Cupping at the ends of ribs is not always rickets

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DESCRIPTION

A 7-week-old baby boy presented with a history of cough, loose stools and respiratory distress since last 7 days. At admission he had a respiratory rate of 64/min, a heart rate of 144/min and an oxygen saturation of 56%. Chest examination revealed crepitations in both lung fields. Rest of the examination was unremarkable. Investigations revealed haemoglobin 82 g/L; white cell count 11.2×10^9 /L (differential counts: polymorphs 68%, lymphocytes 1%, monocytes 26% and eosinophils 5%); absolute lymphocyte count 0.11×10⁹/L; platelet count 102×10⁹/L and C reactive protein 239 g/L. Chest X-ray showed non-homogenous opacities in bilateral lung fields (more on right side) with an absent thymic shadow, cupping at the anterior end of ribs

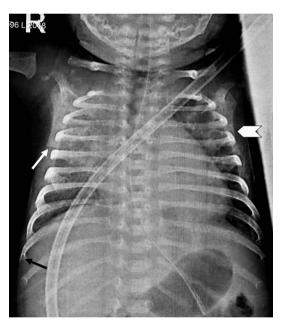


Figure 1 Cupping at the anterior end of ribs (black arrow), flattening of the lower end of the right scapula (white arrow) and a spur at the inferior-lateral angle of the left scapula (white arrow head).

Learning points

- Severe combined immunodeficiency (SCID) is the most severe form of primary immunodeficiency disorder.
- All paediatricians and neonatologists must be aware of the radiological changes of adenosine deaminase (ADA) deficiency SCID.
- Characteristic radiological changes can help in early suspicion and diagnosis of ADA deficiency SCID so that definitive treatment can be instituted.

(black arrow, figure 1), flattening of lower end of the right scapula (white arrow, figure 1) and a spur at the inferior-lateral angle of the left scapula (white arrow head, figure 1). These characteristic radiological changes (ie, cupping at ends of ribs, flattening of the lower end of the scapula and a spur at the inferolateral angle of the scapula) are seen in severe combined immunodeficiency (SCID) due to an underlying adenosine deaminase (ADA) deficiency and provide an important diagnostic clue. He had undetectable levels of immunoglobulins (IgG <202 mg/dL, IgA <17 mg/dL and IgM <25 mg/dL). ADA activity was 0.1 nmol/hour/ mg (normal range 26.4±10), thus suggesting a diagnosis of SCID due to an underlying ADA deficiency.

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