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CASE REPORT

# Addisonian pigmentation associated with acute lymphoblastic leukemia: A rare paraneoplastic phenomenon

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## Abstract

Addisonian pigmentation usually presents with nonspecific symptoms and signs, which are often ignored or misdiagnosed as a sign of other more common diseases. We present a case of 12-year-old child in whom diffuse Addisonian hyperpigmentation of skin was associated with underlying acute lymphoblastic leukemia (B-type), a rare paraneoplastic phenomenon in hematological malignancies.

**KEYWORDS**
acute lymphoblastic leukemia, Addisonian pigmentation, paraneoplastic dermatosis

## 1 | INTRODUCTION

Classical Addisonian pigmentation is characterized by generalized black to brown hyperpigmentation, accentuated on sun exposed areas, over pressure points and palmar creases along with nail and oral mucosal pigmentation. This type of pigmentation is also rarely seen as a paraneoplastic phenomenon in hematological malignancies, such as leukemias and non-Hodgkin lymphoma.[1] It has not been previously reported in acute lymphoblastic leukemia (ALL), to our knowledge.[1–5]

## 2 | CASE REPORT

A 12-year-old male presented with generalized dark brown to black discoloration, developing initially on both hands 2 years previously and gradually generalizing over the previous 6 months (Figure 1). It was associated with generalized weakness and myalgia, loss of appetite, and nausea. His parents reported significant weight loss of ~7 kg in the last 3 months, not associated with orthostatic hypotension or lightheadedness. There was no history of tuberculosis in the child and his family. There was no history of arthritis or dark-colored urine.

On cutaneous examination, diffuse dark brown hyperpigmentation was present all over the body. There was buccal, palatal, and labial hyperpigmentation. Palmar creases and nails were also involved (Figure 2). Differential diagnosis included Addisonian pigmentation, hemochromatosis, alkaptonuria, vitamin B12 deficiency, hyperthyroidism, sarcoidosis, and lichen planus pigmentosus. Detailed laboratory investigation included complete blood count with ESR, vitamin B12 levels, thyroid profile, iron studies, liver and kidney function tests, urinalysis, serum ACTH and serum cortisol levels, triple viral serology including human immunodeficiency virus, hepatitis B and C, serum electrolytes, abdominal ultrasound, electrocardiogram, and chest radiograph. He was found to have anemia (Hb 7 g/dL), leukocytosis (25,000/mm$^3$), elevated ACTH levels, that is, 466 pg/mL (normal 10–50 pg/mL), and decreased 8 AM serum cortisol levels of 2 μg/dL (normal 5–25 μg/dL). The remaining investigations were negative or within normal limits. Skin biopsy from diffuse generalized pigmentation present over the abdomen showed hyperkeratotic epidermis with increased melanin pigmentation in the basal layer and stratum spinosum with a mild perivascular lymphocytic dermal infiltrate, consistent with Addisonian pigmentation (Figure 3). He was further evaluated to rule out underlying etiology. The bone marrow examination showed presence of >90% lymphoblast cells which were diagnostic of acute lymphoblastic leukemia (ALL). Further confirmation was done by flow



  