Acquired universal melanosis (Carbon baby syndrome) in a 4-year old girl

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Conflict of interest: none to declare

INTRODUCTION

Carbon baby syndrome, also known as universal acquired melanosis is one of the causes of diffuse and progressive hyperpigmentation of skin. It involve entire body without any systemic findings. We report the first case of universal acquires melanosis from Iran in a 4-year-old girl.

CASE REPORT

The patient was a 4-year-old female child born to consanguineous parents. The darkening of skin started over the neck and face at the age of 2 months and gradually generalized. There was no history of drug intake prior to the onset of skin lesions, urine discoloration, photosensitivity, infection, or any other systemic complaints.

On the physical examination, the child had generalized dark hyperpigmentation of the skin (Figure 1) including palms and soles while the mucosa was spared. The hair pigmentation, skin texture, and sweat secretion were normal. There was no other evidence of primary or secondary skin changes. Blood pressure was 100/65 mmHg, and she weighed 16kg. The physical and mental development and ophthalmologic examination were normal. Lab tests including CBC, Iron, ferritin, urine amino acids, liver and thyroid function tests, serum adrenocorticotropin hormone, electrolytes, and abdominal ultrasound were reported as normal.

Skin biopsy of the hyperpigmented skin of the trunk revealed excessive melanin pigmentation of the epidermis basal layer with melanophages in the dermis. No alteration in the shape or size of melanocytes was observed. There was no family history of discoloration of skin. After excluding other possible causes of hyperpigmentation, on the basis of clinicohistopathological features, the diagnosis of “carbon baby” was made.

Universal acquired melanosis is a rare cause of diffuse hyperpigmentation of skin and mucosa during childhood. There are only few reported case of this scarce syndrome in medical literature. We report the first case of universal acquires melanosis from Iran in a 4-year-old girl whose skin becomes darker after 2-month of age.

Keywords: Carbon baby, hyperpigmentation, universal acquired melanosis

DISCUSSION

The etiology of Universal acquired melanosis is unknown and while many conditions may present with generalized hyperpigmentation, one needs to rule out other problems before making the diagnosis of carbon baby. As a rare condition and with the lack of knowledge about its genetic inheritance or pathophysiology, its diagnosis is one of the exclusion. Adrenoleukodystrophy, bronze baby syndrome (grey-brown discoloration in the neonates with hepatocellular dysfunction) \(^1\), Addison’s disease, heavy metal toxicity, hemochromatosis, and familial progressive hyperpigmentation can lead to generalized hyperpigmentation \(^2\) and must be ruled out to diagnose carbon baby syndrome.

There are only a few case in English literature that describe the universal acquired melanosis. In 1978, Ruiz-Maldonado et al \(^3\) reported an unusual case of hyperpigmentation in a boy who developed generalized hyperpigmentation at the age of three months and became totally black at the age of four-year. Increased melanin in epidermal basal layer with minimal dermal pigmentation without increase in number of melanocytes were seen in the histological examination. Kaviarasan et al \(^4\) reported an Indian girl born to non-consanguineous parents who developed generalized pigmentation at the age of five-month and became black by the age of three-year. Kumar et al \(^5\) reported a five-year-old Indian girl with progressive generalized hyperpigmentation since three months of age. Chakraborti et al \(^6\) reported this condition in two Indian siblings (brother and sister) of a non-consanguineous parents which started at the of age six year. Shome et al \(^7\) reported this condition in two siblings with consanguineous parents started at the age of five-month and four-month in male and female siblings, respectively. Recently, a nine-year-old boy with universal acquired melanosis started at the age of four-month who was born of non-consanguineous parents was reported from India. Our case was the first case reported from Iran with the same histopathological findings as other reported cases.

Almost all cases are from India and Pakistan which may point to the environmental and genetic factors in pathogenesis of the disease. The condition observed in siblings born to consanguineous and non-consanguineous parents may emphasis the possibility of genetic mutation and some recessive heritage pattern. However, the scarcity of the cases make the precise decision on etiology and heritage pattern impractical. Moreover, prognosis of the disease, its effect on other aspects of health and quality of life, and its association with other abnormalities or conditions is not clear and needs long-term follow-up.
REFERENCES