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A case of immune thrombocytopenic purpura presenting with intracranial hemorrhage

Sinan Akbayram[1*], Fesih Aktar[2], Cihangir Akgün[2], Mehmet Selçuk Bektaş[2], Hüseyin Çaksen[2], Ahmet Faik Oner[1]

[1]Department of Pediatric Hematology, Yuzuncu Yil University Faculty of Medicine, Van, Turkiye

[2]Department of Pediatrics, Yuzuncu Yil University Faculty of Medicine, Van, Turkiye

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ABSTRACT

Immune thrombocytopenic purpura is an acute, generally considered a self-limiting benign disorder with a 60%-80% change of spontaneous recovery occurring usually within a few months after onset. Intracranial hemorrhage is a rare but life-threatening complication of childhood immune thrombocytopenic purpura. We report a 4-year-old girl who admitted with headache, vomiting, bleeding from noise and bruises on the extremities. Her neurological examination was normal. Based on laboratory finding she was diagnosed immune thrombocytopenic purpura and intracranial hemorrhage. We suggest that cranial imaging should be perform in patients with immune thrombocytopenic purpura admitted with bleeding symptoms, vomiting and headache even if they had no abnormal neurological signs.

1. Introduction

Immune thrombocytopenic purpura (ITP) is a disease with an estimated incidence is about 1 in 10 000 children per year[1]. In the majority of children, ITP is an acute, generally considered a self-limiting benign disorder with a 60%-80% change of spontaneous recovery occurring usually within a few months after onset[2]. However, the risk of hemorrhagic manifestations, especially intracranial hemorrhage (ICH), prompts many physicians to consider some form of therapy. Causative factors, predictors, and even outcomes of ICH in ITP have been difficult to ascertain due to ITP with children less than 1% occurrence of this complication[3,4]. Herein, we describe a girl who presented with ICH because of unusual presentation.

*Corresponding author: Dr. Sinan Akbayram, MD, Assistant Professor, Yuzuncu Yıl University, Faculty of Medicine Department of Pediatrics, 65200 Van, Turkiye.
Tel: 0 +90- (505) 272 56 59
E-mail: drsinanakbayram@gmail.com

2. Case report

A 4-year-old girl was admitted to our hospital with headache, vomiting bleeding from noise and bruises on the extremities for 2 d ago. Her vital signs and physical examination were normal with the exception of hemorrhagic manifestations. Complete blood counts were as follows: hemoglobin 8.6 g/dL, white blood cell count 14 350/mm^3, and platelet count 4 000/mm^3. Peripheral blood smear showed thrombocytopenia. Aspiration of bone marrow showed normal erythroid, myeloid cells and megakaryocytes. There were no atypical cells. Cranial computerized tomography (CT) revealed hyperdense areas in the right occipital regions and bilateral basal ganglia, which consisted with hemorrhage (Figures 1).

Based on the clinical and laboratory findings, ITP was diagnosed and a high-dose methyl prednisone (HDMP) treatment (30 mg/kg for 3 d and 20 mg/kg for 4 d), and intravenous immunoglobulin (IVIg; 2 g/kg for 2 d) was given. Thrombocyte suspension was also infused. On the third day of treatment, thrombocyte count increased to 14 000/mm^3, headache and vomiting were gradually regressed. On the 7th day of hospitalization, a thrombocyte count was 57 000/mm^3. The patient discharged from the hospital without any neurological sequel on the seventh day of hospitalization.

3. Discussion

Although it is a rare complication, ICH continues to be the most important cause of significant morbidity and mortality in ITP[3]. A few studies have examined ICH in children with ITP[3]. In a retrospective analysis, the incidence of ICH in children with ITP was found as 0.1%–1.0%[5]. Although there are no definite predictors of which child will suffer an ICH, some factors including history of recent trauma, immunization, viral infection, menstruation, drug ingestion, adolescents with ITP, systemic lupus erythematosis, retinal hemorrhage, 'wet purpura' including other severe muco–cutaneous bleeding, and even cerebral arterio–venous malformation may possible be high risk factors predicting ICH[1,6,7]. In a retrospective study with ICH twelve patients of seventeen cases developed ICH after the onset of ITP however five patients had ICH as our cases on presentation. In the same series, one patient ingested non–steroidal anti–inflammatory drug and four patients had a history of head trauma[2]. We could not identified any predisposing factor in our patient.

Platelet counts of less than 20 000/mm^3 was generally considered risky values for ICH. The threshold platelet count for therapy in ITP is not well known because of the lack of clinical data; however, ICH in children with ITP is not correlated with the severity of bleeding symptoms or with the platelet count at onset[6].

A review between 1975 and 1996 revealed that only 1 of 56 children with complicated by an ICH had a platelet count of more than 20 000/mm^3, 73% of patients had a platelet count of less than 20 000/mm^3 [8]. In our patient, platelet counts were 4 000/mm^3 during ICH. The risk of ICH in patients with ITP cannot be accurately assessed. Instead, we must concentrate our energy on treating patients intensively in whom ICH has occurred. Mortality rate decreased markedly owing to these intensive treatment regimens. Because no therapeutic approach is predominant, platelet transfusions, high–dose steroid and IVIg treatment along with supportive care, splenectomy and neurosurgical intervention are treatment options[1,7]. Our patient received thrombocyte transfusion, high–dose methylprednisolone and IVIg treatment. No surgical intervention was thought.

We suggest that cranial imaging should be performed in patients with ITP admitted with bleeding symptoms, vomiting and headache even if they had no abnormal neurological signs. Secondly, although generally there is no indication in ITP without active or severe bleeding, thrombocyte transfusions can be used in cases of ICH.

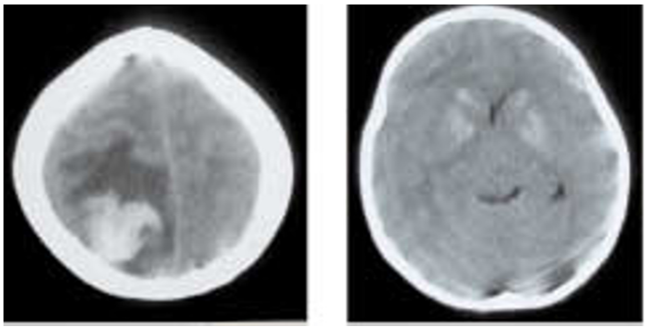

Figure 1. Hyperdense areas in the right occipital regions and bilateral basal ganglia, which consisted with hemorrhage, are shown in A & B.

Conflict of interest statement

We declare that we have no conflict of interest.

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