HENOCH SCHONLEIN PURPURA IN AN ELDERLY LADY : A CASE REPORT AND LITERATURE REVIEW

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Summary

An 81 years old lady presented with recent onset of bilateral lower limbs purpuric rash, bilateral ankle arthritis and low grade fever shortly after an upper respiratory tract infection. Henoch-Schonlein purpura (HSP) was diagnosed on the basis of no infection, accelerated ESR (73 mm/h), nephrotic proteinuria (3.75 g/d), microscopic haematuria, normal platelet count, rise in IgA (7.04 g/L), normal serum complement, leucocytoclastic vasculitis on skin biopsy, and negative searches for RF, ANA, ANCA and anti-GBM.

Case Report

A 81 years old lady with past history of renal impairment (urea 13.6 mmol/l, creatinine 124μ mol/l as in July 1995), atrial fibrillation, congestive heart failure, ischaemic heart disease, pseudogout, osteoarthritis of both knees and left cataract with extra-capsular cataract extraction was admitted to Tuen Mun Hospital via casualty in July 1997 because of bilateral lower limb swelling with skin rash for 2 days. She was on regular enalapril, frusemide, isosorbide dinitrate and dologesic (paracetamol + proproxyphene napsylate) for the past 32 months before the episode. She was a subvented care and attention home inmate. She was able to walk with stick with independent activities of daily living.

She got severe bilateral knee and ankle joint pain not relieved by oral analgesics 2 weeks before admission and symptoms of upper respiratory tract infection 2 - 3 days prior to presentation. She sought advice from a private practitioner and was given senokot, agiolax, amoxicillin, paracetamol, konlax (pridinol methanesulfonate) and some intramuscular analgesic injection. She then developed skin rash over both her lower limbs with bilateral ankle edema. She denied no fever, shortness of breath, abdominal pain nor bleeding tendency.

On admission, her general condition was satisfactory. Her oral temperature measured 37.5℃. She had no pallor, jaundice nor lymphadenopathy. Nonblanchable, maculopapular purpuric skin rashes ranging from 1 mm to few cm were noted over both lower limbs. No lesion was found on mucosa or trunk. There was bilateral ankle arthritis and both knees were deformed with crepitus. She got clinical evidence of congestive heart failure, and her blood pressure was at 160/80 mmHg. Physical examination of the other organ systems was otherwise unremarkable.

Baseline investigations showed normochromic normocytic anaemia (Hb 10.7 g/dL), platelet count was 168 x 10^9/l and white cell count was unremarkable with normal differential count. Erythrocyte sedimentation rate (ESR) was elevated to 73 mm/hr. Renal function was impaired (Na 131 mmol/L K 5.3 mmol/L urea 20.6 mmol/L creatinine 168μ mol/L). Blood glucose level was 5.5 mmol/l. Arterial blood gas, liver function tests and coagulation profile were within normal range. Chest radiograph showed cardiomegaly and congested lung fields, electrocardiogram showed atrial fibrillation with ventricular response of 64/min. Urinalysis revealed proteinuria and microscopic haematuria (Protein ++, RBC ++). There was no dysmorphic red blood cells nor red cell cast. There was no significant growth from the urine, sputum and blood.

Additional investigations showed nephrotic range proteinuria (3.75 g/d) and creatinine clearance 25 ml/min. Autoimmune markers including antinuclear antibody (ANA), rheumatoid factor (RF), antineutophil cytoplasmic antibody (ANCA) and anti-glomerular basement membrane antibody (anti-GBM) were all negative. Serology for Hepatitis B and C were negative. Anti-Streptolysin O titre was < 200 IU/ml. Serum cryoglobulin was negative. Her serum IgA was elevated markedly to 7.04 (0.68-3.78) g/L with normal levels of IgG and IgM. Complements (C3 and C4) level were normal.

Ultrasonography of kidneys showed chronic parenchymal disease without focal lesion. Left leg skin biopsy revealed perivascular mixed inflammatory infiltrates rich in neutrophils and nuclear dusts in the superficial dermis consistent with leucocytoclastic vasculitis. Immunofluorescence stain for IgA was however not performed.

Clinical diagnosis of Henoch-Schonlein purpura was made based on nonthrombocytopaenic purpuric skin rash with biopsy confirmed leucocytoclastic vasculitis, markedly elevated IgA level, nephrotic range proteinuria and microscopic haematuria. During hospitalization, enalapril was taken off because of hyperkalaemia and she was then put on hydrallazine, nitrate and diuretics for the control of congestive heart failure. She developed polyarthralgia involving both knees and ankles that could be controlled by nonsteroidal anti-inflammatory agents. She had one episode of haemoptysis and several episodes of fresh rectal bleeding but subsided spontaneously. She also had several episodes of epigastric pain and upper gastrointestinal endoscopy showed diffuse gastric erosion only. Her condition was complicated by acute renal failure with urea climbing up to 19.3 mmol/l. She had marked fluid retention that could only be controlled with large dose of diuretics. Nephrologist was consulted for renal biopsy but that was withheld because of technical difficulty in an orthopnoeic patient.

Her Henoch Schonlein purpura nephritis was treated with supportive measures. Her heart failure was eventually controlled. Her renal function remained static at urea 15.6 mmol/l and creatinine 192μ mol/l with no more new skin eruption appeared. She was discharged after 3 weeks of hospitalization and was able to walk with quadripod upon discharge.

Discussion

Clinical Profile of Henoch Schonlein Purpura

Henoch Schonlein Purpura is a vasculitic syndrome comprising a characteristic skin rash, abdominal colic, joint pain and glomerulonephritis. It was also known as anaphylactoid purpura [5], purpura rheumatica [2], peliosis rheumatica[2].

The syndrome is mainly a disease of early childhood, with most cases presented under 10 years of age [2] (mean age at onset = 6 years) [1]. It occurs rarely in adults, the incidence in adults over the age of 20 years is 0.1 - 1.2 / million [2,9]. Male was affected twice as commonly as female [2]. Recent history of a respiratory tract infection was reported in 90% of cases [1]. Any of the 4 major components of the syndrome may present in advance of the others, but renal disease usually presents late [2]. Cutaneous manifestation presented as first symptom occurred in more than 50% of cases [19] but in 40% of cases, the rash may be preceded for up to 2 weeks by both abdominal pain and arthralgia[1]. The average duration of the illness was 3.9 weeks (3 days - 2 years) [19]. About a third of patients had symptoms for less than 2 weeks, one-third from 2 to 4 weeks and one-third for more than 4 weeks [19]. Prognosis is excellent without sequel in more than 95%. However, attacks may recur for 2 or more years after initial onset [1].

Classical vasculitic rash appears on the extensor surfaces of the arms and legs and over the buttocks and elbows. However, it had been reported that abdomen and chest wall involvement occurred in 54% of cases [30]. Individual lesions are mostly less than 1 cm in diameter, but they may coalesce to form larger discolored patches and disappearing over about 2 weeks [2].

Two-third of patients had arthralgia. It was the presenting complain in one quarter of cases [2]. The ankles and knees are most frequently affected, followed by elbows and joints of the hands [2]. Children usually had large joint arthralgias but adults had more involvement of small joints [30].

Abdominal manifestations occurred in 50 - 78% of patients [2,31] , presenting as abdominal pain, ileus, melaena or haematemesis. More than 30% of patients experienced diffuse pain described as 'bowel angina' typically occurring after meals and accompanied by bloody diarrhea1. Occasionally, the pain could be colicky and simulated an abdominal emergency. Varying degree of intestinal bleeding was present in up to 80% [2]. Intussusception (most often ileocolic) can occur. Other rare manifestations included ileal stricture, pancreatitis, intestinal perforation, hydrops of gallbladder, pseudomembranous colitis[1].

Glomerulonephritis was the most important aspect of the disease as it might result in end-stage renal failure. Haematuria was the earliest sign of renal involvement [2]. Early studies suggested the severity and likelihood of renal impairment could not be predicted from the severity of the non-renal manifestations [19]. However a recent study showed that a recent infectious history, pyrexia, spread of purpura to the trunk, and biological markers of inflammation were predictive factors for renal involvement [10]. The reported prevalence of renal disease in HSP varied from 12 to 92%, most were between 30 and 60% [2]. Urinary abnormalities was present in 25 to 50% of patients, but serious or progressive nephritis was seen in less than 5% [2].

The central nervous system can also be affected

and the most common symptoms are headache, behavioral changes, and seizures. Others included aphasia, hemi- and quadriplegia, chorea, ataxia, and peripheral nerve lesions. They were usually transient and permanent sequelae were rare. However, it is difficult to ascribe these features to vasculitis of the nervous system or hypertension and metabolic disturbances [2].

Other rare manifestations included conduction abnormalities, congestive heart failure, acute myocardial infarction, pulmonary hemorrhage (more common in older patients) [1], interstitial lung diseases, ureterits which may be complicated by stenosis or perforation [2].

There had been reported differences in the clinical presentations of HSP between adults and children. This is tabulated as in Table 1 [7].

Table 1 Clinical features of HSP : childhood (< 20 years) vs adulthood (> 20 years)
**Modified from Blanco-R, et al.* [7]

	Childhood (≤ 20 years)	*Adulthood (> 20 years)*
At symptom onset		
antecedent URTI	++	+
abdominal pain	++	+/-
fever	++	+/-
joint symptoms	+	++
During clinical course		
renal impairment	++	++++
raised ESR	++	+++
melena	+/-	++
haematuria	++	+++

Diagnosis

Diagnosis of HSP is based on the ACR 1990 criteria for diagnosis of Henoch-Schonlein purpura as shown in table 2 [3, 4].

Table 2 American College of Rheumatology 1990 criteria for diagnosis of Henoch-Schonlein purpura : traditional format

Criterion	*Definition*
Palpable purpura	Raised lesions, not related to thrombocytopaenia
Age ≤ 20 yr	Patient age at onset of disease
Bowel angina	Diffuse abdominal pain that is worse after meals, or bowel ischaemia, usually with bloody diarrhoea
Granulocytes on biopsy	Granulocytes in walls of arterioles or venules

(presence of 2 or more criteria is needed to made a diagnosis of HSP)

This ACR criterion yields a sensitivity of 87.1% and a specificity of 87.7% [4]. The disease manifestation that combined the best sensitivity and specificity was palpable purpura, age at disease onset was identified as the second most important criterion [4]. However, this criterion may not distinguish HSP from such disorders as infectious purpura or allergic reactions, which were not included in the control population with other forms of vasculitis during the proposal of the criterion [3, 4]. Thus the diagnosis of HSP should be made by using the ACR criterion and after excluding infectious disease and allergy.

Etiology

In the majority the etiology is uncertain [31] and a history of an intercurrent infection, usually respiratory, at or shortly before the onset of the illness was common [1, 2]. No particular micro-organism had been proved to be specifically involved in the etiology. Also, allergy to drugs including enalapril had been reported [20, 21, 22, 23, 24, 25, 28]. Other exogenous factors like insect bites [28], cancer [2], exposure to cold [28] and blunt trauma [2] had been incriminated, yet not proven in the etiology of HSP. These various stimuli lead to the synthesis of IgA and the alternative pathway of the complement system is activated leading to deposition of immune complexes in various organ systems [11].

Pathology

The characteristic skin lesion is leucocytoclastic vasculitis [5] with perivascular accumulation of inflammatory cells mostly polymorphonuclear leucocytes (PMNs) and mononuclear cells with occasional eosinophils, surrounding the capillaries and post-capillary venules of the cornium. Immunofluorescent staining reveals the presence of IgA, C3 and fibrin/fibrinogen in vessels and connective tissue of clinically involved skin [2]. In clinically unaffected skin, positive staining for IgA and C3 is seen in some, but not all cases, and then only in capillary walls [2]. However, others reported that immune complexes were deposited with equal frequency in normal-appearing and lesional skin of patients with HSP, biopsy of uninvolved skin for direct immunofluorescence studies may be more helpful in confirming the diagnosis because tissue morphology is of better quality [12].

Renal lesion is characterized by focal and segmental proliferative glomerulonephritis and the proliferative process mainly affects mesangial cells with variable epithelial (extracapillary) proliferation leading to crescent formation. The spectrum of renal involvement is highly variable,

from minimal changes to a fulminant, necrotizing glomerulonephritis with 100% of glomeruli surrounded by large crescents (Table 3) [2].

Immunofluroscent and immunoperoxidase microscopy show mainly IgA deposition present in a predominantly mesangial distribution, but in those with poorer histological gradings, i.e. Grade V and VI, it was mainly pericapillary with very little mesangial deposition [16].

Table 3 Classification of Henoch-Schonlein purpura glomerulonephritis recommended by International Study of Kidney Disease in Childhood (ISKDC)

I	Minimal changes
II	Pure mesangial
	(a) Focal
	(b) Diffuse
III	Mesangial proliferative glomerulonephritis with less than 50% crescents
	(a) Focal
	(b) Diffuse
IV	Mesangial proliferative glomerulonephritis with 50-75% crescents
	(a) Focal
	(b) Diffuse
V	Mesangial proliferative glomerulonephritis with more than 75% crescents
	(a) Focal
	(b) Diffuse
VI	Membranoproliferative (mesangiocapillary) glomerulonephritis

Prognosis

The long term prognosis of HSP is almost entirely determined by the behaviour of the nephritis [28] and most available data indicates that the great majority of patients, both children and adults, recover completely without impairment of renal function [2, 30]. The major predictors of outcome was identified by Meadow et al and was shown in Table 4 [2].

Table 4 Meadow (1972) classification of clinical status at follow-up of patients with HSP

Outcome group	
A	***Normal*** Normal physical examination; no urinary abnormality; normal renal function
B	***Minor urinary abnormality*** Normal physical examination; haematuria (microscopic +/- intermittent macroscopic) and/or proteinuria < 1 g/ 24 hr; normal renal function
C	***Active renal disease*** Proteinuria > 1 g/24 hr+/- hypertension; normal renal function
D	***Renal insufficiency*** GFR < 60 ml/min/1.73 m^2; actual or renal death (dialysis or transplantation)

The most benign presentation was isolated haematuria [13]. The more severe renal presentation would be associated with both worse grades of biopsy change on light microscopy (grade IV to VI) and a clinical outcome in category C or D [2]. Patients with acute nephritic syndrome have a 10 to 20% risk of a category D outcome but those with mixed 'nephritic-nephrotic' features at presentation have the worst prognosis in whom half would be in category C or D and a third in category D [2]. However, patients in any of the clinical categories (other than D) may deteriorate between at 2-6.5 years after onset [14], and there was some reservation about the confidence with which long term predictions can be made from clinical features at presentation. Later studies shown that the creatinine clearance at 3 and 5 years correlated best with renal outcome [15]. No patient with a 3-year creatinine clearance above 110 ml/min/1.73 m2 progressed to end stage renal failure (ESRF), but all patients with a 3-year creatinine clearance below 80 ml/min/1.73 m^2 progressed to ESRF [15] in 14 years. At 5 years, those with creatinine clearance below 95 ml/min/1.73 m^2 eventually developed ESRF [15]. However, these studies were done on pediatric population and whether these data could be extrapolated to the geriatric population was still an enigma.

Renal pathology were also found to be correlated with clinical outcome [28] as 70.8-80% of patients with grade V biopsy changes and 23.8-43% of those in grade IV progressed to category D [2,15]. The presence and number of enveloping crescents on light microscopy being the most reliable predictor of clinical course [2, 27].

Concerning the influence of age on outcome, there is no general consensus yet. Two series mentioned that the complications in adults are no more serious than in pediatric group and renal lesion and course are similar in both age groups [28, 30]. One study reported that elderly (>50 years) more frequently had hypertension and elevated serum creatinine and were likely became dialysis dependent [26].

Treatment

No specific treatment has ever been shown to be of any value in the treatment of any aspect of HSP in a controlled fashion. The majority of cases are mild and need only supportive measures. General measures included appropriate antibiotics for associated bacterial infection, careful fluid and electrolyte balance. For extrarenal manifestations, analgesics should be offered for joint or abdominal pain. Appropriate treatment of possible GIB or

intussussception were mandatory. In a cohort study [19], one-third of patients, particularly those with edema and gastrointestinal signs and symptoms, steroids produced immediate and dramatic benefit. Others even advocated that steroids were useful in the acute stage for treatment of symptoms and a course of steroid not more than 4 weeks should be considered if there was not a prompt improvement, especially if renal function was deteriorating [28]. However, there is general consensus that corticosteroids are of limited value in conditions characterized by leucocytoclastic vasculitis [6]. There were few case reports which mentioned about treatment responsiveness to dapsone (joint pain, skin purpura, abdominal pain) and some authors had recommended trial of dapsone for symptom relief and for the possible life threatening complications of gut purpura and severe glomerulonephritis [6]. Several authors also reported that abdominal symptoms and purpura responded to heat-treated, placenta-derived factor XIII concentrate in patients with severe abdominal complications of HSP who had low activity of factor XIII during the acute phase [8].

As most patients with HSP have either subclinical renal disease or only transient urinary abnormalities, thus no treatment is needed in the majority [2]. Even among those with nephrotic syndrome or acute nephritic syndrome, less than half have a poor long-term outcome [2]. The lack of controlled data and the small number of high risk patients both caused the results of treatment difficult to assess. However, blood pressure (BP) control might be beneficial, both during the acute phase and in long term in those with evidence of incomplete resolution of renal disease (persistent urinary abnormalities and/or reduced GFR) [2]. High dose intravenous pulse methylprednisolone, immunosuppressive agents and plasma exchange did not have conclusive benefit [2]. Some even reported steroid treatment was associated with poorer outcome [14, 29]. Nevertheless, patients with ESRF could be offered cadaveric transplant as the last resort [2].

Conclusion

Deducing from the clinical course, the pathogenesis of HSP in this lady was thought to be related to an antecedant upper respiratory tract infection. The drug, amoxicillin, prescribed by the general practitioner shortly before the onset of skin rash may also be responsible as penicillin was cited in the literature to be implicated in the causation of HSP [2]. Enalapril and dologesic were also considered as possible etiological agents as both of them had been reported to be associated with HSP [2,21]. However, from her case history, enalapril was prescribed since November, 1994 for congestive heart failure and dologesic was offered 2 years previously for a pseudogout attack and she was quite well clinically since then until this admission. Moreover, the onset of HSP from the time of introduction of enalapril was was reported to be 5 weeks [21]. Thus, for this elderly lady, the association of HSP with enalapril or paracetamol was quite remote.

Her clinical course was dominated by renal manifestation of HSP leading to worsening of congestive heart failure. Her attacks of polyarthralgia after hospitalization could be exacerbation of pre-existing osteoarthritis or pseudogout or part of syndrome complex of HSP. Periarticular edema without effusion or enlargement of joint space was cited as the cause of joint swelling in HSP[2] and the failure to obtain joint fluid from diagnostic knee joint tapping indirectly support the role of HSP in causing the polyarthralgia but this would never be sure.

She had one episode of haemoptysis which subsided spontaneously, this was ascribed to uncontrolled heart failure as she was still in congestive heart failure at that juncture and the haemoptysis did not recur when she was out of heart failure although the possibility of pulmonary heamorrhage could not be totally excluded. She had several episodes of mild rectal bleeding that were never severe and per rectal examination/proctoscopy did not reveal any mass or haemorrhoid. This could be due to gastrointestinal involvement by the vasculitic process but other colorectal pathology were also possible as she was not offered a lower gastrointestinal work up.

Our patient had mixed nephritic and nephrotic features at presentation and was clinically in Meadow's category D, her outcome was expected to be ominous. Her renal function had been deteriorated further (latest urea 40.4 mmol/l, creatinine 192μ mol/l, creatinine clearance 7.73 ml/min) despite improvement of proteinuria (0.83 g/d). In fact, she had been repeatedly admitted to hospital thereafter due to worsening renal function with fluid retention and congestive heart failure that was consistent with a guarded prognosis.

What is the take home message from this case? Henoch-Schonlein purpura is a vasculitic syndrome also seen in adults but those over the age of 65 years are only rarely affected [31]. Clinical manifestations are protean but nephritis is the most

important aspect of the disease as it can lead to end-stage renal failure. The disease is usually self-limited and there is at present no effective treatment available. Surveillance of blood pressure seems to be a reasonable approach to prevent deterioration of renal function.

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