Case Report

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Sideroblastic Anemia in Children Due to Lead Poisoning: A Rare Case Presenting as Short Stature

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Abstract

Sideroblastic anemia comprise a heterogeneous group of disorders characterized by 'Ringed sideroblast' in nucleated erythroid precursors of bone marrow. Lead based paint eating in children is more commonly associated with secondary acquired sideroblastic anemia. A 6 year old male child presented with complaints of feeling of weakness, decreased appetite and short stature. History of pica was present. Examination reveals marked pallor with no hepatosplenomegaly. While approaching a child with short stature, detailed investigations should be done and non endocrine causes should also be considered at the back of our mind.

Keywords: Sideroblastic Anemia; Erythroid precursors; Hepatosplenomegaly.

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Introduction

Sideroblastic anemia comprise a heterogeneous group of disorders characterized by 'Ringed sideroblast' in nucleated erythroid precursors of bone marrow.[1] Sideroblastic anemia is categorized into three groups, these are primary aquired sideroblastic anemia, secondary aquired sideroblastic anemia and hereditary sideroblastic anemia.[2]

Primary acquired sideroblastic anemia is a refractory anemia with ringed sideroblast.[3] It is a form of myelodysplasia, occurs in elderly subject.[3] Children eating a lead-based paint is the usual cause of secondary acquired sideroblastic anemia. Hereditary sideroblastic anemia is usually X-linked disease. There is reduced activity of delta amino levulinic acid synthetase, an enzyme in heme biosynthetic pathway.[4]

In sideroblastic anemia, body has sufficient iron but is not able to use it into hemoglobin synthesis which carries oxygen in blood.[5] As a result iron accumulates in mitochondria of red blood cells giving a ringed appearance to the nucleus.

Case report

A 6-year old male child presented to us with complaints of feeling of weakness, decreased appetite and short stature. History of pica was present. Physical examination revealed marked pallor with no hepatosplenomegaly. On anthropometry, height of child was 102 cm and weight was 12 kg. Height of father is 162 cm and of mother is 145 cm. Mid-parental height was calculated, it came to be 162 cm. As the child was born and vaccinated in our medical college hospital, his birth records and height/weight records upto the age of 1 year were available. Length of the patient at birth was 49 cm, weight was 2.7 kg and head circumference was 34 cm. At the age of 1 year length was 74 cm and weight was 8.6 kg.

Laboratory Investigations showed hemoglobin 6.4 gm/dl, total leucocyte counts 7800/Cu mm,

platelet counts 231,000/Cu mm. On peripheral blood smear examination, microcytic hypochromic picture was seen and retic count was 1.6. Serum ferrirtin was 84 ng/ml. Liver function test was done, serum total bilirubin came to be 0.9 mg/ dl, SGPT and SGOT were 28 IU/L and 21 IU/L respectively. Serum creatinine was 0.7 mg/dl and Serum LDH was 181 U/L.

The patient was given empirical trial of hematinics which did not give a significant response. At this stage, Hb electrophoresis, G-6-PD screening, direct Coomb's test, osmotic fragility were advised but revealed no abnormality. Later on, bone marrow examination was done which showed 'Ringed sideroblast'.

On further inquiry, father was painter by occupation and child used to eat lead- based paint.

Blood levels of lead was sent which came to be 42 mcg/dl. Diagnosis of Secondary acquired sideroblastic anemia due to lead-based paint eating was confirmed. Treatment was started with pyridoxine and patient gives significant improvement within few weeks of treatment. On follow up after 1 year, height of patient was 108 cm (increased by 6 cm in 1 year), weight was 14. 2 kg and Hb was 10.2 gm/dl.

Discussion

Sideroblastic anemia is a rare disorder characterized by ' Ringed sideroblast' in bone marrow. Lead based paint eating in children is more commonly associated with secondary acquired sideroblastic anemia. Anemia that accompanies lead poisoning is the result of various inhibitory effects of lead on heme biosynthesis. Most steps in heme biosynthetic pathway are inhibited by lead to a varying degree. It also interferes with breakdown of RNA by inhibiting enzyme pyrimidine 5' nucleotidinase.[6]

Sideroblastic anemias are characterized by fatigue, breathing difficulties and feeling of weakness. Clinical findings include pallor and icterus.

Laboratory findings are suggestive of low Hemoglobin concentrations on CBC. Leukocutes and platelet counts are usually normal. On Perpheral blood smear, microcytic hypochromic picture with basophilic stippling is seen.[5,6] Lead blood level is diagnostic. The upper limit of normal level of lead in blood is 30 mcg/dl. Patient with concentration of 30 to 75 mcg/dl show decreased ALA dehydratase activity, increased urinary excretion of ALA, and increase in protoporphyrin and may have non specific mild symptoms of lead poisoning. Clear symptoms usually are associated with concentrations that exceed 75 mcg/dl. Concentrations in urine greater than 0.1 mg/24 hour also establish the diagnosis lead intoxication.

Bone marrow examination shows iron granules on Prussian blue stain. Some of granuels may encircle the nucleus of erythrocytes to form ringed sideroblast[5]. Erythropoetic activity is bone marrow is increased[5]. Thus marrow is hypercellular but number of circulating reticulocyte is not elevated[5].

Elimination of a toxin or drug and mineral/ vitamin supplementation (copper or pyridoxine) can lead to recovery. To remove excess iron from the body with sideroblastic anemia, the drug desferrioxamine (Desferal) is often used. Desferri oxamine binds excess iron and promotes its excretion from the body.

Conclusion

While approaching a child with short stature, non endocrine causes should also be considered at the back of our mind. Many times a child with short stature is referred to endocrinologist but before that detailed investigations should be done. In our area, microcytic hypochromic anemia is mostly due to iron deficiency as malnutrition and iron deficiency is highly prevalent. Hence it is natural tendency to assume microcytic hypochromic anemia as iron deficiency anemia. Treatment of iron deficiency anemia and sideroblastic anemia is different.

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