

Hyperpigmentation in Primary Adrenal Insufficiency

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A 42-year-old man, Fitzpatrick skin type 5, presented to our clinic with progressive darkening of skin, generalized weakness, recurrent episodes of nausea, vomiting, and abdominal pain for the last 6 months. He also reported an unintentional weight loss of 8 kg. Other personal, family, and drug histories were non-contributory. On clinical examination, pulse rate was 108 beats per minute, blood pressure reading was 96/60 mm of Hg, peripheral oxygen saturation was 95% on room air and capillary blood glucose was 74 mg/dL. Cutaneous examination revealed diffuse greyish-black pigmentation all over the body with accentuation over the dorsae of hands, palmar creases, and knuckles. Similar hyperpigmented coalescent macules were also noted in the hard palate and buccal mucosa (Fig. 1). Other mucocutaneous sites were unaffected. Other systemic examinations were unremarkable. Basic laboratory investigations revealed electrolyte imbalance—sodium level: 112 mmol/L (reference value, 136–145); potassium: 5.6 mmol/L (reference value, 3.5–5.1). Suspecting Addison's disease, specific investigations were carried out—early-morning cortisol level: 67.7 nmol/L (reference range, 140–690); serum corticotropin: 38.4 pmol/L (reference range, 2.2–13.5). Primary adrenal insufficiency or Addison's disease was diagnosed based on corroborative clinical and biochemical findings. Treatment with glucocorticoids (oral hydrocortisone 15 mg/day in two divided doses) and fludrocortisone (0.1 mg/day) was started. Significant amelioration



Fig. 1: Generalized hyperpigmentation more predominant on gingival mucosa and palmar creases

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of presenting signs and symptoms was appreciated on follow-up at 2 months.

In developed nations, Addison's disease is most commonly associated with auto-immunity, but in developing countries such as India, underlying tuberculosis needs to be ruled out in all cases. The condition is typically linked with difficulty swallowing, tiredness, loss of weight, low blood pressure, abdominal discomfort, absence of menstruation, queasiness, vomiting as well as fragile nails, sparse body hair, and thinning hair.¹ In Addison's disease, the lack of negative control over the hypothalamus results in increased release of corticotrophin-releasing hormone and proopiomelanocortin. Consequently, this elevation leads to heightened melanin production by epidermal melanocytes, resulting in skin and mucosal hyperpigmentation.¹ A wide variety of causes like vitamin B12 deficiency, drugs, cancer, HIV infection, and autoimmune diseases need to be ruled out in patients presenting with diffuse melanosis.^{2,3} Generalized hyperpigmentation is a very important diagnostic feature and helps to differentiate primary from secondary adrenal insufficiency. The typical pigmentary feature in Addison's disease is the affliction of sun-exposed areas and pressure points. In patients with darker skin tones, this hyperpigmentation is often missed. Oral pigmentation is commonly observed as

the first indication and tends to emerge before dermatological pigmentation. The systemic symptoms of the disease typically advance gradually and an illness or accident can exacerbate the condition, potentially leading to a life-threatening crisis.^{4,5}

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