

Case Report

Bronze baby syndrome an infrequent complication of phototherapy: a case report

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ABSTRACT

Phototherapy is a common mode of treatment for neonatal hyperbilirubinemia. Bronze baby syndrome is an infrequent complication of phototherapy. The characteristic feature is a grayish-brown discoloration of the skin, plasma and urine. The pigment responsible for the discoloration has not been properly identified and pathogenesis remains still unelucidated. The bronze color disappears spontaneously and the findings are compared to other causes of dusky-hued skin in the neonate. The bronze baby syndrome is usually innocuous but can signal life-threatening disease for which investigations should be performed routinely.

Keywords: Bronze baby syndrome, Neonatal hyperbilirubinemia, Phototherapy

INTRODUCTION

Neonatal jaundice is a very common problem in first 2 weeks of life and phototherapy is the usual mode of treatment. Bronze baby syndrome is a less common complication of phototherapy. Bronze baby syndrome is the greyish brown pigmentation of skin, mucous membrane, and urine following phototherapy.¹ It is assumed that abnormal accumulation of photoisomer of bilirubin and hepatic dysfunction leading to copper porphyrin complex which is photo destroyed during phototherapy leading to brown pigmentation of the skin and mucous membrane.² Sometimes accumulation of biliverdin may also cause this brown pigmentation. It is probably due to a reduction in hepatic excretory function of bilirubin photoproducts.^{3,4}

CASE REPORT

A term 2.49 kg birth weight, female neonate was born to a 29-year-old multipara mother by LSCS in our hospital.

Baby is a product of consanguineous marriage. Antenatal period was uneventful. She cried immediately after birth and didn't require any resuscitation. Baby was noticed to have yellowish discoloration of eye and skin on day 2 of life. Serum total bilirubin level was 16.4 mg/dl with direct bilirubin 1.1 mg/dl. She was treated with single surface phototherapy. Serum bilirubin level increased on next day to a total bilirubin of 18.9 mg/dl with direct component 1.4 mg/dl. So, baby was put under double surface phototherapy. Later towards the end of the day it was noticed that she had a greyish brown discoloration of the entire body (Figure 1).

Phototherapy was continued for 48 hours and was stopped when bilirubin level was below phototherapy range. She was diagnosed to have bronze baby syndrome and was evaluated for a possible cause. She was passing dark colored urine and dark yellow colored stool. Her liver function tests results were within normal range. Ultrasonogram (USG) of the abdomen was performed to rule out any liver and biliary pathology which was

normal. Baby's metabolic parameters were also normal with a normal blood sugar. Her urine was negative for reducing substances. HIDA scan was also normal. CBC result showed Hb=13.2 and rest of the values being normal. Reticulocyte count was 6%.



Figure 1: Greyish brown discoloration of skin during phototherapy.



Figure 2: Normal skin color after 3 weeks.

There is ABO incompatibility in blood grouping with blood group of mother being O+ve and that of baby is B+ve.



Figure 3: Brownish dark color urine during phototherapy.

In peripheral smear, there were no signs of hemolysis. After 48 hr of phototherapy serum bilirubin level was 8.9

mg/dl with direct bilirubin being 1.1 mg/dl which was below phototherapy range. So, baby was discharged and kept in regular follow up. After 3 weeks of discharge there was spontaneous disappearance of the greyish brown pigmentation of the skin with a normal urine color.



Figure 4: Normal color urine in follow up.

DISCUSSION

It is assumed that abnormal accumulation of photoisomer of bilirubin is the cause of bronze baby syndrome.² The second cause postulated is abnormal hepatic function leading to copper porphyrin complex which is photo destroyed leading to brown pigmentation.³

The third explanation is an accumulation of biliverdin leading to brown pigmentation. It is probably due to a reduction in hepatic excretory function of bilirubin photoproducts. The pigment accumulated in the body may be polymerized and forms bilifuscin like substances following a free radical reaction.² It should be differentiated from gray baby syndrome exclusively seen in neonates and very young infants receiving high doses of chloramphenicol with clinical features of cyanosis, acidosis, cold peripheries and marked hypotonia.⁶

No treatment is usually required for bronze baby syndrome as the pigmentation slowly disappears after stopping phototherapy.⁵ The prognosis of bronze baby syndrome depends on the causative liver diseases like bile duct atresia, galactosemia and parenteral nutrition with cholestasis for which investigations should be performed routinely.⁶

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient's parents have given their consent for their baby's images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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