

## Lichen Striatus Following BCG Vaccination in an Infant

For more than 70 years, Bacillus Calmette-Guerin (BCG) vaccines have been administered safely to billions of individuals throughout the world. Local adverse reactions following BCG vaccination usually occur at a rate of 0.1 to 0.5 per 1000 vaccinations, and serious, disseminated complications occur at a rate of less than 1 in a million vaccinations [1]. Physicians should be aware of the various adverse effects of this commonly used vaccine for proper management. We herein present an infant who developed lichen striatus (LS) following BCG vaccination.

A 7-month-old girl was brought with linearly arranged hypopigmented skin lesions of four month duration along the lateral aspect of her left upper limb. The lesions typically followed the lines of Blaschko (*Fig. 1*). She was asymptomatic and there was neither any history of pruritis, drug intake nor any infection preceding the eruption. The birth history was normal. There was history of atopic dermatitis in the mother. She was given BCG vaccine at two and half months of age and the skin lesions started two weeks after immunization. The patient's relatives did not give consent for a skin biopsy. A diagnosis of lichen striatus was made on the basis of her clinical history and physical examination. The WHO causality assessment criteria for adverse events following immunization suggests that the adverse event (lichen striatus) was *probably* related to BCG vaccination [2]. We treated her with emollients only, without any other topical therapy. She is well on follow up after 2 months with the skin lesions still persisting.

Lichen striatus is a rare, benign, self-limited linear dermatosis that predominantly affects children between 5 months and 15 years of age [3]. It has been reported to occur after infections and following immunization with MMR or hepatitis B. These act as potentially triggering factors with the skin lesions usually appearing over 2 to 3 weeks after the event [3]. The lesions are usually pink or flesh-coloured,



**FIG. 1** Linearly arranged hypopigmented skin lesions along the lateral aspect of left upper limb typically following the lines of Blaschko.

lichenoid papules that are arranged in continuous or interrupted bands. Rarely, patients can have hypopigmented macules and papules, as seen in our patient. Such lesions are classified as lichen striatus albus [4]. Lichen striatus is clinically diagnosed on the basis of its appearance and characteristic developmental pattern following the lines of Blaschko, which are thought to correspond to the migration of embryonic skin cells [3,4]. The etiology of LS is unknown. Atopy may be a predisposing factor and upto 85% of patients with LS have a positive history of atopic disorders [3]. The lesions are most commonly located on a proximal extremity and less commonly on the trunk, head, neck, or buttock. Lesions are usually asymptomatic with pruritis being a rare complaint. The mean duration of the disease is 6 to 9 months [3,4]. No specific treatment is required. Some studies have mentioned the beneficial role of topical corticosteroids and tacrolimus ointment in the treatment of lichen striatus with prolonged course or multiple lesions [4,5]. The caregivers should be reassured about the benign nature of the disease.

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### REFERENCES

1. Grange JM. Complications of Bacille Calmette-Guérin (BCG) vaccination and immunotherapy and their management. *Commun Dis Public Health*. 1998;1:84-8.
2. Collet JP, MacDonald N, Cashman N, Pless R. Monitoring signals for vaccine safety: the assessment of individual

- adverse event reports by an expert advisory committee. Advisory Committee on Causality Assessment. Bull World Health Organ. 2000;78:178-85.
3. Dragos V, Mervic L, Zgavec B. Lichen striatus in a child after immunization. A case report. Acta Dermatovenerol Alp Panonica Adriat. 2006;15:178-80.
  4. Moore K, Lam JM. The toddler with 1 striped leg: a linear papular rash. CMAJ. 2009; 28;180:947-8.
  5. Vukiaevia J, Milobratovia D, Vesia S, Milosevicia Jovacia N, Ciriã D, Medenica L. Unilateral multiple lichen striatus treated with tacrolimus ointment: a case report. Acta Dermatovenerol Alp Panonica Adriat. 2009;18:35-8.

## Propionic Acidemia Presenting as Diabetic Ketoacidosis

Propionic acidemia is a rare, autosomal recessive, inherited inborn error of propionate metabolism presenting as life threatening ketoacidosis progressing rapidly to coma and death [1]. Very few cases presenting with hyperglycemia have been described [2,3].

We report a 11 month old girl with propionic acidemia appearing as diabetic ketoacidosis. This child was referred to us for further care with diagnosis of diabetic ketoacidosis. Before coming to us she was admitted with fever, breathlessness and altered sensorium. There was history of failure to thrive and frequent vomiting. She was drowsy and had acidotic breathing, weak peripheral pulses and delayed capillary refill. Investigations revealed hemoglobin of 8.7 g/dL, normal WBC and differential count and low platelet count of 57000/cmm. The blood glucose was 466 mg/dL and urine ketones. Blood gas revealed severe metabolic acidosis (pH were positive -7.18, bicarbonate -4.4 mmol/l) with increased anion gap and increased serum lactate. Glycosylated Hb was high (16.2%). She received treatment with insulin drip, IV fluids and antibiotics. Acidosis and sensorium improved over the next 24 hours after which she was referred to our institution. She weighed 6 kg (<3rd centile) and measured 72 cm in length. She was given subcutaneous regular insulin of 0.5 u/kg/day with daily monitoring of blood glucose. Insulin had to be gradually decreased and then stopped within 7 days

as she had frequent hypoglycemia. She was re-admitted within 7 days for vomiting, dehydration, tachypnea and drowsiness. Investigations revealed WBC of 3400/cmm with normal platelet count, metabolic acidosis, blood glucose of 349 mg%, urine positive for ketones and high serum ammonia of 202 mcg/dL. She was treated with IV fluids and subcutaneous insulin after which she improved within 24 hours. Urine was analysed with a suspicion of organic acidemia by gas chromatography/mass spectrometry, revealed increased excretion of glycine, 3-hydroxypropionic acid, 3-hydroxybutyric acid, 3-hydroxyvalerate, propionyl glycine, tiglylglycine and methylcitrate, which are the biochemical marker compounds of propionic acidemia. Biotin (10mg) and carnitine was added to the therapy and diet with low proteins (1 g/kg/day) was given.

Our initial diagnosis in this patient was type 1 diabetes mellitus. However, rapid response and recurrent hypoglycemia on insulin therapy, leucopenia, thrombocytopenia and hyperammonemia made us suspect an organic acidemia. Most patients with propionic acidemia present with acute episodes of metabolic acidosis, hypoglycemia, hyperammonemia and characteristic organic aciduria due to the decreased activity of intramitochondrial enzyme propionyl CoA carboxylase before they are a few months old.

In our patient, severe hyperglycemia with ketoacidosis was the first life threatening presentation of propionic acidemia to emphasize that hyperglycemia with ketoacidosis is not always diabetes. One should think of propionic acidemia, especially if there is young age of presentation, hyperammonemia, leukopenia, thrombocytopenia and insulin therapy gets withdrawn rapidly.