Case report **UDC: 616.16-002:[616.98:578.834**
doi: 10.5633/amm.2024.0311

AN ADULT WITH HENOCH–SCHÖNLEIN PURPURA SECONDARY TO CORONAVIRUS DISEASE INFECTION

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Previous upper respiratory tract infection has been identified as the most common factor causing Henoch–Schönlein purpura (HSP). The most common causes of infection are streptococci, followed by viral infections. Upper respiratory tract infection with coronavirus disease (COVID-19) could be an HSP-triggering virus.

We present a case of a 39-year-old male who developed HSP in the setting of COVID-19 infection. HSP occurred 14 days after COVID-19 diagnosis and it exhibited itself in the form of the lower extremities and buttocks palpable purpura, lower abdominal pain, nausea and hematuria. The patient was treated with methylprednisone, and meprednisone, which led to rapid clinical improvement. Endothelial damage in patients with COVID-19 viral infection occurs as a consequence of a severe inflammatory reaction. Extremely important place in the inflammatory reaction of the endothelium is occupied by IgA, which can be deposited within the endothelium. This activates other cytokines that can lead to HSP occurrence.

Acta Medica Medianae 2024;63(3):90–93.

Key words: *coronavirus disease, Henoch–Schönlein purpura, adults*

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Introduction

Henoch–Schönlein purpura (HSP) belongs to the group of small blood vessel vasculitis caused by the deposition of the IgA-immune complex. Non-thrombocytopenic palpable purpura, abdominal pain, arthritis and renal involvement are clinical tetrads characterized by Henoch–Schönlein purpura (1). Children account for over 90% of all cases, while in adults it occurs much less frequently with a prevalence of 3.4 to 14.3 cases per million. Such a low incidence in adults may be due to misdiagnosis or insufficient diagnosis (2).

The COVID-19 pandemic was initially described as the common cold. Still, later the clinical picture changed with the appearance of many other clinical symptoms, of which pneumonia occupies the most significant place. Numerous biological abnormalities occur in patients with COVID-19 infection, including hypoxemia, hyperinflammation, hemophagocytosis, high creatinine levels, electrolyte imbalance, disorders of the renin-angiotensin-aldosterone system and cytopenia (3). Consequently, COVID-19 infection can lead to various complications affecting the cardiovascular, respiratory and other systems as well as the skin (4).

We present a case of a 39-year-old male who developed Henoch–Schönlein purpura in the setting of COVID-19 infection.

Case Presentation

A 39-year-old patient with a fever that lasted for two weeks, dry cough, shortness of breath, sore throat, diarrhoea and headache was referred to the Clinical Centre Niš, Serbia for treatment. COVID-19 infection was confirmed by PCR after nasopharyngeal and oropharyngeal swabs.

At the patient's admission, the body temperature was 37.4 °C, respiratory rate was 40 breath/min, pulse rate was 106 beats/minute and blood pressure was 140/90 mmHg. Radiological examination diagnosed bilateral severe COVID-19 pneumonia.

Upon admission to the hospital, the patient received the following therapy: intravenous Ceftriaxone 1 gram every 12 hours, azithromycin 500 mg/24h, lopinavir/ritonavir 400 mg every 12 hours, vitamin C 200 mg every 8 hours, zinc

sulphate 220 mg/24 h and subcutaneous low molecular weight heparin of 40 mg daily. The patient was admitted to intranasal oxygen therapy at the rate of 5 litres/min.

On the tenth day from the beginning of hospitalisation, a skin rash appeared, and it was characterised by non-pruritic palpable purpura over the buttocks, lower extremities, upper extremities and the trunk of the body, showing signs of several weeks duration (appearance of the eruption is shown in Figures 1A and 1B). This was accompanied by intermittent, crampy lower abdominal pain, nausea and hematuria. The patient had a fever and arthralgia. There were no clinical signs of superficial thrombophlebitis or deep vein thrombosis.

The absence of thrombocytopenia was confirmed on several occasions by haematological examination. Microscopic haematuria and proteinuria were confirmed by urinalysis.

The value of serum IgA was 787 mg/dL (normal range of 70–400 mg/dL), while tests for vasculitis, and lupus were negative. Other diagnostic methods were not used because the conditions in the COVID hospital did not allow it.

For that reason, the patient started treatment with IV methylprednisolone, 3 g (1 g/day for 3 days), and then meprednisone, 1 mg/kg/day for 10 days. After the applied therapy, the clinical picture improved as well as kidney function and urinary sediment analysis.

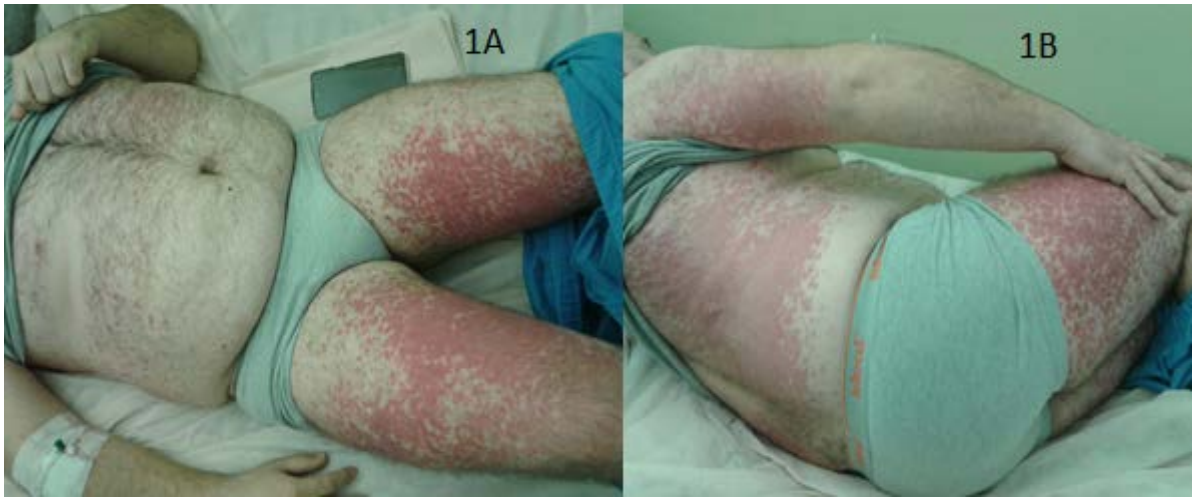

Figure 1. Purpuric rash on the lower limbs of the patient (1A); Purpuric rash on the trunk of the body (1B)

Discussion

Despite a great deal of research, the aetiology of Henoch–Schönlein purpura has not been adequately elucidated to date, however, there are suggestions that several causative agents may be responsible for its occurrence (5). Previous upper respiratory tract infection has been identified as the most common factor causing Henoch–Schönlein purpura. The most common cause of infection is streptococcus, followed by viral infections (6). Upper respiratory tract infection COVID-19 could be an HSP-triggering virus.

IgA vasculitis associated with COVID-19 infection in adults has been reported in several studies. Suso et al. (6) showed the presence of cutaneous vasculitis, nephritic syndrome, and arthritis three weeks after respiratory infection due to PCR-confirmed COVID-19 infection in a 78-year-old man.

Allez et al. (7) describe a 24-year-old man with cutaneous, musculoskeletal and gastrointestinal manifestations of HSP. The patient had an asymptomatic form of COVID-19 infection that was confirmed after PCR testing. Both patients responded successfully to systemic corticosteroid therapy.

IgA vasculitis and COVID-19 infection have also been reported in children. AlGhoozi and AlGhoozi. (8) reported on the clinical presentation of HSP in a four-year-old child who had recently recovered from COVID-19 upper respiratory tract infection.

When the SARS-CoV-2 virus enters the human cell, a systemic inflammatory response is subsequently induced. SARS-CoV-2 IgA is the first immunoglobulin to rise following infection, which may indicate the possible connection between IgA vasculitis and COVID-19 infection (8).

According to the available data, the presence of COVID-19 infection and HSP in adults is exceptionally rare. The published work referred mostly to children and the younger population.

Our patient had the clinical signs and symptoms of HSP, meeting the criteria set forth by the European League Against Rheumatism, the Paediatric Rheumatology International Trials Organization and the Paediatric Rheumatology European Society (9) in 2010.

The patient had non-pruritic palpable purpura predominantly on the lower torso and legs, abdominal pain, arthralgia and impaired renal function.

The IgA serum had a high value, as well. Based on the presented data, it appears that there is a causal relationship between COVID-19 and postinfectious vasculitis.

Conclusion

Endothelial damage in patients with COVID-19 viral infection occurs as a consequence of severe inflammatory reaction. The extremely important place in the inflammatory reaction of the endothelium is occupied by IgA which can be deposited within the endothelium. This activates other cytokines that can lead to HSP occurrence.

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Prikaz bolesnika **UDC: 616.16-002:[616.98:578.834**
doi: 10.5633/amm.2024.0311

HENOH–ŠENLAJNOVA PURPURA KAO POSLEDICA INFEKCIJE COVID-19

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Prethodne infekcije gornjeg respiratornog trakta najčešći su faktor nastanka Henoh–Šenlajnove purpure (*Henoch–Schönlein purpura* – HSP). Najčešći uzročnici infekcije su streptokoke, a za njima slede virusne infekcije. Infekcija gornjih disajnih puteva koju je izazvao COVID-19 mogla bi biti predisponirajući faktor koji izaziva HSP.

Predstavljamo slučaj tridesetdevetogodišnjeg muškarca koji je razvio HSP tokom infekcije COVID-19. HSP se javio 14 dana nakon dijagnoze COVID-19 i ispoljio u vidu palpabilne purpure donjih ekstremiteta i zadnjice, bolova u donjem delu abdomena, mučnine i hematurije. Bolesnik je lečen metilprednizonom i meprednizonom i to je dovelo do brzog kliničkog poboljšanja. Oštećenje endotela kod bolesnika sa virusnom infekcijom COVID-19 nastaje kao posledica teške upalne reakcije. Izuzetno važno mesto u inflamatornoj reakciji endotela zauzima imunoglobulin A (IgA), koji se može deponovati unutar endotela. Ovo aktivira druge citokine koji mogu dovesti do pojave HSP-a.

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***Ključne reči**: koronavirusna bolest, Henoh–Šenlajnova purpura, odrasla osoba*