

# Predictive Factors for Renal Sequelae in Adults with Henoch-Schönlein Purpura

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**ABSTRACT. Objective.** To examine the outcome and risk factors for renal sequelae in an unselected population of adults with Henoch-Schönlein Purpura (HSP).

**Methods.** Retrospective study of adult patients (> 20 years) with biopsy proved cutaneous vasculitis diagnosed as having HSP seen at a single center between 1984 and 1998. Patients were classified as having HSP according to proposed criteria. Only those patients with a followup of at least 1 year were included in this study of renal sequelae.

**Results.** Twenty-eight patients with a mean followup of 5.5 years fulfilled the inclusion criteria. When the study was concluded, 10 patients (36%) had renal sequelae and 2 (7%) had renal insufficiency. Men outnumbered women. However, neither a previous history of drugs, gender, nor age at disease onset was associated with a higher risk of permanent renal involvement. Patients with hematuria at disease onset or renal involvement during the course of the disease more commonly developed renal sequelae ( $p < 0.001$ ). The presence of anemia ( $p = 0.05$ ) at the time of diagnosis and the onset in summer ( $p < 0.05$ ) were also more common in those with permanent renal involvement (renal sequelae). Patients with relapses had also a higher trend to develop renal sequelae ( $p = 0.07$ ). All patients who fulfilled more than 2 of these 5 risk factors developed permanent renal involvement. With this model we were able to predict renal sequelae in 8 of the 10 patients who had this complication. The Goodman-Kruskal gamma test value was 0.92 (95% CI 0.78–1.00).

**Conclusion.** In unselected adults with HSP, permanent renal involvement (renal sequelae) is not uncommon. Hematuria at disease onset and persistence of renal manifestations during the course of the disease are significant indicators of possible development of renal sequelae. These manifestations plus other features such as onset in summer, anemia at disease onset, or relapses of the disease may predict the development of renal sequelae in most patients. (J Rheumatol 2001;28:1019–24)

## Key Indexing Terms:

CUTANEOUS VASCULITIS                      LEUKOCYTOCLASTIC VASCULITIS                      ADULTS  
HENOCH-SCHÖNLEIN PURPURA                      RENAL COMPLICATIONS                      OUTCOME

Henoch-Schönlein purpura (HSP) is a vasculitis due to IgA mediated inflammation of small vessels, characterized by leukocytoclastic angiitis and predominant cutaneous involvement<sup>1</sup>. HSP is the most common vasculitis in children and an infrequent condition in adults. The pediatric form of the disease has been extensively discussed and has been generally considered a benign and self-limited disorder<sup>2</sup>. There have been a few reports regarding this condition in adults<sup>3-8</sup>, and a high longterm risk of renal disfunction was found in some<sup>3-6</sup>. Most of these studies

involved a small series of selected patient populations with kidney dysfunction attended in reference centers. We have analyzed the renal outcome in adults with HSP based on a series of unselected cutaneous vasculitis seen at a single referral hospital for Lugo region (Northwestern Spain).

## MATERIALS AND METHODS

We retrospectively studied the case records of all adult patients diagnosed with HSP in the Hospital Xeral-Calde (Lugo, Spain) from January 1984 through December 1998. The main characteristics of the Lugo population have been reported<sup>9-11</sup>. The Hospital Xeral-Calde is the single referral center for a mixed rural (60%) and urban population of about 250,000 people living in the Lugo region of Northwestern Spain.

Partial information about adults with HSP diagnosed between 1988 and 1997 has been reported<sup>11</sup>. Patients older than 20 years were considered as adults<sup>10-14</sup> and were included in this study if they had a skin biopsy consistent with cutaneous leukocytoclastic vasculitis.

Adults with cutaneous leukocytoclastic vasculitis other than HSP were excluded. To differentiate HSP from hypersensitivity vasculitis, the traditional format of the criteria proposed by Michel, *et al* was used<sup>14</sup>. According to this format, primary cutaneous vasculitis were classified as HSP if 3 or more of the following criteria were fulfilled: (1) cutaneous palpable purpura; (2) bowel angina; (3) gastrointestinal bleeding; (4) presence of hematuria (gross or microhematuria); (5) age at onset of the disease equal to or younger than 20 years; and (6) absence of medications. Between

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June and November 1999, those adults with HSP and less than one year followup were asked to attend the hospital for further evaluation (clinical history, especially looking for relapses, and laboratory analysis including full blood cell count and routine blood and urine biochemistry tests). Those patients with less than one year followup who did not attend the hospital for further evaluation (n = 5) were excluded.

If the Chapel Hill Consensus Conference (CHCC) definitions had been used, some patients who were classified as HSP according to the Michel criteria would have also fulfilled CHCC definitions for microscopic polyangiitis (MPA)<sup>15</sup>. However, only one of the 3 patients who, during the course of the disease, developed renal insufficiency had fulfilled these definitions for MPA. In 2 of the 3 patients who developed renal insufficiency and met CHCC definitions for HSP, a renal biopsy showed glomerulonephritis with mesangial IgA deposits by immunofluorescence. In addition, the ANCA test was negative by indirect immunofluorescence on alcohol fixed neutrophils in the 9 patients who were tested, although we did not have complete ANCA data (some cases were diagnosed before ANCA was routinely available and the methodology changed during the study period).

**Clinical definitions.** Clinical definitions in patients diagnosed with HSP in the Lugo region between 1988 and 1997 have been described<sup>10,11</sup>. Briefly, drug intake history and infections (most involving the upper respiratory tract, in which treatment for the infection did not cause the resolution of the HSP) were considered to be precipitating events if there was a close temporal relationship (less than a week) between the initiation of therapy or the infection and the onset of HSP. Fever was considered if temperature was > 37.7°C. Nephritis was defined as reported<sup>10,11,16,17</sup>: (1) mild nephropathy if hematuria ( $\geq 5$  red blood cells/hpf) and/or proteinuria (> 300 mg/24 h) without nephrotic range was present; (2) severe nephropathy if nephrotic syndrome (i.e., 1 g/day/m<sup>2</sup> body surface area or > 3.5 g/day proteinuria with plasma albumin  $\leq 25$  g/l), with or without edema and/or acute nephritic syndrome (i.e., hematuria with at least 2 of the following: hypertension, raised plasma urea or creatinine, and oliguria) was present. Renal insufficiency was considered present if the plasma creatinine concentration was above 125% of the upper limit of normal. Renal manifestation was defined as the presence of any event over the course of the disease. Renal sequelae were considered to be present if, at last followup, patients had any of the renal complications described above. However, a patient who had had proteinuria, hematuria, or renal insufficiency during the course of the disease but at last followup had normal urine and renal function was not classified within the group of patients who had renal sequelae. Therefore, patients were only classified with renal sequelae if there was permanent renal involvement at last followup. If patients complained of well defined arthralgia or synovitis was observed on examination<sup>11</sup> then they were considered to have joint manifestations. Gastrointestinal manifestations included: (1) bowel angina: diffuse abdominal pain that worsened after meals or bowel ischemia usually including bloody diarrhea, (2) gastrointestinal bleeding: melena, hematochezia, or positive test for occult blood in the stool<sup>11,14</sup>. If the hemoglobin was lower than 11 g/dl, the patient was considered anemic. Leukocytosis required a white blood cell count > 11,000/mm<sup>3</sup>. Elevation of the erythrocyte sedimentation rate (ESR) (Westergren) required values > 15 mm/h in men and 20 mm/h in women. Serum IgA values > 450 mg/dl constituted an increased level. We used the following definition for relapse: if a patient was diagnosed with HSP, was asymptomatic for at least one month, and presented again a new flare of skin lesions or other systemic complications<sup>11,16</sup>.

**Statistical analysis.** Continuous data were described as mean  $\pm$  standard deviation (mean  $\pm$  SD) and categorical variables as percentages. Comparisons were made using Student's t test for continuous variables. To analyze categorical data, we performed the chi-square test or Fisher's exact test when minimum expected value was less than 5. Statistical significance was defined as  $p \leq 0.05$ . To predict renal sequelae, a score was made. The rationale for this score was to assign one point to any variable, using only those variables associated with renal sequelae with a p value < 0.1. For this purpose logistic regression analysis could not be performed as some vari-

ables (for example, renal manifestations during the course of the disease) produced a singular matrix. The Goodman-Kruskal gamma test was performed. This test must be considered as a correlation coefficient for ordinal variables when these variables are ordered. It takes values between -1 and +1. A value near +1 means that the higher the score obtained, the higher the risk for permanent renal damage (renal sequelae). Calculations were performed with the statistical package Stata Intercooled, release 6.0 (Stata Corp., College Station, TX, USA).

## RESULTS

**Clinical features and etiological factors in adults with biopsy proved HSP and followup of at least one year.** The main demographic and etiological data are shown in Table 1. In adults with HSP and onset of disease between 1984 and 1998 the vasculitis was more common in men during the fifth decade. In most cases there was no history of drug intake or infections prior to disease onset.

Skin lesions were the most frequent clinical manifestation at disease onset. Joint manifestations and hematuria or proteinuria were also frequently observed at the onset of vasculitis (Figure 1). Although IgA levels in the serum are often increased in acute phases of the disease, as described in other series<sup>13,16</sup>, raised IgA serum levels were only observed in 7 of 24 (29%) patients in whom they were tested. Renal sequelae were only observed in 3 of the 7 patients with increased IgA serum levels.

The main clinical features during the course of the disease are summarized in Table 2. All patients developed palpable purpura involving the lower extremities. Gastrointestinal manifestations occurred in most of the patients. Renal disease was found in 54% of patients. Most of them had mild or severe renal involvement and only 3 (11%) developed renal insufficiency (one with oliguria).

**Outcome of adults with biopsy proved HSP.** After a mean followup of 5.5 years, almost 30% of patients had persistent hematuria or proteinuria and 7% had renal insufficiency

Table 1. Epidemiological data and etiological factors in 28 adults (age  $\geq 20$  yrs) with biopsy proved Henoch-Schönlein purpura and followup of at least 12 months.

Age (yrs)		
Mean age $\pm$ SD		45.8 $\pm$ 17.8
Range		21-88
Median		47
Sex	Male/female	19/9
	Proportion of men (%)	67.9
Seasonal pattern (%)		
	Summer	7 (25.0)
	Fall	7 (25.0)
	Winter	9 (32.1)
	Spring	5 (17.9)
Etiological factors (%)		
	Unknown	19 (67.9)
	URT infection	6 (21.4)
	Drugs	8 (28.6)
	URT infection + drugs	5 (17.9)

URT: upper respiratory tract.

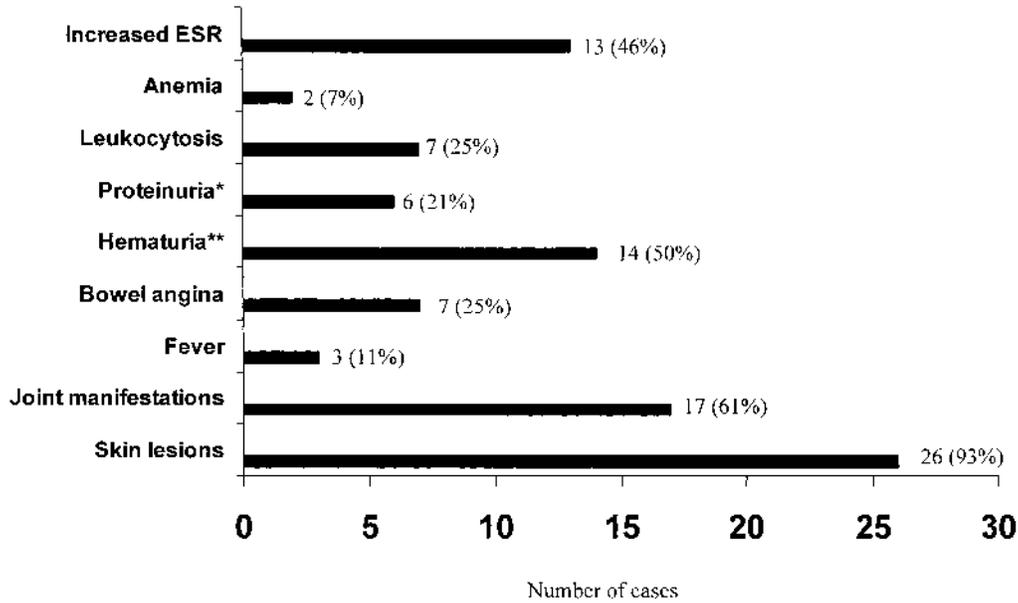


Figure 1. Initial manifestations (at disease onset) and routine laboratory data at the time of diagnosis in 28 adults (> 20 yrs) with HSP and followup of at least 12 months. \*Proteinuria: > 300 mg/24 h; \*\*hematuria:  $\geq$  5 red blood cells/hpf.

Table 2. Clinical and laboratory features in 28 adults (> 20 yrs) with Henoch-Schönlein purpura during the clinical course of the disease.

Variable	n (%)
Predominant Skin Lesion	
Palpable purpura	28 (100.0)
Maculopapular rash	0 (0.0)
Urticarial rash	0 (0.0)
Location of Skin Lesions	
Upper extremities	14 (50.0)
Trunk	11 (39.3)
Lower extremities	28 (100.0)
Joint Manifestations	18 (64.3)
Arthralgia without arthritis	12 (42.9)
Monoarthritis	1 (3.6)
Oligoarthritis	5 (17.9)
Gastrointestinal Manifestations	24 (85.7)
Bowel angina	24 (85.7)
Gastrointestinal bleeding	12 (42.9)
Renal Involvement	
0 None	13 (46.4)
1 Mild*	6 (21.4)
2 Severe**	6 (21.4)
3 Renal insufficiency***	3 (10.7)

\*Mild nephropathy: hematuria ( $\geq$  5 red blood cells/hpf) and/or proteinuria (> 300 mg/24 h) without nephrotic range. \*\*Severe nephropathy: nephrotic syndrome (i.e., 1 g/day/m<sup>2</sup> body surface area or > 3.5 g/day proteinuria with plasma albumin  $\leq$  25 g/l), with or without edema and/or acute nephritic syndrome (i.e., hematuria with at least 2 of the following: hypertension, raised plasma urea or creatinine, and oliguria). \*\*\*Renal insufficiency: plasma creatinine concentration above 125% upper limit of normal<sup>10,11,16,17</sup>.

(one of the 3 patients with renal insufficiency during the course of the disease had improved filtration function). Relapses were observed in 21% of the cases, especially in those who developed renal sequelae (Table 3).

*Epidemiological and clinical differences between patients with and without renal sequelae.* No significant differences in age at disease onset were found. In those adults with persistent renal involvement at last followup, the onset of the disease was more common in summer ( $p = 0.03$ ) (Table 4). A history of upper respiratory tract infections or drugs prior to onset of the disease was not associated with a poor outcome. As expected, hematuria at the onset of disease or renal manifestations during the course of disease frequently observed in patients with relapses of vasculitis were the

Table 3. Outcome of adults with biopsy proved HSP and followup of at least 12 months.

Followup (yrs)	
Mean $\pm$ SD	5.5 $\pm$ 3.6
Median	4.9
Range	1.3–14.3
Relapses	
No. of patients (%)	6 (21.4)
Median relapses*	2.5
Mean $\pm$ SD*	2.3 $\pm$ 1.2
Range*	1–4
Complete recovery (%)	18 (64.3)
Persistent hematuria and/or proteinuria (%)	8 (28.6)
Renal insufficiency (%)	2 (7.1)

\*Based on patients who had at least one relapse.

**Table 4.** Epidemiological differences between biopsy proved Henoch-Schönlein purpura in adult patients (> 20 years) with and without renal sequelae at last followup (at least 1 year).

	With Renal Sequelae (%)	Without Renal Sequelae (%)	p
No. of patients	10/28 (35.7)	18/28 (62.3)	
Age (yrs), mean age ± SD	40.0 ± 28.6	48.9 ± 22.2	0.21
Sex (male/female)	8/2	11/7	0.31
Proportion of male, %	80.0	61.1	
Seasonal pattern			
Summer	5 (50.0)	2 (11.1)	0.03
Fall	1 (10.0)	6 (33.3)	0.17
Winter	3 (30.0)	6 (33.3)	0.38
Spring	1 (10.0)	4 (22.2)	0.79
Etiological Factors			
URT infection	2 (20.0)	4 (22.2)	0.89
Drugs	4 (40.0)	4 (22.2)	0.32
URT infection + drugs	2 (20.0)	3 (16.7)	0.83

URT: upper respiratory tract.

main risk factors of renal sequelae ( $p < 0.001$ ) (Table 5). Interestingly, the only 2 patients with anemia and hematuria at the time of diagnosis persisted with hematuria after a followup of 11.4 and 3.9 years, respectively. Finally, there was a trend toward more relapses in patients with permanent renal involvement (renal sequelae) ( $p = 0.07$ ) (Table 5).

*Predictive model for permanent renal involvement (renal sequelae) in adults with HSP.* Based on those variables with a p value lower than 0.1, a predictive model was designed (Table 6). Variables in the model included the following: disease onset in summer, hematuria at disease onset, anemia at time of diagnosis, presence of renal manifestations during the course of disease, and a history of relapses of HSP. Our

**Table 6.** Predictive model for permanent renal involvement (renal sequelae) in adults with Henoch-Schönlein purpura. Variables included in this model were the following: onset in summer, hematuria at disease onset, anemia at the time of diagnosis, renal complications during the course of the disease, and relapses.

Score	Patients With Renal Sequelae, n = 10	Patients Without Renal Sequelae, n = 18
0	0	10
1	0	3
2	2	5
3	5	0
4	3	0
5	0	0

**Table 5.** Clinical and laboratory differences between biopsy proved Henoch-Schönlein purpura in adult patients (> 20 years) with and without renal sequelae at last followup (at least 1 year).

	With Renal Sequelae (%)	Without Renal Sequelae (%)	p
No. of patients	10/28 (35.7)	18/28 (62.3)	
Arthralgia without arthritis	5 (50.0)	7 (38.9)	0.57
Arthritis	3 (30.0)	3 (16.7)	0.41
Bowel angina	8 (80.0)	16 (88.9)	0.52
Gastrointestinal bleeding	3 (30.0)	9 (50.0)	0.31
Patients with leukocytosis	2 (20.0)	5 (27.8)	0.65
Patients with anemia	2 (20.0)	0 (0.0)	0.05
Increased ESR	5 (50.0)	8 (44.4)	0.78
Increased IgA levels (serum)*	3 (33.3)	4 (26.7)	0.73
Hematuria	10 (100.0)	4 (22.2)	< 0.0001
Proteinuria	3 (30.0)	3 (16.7)	0.41
Renal manifestations	10 (100.0)	5 (27.8)	0.0002
Relapses	4 (40.0)	2 (11.1)	0.07

\*Performed in 24 patients

Hematuria: ( $\geq 5$  red blood cells/hpf) at the onset of the disease

Proteinuria: ( $> 300$  mg/24 hours) at the onset of the disease

Renal manifestations: Renal involvement during the course of the disease

Leukocytosis was defined as leukocyte count  $\geq 11,000/\text{mm}^3$ ; anemia as hemoglobin  $< 11$  gm/dl; erythrocyte sedimentation rate (ESR) was considered elevated if values were  $> 15$  mm/h in men and  $> 20$  mm/h in women. An increase of serum IgA was considered if the values were  $> 450$  mg/dl.

model gave one point to each one of these 5 variables associated with renal sequelae. As shown in Table 6, there was a strong correlation between the score obtained and the presence of renal sequelae ( $p < 0.001$ ). None of the 13 patients with a score of zero or one had renal sequelae. In contrast, all patients with a score of 3 or 4 (in all cases with presence of renal manifestation during the course of the disease) who fulfilled more than 2 of the 5 variables developed renal sequelae. With this model, we were able to define a risk of persistent renal involvement in 8 of the 10 patients with this complication. However, the renal outcome could not be predicted in people who met only 2 clinical variables. With the Goodman-Kruskal gamma test a value of 0.92 (95% confidence interval 0.78–1.00) was obtained.

## DISCUSSION

Classically, renal involvement constitutes the most serious feature of HSP. It has been reported to occur in 30 to 80% of patients with HSP<sup>18</sup>. However, many of the previous series that reported a relatively high frequency of chronic renal failure in children and adults were based on selected patient populations, usually sent to referral centers because of kidney dysfunction<sup>3,4,19-21</sup>. Also, although severe renal involvement is considered to be more common in adults than in children with HSP<sup>22</sup>, Blanco, *et al* reported a good outcome in adults with HSP, with complete recovery in the majority of their patients<sup>16</sup>. Similarly, in their series of patients older than 15 who were selected due to the presence of cutaneous IgA deposits and purpura, Tancrede-Bohin, *et al*<sup>7</sup> also observed a good outcome.

Renal involvement generally occurs within the first 3 months after the onset of features of the disease. However, it may be observed later, generally in the setting of relapses of palpable purpura. Nephropathy in patients with HSP usually presents with hematuria that may persist for several weeks, months, or even years. Recurrences of hematuria may be observed in association with new relapses of skin lesions. More rarely, recurrences of hematuria may occur long after other manifestations of HSP have been resolved. As in our series, hematuria may be isolated or in association with proteinuria.

In our series of adults with HSP permanent renal involvement was present in 36% of the patients. The presence of hematuria at disease onset and the persistence of renal manifestations during the course of the disease were significant indicators of the possible development of renal sequelae. These manifestations along with other features such as onset in summer, anemia at disease onset, or relapses of the disease allowed us to predict the development of renal sequelae in most of our patients. In keeping with our findings, Goldstein, *et al*<sup>17</sup> and Kraft, *et al*<sup>23</sup> reported that the presence of hematuria at the onset of vasculitis was frequently associated with progression to permanent renal involvement. In contrast, Fogazzi, *et al*<sup>3</sup> did not find features

at presentation that predicted the course of the disease, and most of their patients with normal renal function at disease onset developed renal insufficiency subsequently. More specifically, in children with HSP, Kaku, *et al*<sup>18</sup> observed that onset age  $> 7$  years, the presence of persistent proteinuria, and decreased activity of coagulation factor XIII increased the risk of developing renal insufficiency. Recently, Mata-Arnaiz, *et al* reported a higher risk of renal sequelae in those patients with previous antibiotic intake in an unselected population in which children and adults with HSP were analyzed together<sup>24</sup>. In our series of adults, neither history of drug intake nor older age at disease onset were associated with a higher risk of renal sequelae.

Our results support a basic procedure including a careful clinical history and physical examination, with special search for relapses, full blood cell count, and urinalysis looking for anemia and hematuria, and a followup of at least one year. This procedure may be sufficient to predict longterm renal sequelae in most adults with HSP. However, further studies based on other unselected populations of adults with HSP are required.

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## REFERENCES

1. González-Gay MA, García-Porrúa C. Henoch-Schoenlein purpura. In: Vasculitis. Ball GV and Bridges SL, editors. New York: Oxford University Press (in press).
2. Cassidy JT, Petty RE. Henoch-Schönlein purpura. In: Cassidy JT, Petty RE, editors. Textbook of pediatric rheumatology. 3rd edition. Philadelphia: W.B. Saunders; 1995:384-8.
3. Fogazzi GB, Pasquali S, Moriggi M, et al. Long-term outcome of Schönlein-Henoch nephritis in the adult. *Clin Nephrol* 1989; 31:60-6.
4. Roth R, Wilz DR, Theil GB. Schönlein-Henoch syndrome in adults. *Q J Med* 1985;55:145-59.
5. Lee HS, Koh HI, Kim MJ, Rha HY. Henoch-Schoenlein nephritis in adults: a clinical and morphological study. *Clin Nephrol* 1986;26:125-30.
6. Cream JJ, Gample JM, Peachey RDG. Schönlein-Henoch purpura in the adult. *Q J Med* 1970;39:461-82.
7. Tancrede-Bohin E, Ochonisky S, Vignon-Pennamen M, Flaheul B, Morel P, Rybojad M. Henoch-Schönlein purpura in adult patients. *Arch Dermatol* 1997;133:438-42.
8. Lahita RG. Influence of age on Henoch-Schönlein purpura. *Lancet* 1997;350:1116-7.
9. González-Gay MA, Blanco R, Abreira V, et al. Giant cell arteritis in Lugo (Spain) is associated with low longterm mortality. *J Rheumatol* 1997;24:2171-6.
10. González-Gay MA, García-Porrúa C. Systemic vasculitis in adults in Northwestern Spain, 1988-1997: Clinical and epidemiologic aspects. *Medicine (Baltimore)* 1999;78:292-308.
11. García-Porrúa C, González-Gay MA. Comparative clinical and

- epidemiological study of hypersensitivity vasculitis versus Henoch-Schönlein purpura in adults. *Semin Arthritis Rheum* 1999; 28:404-12.
12. García-Porrúa C, González-Gay MA. Cutaneous vasculitis as a paraneoplastic syndrome in adults. *Arthritis Rheum* 1998; 41:1133-5.
  13. Mills JA, Michel BA, Bloch DA, et al. The American College of Rheumatology 1990 criteria for the classification of Henoch-Schönlein purpura. *Arthritis Rheum* 1990;33:1114-21.
  14. Michel BA, Hunder GG, Bloch DA, Calabrese LH. Hypersensitivity vasculitis and Henoch-Schönlein purpura: A comparison between the 2 disorders. *J Rheumatol* 1992;19:721-8.
  15. Jennette JC, Falk RP, Andrassy K, et al. Nomenclature of systemic vasculitides. Proposal of an international consensus conference. *Arthritis Rheum* 1994;37:187-92.
  16. Blanco R, Martínez-Taboada VM, Rodríguez-Valverde V, García-Fuentes M, González-Gay MA. Henoch-Schönlein purpura in the adulthood and in the childhood: two different expressions of the same syndrome. *Arthritis Rheum* 1997;40:859-64.
  17. Goldstein AR, White RHR, Akuse R, Chandler C. Long-term follow-up of childhood Henoch-Schönlein nephritis. *Lancet* 1992;339:280-2.
  18. Kaku Y, Nohara K, Honda S. Renal involvement in Henoch-Schönlein purpura: a multivariate analysis of prognostic factors. *Kidney Int* 1998;53:1755-9.
  19. Meadow SR, Glasgow EF, White RHR, Moncrieff MW, Cameron JS, Ogg CS. Schönlein-Henoch nephritis. *Q J Med* 1972;41:241-58.
  20. Counahan R, Winterborn MH, White RHR, et al. Prognosis of Henoch-Schönlein purpura in children. *BMJ* 1977;2:11-4.
  21. Lee HS, Koh HI, Kim MJ, Rha HY. Henoch-Schoenlein nephritis in adults: a clinical and morphological study. *Clin Nephrol* 1986;26:125-30.
  22. Uthman I, Kassak K, Nasr FW. Henoch-Schönlein purpura in adulthood and childhood [letter; comment]. *Arthritis Rheum* 1998;41:1518-20.
  23. Kraft DM, McKee D, Scott C. Henoch-Schönlein purpura: A review. *Am Fam Physician* 1998;58:405-8.
  24. Mata-Arnaiz C, Blanco R, Martínez-Taboada VM, García-Fuentes M, Rodríguez-Valverde V. Nephropathy in Henoch-Schönlein purpura: outcome and predictive factors for residual renal insufficiency [abstract]. *Arthritis Rheum* 1999;42 Suppl:1461.