Carbon Baby Syndrome - A Rare Case

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ABSTRACT

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Carbon baby syndrome, also known as universal acquired melanosis is one of the causes of diffuse hyperpigmentation of skin and mucosa with only a limited number of reported cases in the literature. Here we report a case of universal acquired melanosis with a early presentation. A 26 day old male child born of nonconsanguineous marriage was brought to our department with complaints of progressive darkening of whole body since birth. His mother had uneventful prenatal, natal and postnatal periods. The increased pigmentation, which the parents noticed, started in the face and hands at the age of 20 days and then gradually progressed over the next 6 days to involve the whole body.

On examination, the child had generalized diffuse hyperpigmentation of entire body. Palms and soles were also involved. Histopathology of skin showed increased melanin pigmentation in basal and suprabasal layers of epidermis and a few melanophages in the dermis suggestive of carbon baby syndrome.

Keywords: Acquired universal melanosis, Neonate, Melanin

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INTRODUCTION

Acquired universal melanosis, an extremely rare condition is characterized by progressive pigmentation of the skin during childhood, resembling that seen in black races. The major determinant of normal skin colour is the melanin, produced by melanocytes of basal layer of epidermis. Melanin production normally is not maximal in the newborn skin. Baby skin is slightly tanned for few days after birth and it turns into racial colour after few months due to repeated sun exposure.[1] As far as we know the reporting of this rare condition, universal acquired melanosis, at such an early age, has not been reported in literature.

A 26 day old male child born of consanguineous marriage was brought to our department with complaints of progressive darkening of whole body. His mother had uneventful prenatal, natal and postnatal periods. The increased pigmentation was noticed by the parents in the face and hands at the age of 20 days and then gradually progressed over the next 6 days to involve the whole body. There was no history of fever, skin infection, darkening of urine, photosensitivity or any other systemic complaints. History of any prior drug intake was absent. Family history was not present.

On examination, the child had generalized diffuse hyperpigmentation of entire body **(figure 1)** Palms and soles were also involved. The baby had branded marks on the abdomen inflicted with a hot iron rod when the baby was taken to the tribal chief as a method of curing this condition. No abnormality of hair and nails was detected. Hyperpigmentation was more pronounced over sun-exposed areas. Growth parameter and developmental milestones were within normal limits. No other system was involved. On ophthalmoscopic examination, retina and fundus were normal. Routine blood and urine examinations were within normal range. No abnormalities were detected in liver function tests and thyroid function tests. Serum corticotrophin was in normal range and there was no diurnal

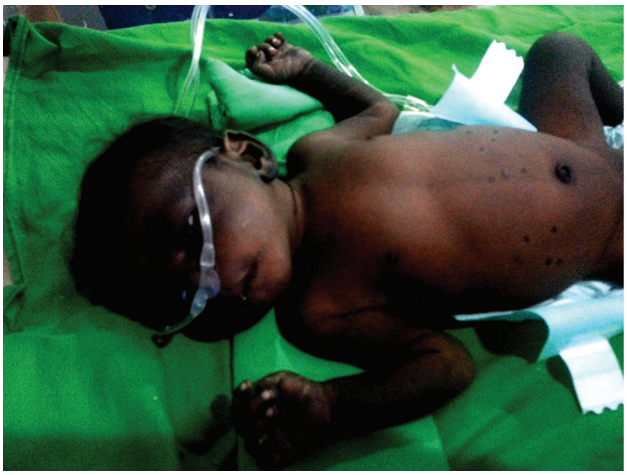

Figure 1. **Generalized diffuse hyperpigmentation of entire body**

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variation. Histopathology of skin showed increased melanin pigmentation in basal and suprabasal layers of epidermis and a few melanophages in the dermis. The clinico-histopathological features were suggestive of carbon baby syndrome.

Our patient did not have any evidence of inflammatory condition. We also excluded systemic conditions like Addison's disease, heavy metal toxicity and hemochromatosis from the history, clinical features and laboratory findings. Since early presentation (around 26 days) is not reported in any of the literature, we find it unique of reporting this case.

DISCUSSION

There are multiple causes of diffuse hyperpigmentation of the skin in infancy. Classifications based on both clinical and histological findings increase the accuracy of diagnosis.[1,2]

Ruiz-Maldonado described a single case of progressive hyperpigmentation in which a child developed pigmentation at the age of three months and became jet black by the age of 4 years. He described him as "carbon baby".

Histological examination of this patient showed heavy melanin deposition throughout the epidermis with minimal dermal pigmentation. There was no increase in the number of melanocytes[3]. Our patients showed similar clinical and histopathological features.

Kaviarasan et al.[9] reported a similar case in a 3-year-old Indian girl who developed progressive diffuse hyperpigmentation by the age of 5 months. Histopathology revealed increased melanin deposition in epidermal basal layer.

Furuya and Mishima[4] reported a Japanese child with progressive pigmentary disorder. This child developed hyperpigmentation at the age of 3 months. At 4 years of age the child was mentally retarded with partial hyperpigmentation of the body. Biopsy revealed hyperkeratosis, papillomatosis and proliferation of melanocytes.

Familial progressive hyperpigmentation has been described in kindred. This condition is characterized by hyperpigmented patches that are present since birth and increase in size and number as the infant grows. Most of the skin and mucous membrane surface show increased pigmentation. Microscopically, the melanin granules are more numerous and larger than normal.[5] Familial progressive hyperpigmentation was excluded in our patient as the skin lesions were not seen at birth.

Kint et al.[6] described two cases of congenital diffuse melanosis in which the patients developed hyperpigmentation shortly after birth which invaded progressively the trunk and limbs. The pigmentation was diffuse on the abdomen but reticulated on the neck and groin. On electron microscopy, they found the melanosomes were not grouped within the keratinocytes but dispersed within the cytoplasm of the epidermal cells.[6]

Bronze baby syndrome is a rare acquired generalized pigmentary disorder which occurs in the neonates. It is characterized by gray brown discoloration and occurs in patients with hepatocellular dysfunction undergoing phototherapy. Porphyrin compound undergoes photo destruction which results in a brown substance that is deposited in the skin.

Adrenoleukodystrophy,[7] an X-linked acquired neurodegenerative disease characterised by generalized hyperpigmentation with a slowly progressive involvement of the brain and adrenals. Patient develops uniform macular hyperpigmentation which spares the palms and groin area. This disease is characterized by the accumulation of unsaturated fatty acids with a chain of 24-30 carbons, particularly hexacosanoate in the adrenal cortex and in certain sphingolipids of the brain.[8]

Our patient did not have any evidence of inflammatory condition. We also excluded systemic conditions like Addison's disease, heavy metal toxicity and hemochromatosis from the history, clinical features and Laboratory findings.

END NOTE

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