

²Department of Chest Diseases, Abant Izzet Baysal University Faculty of Medicine, Bolu, Turkey

³Department of Thoracic Surgery, Atatürk Chest Diseases and Thoracic Surgery Training and Research Hospital, Kecioren, Ankara, Turkey

⁴Department of Pathology, Gazi University Faculty of Medicine, Ankara, Turkey

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Corresponding Author: Ersin Gunay, Ataturk Gogus Hastaliklari ve Gogus Cerrahisi Egitim ve Arastirma Hastanesi, Kecioren, Ankara, Turkey, Phone: +90.312.3552110-1050, Mobile: +90.505.5023639, Fax: +90.312.3552135, e-mail: ersingunay@gmail.com

Chickenpox-associated immune thrombocytopenic purpura

Dear Editor,

Thrombocytopenia is a well-known complication of varicella infections. Although various mechanisms have been implicated in its pathogenesis, including decreased bone marrow production of platelets, disseminated intravascular coagulation and virally-induced platelet aggregation followed by phagocytosis or lysis, the main mechanism implicated is immune-mediated platelet destruction. A 5-year-old boy was referred to us due to petechiae and bruises. He was initially diagnosed with chickenpox 6 days earlier. On admission, he had several dozens crusted skin lesions and few drying lesions of varicella. Additionally, he had prominent bruises, mainly on his legs, along with countless petechiae. No wet purpura was noted, while the liver and spleen were not palpable. An initial hemogram showed leukocytes 8,590/ μ l, hemoglobin 12.3g/dl, and platelets 11,000/ μ l. Urinalysis was normal. Initially, no treatment was administered, but next morning due to worsening of the thrombocytopenia (platelets 8,000/ μ l) with increasing hemorrhagic manifestations, he received a single dose of intravenous immunoglobulin at approximately 550mg per kilogram over 2 hours. The next morning, no new hemorrhagic lesions had developed, and since the platelet count had risen to 35,000/ μ l he was discharged home. Three days later, he had a platelet count of 47,000/ μ l. Five weeks later, he had a normal hemogram. Ziebold et al studied non-immunocompromised children and adolescents up to the age of 16 years who were hospitalized with complications of varicella¹. Of the 119 cases that met their case definition, 6 children developed hematological complications, 5 of whom had thrombocytopenia that was persistent in 1. Bovill et al reviewed 613 patients who were admitted with chickenpox over three 5-year periods in London². Platelet counts were recorded in 44 children. Only one boy had a platelet count < 100,000/ μ l. Regarding treatment of varicella-induced thrombocytopenia both corticosteroids and IVIg can be used, the former past the incubation period of chickenpox, because corticosteroids at doses ≥ 0.5 mg/kg/day during the incubation period of varicella can lead to development of visceral varicella. We elected to use low-dose IVIg as a single infusion, although IVIg is traditionally used at higher doses for the treatment of ITP (0.8g-2g), because lower doses have been successfully used and can safely increase the platelet counts at a substantially lower cost. Almost all reports of varicella-induced thrombocytopenic purpura describe quick recovery. However, Ware et al described 3 girls who developed chronic thrombocytopenia in association with varicella³. Hence, in all likelihood varicella-induced ITP is similar to 'idiopathic ITP' of childhood, a disease with an excellent outcome and a 28% likelihood of persistent thrombocytopenia for >6-12 months after diagnosis⁴. To conclude, although thrombocytopenia can be a finding of severe varicella, isolated thrombocytopenia in otherwise healthy children who are recovering from chickenpox has an excellent prognosis.

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Mantadakis E, Thomaidis S, Chatzimichael A

Department of Pediatrics, University General Hospital of Alexandroupolis and Democritus University of Thrace Medical School, Alexandroupolis, Thrace, Greece

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Corresponding author: Elpis Mantadakis, Democritus University of Thrace Medical School and University General District Hospital of Alexandroupolis, 6th Kilometre Alexandroupolis-Makris, 68 100 Alexandroupolis, Thrace, Greece, Telephone: +30-25510-74411, e-mail: eman-tada@med.duth.gr