

Autoimmune thrombocytopenic purpura after mumps infection

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SUMMARY: Ünal Ş, Yetgin S, Kara A, Kanra G. Autoimmune thrombocytopenic purpura after mumps infection. Turk J Pediatr 2005; 47: 270-271.

Autoimmune thrombocytopenic purpura is estimated to be one of the most common acquired bleeding disorders of children. The pathogenesis involves the generation of autoantibodies against the normally expressed glycoproteins on the platelet membranes. These antibody-coated platelets in turn are destroyed by the spleen and other reticuloendothelial organs. Although the disease can occur without an identifiable etiology, many underlying pathologies, including infections, can be found. We report the mumps virus as a rare etiology of secondary autoimmune thrombocytopenic purpura.

Key words: autoimmune thrombocytopenic purpura, mumps, toddler.

Thrombocytopenia is defined as a platelet count of less than 150,000/ μ l. One of the most common acquired causes of thrombocytopenia is autoimmune thrombocytopenic purpura, which occurs when platelets are exposed to premature destruction as a result of autoantibody or immune complex depositions on their membrane surfaces. Although this autoimmune event is secondary to infections, collagen vascular diseases, lymphoproliferative disorders or drugs¹, in most cases no identifiable etiologic factor can be found, and it is thus termed idiopathic thrombocytopenic purpura. Among the many infectious causes of secondary autoimmune thrombocytopenic purpura, Epstein-Barr virus², varicella zoster, human immunodeficiency virus, rubella³, measles, Parvovirus B19⁴, hepatitis viruses and tuberculosis⁵ are the most well known; mumps virus, on the other hand, is a rare preceding pathogen.

The diagnosis of autoimmune thrombocytopenic purpura is made clinically and by exclusion of other causes of thrombocytopenia. The secondary causes of the disease, including infections, drugs and collagen vascular diseases, should be excluded by physical and laboratory findings. The presence of hepatosplenomegaly, lymphadenopathy, bone pain or weight loss in the history, or leukocytosis, leukopenia, anemia or atypical leukocytes in the peripheral blood smear should trigger a malignancy suspicion in a thrombocytopenic patient. These patients, together with the patients who will receive steroid for treatment, should be evaluated with bone marrow aspiration.

The treatment of autoimmune thrombocytopenic purpura in children is controversial because of the high spontaneous resolution rate. The decision is made by the determination of intracranial hemorrhage risk. Treatment includes steroid, intravenous immunoglobulin or anti-D immunoglobulin⁶. All patients should be instructed to avoid activities predisposing to trauma and drugs impairing platelet function. In this report, we present a case with autoimmune thrombocytopenia following mumps infection.

Case Report

A three-year-old girl presented with a head trauma after falling down the stairs. Her past medical history was significant for mumps infection 40 days previously and large ecchymosis after trivial injuries in the last 30 days. On physical examination, her vital signs were normal. Neurologic examination was also normal. Multiple ecchymoses ranging in size from 1 to 1.5 cm were noticeable on all extremities. Laboratory data revealed hemoglobin: 13.6 g/dl, platelet count: 42,000/ μ l, and total white count: 10,300 cells/ μ l. The blood smear was normal except for decreased thrombocytes. The mumps IgM and IgG levels were found to be 2.4 (0-1.1) and 7.4 (0-1.1), respectively, indicating a recent mumps infection. Cytomegalovirus, Epstein-Barr virus, hepatitis A and C, Parvovirus B19, toxoplasma serologies and anti-nuclear antibody, anti-double stranded DNA antibodies were all negative. Hepatitis B immunization was made at 2, 3 and 9 months

of age, whereas no mumps vaccination was present in her medical records. Cranial computed tomography imaging was obtained in order to exclude a possibility of intracranial hemorrhage, since there was a history of head trauma, and no sign of hemorrhage could be identified. The patient's clinical and blood smear findings did not suggest a malignancy, so a bone marrow aspiration was not obtained.

On a follow-up visit after two months her platelet count was found to be 181,000/ μ l without any specific treatment such as intravenous immunoglobulin or steroid.

Discussion

Viral infections may affect hematopoietic cells in very different ways. For example, Parvovirus B 19 infection has recently been associated with transient blastic morphology and CALLA +B cell immunophenotype of acute lymphoblastic leukemia⁷; however, a more common association of viral infections with blood cells is encountered in autoimmune thrombocytopenia. Among the many secondary infectious causes of autoimmune thrombocytopenias, mumps virus is a rare pathogen. However, Tucci et al.⁸ demonstrated a mild subclinical thrombocytopenia in 25% of mumps infections without any hemorrhagic manifestations, in which platelet counts returned to normal spontaneously. The proportion of children whose idiopathic thrombocytopenic purpura was associated with documented acute viral infection was found to be 13.3% in a previous report by Yenicesu et al.⁹ On the other hand, occasional cases of thrombocytopenic purpura have also been reported after vaccination with measles, mumps and rubella^{10,11}. In a case report by Hayashi et al.¹², an adult patient with autoimmune thrombocytopenic purpura following mumps infection was administered a five-day infusion of gammaglobulin and the patient recovered in 35 days. Because of the high rate of spontaneous recovery of autoimmune thrombocytopenic purpura in children and the 42,000/ μ l platelet count in the hemogram (indicating a low spontaneous hemorrhage risk), our patient did not receive any specific treatment and platelet count increased spontaneously without the development of any complication of thrombocytopenia despite her head trauma. The case we report also indicates that many infectious etiologies besides those

that are well known must be evaluated in the history of the patient together with the microbiologic studies. Our patient presented with a 30-day history of widespread ecchymosis on the body, which may suggest that the thrombocytopenia was present before admission to hospital following the head trauma. Although subclinical thrombocytopenia is not uncommon during mumps virus infection like that seen in other viral infections, thrombocytopenia following the infection with clinical findings is rare. Families must be informed about the risk of thrombocytopenia after mumps infection in early ages.

REFERENCES

1. Lee GR, Foerster J, Lukens J, et al. *Wintrobe's Clinical Hematology*, 10th ed, Vol. 2. Philadelphia, PA: Lippincott, Williams and Wilkins; 1999: 1583-1597.
2. Kappers-Klunne MC, van Vliet HH. IgM and IgG platelet antibodies in a case of infectious mononucleosis and severe thrombocytopenia. *Scand J Haematol* 1984; 32: 145-148.
3. Kahane S, Dvilansky A, Estok L, et al. Detection of antiplatelet antibodies in patients with idiopathic thrombocytopenic purpura (ITP) and in patients with rubella and herpes group viral infections. *Clin Exp Immunol* 1981; 44: 49-56.
4. Aktepe O, Yetgin S, Olcay L, Ozbek N. Human Parvovirus B 19 associated idiopathic thrombocytopenic purpura. *Pediatr Hematol Oncol* 2004; 21: 421-426.
5. Al-Majed SA, Al-Momen AK, Al-Kassimi FA, et al. Tuberculosis presenting as immune thrombocytopenic purpura. *Acta Haematol* 1995; 94:135-138.
6. Yetgin S, Olcay L, Ozsoylu S, Hicsonmez G, Gurgey A, Tuncer AM. Retrospective analysis of 78 children with idiopathic chronic thrombocytopenic purpura: follow-up from 1976 to 1996. *Pediatr Hematol Oncol* 1997;14: 399-412.
7. Yetgin S. Parvovirus B 19 infection presenting in two features: transient and progressive blastic morphology with CALLA +B cell immunophenotype of acute lymphoblastic leukemia in children. *Am J Hematol* (Submitted).
8. Tucci PL, Tucci F, Peruzzi PF. The behaviour of platelets in some viral infectious diseases in childhood (author's translation). *Ann Sclavo* 1980; 22: 431-437.
9. Yenicesu I, Yetgin S, Ozyurek E, Aslan D. Virus-associated immune thrombocytopenic purpura in childhood. *Pediatr Hematol Oncol* 2002; 19: 433-437.
10. Autret E, Jonville-Bera AP, Galy-Eyraud C, et al. Thrombocytopenic purpura after isolated or combined vaccination against measles, mumps and rubella. *Therapie* 1996; 51: 677-680.
11. Yenicesu I, Yetgin S, Ozyurek E. Vaccination associated immune thrombocytopenic purpura in five children. *Pediatr Hematol Oncol* 2001; 18: 547-549.
12. Hayashi K, Kawada E, Shinonome S. A case of adult idiopathic thrombocytopenic purpura associated with atypical mumps virus infection. *Rinsho Ketsueki* 1997; 38: 696-698.