CASE REPORTS

UDC: 616-053.2:[616.13/.16-002:616.61
DOI: 10.2298/VSP140821028J

Henoch-Schönlein purpura associated with *Strongyloides stercoralis* infection

Henoch-Schönlein purpura udružena sa infekcijom *Strongyloides stercoralis*

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Abstract

Introduction. Henoch-Schönlein purpura (HSP) is a small blood vessel vasculitis, which usually manifests during childhood. The exact cause of the disease is unknown. **Case report.** We reported a 14-year-old girl who had been admitted to our clinic due to the appearance of red macules on her extremities and face, vomiting, and pain in the abdomen and joints. The patient was initially diagnosed with Henoch-Schönlein purpura. At the end of the fourth week of illness, larvae of *Strongyloides stercoralis* were detected in stool samples. The patient was therefore treated with mebendazole, after which all symptoms permanently withdrew. About a month later laboratory examinations were repeated demonstrating increasing signs of renal damage. Kidney biopsy was performed, showing mesangioproliferative glomerulonephritis with crescents and IgA and C3 positive staining in the mesangium. Upon reviewing the clinical presentation, biochemically demonstrated progressive renal damage and biopsy results, the patient was diagnosed with HSP nephritis. **Conclusion.** The time course of the disease and present knowledge concerning the pathogenic mechanisms of HSP suggest that *Strongyloides stercoralis* infection could have caused HSP in the presented patient, which was complicated by nephritis.

Key words:
purpura, shoenlein-henoch; nephritis; strongyloidiasis; diagnosis, differential.

Apstrakt

Uvod. Henoch-Schönlein purpura (HSP) je vaskulitis malih krvnih sudova i obično se manifestuje u detinjstvu. Tačan uzrok bolesti nije poznat. **Prikaz bolesnika.** Prikazali smo 14-godišnju devojčicu koja je hospitalizovana na našoj klinici zbog pojave crvenih makula na ekstremitetima i licu, povraćanja i bola u trbuhu i zglobovima. Kod bolesnice je inicijalno postavljena dijagnoza Henoch-Schönlein purpure. Krajem četvrte nedelje bolesti, detektovane su larve nematode *Strongyloides stercoralis* u uzorcima stolice. Bolesnica je stoga lečena mebendazolom, nakon čega su se trajno povukli svi simptomi. Oko mesec dana kasnije ponovljeno je laboratorijsko ispitivanje koje je pokazalo izražene znake bubrežnog oštećenja. Učinjena je biopsija bubrega i uočen mezangioproliferativni glomerulonefritis sa polumesecima uz pozitivno bojenje na IgA i C3 u mezangijumu. Nakon analize kliničke slike, biohemijski potvrđenog progresivnog oštećenja bubrega i rezultata biopsije, kod bolesnice je postavljena dijagnoza HSP nefritisa. **Zaključak.** Vremenski sled događaja i trenutna saznanja o patogenetskim mehanizmima u HSP, ukazuju na to da je infekcija strongiloidesom mogla izazvati HSP koja se komplikovala razvojem nefritisa.

Ključne reči:
purpura, šenlajn-henohova; nefritis; strongiloidijaza; dijagnoza, diferencijalna.

Introduction

Henoch-Schönlein purpura (HSP) is a small blood vessel vasculitis, which usually manifests during childhood and is characterized by the presence of immunoglobulin A1 (IgA1) deposits [1, 2]. HSP is a self-limiting, systemic, non-granulomatous vasculitis with multiorgan manifestations. The exact cause of the disease is unknown. HSP is the most common vasculitis in childhood [3–5]. Renal affection is the most important aspect of the disease, which determines the outcome and is most responsible for HSP morbidity and mortality rates. Approximately 40% of HSP patients will develop nephritis during the first 4 to 6 weeks of illness [2]. HSP in children progresses to end-stage renal failure in 1% of patients [6–8].

Case report

A 14-year-old girl was admitted to our clinic, due to the appearance of red macules on her extremities and face, vomi-

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ting, and pain in the abdomen and joints. Skin changes soon became partially confluent, transforming into dark purple papules, which did not blanch on compression and did not cause itching. The patient was previously healthy, and from *anamnesis vitae* we learned about allergic reactions to pollen, nutritive allergens and medications. Additionally to the purpuric rash, the physical exam revealed diffuse abdominal tenderness and tenderness of her large joints in the lower limbs. Laboratory tests performed on admission, showed elevated leukocyte count (14.0×10^9/L) with a polymorphonuclear predominance in the formula (94.1%), and increased erythrocyte sedimentation rate (25 mm/h). Basic biochemistry, urine test, ultrasound and radiography examination findings were normal. Due to intense abdominal pain, surgical treatment was considered on several occasions, but physical exam and additional testing did not justify surgical intervention. During the first week of illness, we found elevated blood pressure (above 95 percentile for sex, age and height), elevated level of total serum IgE (182.90 kIU/L), positive proteins (1+) in the mornig urine sample along with microhematuria. In the second week of illness, fresh blood was noted in her stool, which did not appear in the later course of disease. Upon admission the patient was treated with corticosteroids, initially with methylprednisolone, then prednisone, with antihistamine and hypoallergenic diet. Introduction of corticosteroids mitigated abdominal and joint pain. During the third week of illness skin changes completely withdrew and the patient had no other symptoms. In the beginning of the fourth week of the illness, the patient had another burst of purpuric rash on her face, forearms and feet, and complained of abdominal pain, while still on corticosteroid treatment. Repeated tests showed reduced levels of complement components in serum (C3 0.02 g/L, C4 0.0 g/L) and significant proteinuria, (1.6 g/24h) with normal creatinine clearance in the 24-hour urine collection test. Total blood cholesterol, proteins and other biochemical test values were within the reference range. Serum levels of α, γ and μ class of immunoglobulins were also normal each time they were measured. At the end of the fourth week of illness, rhabditiform and filariform larvae of *Strongyloides stercoralis* were detected by direct microscopic examination of stool samples. Mebendazole therapy was initiated accordingly to these findings. After mebendazole treatment abdominal symptoms withdraw completely and there were no new bursts of purpuric rash. About a month later the patient received another course of mebendazole therapy and laboratory examinations were repeated demonstrating increased serum cholesterol level (7.42 mmol/L), positive proteins in the morning urine sample (2+), increased proteinuria (2.92 g/24h) with normal creatinine clearance in the 24-hour urine collection test, and normal serum levels of C3 and C4. Based on these results a kidney biopsy was performed, showing mesangioproliferative glomerulonephritis with crescents and immunohystochemical staining positive for IgA and C3 deposits in the mesangium. Upon reviewing the clinical presentation, biochemically demonstrated progressive renal damage and biopsy results, the patient was diagnosed with Henoch-Schönlein purpura nephritis. The patient was then treated with combined therapy, consisting of prednisone, azathioprine and enalapril. After a month of treatment a complete urinary remission of the disease was achieved, while her cholesterol levels were still elevated. Later evaluation demonstrated normal biochemistry results, apart from persistently increased serum IgE level (256.25 kIU/L).

Discussion

The presented patient was initially diagnosed with Henoch-Schönlein purpura according to the 2010 EULAR/PRINTO/PRES[1] (EULAR – The Europen League Against Rheumatism; PRINTO – Paediatric Rheumatology International Trials Organisation; PRES – Paediatric Rheumatology European Society) criteria [9], based on characteristic palpable purpuric rash without thrombocytopenia, associated with pain in the abdomen and joints and signs of kidney damage. The disease manifested itself dominantly by pronounced gastrointestinal signs, along with biochemically demonstrated progressive renal damage. The treatment with systemic corticosteroids significantly mitigated gastrointestinal symptoms, but could not prevent the development of nephritis as demonstrated in previously published reports [10]. It is well known that corticosteroids can alleviate the symptoms but do not affect the course of the disease, thus prednisone use is not recommended for prevention of persistent renal disease [11, 12]. Henoch-Schönlein purpura aetiology is unclear, but possible causes might be bacterial, viral and parasitic infections [13–17], alterations in secretion of interleukins (interleukin 1 and 6) [18] or growth factors (platelet derived growth factor, transforming growth factor β) [19], as well as vaccination (vaccines for cholera, measles, yellow fever, *Salmonella tiphy* and *paratiphi* A and B) [20]. There are evidence to support the correlation of disease severity with increased serum levels of thrombin-antithrombin complexes, prothrombin factors 1 and 2, von Willebrand antigen and D-dimer [21]. Certain alleles are attributed to increasing likelihood of HSP [22]. The presented patient had several epizodes of rash associated with abdominal pain and a brief period of intermittent appearance of fresh blood in the stool. After detection of *S. stercoralis* larvae in stool samples and upon administering mebendazole, there were no further bursts of rash nor gastrointestinal complaints. Based on the present knowledge, *S. stercoralis* may be considered as the initiator of HSP in the presented patient.

The key role in the pathogenesis of HSP most probably belongs to abnormal IgA1 [23]. The main origin of aberrantly glycosylated IgA1 are the mucosal tissues and bone marrow [24–27]. Therefore, we suggest that infection caused by *S. stercoralis* provoked the production of IgA1 in the intestinal mucosa, which preceded the forming and precipitation of immune complexes containing aberrantly glycosylated IgA1 leading to the development of vasculitis and clinical manifestations of HSP. However, our patient's urine examination showed microhematuria and increasing proteinuria in the 24-hour urine collection test. The presence of galactose deficient gA1 (Gd-IgA1) makes the best distinction between HSP patients who will develop nephritis and those who will not. HSP patients with nephritis have been shown to have increased serum levels of IgG specific for Gd-IgA1, as oppose to

HSP patients without nephritis and healthy controls [28, 29]. The present view is that antiglycan antibodies recognize Gd-IgA1 and form immune complexes that precipitate in the mesangium and then give rise to the process of renal damage by activating complement cascade and initiating leukocytoclastic vasculitis, a process indistinguishable from that in IgA nephropathy [30].

Decreased expression of β1,3-galactosyltransferase and increased expression of α2,6-sialyltransferase has been detected in HSP patients with nephritis, unlike HSP patients without nephritis [31, 32]. IgA1 molecules of healthy people have mono- or disialylated galactosamine-N-acetylgalactosamine (Gal-GalNAc) disaccharide, thus it is possible IgA1 with non-sialylated galactose or N-galactosamine participate in the pathogenesis of HSP nephritis [33, 34] since the clearance of immune complexes containing IgA1 depends on asialoglycoprotein receptor expressed by Kupffer cells [35]. Propensity towards the development of nephritis is linked to HLA-B35 allele [36]. According to previously stated research results, there is a certain genetic predisposition of some HSP patients to develop nephritis.

Recent papers describe cases with *S. stercoralis* infection leading to renal damage and the development of nephrotic syndrome [37, 38], but direct infection of renal parenchym has not been demonstrated in neither of those cases. Pathohistological findings of our patient's kidney biopsy matches those seen in HSP nephritis [39], thus damage by direct infection is unlikely. Later reevaluation of the presented patient's medical status reveals that serum IgE level was still increased, which could be attributed to her atopic constitution. Total IgE is frequently elevated in HSP patients [40], and association of HSP with type 1 hypersensitivity reactions has been so far documented in several studies [41, 42]. During the course of her illness, the patient never had loose stools, maldigestion, eosinophilia, nor any other symptom of infection with an intestinal parasite except abdominal pain, so the abdominal pain might be considered as the only symptom of *S. stercoralis* infection. Abdominal pain is one of the most common manifestations of HSP and about two-thirds of patients complain of this symptom [43], so if we attribute this symptom to HSP, the infection itself could be considered asymptomatic. The presented patient complained of abdominal pain in spite of early introduction of corticosteroids, and the complete withdrawal of symptoms was achieved only after treatment with mebendazole, indicating the significance of strongyloidiasis for the development of HSP in the presented case. Corticosteroids provide for quick resolution of symptoms in HSP patients [44–46], however, patients with strongyloides infection receiving immunosuppressive drugs, and especially corticosteroids, may develop severe hyperinfection or disseminated infection syndrome [47–49]. *S. Stercoralis* is a nematode leading a complex life cycle. It is the only helminth to secrete larvae in faeces and continuously reproduces within the host, therefore *S. Stercoralis* is able to maintain long-lasting persistence by the process of autoinfection causing mild symptoms (cough, bloating, loss of appetite) or no symptoms at all in the majority of infected people [50]. Corticosteroids contribution to the propagation of the autoinfection process could not be ruled out in the presented patient. It also needs to be emphasized that the use of corticosteroids did not lead to clinical presentation of severe hyperinfection or disseminated infection syndrome, but it is probable that corticosteroid treatment created the conditions for *S. Stercoralis* reproduction and increasing their number, which made possible microbiological detection of larvae in stool samples.

S. Stercoralis can persist for more than 30 years without developing a clinically notable disease [51–53]. The risk for severe strongyloidiasis exists for all patients treated with corticosteroids that come in contact with this nematode, while the duration of corticosteroid use is highly variable (4 days to 20 years), as shown by Fardet et al. [47] and is therefore not a major determinant. Having in mind the life cycle and the lack of symptoms in most infected people, we could not know when the presented patient became infected. Laboratory examinations of the presented patient demostrated biochemical signs of renal damage during the first week of illness, and since strongyloidiasis clinical course could be with no symptoms, it is possible that pre-existing strongyloides infection could have caused nephritis, although there are no known mechanisms for parasitic infection to influence the early onset of nephritis in HSP. Some published reports demonstrate remission of nephrotic syndrome after strongyloides eradication [37, 54, 55], implying a causal link between strongyloidiasis and glomerulonephritis.

Conclusion

Strongyloidiasis is not a severe disease in immunocompetent people and its association with HSP has not been previously described. Resolution of clinical signs of HSP in our patient was achieved after treatment with mebendazole, but this intervention did not stop the development of nephritis. The time course of the disease and present knowledge concerning the pathogenic mechanisms of HSP, suggest that *Strongyloides stercoralis* infection could have caused HSP in the presented patient, which was further complicated by nephritis.

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Received on August 21, 2014.
Accepted on March 9, 2015.
Online First March, 2016.