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Case Report

Severe iron deficiency anemia in an infant: A case report

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ABSTRACT

Iron deficiency anemia (IDA) is the most common nutritional deficiency disorder in children and is worldwide in distribution. In fact, iron deficiency is the only micronutrient deficiency that is prevalent in virtually all developed countries. An infant is predominantly fed on milk, bioavailability of breast milk is much better than cow's milk; although both are deficient in iron content. The peak prevalence of nutritional IDA occurs in late infancy and very rarely seen before the age of 6 months in a term baby who is exclusively breastfed. We report a case of severe IDA in a 6-month-old child in whom no other obvious cause was found.

Key words: *Breast-fed babies, Iron stores at birth, Infant, Microcytic hypochromic anemia, Severe iron deficiency*

Iron deficiency anemia (IDA) is a major public health problem in infants and young children worldwide, in both developed and developing countries. According to the World Health Organization (WHO), there are two billion people with anemia in the world and half of the anemia is due to iron deficiency [1]. Infancy is a period of rapid growth, and consequently, high iron requirements. Worryingly, iron deficiency with or without anemia in early childhood has been shown to have long-term consequences for cognitive, motor, and behavioral development [2]. From 3 months to 3 years, poor dietary intake with excess cow's milk diluted or undiluted is the usual cause of IDA, and nutritional IDA very rarely occurs before the age of 6 months in exclusively breast-fed baby [3]. We report a case of severe IDA in an exclusively breast-fed child at the age of 6 months.

CASE REPORT

A 6-month-old male child presented to us with a history of decreased oral intake and progressively increasing pallor for the past 3 weeks and increasing irritability for 1 week. The child was the product of non-consanguineous marriage, delivered vaginally at full term with birth weight of 2.7 kg. There was no history of neonatal jaundice or any other blood loss. Child was exclusively breastfed till 5 months of age and started on complementary feeding for 1 month. The past history was not significant, and his development was normal. His family history was not contributory.

On examination, child had severe pallor, heart rate was 130/min, respiratory rate 40/min, and his weight was 6 kg at 3rd percentile and length 59 cm (<3rd percentile). On general examination, no icterus, bruising, petechiae, rash, or lymphadenopathy was seen. Extremities were well perfused without any edema. Neither facial dimorphism nor any other musculoskeletal anomaly was seen. His systemic examination was normal without any evidence of congestive heart failure or hepatosplenomegaly.

Hematological investigation revealed hemoglobin (Hb) 3.5 g%, mean cell volume 49.4 fl, and red cell distribution width (RDW) increased to 25.40%. Peripheral smear revealed severely microcytic hypochromic blood picture and reticulocyte count of 1.5%. The total leucocyte count was normal, and mild thrombocytosis was present. Serum ferritin was <1.5 ng. Stool (occult blood) and urine examination did not reveal any blood loss. Hb electrophoresis, HbF, HbA_2 estimation, G-6-PD screening, and direct Coomb's test did not reveal any abnormality. Liver function and renal functions were also normal. We did not have extra pre-transfusion samples to do complete iron studies as a child was very pale and minimum sampling was planned.

The child had a history of kajal application in the eyes throughout 6 months, so blood lead levels were done and it was 4 µg/dL less than the level of concern which is 5 µg/dL. In view of introduction of wheat diet, TTG was done, but it was negative (1U/mL). Hence, a final diagnosis of Iran deficiency anemia was made, patient received packed RBC transfusions and discharged on hematinic. On the day 5 of hematinic therapy, reticulocyte percentage increased to 5.5%. Repeat HB done after 1 month of hematinic showed a rise of 1 g%. Follow-up iron studies are planned, and the patient has been kept in follow-up. The plan was to repeat studies after 90 days of blood transfusion.

DISCUSSION

The predominant cause of microcytic hypochromic anemia in infancy and child age group is IDA. IDA and beta thalassemia trait are close differentials for microcytic hypochromic anemia, and coexistence of both the conditions is not uncommon. Low serum

ferritin in association with high RDW is a helpful parameter to distinguish IDA from thalassemia, and high-performance liquid chromatography (HPLC) confirms the state of thalassemia if present. After the correct diagnosis of IDA, it remains a challenge to identify the underlying cause. In infants, nutritional iron deficiency, for example, due to prolonged (>6 months) breastfeeding, is frequent. Prolonged exclusive breastfeeding (EBF) has been associated with IDA at 9 months of age and later and for low birth weight infants, at 6 months of age [4]. Term babies are iron replete at birth with an iron concentration of around 80 mg/kg body weight compared with 50-55 mg/kg body weight of adult male [5].

Due to rapid growth, there is increased demand for iron in infants, and therefore, they are vulnerable to develop IDA at this point of time. Prematurity, neonatal infection, blood loss, repeated samplings, neonatal hyperbilirubinemia, cow's milk intake, and bottle feeding are a few predisposing factors for the development of IDA in infants before 6 months. Cow's milk may contribute the iron deficiency because of poor bioavailability, replacement of iron rich food in diet, presence of inhibitors of iron absorption in milk itself, and sometimes causing low-grade but chronic hemorrhage by irritating gastrointestinal (GI) lining. Nutritional iron deficiency is never responsible for anemia in term infants before the age of 6 months as per Brugnra et al. study [3]. Nutritional IDA is often compounded by chronic blood loss from parasitic infestation and malaria in endemic regions in later infancy and toddlers. In IDA, lead absorption is increased, and lead inhibits absorption of iron compounding severity of anemia.

Our patient was exclusively breastfed till 5 months never received cow's milk and did not have any neonatal risk factors for the development of anemia. Stool occult blood was negative on two occasions ruling out possibility of chronic GI blood loss. There was no evidence of hemolysis and family history, and HPLC ruled out hemoglobinopathy. Although the literature suggests that nutritional iron deficiency rarely manifests clinically before 6 months of age, Calvo et al. [6] suggested in breast-fed babies even before 6 months of age, IDA can occur as: (1) Depletion of body iron stores occurs at around 4 months of age unless replenished by adequate exogenous supply of iron, and (2) introduction of semisolid food has been implicated as a cause of marked decrease in bioavailability of iron from breast milk because of presence of inhibitors in it. Our patient was started on semisolids since the age of 5 months and did not receive any iron supplementation compounding the iron deficiency. We could not do complete iron studies, but low serum ferritin in combination with high RDW is the most accurate indicator of IDA. Being acute phase reactant, high ferritin level requires correlation with other parameters for diagnosing the cause of microcytic hypochromic anemia, but low levels are specific for iron deficiency.

Iron-refractory IDA, a rare genetic disorder with autosomal recessive inheritance where patient exhibits increased predisposition to develop iron deficiency and present at an earlier age. These patients have nearly complete lack of response to oral iron and slow incomplete response to parenteral iron therapy [7]. Diagnosis is suspected in the absence of response to oral iron and persistence of severe anemia requiring multiple transfusions. Genetic mutational analysis establishes diagnosis. Keeping the diagnostic possibility in mind, the patient has been kept in close follow-up. This case emphasizes the role of iron supplementation in term and preterm babies for prevention of anemia. The American Academy of Pediatrics and WHO recommend iron to be started in all term breast-fed babies at the age of 4 months at 1 mg/kg/day and continued till iron rich food introduced till around 6 months of age and in preterm babies at dose of 2-4 mg/kg/day since the age of 2 weeks until 6 months [8,9]. Iron fortified supplementary food products, promotion of EBF till 6 months of age, and delayed introduction of cow's milk and complimentary feeding not before 6 months are other measures suggested for the prevention of IDA.

CONCLUSION

Low serum ferritin in combination with high RDW is a reliable indicator of IDA, and these may indicate IDA even in breast-fed infants. This case emphasizes the role of iron supplementation in term and preterm babies for the prevention of anemia.

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