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A 20-Year-Old Man with Eosinophilia and Easy Bruisability

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ABSTRACT

Acquired platelet dysfunction with eosinophilia is a rare, benign self-limiting disorder characterised by platelet dysfunction and an association with parasitic infections. A 20-year-old national serviceman who presented with easy bruising and spontaneous epistaxis is reported. Investigations revealed eosinophilia and an abnormal platelet function test similar to that seen in patients with Glanzmann's thrombasthenia infections. He also had evidence of hookworm infestation. Following a course of mebendazole, his symptoms and platelet dysfunction resolved. Hence, this disorder of acquired platelet dysfunction with eosinophilia should be considered in patients with bleeding disorders secondary to platelet dysfunction.

Keywords: acquired platelet dysfunction, eosinophilia, Glanzmann thrombasthenia, parasite

CASE REPORT

A 20-year-old, previously healthy Chinese national serviceman was referred for problem of easy bruising and spontaneous epistaxis for 1 month. He had no past medical or surgical history of note, no family history of bleeding disorders and no history of recent drug or Chinese herb ingestion. The only significant abnormalities noted on physical examination were ecchymoses over his left forearm and knee and right shin.

His initial results were: haemoglobin 13.9g/dL; leucocyte count 8,400/uL with 57% polymorphonuclear cells, 15% lymphocytes, 8% monocytes, 2% basophils and 18% eosinophils; platelet count was 169,000/uL. Blood urea nitrogen, creatinine, protein, albumin, serum for aspartate aminotransferase and alanine aminotransferase were all normal. Tests for prothrombin time, activated partial thromboplastin time and thrombin clotting time were normal. Bleeding time was grossly prolonged at more than 20 minutes and a platelet function test performed on the Cronolog aggregonometer showed decreased aggregation to ADP, epinephrine, arachidonic acid and collagen but normal aggregation to ristocetin. Serum IgE concentration was markedly elevated at 2213 IU/L. Stool examination for parasites was positive for hookworm lava. Thus the results of his investigation indicated a platelet dysfunction disorder. At the same time, he had eosinophilia which was probably related to his parasitic infection. He was treated with a course of mebendazole 100 mg twice a day for three days. Two months later, on review, he no longer had any easy bruisability and had stopped having epistaxis. His repeat blood tests showed disappearance of eosinophilia and normalisation of bleeding time (Table I). A repeat stool examination showed no parasites.

DISCUSSION

An interesting case of acquired platelet dysfunction with eosinophilia is presented here. Initially, a differential diagnosis of Glanzmann's thrombasthenia was also considered. This is an inherited disorder of platelet function characterised by the presence of a normal platelet count and morphology; normal plasma coagulation studies and characteristically prolonged bleeding time with absent or severely diminished platelet aggregation by adenosine diphosphate and other agonists and normal platelet aggregation by ristocetin[1]. Patients with Glanzmann's thrombasthenia classically have gycoprotein IIb-IIIa deficiencies which can be determined by flow cytometry.

However, in view of the relatively recent onset of symptoms of bleeding, presence of eosinophilia and identification of a parasitic infection in the patient, the diagnosis of acquired platelet dysfunction with eosinophilia (APDE) was considered more likely. This is a rare, benign, self-limiting disorder characterised by spontaneous or easy bruising, varying severity of mucosal bleeding, normal platelet count with variable aggregation defects and eosinophilia. It was first described in Thai children and has subsequently been reported in indigenous children and adults in Southeast Asia and India[1-4]. It has also been reported in Caucasian children who had previously visited Southeast Asia[4]. The pathogenesis is unknown although the association with parasitic infections in 50% of the cases suggest that parasitism may be related to the underlying pathophysiology. The prognosis is excellent and the bleeding diathesis in APDE

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Table I – Platelet function test results

Platelet aggregation with	At presentation	8 weeks later
Collagen (3 ug/mL)	absent	normal
ADP (10 umol/L)	markedly decreased	decreased
Epinephrine (338 umol/L)	markedly decreased	normal
Arachidonic acid (2 mmol/L)	absent	normal
Ristocetin (1.5 mg/mL)	normal	normal
Bleeding time (minutes)	> 20 minutes	7 minutes

usually resolves spontaneously in 6 to 12 months[1,3]. The patient was reassessed after a course of anti-parasitic treatment and showed disappearance of initial platelet function abnormalities which confirms the acquired nature of his illness.

This case illustrates the fact that although a patient may have the classically described features of Glanzmann's thrombasthenia, APDE should be considered in the differential diagnosis. Flow cytometry to detect the amount of glycoprotein IIb/IIIa can be easily performed to distinguish the two conditions. Patients with Glanzmann's thrombasthenia have low or absent glycoprotein IIb/IIIa while patients with APDE have normal levels.

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