Henoch-Schönlein Purpura in a Child Following Varicella

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Summary

Henoch-Schönlein purpura (HSP) is one of the most common vasculitides of childhood. It is characterized by nonthrombocytopenic palpable purpura, arthritis, renal and gastrointestinal system (GIS) involvement. HSP is usually triggered by an antigenic stimulus including infectious agents, drugs, cold, insect bite or food. HSP is rarely triggered by Varicella zoster infection. We herein presented a case with HSP following varicella.

Introduction

Henoch-Schönlein purpura (HSP) is a vasculitis of small vessels. It is the most common cause of nonthrombocytopenic purpura in children. The cause remains unknown although there is often an antecedent respiratory infection. Bacterial infections have been implicated but other organisms including virus and mycoplasma have also been reported to precede HSP.1

Case Report

A seven-year-old boy was admitted with purpuric rashes predominantly on the extensor surface of his legs. The bilateral ankle joints were swollen and tender. Ten days prior to the admission, he had been diagnosed as varicella by another physician. Consequently, scattered and crusted lesions of varicella were noted over the face, trunk and legs.

Laboratory examinations showed white blood cell count was 8300/mm³, hemoglobin 10.5 g/dl, platelet count was 422 000/mm³, erythrocyte sedimentation rate 74 mm/h. ASO: 200 U/L. C-reactive protein was within normal limits. Urinalysis and renal function tests were within normal limits. Occult blood test in stool was negative. Prothrombine time (PT) and partial thromboplastin time (PTT) were normal. Immunoglobulin A (IgA), complement levels, anti-nuclear antibody and anti-dsDNA were within normal limits. Urine and throat cultures were negative. Varicella zoster IgM antibody was studied in sera and the result was positive. The skin biopsy from the purpuric lesions revealed leukocytoclastic vasculitis. The purpuric lesions disappeared during the following week. So the patient showed uneventful recovery without signs of renal involvement and gastrointestinal system (GIS). At the end of a three-month follow-up, the patient showed no sign of complication.

Discussion

The pathogenetic mechanisms underlying HSP are poorly understood. However, either infection agents, drugs or allergy substances, by causing changes in patients' mucosa, each may lead to abnormalities in IgA synthesis. As a result, at the capillaries, venules and arterioles of the target organs (such as skin, kidney and GIS), neutrophil, eosinophil, and fibrin accumulate and so they cause leukocytoclastic vasculitis.1

Numerous microorganisms have been implicated as the triggering factor among which the most commonly known are streptococcus, parvovirus, Epstein-Barr virus, Yersinia, and adenovirus. Varicella zoster virus (VZV), however, has been rarely reported as the triggering factor of HSP. Among the rare complications of varicella that are most often reported are encephalitis, Reye syndrome, and disseminated varicella and secondary bacterial infections such as sepsis, pneumonia, osteomyelitis, and impetigo.2 Purpura fulminance, which is highly rarely reported but a serious complication of varicella, was differentiated from HSP in our case as a result of typical HSP clinical findings, PT, PTT normality, and the finding of leukocytoclastic vasculitis at his skin biopsy.3

In the literature so far five cases with HSP in relation with VZV were reported, four following...
varicella \(^4\text{–}^7\) and one during varicella. \(^8\) In our case, the patient was diagnosed as HSP 10 days after the varicella infection and so this report suggests it should be borne in mind that the VZV is a rare agent that triggers HSP. As a conclusion, further study is needed to find out which strains of VZV trigger HSP.

**References**