 (7)
<i>p</i>	0.23	0.29	0.31

**Table 4.** Gender-based clinical presentation profile of patients

	Male (n = 63) No. (%)	Female (n = 44) No. (%)	<i>p</i>
Hematuria or proteinuria	17 (27)	13 (30)	0.77
Hematuria	16 (25)	12 (27)	0.82
Proteinuria	5 (8)	5 (11)	0.53
Hematuria and proteinuria	4 (6)	4 (9)	0.56

We performed endoscopy on those who were agreeable to the procedure. Results showed that 20/23 had the typical changes of GI vasculitis — coalescing purpuric lesions, punctuated erythematous changes, and mucosal edema. Microscopic findings revealed perivascular infiltrates of polymorphonuclear leukocytes and lymphocytes around small blood vessels in the GI tract. Of the other 3 cases, 1 failed due to sedation and 2 presented negative findings. Four patients (4/47; 8.5%) suffered from scrotum swelling or pain, but all resolved spontaneously within 1 week.

Supportive care and corticosteroid treatment to all patients led to the relief of abdominal colic and joint pain, although the latter was not the rationale behind corticosteroid usage.

Patients with crescentic glomerulonephritis and decreased renal function were treated with oral prednisolone and dipyridamole for 3-4 months. However, the patient with acute renal failure was treated with hemodialysis and methylprednisolone pulse therapy, cyclophosphamide and azathioprine, followed by oral prednisolone and dipyridamole.

## Discussion

HSP is an IgA-mediated, non-thrombocytopenic, purpuric, systemic vasculitis of childhood. It is associated with a variety of abnormalities involving IgA, including the levels of serum IgA, serum polymeric IgA and IgA containing circulating immune complexes, as well as the presence of IgA rheumatoid factor [15]. The IgA abnormalities seen in HSP are associated with IgA1 alone, and not IgA2 [15]. Vascular endothelial growth factor may play a crucial role in the morphological and functional changes of the vascular bed and inflammatory reactions seen in HSP [16]. Serum IgA analysis by nephelometry showed no cases of elevated serum IgA levels. This observation is inconsistent with most published papers, which report readings to be usually 40-50% higher in HSP patients [10]. The underlying reasons for this irregularity are as yet unclear, and may

include racial factors among others. HSP has an incidence of 14 cases per 100,000 people [17-19]. The male:female ratio found in our study (1.00:0.70) was comparable to those of previous reports (1.20 to 2.00:1) [6,8]. Previous studies have reported the age of HSP patients to range from 6 months to 86 years, with the mean peak age being 4 to 7 years [8,10,20]. Similarly, we found that the majority of subjects were less than 7 years old (range, 2 to 13 years), with a mean of  $6.2 \pm 2.5$  years.

Since there are no diagnostic laboratory tests for HSP, previous studies were taken as a guide for excluding other conditions that resembled HSP [10]. Thus, diagnoses were mainly made based on clinical judgment.

The main clinical features of HSP included purpuric rashes, GI symptoms, arthralgia, and kidney involvement. Skin rashes presented in a total of 102/107 cases (95.3%), but were seen as a first manifestation in only 60/107 cases (56.1%). This finding was again comparable to earlier studies [3]. Rashes were often seen on the lower extremities and buttocks. Classical lesions consisted of urticarial wheals, erythematous maculopapules, and larger palpable ecchymosis-like lesions [17], which could persist from days to weeks and recurred in some patients [6].

GI symptoms and signs preceded the typical rash for a period of days or even up to 2 weeks in 20% of the patients [15]. The most common complaint of GI involvement was abdominal pain, often in the form of colic, at times associated with vomiting. Stools could show gross or occult blood, and hematemesis was also noticed in some. Joint involvement was characterized by warmth, tenderness, and swelling of the joints, particularly the large joints (specifically the ankles and knees) [13]. Joint involvement left no permanent deformities [21]. Preceding URI symptoms and signs were found in 28% (30/107) of our cases, which is lower than previous reports (54-90%) [6]. Arthritis has also been reported to precede purpura by up to 1 week in 25% of patients [15].

The issue of whether HSP patients should be treated is still controversial. A previous study reported that abdominal pain resolved within 24 h in 44% of patients treated with steroids, but in only 14% of the non-steroid treatment group. At 48 h, resolution was 65% in the steroid group and 45% in the other, although the difference was not statistically significant. By 72 h following admission, resolution of pain was similar in both groups (75%) [22]. Corticosteroid administration

may hasten the resolution of abdominal pain, but may not prevent recurrence. Controlled trials need to be undertaken to study this in detail [22,23].

Nephritis occurs in 20-50% of children with HSP, and a substantial proportion of the patients show a delay of weeks or months in the onset of renal involvement [24]. Hematuria was reported as a common initial manifestation of renal disease [14], which is similar to our findings (isolated hematuria, 20/30). Early corticosteroid therapy was shown not to prevent delayed nephritis in children with HSP [24-26], and no other specific treatment has been reported to ease the symptoms of nephritis [26]. Fortunately, less than 1% of those with kidney involvement progress to end-stage renal disease [17].

Although its cause is still unknown, HSP is often associated with infectious agents, such as group A *Streptococcus*, *Mycoplasma pneumoniae*, hepatitis B virus, human immunodeficiency virus, adenovirus, herpes simplex virus, *Helicobacter pylori*, *Toxocara canis*, and human parvovirus B19 among others [10, 27-32]. Nevertheless, few studies have examined the incidence of infections by specific pathogens in children with HSP in comparison to controls [10]. HSP has also been reported to be associated with food reactions, exposure to cold, insect bites, and drug allergies [13,17, 33-36]. However, our studies did not find any obvious association between HSP and these factors — not even the most widely discussed infectious pathogens, i.e., group A beta-hemolytic *Streptococcus* (0/32) and *M. pneumoniae* (2/28). Moreover, although 18 patients received virus culture examinations, none showed positive findings. While some previous studies indicated high association between group A beta-hemolytic streptococci and HSP [10,36-38], some have shown no association at all [10].

The mechanism involved in the pathogenesis of HSN is still not well understood, but several lines of evidence suggest that immunological factors, including immune complexes might be implicated [39]. The presence of IgA (especially IgA1) [10,40,41] and IgM immune complexes, in the glomerular mesangium of patients with HSN is strongly suggestive of a key role of these antibodies in the pathogenesis of renal damage [39]. One previous study also reported that patients with high serum IgA levels had a predisposition for renal involvement with poor prognosis [6]. However, neither the elevated serum IgA levels noted in our study nor the suppositions mentioned above could account for the fact that prognosis in patients with kidney involvement

was not worse than in the others. Furthermore, only 1 case of IgM nephropathy was found in our study.

Though acute morbidity is primarily caused by GI complications, such as massive bleeding and intussusception [24], literature reviews indicate that long-term prognosis of HSP is determined by the level of renal involvement [6,9,42-44]. Since a small percentage of patients can progress to renal failure [17], it is important to repeat urinalysis during the follow-up period. In most cases, however, HSP is a benign, self-limited condition [17]. In this study, the overall prognosis of the cases studied was good, even in those with kidney involvement.

## References

1. Cassidy JT, Petty RE. Vasculitis. In: Cassidy JT, Petty RE, eds. Textbook of pediatric rheumatology. 3rd ed. Philadelphia: WB Saunders; 1995:365-422.
2. Canoso JJ. The vasculitides. In: Canoso JJ, ed. Rheumatology in primary care. Philadelphia: WB Saunders; 1997;16:115-34.
3. Bagga A, Kabra SK, Srivastava RN, Bhuyan UN. Henoch-Schönlein syndrome in northern Indian children. Indian Pediatr. 1991;28:1153-7.
4. Meadow SR, Glasgow EF, White RH, Moncrieff MW, Cameron JS, Ogg CS. Schönlein-Henoch nephritis. Q J Med. 1972;41:241-58.
5. Chen WP, Lin CY, Cheng JH, Hwang BT. Purpura nephritis in Chinese children from northern Taiwan. Child Nephrol Urol. 1988-1989;9:331-6.
6. Wang YJ, Chi CS, Shian WJ. Clinical studies of Henoch-Schönlein purpura in Chinese children. Zhonghua Yi Xue Za Zhi (Taipei). 1993;51:345-9.
7. Kobayashi O, Wada H, Okawa K, Takeyama I. Schönlein-Henoch's syndrome in children. Contrib Nephrol. 1975;4: 48-71.
8. Robson WL, Leung AK. Henoch-Schönlein purpura. Adv Pediatr. 1994;41:163-94.
9. Stewart M, Savage JM, Bell B, McCord B. Long term renal prognosis of Henoch-Schönlein purpura in an unselected childhood population. Eur J Pediatr. 1988;147:113-5.
10. Saulsbury FT. Henoch-Schönlein purpura in children. Report of 100 patients and review of the literature. Medicine (Baltimore). 1999;78:395-409.
11. Saulsbury FT. Henoch-Schönlein purpura. Pediatr Dermatol. 1984;1:195-201.
12. Powell KR. Rheumatic disease of childhood. In: Behrman RE, Kliegman R, HB Jenson, eds. Nelson textbook of pediatrics. 16th ed. Philadelphia: WB Saunders; 2000;15:698-735.
13. Heptinstall RH. IgA Nephritis and Schönlein-Henoch syndrome. In: Heptinstall RH, ed. Pathology of the kidney.

- 4th ed. Boston: Little Brown; 1992;1:430-76.
14. Tang RB, Tsai LC, Chang YF, Hsiao IS, Wang HC, Han SH. Plasma levels of immunoglobulins and complement components in normal Chinese children. *Chin Med J (Taipei)*. 1982;29:360-3.
  15. Saulsbury FT. Henoch-Schönlein purpura. *Curr Opin Rheumatol*. 2001;13:35-40.
  16. Topaloglu R, Sungur A, Baskin E, Besbas N, Saatci U, Bakkaloglu A. Vascular endothelial growth factor in Henoch-Schönlein purpura. *J Rheumatol*. 2001;28:2269-73.
  17. Kraft DM, Mckee D, Scott C. Henoch-Schönlein purpura: a review. *Am Fam Physician*. 1998;58:405-8, 411.
  18. Jennette JC, Falk RJ. Small-vessel vasculitis. *N Engl J Med*. 1997;337:1512-23.
  19. Trujillo H, Gunasekaran TS, Eisenberg GM, Pojman D, Kallen R. Henoch-Schönlein purpura: a diagnosis not to be forgotten. *J Fam Pract*. 1996;43:495-8.
  20. Tancrede-Bohin E, Ochonisky S, Vignon-Pennamen MD, Flageul B, Morel P, Rybojad M. Schönlein-Henoch purpura in adult patients. Predictive factors for IgA glomerulonephritis in a retrospective study of 57 cases. *Arch Dermatol*. 1997;133:438-42.
  21. Shetty AK, Desselle BC, Ey JL, Correa H, Galen WK, Gedalia A. Infantile Henoch-Schönlein purpura. *Arch Fam Med*. 2000;9:553-6.
  22. Kirschner BS. Undetermined colitis and other inflammatory diseases. In: Walker WA, Goulet OJ, Kleinman RE, Sherman PM, Shneider BL, Sanderson IR, eds. *Pediatric gastrointestinal disease: pathophysiology, diagnosis, management*. 4th ed. Philadelphia: BC Decker; 2004;855-9.
  23. Chang WL, Yang YH, Lin YT, Chiang BL. Gastrointestinal manifestations in Henoch-Schönlein purpura: a review of 261 patients. *Acta Paediatr*. 2004;93:1427-31.
  24. Saulsbury FT. Corticosteroid therapy does not prevent nephritis in Henoch-Schönlein purpura. *Pediatr Nephrol*. 1993;7:69-71.
  25. Austin HA, Balow JE. Henoch-Schönlein nephritis: prognostic features and the challenge of therapy. *Am J Kidney Dis*. 1983;2:512-20.
  26. Iijima K, Ito-Kariya S, Nakamura H, Yoshikawa N. Multiple combined therapy for severe Henoch-Schönlein nephritis in children. *Pediatr Nephrol*. 1998;12:244-8.
  27. Hall TN, Brennan B, Leahy MF, Woodroffe AJ. Henoch-Schönlein purpura associated with human immunodeficiency virus infection. *Nephrol Dial Transplant*. 1998;13:988-90.
  28. Hamidou MA, Gueglio B, Cassagneau E, Treweek D, Grolleau JY. Henoch-Schönlein purpura associated with *Toxocara canis* infection. *J Rheumatol*. 1999;26:443-5.
  29. Maggiore G, Martini A, Grifeo S, De Giacomo C, Scotta MS. Hepatitis B virus infection and Schönlein-Henoch purpura. *Am J Dis Child*. 1984;138:681-2.
  30. Reinauer S, Megahed M, Goerz G, Ruzicka T, Borchard F, Susanto F, et al. Schönlein-Henoch purpura associated with gastric *Helicobacter pylori* infection. *J Am Acad Dermatol*. 1995;33:876-9.
  31. Schwarz TF, Bruns R, Schröder C, Wiersbitzky S, Roggendorf M. Human parvovirus B19 infection associated with vascular purpura and vasculitis. *Infection*. 1989;17:170-1.
  32. Veraldi S, Rizzitelli G. Henoch-Schönlein purpura and human parvovirus B19. *Dermatology*. 1994;189:213-4.
  33. Finkel TH, Török TJ, Ferguson PJ, Durigon EL, Zaki SR, Leung DY, et al. Chronic parvovirus B19 infection and systemic necrotising vasculitis: opportunistic infection or aetiological agent? *Lancet*. 1994;343:1255-8.
  34. Lind KM, Gaub J, Pedersen RS. Henoch-Schönlein purpura associated with *Campylobacter jejuni enteritis*. Case report. *Scand J Urol Nephrol*. 1994;28:179-81.
  35. Szer IS. Henoch-Schönlein purpura. *Curr Opin Rheumatol*. 1994;6:25-31.
  36. Abdel-Al YK, Hejazi Z, Majeed HA. Henoch Schönlein purpura in Arab children. Analysis of 52 cases. *Trop Geogr Med*. 1990;42:52-7.
  37. Nielsen HE. Epidemiology of Schönlein-Henoch purpura. *Acta Paediatr Scand*. 1988;77:125-31.
  38. al-Sheyab M, el-Shanti H, Ajlouni S, Batieha A, Daoud AS. Henoch-Schönlein purpura: clinical experience and contemplations on a streptococcal association. *J Trop Pediatr*. 1996;42:200-3.
  39. Oner A, Tinaztepe K, Erdogan O. The effect of triple therapy on rapidly progressive type of Henoch-Schönlein nephritis. *Pediatr Nephrol*. 1995;9:6-10.
  40. Moja P, Quesnel A, Resseguier V, Lambert C, Freycon F, Berthoux F, et al. Is there IgA from gut mucosal origin in the serum of children with Henoch-Schönlein purpura? *Clin Immunol Immunopathol*. 1998;86:290-7.
  41. Saulsbury FT. Heavy and light chain composition of serum IgA and IgA rheumatoid factor in Henoch-Schönlein purpura. *Arthritis Rheum*. 1992;35:1377-80.
  42. Goldstein AR, White RH, Akuse R, Chantler C. Long-term follow-up of childhood Henoch-Schönlein nephritis. *Lancet*. 1992;339:280-2.
  43. Farine M, Poucell S, Geary DL, Baumal R. Prognostic significance of urinary findings and renal biopsies in children with Henoch-Schönlein nephritis. *Clin Pediatr (Phila)*. 1986;25:257-9.
  44. Bunchman TE, Mauer SM, Sibley RK, Vernier RL. Anaphylactoid purpura: characteristics of 16 patients who progressed to renal failure. *Pediatr Nephrol*. 1988;2:393-7.