Austin Journal of Anatomy

Open Access

Austin Publishing Group

Review Article

Henoch-Schönlein Purpura (HSP) – What can we Learn from Biomedical and Clinical Anatomy Practice on this Multidisciplinary Disease?

Muhammad SN[1,2]* and Abdel Meguid E[3]

[1]The University of the West of England (UWE), Department of Health and Applied Sciences (HAS), UK

[2]The Renal Patient Support Group (RPSG), UK

[3]Queen's University Belfast, School of Medicine and Dentistry and Biomedical Sciences, UK

***Corresponding author:** Muhammad SN, The University of the West of England (UWE), Department of Health and Applied Sciences (HAS), The Renal Patient Support Group (RPSG), UK

Received: April 06, 2018; **Accepted:** April 25, 2018; **Published:** May 02, 2018

Abstract

Background: Henoch-Schönlein Purpura (HSP) is the most common vasculitis in children, characterized by triad of symptoms; 1) palpable purpura without thrombocytopenia, 2) abdominal pain, and 3) arthritis. Renal involvement also often occurs in children with HSP. Henoch-Schönlein Purpura (HSP) is the most common vasculitis in children, occurring in 8 to 20.4 per 100,000 children per year.

Epidemiology: HSP usually occurs in children aged between 2-10 years, with 50% of all cases occurred in children aged <5 years, mostly in children aged 4-6 years and occurs more frequently in male. Although it is generally a self-limiting condition, HSP can cause renal manifestations with various incidences. The guidelines and treatment for managing care in HSP patients ranges between centers.

Aims: 1) To provide practical understanding of Henoch-Schonlein Purpura (HSP), 2) Highlight the importance of multidisciplinary laboratory practice for patients with HSP, and 3) Highlight what laboratory practices could be enhanced to support the development of HSP guidelines.

Practice: HSP is a multi-specialty disease wherein a patient's care plan will have laboratory involvement from a variety of disciplines. Laboratory investigations suggest that HSP is not a self-limited disease and patients eventually develop CKD.

Research Stance: In a retrospective study of 141 patients with HSP, Zhao et al. 2015 (9) demonstrated that abdominal pain was not related to HSP Nephritis (HSPN). However, 45% of the patients were complicated with obesity and 29.8% of them had a long disease course. Multidisciplinary laboratory perspectives are paramount in disease follow-up and to help inform clinical decision-making.

Discussion: Future biomedical/ laboratory practices can help tighter clinical decision-making in the care of young people with HSP. More case and longitudinal studies would be helpful to understand whether patients would benefit different care plan options.

Conclusion: Evidence informs that male gender, age greater than 10 years, have symptoms of severe gastrointestinal involvement, arthritis/arthralgia. So, what can we learn from this multidisciplinary disease? Certainly, well-designed and conventionally reported studies in histology, microbiology and hematology laboratory collaborations are important to identify HSP disease development and progression.

Keywords: Henoch-Schönlein Purpura (HSP); Hematology; Anatomy; Histology; Microbiology; Renal

Introduction

Henoch-Schönlein Purpura (HSP) is, characterized by triad of symptoms; 1) palpable purpura without thrombocytopenia, 2) abdominal pain, and 3) arthritis. Renal involvement also often occurs in children with HSP [1]. It is more frequently in male [1,2]. In spite of its being a self-limiting condition, HSP can cause renal manifestations with various incidences [3-9].

The guidelines and treatment for managing care in such patient's ranges between centres [2,3-9]. Long-term prognosis depends mainly on the severity of renal involvement, which may manifest as persistent haematuria, proteinuria, nephritic or nephrotic syndrome, or even renal failure [10]. To prevent or delay end-stage renal disease, identification of early-stage nephritis is crucial. The risk factors associated with renal involvement in HSP are not clear although epidemiologic and clinical features as well as some abnormal

Citation: Muhammad SN and Abdel Meguid E. Henoch-Schönlein Purpura (HSP) – What can we Learn from Biomedical and Clinical Anatomy Practice on this Multidisciplinary Disease?. Austin J Anat. 2018; 5(2): 1083.

Table 1: HSP defining features.

Defining Features
Most common systemic vasculitis in children
Vomiting
Abdominal pain with Gastro-Intestinal bleeding
Arthralgias (Joint Pain)
Haematuria
Proteinuria
Nephrotic syndrome
Systemic small vessel leukocytoclastic vasculitis
Purpuric skin lesions on extensor arms, legs and buttocks
Table adopted from (21)

Table 2: HSP criteria for hospitalization.

Criteria for Hospitalization
• Inability to maintain adequate hydration with oral intake • Severe abdominal pain or significant GI bleeding • Changes in mental status • Severe joint involvement limiting ambulation and/or self-care • Renal insufficiency (elevated creatinine), hypertension, nephrotic syndrome
Table adopted from (21)

laboratory findings have been suggested to have a predictive role [11].

Aims

The aims of this review are: 1) To provide practical understanding of HSP, 2) To provide an understanding of what further multidisciplinary care is required for patients with HSP and 3) To highlight how laboratory practices could be enhanced to support the development of HSP care guidelines.

Epidemiology

Henoch-Schönlein Purpura (HSP) is the most common vasculitis in children, occurring in 8 to 20.4 per 100,000 children per year [5-7]. HSP usually occurs in children aged between 2–10 years, with 50% of all cases occurred in children aged <6 years, mostly in children aged 4–6 years [5,6]. Although it is generally a self-limiting condition, HSP can cause acute gastrointestinal manifestations (bleeding, intussusception), and/or renal manifestations with various incidence [7-8].

Age of onset is a suspect risk factor for renal manifestations in HSP [8]. Aetiology of HSP is unknown, although a variety of antigens such as infections, vaccinations, drugs, foods and insect bites can trigger the onset of HSP [9-11].

The disease is characterized by deposits of Immunoglobulin A (IgA) which contained immune complexes and complement components in small blood vessel walls [12]. It is generally agreed that the incidence of HSP decreases with age [12]. The proportion of children presenting with renal involvement reported in studies varies from 20-10 percent.

Biomedical Markers

There are no specific diagnostic markers of HSP. If present, anaemia is usually a result of gastrointestinal bleeding or associated with acute nephritis. Thrombocytosis is a feature that may be related to disease severity. Renal involvement results in haematuria with or without proteinuria, which may be in the nephrotic range. A rapidly progressive nephritis may result in renal insufficiency and requires histological assessment. Von-Willebrand factor antigen is synthesized by and is present in endothelial cells. High concentrations have been detected in patients with HSP as with other vasculitic diseases probably because of vascular endothelial damage [13-15].

An association with disease activity has a been noted. This may be a useful marker of disease severity. Soluble thrombomodulin is derived from damaged endothelium which may reflect endothelial injury. Raised serum thrombomodulin concentrations have been demonstrated in patients with HSP nephritis [16,17]. Routine coagulation tests are normal while factor XIII activity has been reported to be low, especially in patients with severe gastrointestinal disease [18].

Chronic Kidney Disease (CKD)

Renal involvement rarely precedes the appearance of the purpura and usually occurs within the first 3-weeks of the illness. It ranges from isolated microscopic haematuria, proteinuria, or nephritic-nephrotic syndrome (> 3 g/24 h in adults and > 40 mg/m^2/h in children) to acute, rapidly progressive glomerulonephritis. It can be associated with either high blood pressure or renal failure, or both. Depending on the diagnostic criteria, the frequency of renal involvement varies from 20% to 100% [19]. Overall, an estimated 2% of children with HSP experience renal failure.

In one study, the frequency of chronic kidney disease in adults was estimated to be 1% and that of end-stage renal disease to be < 1%. The frequency of renal failure was reported to be 10% to 20% in adults and 1% in children [19].

Retrospective cohort studies revealed some degree of initial renal involvement in about 20–54% of HSP patients, ranging from isolated haematuria and/or proteinuria (without any abnormality in renal function and blood pressure) to acute nephropathy with renal impairment [20]. Table 1 below summarizes the defining features of HSP. Renal manifestations generally occur over a period of 28 days after the initial presentation of HSP. Gross or microscopic haematuria, proteinuria or nephritic syndrome may occur [7,20]. Table 2 below highlights HSP criteria for hospitalization.

The importance of multidisciplinary laboratory efforts

Streptococcus, vaccines, viral infections (measles, varicella, rubella, hepatitis A, B), tuberculosis, mycoplasma, Bartonella, helicobacter pylori are stated as the triggering factors in the literature [22-25]. This is a multidisciplinary disease and affects the skin, joints, Gastrointestinal System (GIS), kidneys, and in addition more rarely can have an effect on Central Nervous System (CNS), heart and scrotum. Its pathogenesis and causal factors have not been accurately identified yet [22]. Viral and bacterial infections, drugs, vaccines, food allergy and even insect bites have been considered in the aetiology of HSP. It is thus important to appreciate that where supporting clinicians with laboratory diagnosis – HSP requires Multidisciplinary Team (MDT) laboratory practice. Table 3 below provides a summary of the importance of MDT laboratory practice. Table 4 below summarizes HSP clinic-pathological findings.

Table 3: HSP MDT laboratory practice.

Laboratory Investigation	Typical Finding
Full Blood Count (FBC)	FBC may indicate Anaemia and Leukocytosis
Erythrocyte Sedimentation Rate (ESR)	Normal or slightly raised (not indicative for HSP alone)
Coagulation	Normal
Biochemistry	Renal Function may be abnormal with a raised creatinine. Low albumin (might be related to Gastrointestinal Disease)
Abti-Streptolysin O Titre Anti0deoxyribonuclease B (anti-DNase)	Test used to confirm preceding streptococcal infection (this does not exclude HSP as it may precede HSP
Urine Dipstick and protein-creatinine ratio	Haematuria, Proteinuria
Septic Screen	If diagnosis is unclear and purpura present
Table adapted from (21)	

Table 4: HSP clinico-pathological findings.

HSP - Clinico-Pathological Findings
• Significant clinico-pathological differences with IgA nephropathy (25)
• Renal symptoms in 30-70%; some adults develop rapidly progressive glomerulonephritis 70% are ages 2-11 years; rare in adults or infants 1 year or less.
• Higher rate of renal involvement in children ages 10-18 (26). Associated with atopy in 1/3; respiratory infection may follow.
• Related to IgA nephropathy, due to elevated serum IgA, circulating immune complexes with IgA, creates similar kidney
• lesions (27).
• High serum galactose-deficient immunoglobulin A1 level is also demonstrated (28).
• Hypertension, serum creatinine, proteinuria, cellular crescents, glomerular necrotizing lesions and chronic renal lesions are associated with the development of renal failure (29).
• There are variable recurrence rates (12-69%) after renal transplant; however it is not clinically important (30).

Table 5: HSP Microanatomy of the kidney and the small blood vessels.

HSP - Microanatomy of the kidney and small blood vessels	
Acute	
·	Leukocytoclastic vasculitis of small vessels due to deposition of IgAimmune complexes
·	Diffuse proliferation of mesangial cells and matrix without significant involvement of capillary walls or lumin
·	Segmental necrotizing lesions (50%), endocapillary proliferation (13%), cellular crescents, glomerular acute and chronic inflammatory infiltrate
Chronic	
·	Glomerular sclerosis, tubular loss, interstitial fibrosis and hyaline arteriolosclerosis
Skin	
·	Haemorrhage and necrotizing vasculitis in dermal small vessels, which contain IgA
·	Vasculitis is present in other organs but usually NOT kidney
Table adapted from (31)	

Biomedical and clinical anatomy practice

Results of most of the routine laboratory investigations remain within normal limits. A normal platelet count rules out idiopathic thrombocytopenic purpura. A normal platelet count and normal coagulation study results rule out thrombotic thrombocytopenic purpura. A normal lipase level makes acute pancreatitis unlikely. Anti-nuclear factor testing, serum immuno-electrophoresis, anti-neutrophil cytoplasm antibody testing, complement C3 and C4 testing and genetic typing are performed only when overlap syndromes are suspected [19]. When there is palpable purpura, gastrointestinal symptoms, and arthralgia, with or without proteinuria or haematuria the diagnosis of HSP is straight forward.

In Atypical presentations, severe and varied complications, recurrences are more common in adults and children<2 years of age [19].

Reduced glomerular filtration rate is linked with a worse long-term renal prognosis [22-23]. Early identification of impaired renal function is an important part of biomedical practice and disease progression. Haematuria is commonly present in HSP nephritis and usually runs a benign course when isolated [19]. Macroscopic haematuria for >7 days in our cohort was rare and would require further investigation to explore differential diagnoses. Table 5 summarizes the critical microanatomy of HSP so it is better to take into great consideration disease progression and prognosis.

Biomedical practices to support the development of HSP guidelines

Biopsy and histological lesions of HSP nephritis have been grouped by the International Study of Kidney Disease in Children classification into six categories (I, II, III, IV, V, and IV) according to the presence and number of crescents [22].

There is, however, a lack of consensus regarding the utility of crescents as a long-term predictor. Some studies have shown that a high percentage of crescents predicts adverse renal outcomes, [22,23] while others did not [22-25]. Therefore, there is need for a reliable, proven biomedical practice that can support both the development of HSP guidelines and support for clinicians to make more accurately formulated care plans for patients with HSP nephritis [26,27].

In a systematic review by Narchi (2005) [32], children were followed up with the HSP and found to have an excellent overall renal prognosis. The review examined different lengths of follow up after the initial presentation of HSP and showed that only 21 patients (which accounted for 1.8% of the total study population) developed subsequent renal impairment. Follow up with urinalysis and BP measurement is recommended, however the ideal duration of follow up is unclear. Blanco et al. (1995) [33] revealed a favourable short-term prognosis for initial renal impairment in most HSP patients, with complete resolution of 94% in pediatric patients and 89% in adult patients at an average of 18-months follow up.

Discussion

The pathogenesis of HSP remains unknown; however, it is generally considered to be an immune complex-mediated disease characterized by the presence of polymeric IgA1 (pIgA1) containing immune complexes predominantly in dermal, gastrointestinal, and glomerular capillaries [22-25]. The pathognomonic granular IgA and C3 deposits in the mesangium are indistinguishable from those seen in IgA nephropathy. Similar immunohistological findings have also been observed in the kidneys of patients with liver cirrhosis, dermatitis herpetic form is, celiac disease, and chronic inflammatory disease of the lung [22-25]. Where light microscopy is concerned, renal morphology in HSP cannot be distinguished definitively from that in IgA nephropathy, as the glomerular lesions are similar, and vasculitis is virtually never observed within the kidney [34]. With the use of more proliferative lesions there may be more intense staining, often with segmental IgA in capillary walls. Fibrinogen can be seen in the urinary space when crescents are present [35,36].

Using electron microscopy, there are variable visceral epithelial cell foot processes effacements and capillary basement membrane thinning, thickening, and lamellation depending on the degree of glomerular injury [36]. Future biomedical/ laboratory practices can help tighter clinical decision-making in the care of young people with HSP. More case and longitudinal studies would be helpful to understand whether patients would benefit different care plan options [30]. Prognosis is excellent in children (50% have spontaneous remission), but poorer in adults [31] or with nephrotic syndrome; often difficult to predict [12].

Conclusion

Whilst biomedical collaborations are imperative, the most accurate prognostic factors are histological. The percentage of crescents, the presence of interstitial fibrosis, and extension of mesangial deposits correlate with the risk of CKD [36,37]. The risk is highest in children with crescents in more than half of the glomeruli [37]. In adults even fewer than 50% crescents augurs an unfavourable course. The course of persistent renal sequelae and further renal flare-ups cannot be precisely predicted by histology, and long-term follow up is warranted [36,37]. Evidence informs that male gender, age greater than 10 years, have symptoms of severe gastrointestinal involvement, arthritis/arthralgia. So, what can we learn from Biomedical Practice on this MDT disease? Much like Hemolytic Uraemic Syndrome (HUS) where it is a triad clinical syndrome [38], HSP has similar pathological consequences and 'heavy' laboratory involvement. Certainly, well-designed and conventionally reported studies in histology, microbiology and hematology laboratory collaborations are still needed to identify HSP disease development and progression [39-49].

Declarations

Ethical approval and consent to participate

This is a review of Henoch-Schönlein Purpura (HSP) and what can we learn from Biomedical and Clinical Anatomy Practice on this Multidisciplinary Disease. Ethical approval and consent is not required for this work. No participants were involved in this work.

Consent for publication

This is a collaborative review of this important subject entity. Both colleagues have been working together to help inform this review and have agreed between each other/ departments that this work would be invaluable to help inform current practice.

Availability of Supporting Data

This is a review of Henoch-Schönlein Purpura (HSP) and what can we learn from Biomedical and Clinical Anatomy Practice on this Multidisciplinary Disease. Any information and context relating to epidemiology and this has been retrieved through citing the literature, respectively.

Acknowledgement

We would like to acknowledge and dedicate this work to a dear friend/ Renal Patient Support Group (RPSG) co-founder, John Gardner – The best example of what it means to live with HSP.

Authors Information

Shahid is a Specialist Biomedical Scientist and member of the Healthcare Professions Council (HCPC). Shahid is a Fellow for the Institute of Biomedical Sciences (FIBMS) and a Registered Scientist with the Science Council (RSci). Shahid is also a member for the British Blood Transfusion Society (BBTS). Shahid has built experience in laboratory and health research practices and his understanding surrounds the biomedical/clinical and social sciences. Shahid has provided multidisciplinary team working perspectives from laboratory to community and wider through several key publications. Shahid co-founded the Renal Patient Support Group (RPSG) in (2009) which is an evidence-based support group for patients and careers in Chronic Kidney Disease (CKD); the group now has nearly 8000 members globally.

Summary Points

- HSP is truly a biomedical multidisciplinary disease
- HSP is still considered rare, systemic, non-thrombocytopenic vasculitis, affecting mostly children between the ages of 2 and 10 years old. In this age group, the outcome is almost always excellent and requires only supportive care.

- In other age groups atypical presentations are common. Renal complications and, to a lesser extent, gastrointestinal, pulmonary, and neurologic complications can be severe and difficult to diagnose without a biopsy of the affected system.

- Urinalysis is the only investigation required in a classic presentation of HSP. Further investigations may be required if the diagnosis is unclear, abdominal symptoms are severe or where there is evidence of significant renal involvement (hypertension, macroscopic haematuria or proteinuria)

- Most cases are self-limiting and require only symptomatic management. Close follow-up is critical to identify significant renal involvement which request intervention. Such renal involvement can be asymptomatic [28-30].

References

1. Gardner-Medwin JM, Dolezalova P, Cummins C, Southwood TR. Incidence of Henoch-Schönlein purpura, Kawasaki disease, and rare vasculitides in children of different ethnic origins. 2002; 360: 1197-1202.

2. Aalberse J, Dolman K, Ramnath G, Pereira RR, Davin JC. Henoch Schonlein purpura in children: an epidemiological study among Dutch paediatricians on incidence and diagnostic criteria. Ann Rheum Dis. 2007; 66: 1648-1650.

3. Reamy BV, Williams PM, Lindsay TJ. Henoch-Schönlein purpura. Am Fam Physician. 2009; 80: 697-704.

4. Lau KK, Suzuki H, Novak J, Wyatt RJ. Pathogenesis of Henoch- Schönlein purpura nephritis. Pediatr Nephrol. 2010; 25: 19-26.

5. Sinclair P. Henoch-schönlein purpura: a review. Curr Allergy Clin Immunol. 2010; 23: 116-120.

6. Ozen S, Pistorio A, Iusan SM. Paediatric Rheumatology International Trials Organisation (PRINTO). EULAR/ PRINTO/PRES criteria for Henoch-Schönlein purpura, childhood polyarteritis nodosa, childhood Wegener granulomatosis and childhood Takayasu arteritis: Ankara. Part *II*: Final classification criteria. Ann Rheum Dis. 2010; 69: 798-806.

7. 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: 143-153.

8. Peru H, Soylemezoglu O, Bakkaloglu SA, Elmas S, Bozkaya D, Elmaci AM, *et al*. Henoch Schonlein purpura in childhood Clinical analysis of 254 cases over a 3-year period. Clin Rheumatol. 2008; 27: 1087-1092.

9. Clinical analysis of 254 cases over a 3-year period. Clin Rheumatol. 2008; 27: 1087-1092.

10. Zhao YL, Liu ZJ, Bai XM, Wang YC, Li GH, Yan XY. Obesity increases the risk of renal involvement in children with Henoch-Scholein purpura. J European journal of paediatrics. 2015; 1-7.

11. Ronkainen J, Nuutinen M, Koskimies O. The adult kidney 24 years after childhood Henoch-Scho Ènlein purpura: a retrospective cohort study. Lancet. 2002; 360: 666-700.

12. Stewart M, Savage JM, Bell B, McCord B. Long term renal prognosis of Henoch-Schonlein purpura in an unselected childhood population. Eur J Pediatr. 2010; 147: 113-115.

13. Davin JC. Henoch-Schonlein purpura nephritis: pathophysiology, treatment, and future strategy. Clin J Am Soc Nephrol. 2011; 6: 679-689.

14. Fujieda M, Oishi N, Naruse K, Hashizume M, Nishiya K, Kurashige T, *et al*. Soluble thrombomodulin and antibodies to bovine glomerular endothelial cells in patients with Henoch-Schönlein purpura. Arch Dis Child. 1998; 78: 240-244.

15. Hughes FJ, Wolfish NM, McLaine PN. Henoch-Schönlein syndrome and IgA nephropathy: a case report suggesting a common pathogenesis. Pediatr Nephrol. 1998; 2: 389-392.

16. Meadow SR, Scott DG. Berger disease: Henoch-Schönlein syndrome without the rash. J Pediatr. 1985; 106: 27-32.

17. Smith GC, Davidson JE, Hughes DA, Holme E, Beattie TJ. Complement activation in Henoch-Schönlein purpura. Pediatr Nephrol. 1997; 11: 477-480.

18. Ates E, Bakkaloglu A, Saatci U, Soylemezoglu O. Von Willebrand factor antigen compared with other factors in vasculitic syndromes. Arch Dis Child. 1994; 70: 40-43.

19. De Mattia D, Penza R, Giordano P, Del Vecchio GC, Aceto G, Altomare M, *et al*. Von Willebrand factor and factor *XIII* in children with Henoch-Schönlein purpura. Pediatr Nephrol. 1995; 9: 603-605.

20. Nguyen-Ho P, Jewell LD, Thomson AB. Hemorrhagic intestinal Henoch-Schönlein purpura complicated by cytomegalovirus infection. Can J Gastroenterol. 1998; 12: 71-74.

21. Calviño MC, Llorca J, García-Porrúa C, Fernández- Iglesias JL, Rodriguez-Ledo P, *et al*. Henoch-Schönlein purpura in children from northwestern Spain: a 20-year epidemiologic and clinical study. Medicine. 2001; 80: 279-290.

22. McCarthy HJ, Tizard EJ. Clinical practice: Diagnosis and management of Henoch-Schönlein purpura. Eur J Pediatr. 2010; 169: 643-650.

23. Petty RE, Laxer RM, Lindsley CB, Wedderburn LR. Textbook of Pediatric Rheumatology - 7th edition. Elsevier. 2016.

24. Islek I, Kalayci AG, Gok F, Muslu A. Henoch-Schönlein purpura associated with hepatitis A infection. Pediatr Int. 2003; 45: 114-116.

25. Islek I, Muslu A, Totan M, Gok F, Sanic A. Henoch-Schonlein purpura and pulmonary tuberculosis. Pediatr Int. 2002; 44: 545-546.

26. Guissa VR, Aragao PA, Marques HH, Jacob CM, Silva CA. Chronic active Epstein-Barr virus infection mimicking Henoch-Schönlein purpura. Acta Reumatol Port. 2010; 35: 513-517.

27. Guo HX, Zhang JJ, Shi PP, Fu SQ, Zhang LG, Wang M, *et al*. [A clinico-pathological comparison between Henoch-Schonlein purpura nephritis and IgA nephropathy in children]. Zhongguo Dang Dai Er Ke Za Zhi. 2012; 14: 506-509.

28. Tabel Y, Inanc FC, Dogan DG, Elmas AT. Clinical features of children with Henoch-Schonlein purpura: risk factors associated with renal involvement. Iran J Kidney Dis. 2012; 6: 269-274.

29. Stevenson JA, Leong LA, Cohen AH, Border WA. Henoch-Schönlein purpura: simultaneous demonstration of IgA deposits in involved skin, intestine, and kidney. Arch Pathol Lab Med. 1982; 106: 192-195.

30. Kiryluk K, Moldoveanu Z, Sanders JT, Eison TM, Suzuki H, Julian BA, *et al*. Aberrant glycosylation of IgA1 is inherited in both pediatric Ig A nephropathy and Henoch-Schönlein purpura nephritis. Kidney Int. 2011; 80: 79-87.

31. Szeto CC, Choi PC, To KF, Li PK, Hui J, Chow KM, *et al*. Grading of acute and chronic renal lesions in Henoch-Schönlein purpura. Mod Pathol. 2001; 14: 635-640.

32. Sangle N. Kidney nontumor Associated with systemic conditions Henoch-Schonlein purpura. 2012.

33. Narchi H. Risk of long term renal impairment and duration of follow up recommended for Henoch-Schonlein purpura with normal or minimal urinary findings a systematic review. Arch Dis Child. 2005; 90: 916-920.

34. Blanco R, Martinez-Taboada VM, Rodriguez-Valverde V. Henoch-Schonlein purpura in adulthood and childhood: Two different expressions of the same syndrome. Arthritis Rheum. 1997; 40: 859-864.

35. Danilewicz M, Wagrowska-Danilewicz M. Morphometric analysis of glomerular and interstitial lesions in idiopathic IgA nephropathy and Schoenlein-Henoch nephritis. Pol J Pathol. 1997; 49: 303-307.

36. Sinniah R, Feng PH, Chen BT. Henoch-Schoenlein syndrome: A clinical and morphological study of renal biopsies. Clin Nephrol. 1978; 9: 219-228.

37. Rieu P, Noel LH. Henoch-Schonlein nephritis in children and adults: Morphological features and clinico-pathological correlations. Ann Med Interne. 1999; 150: 151-159.

38. Muhammad SN, Abdel Meguid E, Novo R. Haemolytic Uraemic Syndrome (HUS): Clinical Medicine *Versus* Clinical Anatomy. Austin J Anat. 2017; 4: 1064.

39. Yoshikawa N, White RH, Cameron AH. Prognostic significance of the glomerular changes in Henoch-Schoenlein nephritis. Clin Nephrol. 1981; 16: 223-229.

40. Coppo R, Andrulli S, Amore A. Predictors of outcome in Henoch-Schonlein nephritis in children and adults. Am J Kidney Dis. 2006; 47: 993-1003.

41. Pillebout E, Thervet E, Hill G. Henoch-Schonlein Purpura in adults: outcome and prognostic factors. J Am Soc Nephrol. 2002; 13: 1271-1278.

42. Shrestha S, Sumingan N, Tan J. Henoch Schonlein purpura with nephritis in adults: adverse prognostic indicators in a UK population. QJM. 2006; 99: 253-265.

43. Ronkainen J, Nuutinen M, Koskimies O. The adult kidney 24 years after childhood Henoch-Schonlein purpura: a retrospective cohort study. Lancet. 2002; 360: 666-670.

44. Haas M. IgA nephropathy and Henoch-Scho¨nlein purpura, Pathology of the Kidney, 6th edn. Phildelphia. 2007; 423-486.

45. Coppo R, Mazzucco G, Cagnoli L. Long-term prognosis of Henoch-Schonlein nephritis in adults and children. Italian Group of Renal Immunopathology Collaborative Study on Henoch-Schonlein purpura. Nephrol Dial Transplant. 1997; 12: 2277-2283.

46. Schaier M, Freitag J, Dikow R, Sommerer C, Gross-Weissmann ML, Waldherr R, *et al*. Henoch-Schönlein purpura in adults is not uncommon in elderly patients with an adverse prognosis. Clin Nephrol. 2011; 76: 49-56.

47. Clinical Practice Guideline on Henoch-Schonlein Purpura. Royal Children's Hospital Melbourne.

48. Lambers Heerspink HJ, Gansevoort RT, Brenner BM, Cooper ME, Parving HH. Comparison of different measures of urinary protein excretion for prediction of renal events. J Am Soc Nephrol. 2010; 21: 1355-1360.

49. Edstrom Halling S, Soderberg MP, Berg UB. Predictors of outcome in Henoch-Schonlein nephritis. Pediatric nephrology. 2010; 25: 1101-1108.

Austin J Anat - Volume 5 Issue 2 - 2018
ISSN : 2381-8921 | www.austinpublishinggroup.com

Citation: Muhammad SN and Abdel Meguid E. Henoch-Schönlein Purpura (HSP) – What can we Learn from Biomedical and Clinical Anatomy Practice on this Multidisciplinary Disease?. Austin J Anat. 2018; 5(2): 1083.