

Case Report

A rare case of isoniazid induced sideroblastic anemia

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ABSTRACT

Sideroblastic anemia is a rare cause of anemia. Most of it accounts for the genetic cause, while drug induced is still uncommon. Our patient, a 20 year old female, is a known case of right frontal tuberculoma on ATT presented with complaints of generalized weakness and loss of appetite. On evaluation, she was found to have severe anemia and bone marrow studies confirmed it to be sideroblastic anemia. On revisiting the history, it was noted that she was not taking pyridoxine supplements as advised along with antitubercular drugs. Our patient is one among the few documented cases of Isoniazid induced sideroblastic anemia. This case needs attention because it is a preventable cause of anemia and the clinicians need to be aware about the compliance of the patient with the supplementary drugs.

Keywords: Antitubercular treatment, Isoniazid, Pyridoxine, Sideroblastic anemia

INTRODUCTION

Sideroblastic anemias comprise a heterogeneous group of disorders characterized by amorphous iron deposits in the erythroblast mitochondria giving rise to ringed sideroblast. It occurs due to defective heme synthesis.¹ Sideroblastic anemia can be hereditary, acquired or secondary.² Isoniazid induced sideroblastic anemia is a rare type of secondary sideroblastic anemia.³ This case is being reported with the objective of raising awareness among the clinicians in order to prevent development of sideroblastic anemia in patients on antitubercular treatment.

CASE REPORT

A 20 year old female with history of right frontal tuberculoma status post craniotomy, on anti-tubercular treatment (ATT) for 1 year presented with complaints of severe generalized weakness, loss of appetite, dark colored

stools and weight loss since the last 3 months. On physical examination, she was afebrile and she had pallor. Systemic examination did not yield any positive findings. Laboratory investigations revealed severe anemia (Hb-3.4 g/dl), peripheral smear showed microcytic hypochromic picture, bilirubin levels were within normal limits, anemia profile showed high iron levels (255.8 µg/dl), normal vitamin B12 (736 pg/dl) and folic acid levels (16.7 ng/ml). Direct and indirect Coomb's tests were negative. Reticulocyte count was normal (2.5%). Stool occult blood sample sent, one out of the three samples was positive. Upper GI endoscopy and colonoscopy was done to evaluate the cause of melaena but both came normal. Duodenal biopsy was normal. To further evaluate for the cause of anemia bone marrow biopsy was done to establish the cause of anemia, which showed erythroid hyperplasia with dyserythropoiesis, iron stores 3+ with presence of ring sideroblasts with ineffective erythropoiesis, these features were likely to be drug induced. After receiving the bone marrow report, history was reviewed, it was noted

that she was not taking pyridoxine supplements as advised along with ATT. Based on the history, bone marrow findings and other supportive laboratory results, a diagnosis of Isoniazid induced sideroblastic anemia was made. Patient received multiple blood transfusions, subsequently her hemoglobin levels rose to 10.2 g/dl on discharge. Isoniazid was then stopped and the ATT regimen was modified and the patient was put on pyridoxine supplements.

DISCUSSION

Sideroblastic anemias are a heterogeneous group of disorders characterized by amorphous iron deposits in the erythroblast mitochondria giving rise to the ring sideroblasts, pathognomonic of the disease. Eventually leading to an iron overload state (erythropoietic hemochromatosis).

Sideroblastic anemia can be caused due to hereditary, acquired or secondary causes.⁴

Drugs like isoniazid, pyrazinamide and cycloserine interfere with vitamin B6 metabolism leading to drug induced SA, which is a reversible cause of secondary sideroblastic anemia.^{5,6} Isoniazid inhibits ALA synthase, which is the rate limiting enzyme of heme synthesis (by blocking pyridoxine which is a cofactor of the enzyme) hence dysregulating heme synthesis, eventually leading to accumulation of iron in mitochondria of nucleated RBCs (sideroblasts) and hence anemia.⁷

Isoniazid induced SA incidence is very rare and there are only a few cases reported in the literature.³ Isoniazid induced sideroblastic anemia is rarely seen due to the co administration of pyridoxine supplements.⁸ In our case, the patient's malcompliance to the pyridoxine might have led to sideroblastic anemia in her. A similar case of isoniazid induced sideroblastic anemia was reported by Piso et al in Switzerland, where when the patient was improved on pyridoxine supplementation.⁹ Haden et al have showed regular increase of hemoglobin levels in patients receiving isoniazid on supplementation of pyridoxine.¹⁰ All these studies emphasize the need of ensuring the compliance of the patient's towards pyridoxine tablets.

CONCLUSION

Isoniazid induced sideroblastic anemia, though rare, is a potential cause leading to severe anemia. This case is being

reported with an intention that even though isoniazid induced sideroblastic anemia is a treatable rare cause of anemia, clinicians should be judicious enough to check the compliance of patients' to pyridoxine tablets.

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