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BCG reactivation: a useful diagnostic tool even for incomplete Kawasaki disease

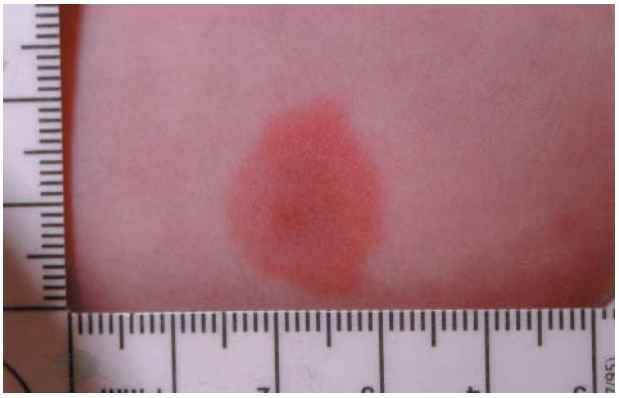
A 16 week old child of Chinese origin presented with a history of persistent fever for three days. She was very irritable and had bright red lips and few maculopapular spots on the trunk. She did not have any significant cervical lymphadenopathy, but did have red eyes. In view of the age a full septic screen was performed and intravenous antibiotic was started. Investigations revealed a raised white blood cell count, C reactive protein, erythrocyte sedimentation rate, and liver enzymes, but normal chest *x* ray, cerebrospinal fluid, and urine. She continued to have a very high spiking temperature even at 48 hours despite negative blood culture. Subsequently marked redness with some induration was noticed around the BCG site.

Due to the presence of fever for over five days, conjunctivitis, red lips, and irritability, incomplete Kawasaki disease was postulated. This hypothesis was further strengthened by the development of erythema around the BCG scar.

The child was started on intravenous immunoglobulin in accordance with a recent recommendation of the American Heart Association.[1] Fever subsided within 36 hours and the erythema around the BCG site disappeared. Her initial echocardiogram was normal and she is under cardiac follow up.

Any child with irritability and persisting fever (⩾5 days) not responding to antipyretics should be suspected to have Kawasaki disease. All criteria need not be fulfilled; incomplete Kawasaki disease may be present.[1] In view of the reported higher incidence of coronary involvement in infancy,[2] an early diagnosis and prompt treatment are essential. Erythema at the site of BCG inoculation is rare, but it is a specific sign of Kawasaki disease[3] and hence can be used as a tool for an early diagnosis.

Children have been diagnosed early by looking at the BCG scar on admission.[4] This should be particularly useful in communities where BCG vaccination is universal. This phenomenon has been hypothetically ascribed to cross-reactivity between mycobacterial heat shock protein (HSP) 65 and human homologue HSP 63.[5]



Consent was obtained for publication of this figure.

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