Acquired platelet dysfunction with eosinophilia (APDE)

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(Key words: Acquired platelet dysfunction, eosinophilia, APDE)

Abstract

Objective To study a series of cases of acquired platelet dysfunction with eosinophilia (APDE).

Design A prospective study of cases of APDE admitted to a paediatric unit from August 1994 to December 2000.

Setting Lady Ridgeway Hospital for Children, Colombo.

Method Children with suspected APDE were investigated and analysed. Investigations included haemoglobin, white cell count, bleeding time, clotting time, platelet count, prothrombin time, partial thromboplastin time, ESR, peripheral blood film, clot retraction, platelet aggregation test, stools full report, filarial antibody test and toxocara antibody test. The platelet aggregation test was repeated after 6 months.

Results There were 12 children with suspected APDE. The male: female ratio was 1.4:1. The ages ranged from 16 months to 10 years. Hess test was negative in all children. The absolute eosinophil count ranged from 1.9-7.8 x 10^9/l. The bleeding time was prolonged in 58% cases. The platelet aggregation test was abnormal in all cases. The filarial antibody test was positive in 17% cases. All other investigations were normal. The platelet aggregation test normalised after 6 months in all cases.

Conclusion APDE is a not uncommon cause of non-thrombocytopenic purpura in Sri Lanka. It is a benign condition with a mild, transient haemorrhagic diathesis and spontaneous recovery occurs within 6 months.

Introduction

Abnormalities of platelet functions, either congenital or acquired defects, can lead to a generalised haemorrhagic diathesis despite a normal number of platelets[1]. Congenital platelet dysfunctions are rare, characterised by lifelong bleeding diathesis and the family history is usually positive[2]. Acquired platelet dysfunctions are more common, the bleeding tendency varying from mild to severe. Most acquired platelet function defects are associated with other pathologic conditions or are due to pharmacologic agents[3]. Acquired platelet dysfunction with eosinophilia (APDE), a syndrome caused by a transient acquired platelet function defect was first described by Mitrakul in 1975[4]. The first fully documented case of APDE in Sri Lanka was published in the Ceylon Medical Journal in 1996[5]. I wish to analyse a series of 12 fully documented cases of APDE seen by me from 1994 to 2000. Though several similar cases were seen by me after 2000, I was unable to document them fully as platelet functional tests were not freely available.

Patients and methods

Children with purpura and eosinophilia with normal platelet counts, admitted to a paediatric unit of the Lady Ridgeway Hospital, Colombo from 1994 to 2000, formed the study population. The following investigations were done; haemoglobin, white cell count (WBC/DC), bleeding time, clotting time, platelet count, prothrombin time, partial thromboplastin time, ESR, peripheral blood film, clot retraction test, platelet aggregation test and stools full report. The filarial antibody test was done in all cases and the toxocara antibody test in 2 cases. The platelet aggregation test was repeated after 6 months in all cases.

Results

12 children presenting with non-thrombocytopenic purpura and eosinophilia were suspected to be cases of APDE. In all cases there was widespread purpura on the trunk, face and extremities. Except for the bruising, all were in good health and asymptomatic with no epistaxis or gum bleeding. Splenomegaly was not present in any of the cases. 7 were male and 5 were female. 5 children were in the 1-3 year age group, 4 in the 4-6 year age group, 2 in the 7-9 year age group and 1 in the 10-12 year age group. The youngest was 16 months and the oldest 10 years with

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a mean age of 4.8 years. There was no past history of bleeding episodes or family history of bleeding and no parental consanguinity in any of the cases. There was no history of recent drug ingestion.

The Hess test was negative in all 12 children. The absolute eosinophil count ranged from 1.9-7.8 x 10^9/l with a mean of 3.9 x 10^9/l. The bleeding time was prolonged in 7 cases and normal in 5 cases. The haemoglobin, clotting time, platelet count, prothrombin time, partial thromboplastin time, ESR, clot retraction and stools full report were normal in all cases. The filarial antibody test was positive in 2 cases. The toxocara antibody test was done only in 2 cases and was negative in both of them. The platelet aggregation test was abnormal in all cases; all of them had absent or reduced aggregation with collagen and ADP and 3 had reduced aggregation with ristocetin. The repeat platelet aggregation test done after 6 months was normal in all cases. All 12 children were empirically given a 2 week course of diethylcarbamazine and a 3 day course of mebendazole. Although fresh purpuric patches appeared in a few cases, all 12 children were completely free of purpura within 6 months of onset.

Discussion

APDE is the most common form of purpura in Thai children and the largest study to date, consisting of 62 cases, was carried out in Thailand by Suvatte et al. in 1979[6]. In this study, the male: female ratio was 1.4:1[6]. In my study, too, the male: female ratio was 1.4:1. In the Thai study, the ages ranged from 19 months to 11 years with a mean age of 6.5 years[3]. In my study, the ages ranged from 16 months to 10 years with a mean age of 4.8 years. Just as in the Thai study, all 12 children in my study were in good health except for the bruising with no history of recent drug intake. In the Thai study, epistaxis was present in 48% children and bleeding from the gums in 13% children[3]. In contrast, epistaxis and gum bleeding were not a feature of my series.

In the Thai study, the absolute eosinophil count ranged from 0.6-28.5 x 10^9/l with a mean of 3.6 x 10^9/l. In my study absolute eosinophil count ranged from 1.9-7.8 x 10^9/l with a mean of 3.9 x 10^9/l. In the Thai study, 67% cases had a prolonged bleeding time and 28% cases had a positive Hess test[6]. In my study, 58% cases had a prolonged bleeding time and none had a positive Hess test. In the Thai study, platelet aggregation to ADP, collagen and ristocetin were abnormal in 68%, 87% and 75% respectively[6]. In my study, platelet aggregation to ADP, collagen and ristocetin were abnormal in 100%, 100% and 25% respectively. In both studies the platelet aggregation to ADP, collagen and ristocetin became normal within 6 months in every case.

The exact mechanism of platelet dysfunction in this syndrome is not known. There are many drugs and chemicals which can cause transient platelet dysfunction such as aspirin, phenylbutazone, indomethacin, antihistamines and dextran[3]. However, none of the children had a history of any drug ingestion or exposure to chemical agents. The possibility of platelet dysfunction due to other pathologic conditions such as uraemia, cirrhosis, glycogen storage could be readily excluded since all children were in good health. The only consistent abnormality found in these children was the peripheral blood eosino-philia. In the Thai study, various intestinal parasites such as roundworm, threadworm and hookworm were detected in the stools of 58% of these children[6]. It has been postulated that children with APDE might have some unique reaction to parasitic infection and produce special immunologic reaction which may cause platelet dysfunction[6]. In my study, the stools full report was normal in all cases. However, the filarial antibody test was positive in 17% of my cases. In a paper on unusual manifeststions of toxocariasis, 5 children, presenting with purpura, prolonged bleeding time, normal platelet count and eosinophilia had abnormal platelet aggregation tests[7]. These were presumably cases of APDE. Unfortunately, in my study I was able to do the toxocara antibody test in only 2 children and it was negative in both of them.

Conclusion

APDE is a not uncommon cause of non thrombocytopenic purpura.in Sri Lanka. It is a benign condition with a mild, transient haemorrhagic diathesis and spontaneous recovery occurs within 6 months.

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