however, paper, foam and powders have also been reported.1,4 Iron deficiency anaemia is also a frequent finding in Coeliac disease and may be the presenting feature.3

Without thorough attention to the complete history, the diagnosis of Coeliac disease could have been overlooked in the presence of another distracting diagnosis.

Yours faithfully,

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Dear Editor,

BCG SITE INFLAMMATION: A USEFUL DIAGNOSTIC SIGN IN INCOMPLETE KAWASAKI DISEASE

Kawasaki disease (KD) is an acute, systemic vasculitis of unknown aetiology that occurs predominantly in infants and young children. Coronary artery involvement with aneurysms or ectasia occurs in 15–25% of untreated cases, with sudden death from myocardial infarction in 1%.1 Although diagnostic criteria for KD, endorsed by the American Heart Association, are widely used in Australasia, incomplete cases in young infants with coronary abnormalities have been reported.2,3

We report a case of incomplete KD in which reactivation and inflammation of a previous Bacille Calmette-Guerin (BCG) immunisation site was a clue to the correct diagnosis.

A previously well 11-month-old girl was treated for right otitis media by her General Practitioner. Immunisations were up to date, including BCG at birth. Despite successive courses of amoxicillin and cotrimoxazole, she remained febrile. On day 11 of her illness, she was noted to be febrile, 38°C, and very irritable. There was mild pharyngitis, a non-pruritic macular erythematous rash on her trunk, and redness of her palms and soles. She was referred to a hospital.

Upon examination, she was afebrile but miserable. In addition to the findings mentioned earlier, we noted redness of her upper eyelids but no conjunctivitis or other mucosal changes. There were multiple small (<0.5 cm) lymph nodes in her neck, no hepatosplenomegaly and no arthritis. The admitting doctor documented ‘a large BCG scar, left upper arm, inflamed’.

Laboratory investigations revealed normal haemoglobin concentration of 124 g/L (110–140), normal white cell count of 8.9 × 10⁹/L (5–12), normal differential count and an elevated platelet count of 644 × 10⁹/L (150–400). C-reactive protein was <3 mg/L (<5). A tentative diagnosis of Viral infection NOS was made and she was discharged on symptomatic treatment.

At follow-up on day 18 of her illness, she remained afebrile and irritable. The rash had resolved. Fine scaling desquamation of fingers, toes and BCG scar was seen. Further investigation revealed a raised serum gamma glutamyl transpeptidase (GGT) of 67 U/L (15–30), normal platelet count of 373 × 10⁹/L (150–400), CRP of <3 mg/L (<5) and ESR of 7 mm/h (1–10). Sterile pyuria (100–500 leukocytes/high power field) was found on urinalysis. Streptococcal serology was negative.

A paediatrician reviewing her case and aware of the possible significance of BCG scar changes, diagnosed probable incomplete KD. Echocardiogram confirmed aeurysmal dilatation of the proximal left main coronary artery with an internal diameter of 3 mm (Z-score + 2.5) consistent with KD. In view of her clinical improvement and normal inflammatory markers, she was treated with low dose aspirin only. Intravenous gamma globulin was not given. Repeat echocardiogram at 6 weeks was normal.

To our knowledge, this is the first published Australasian case of KD, diagnosed after BCG site changes. Other recent case reports from India,4 Canada3 and the United Kingdom4 have highlighted the specificity of BCG site reactivation and inflammation as a sign of KD. The reason for the reaction is unclear but studies suggest molecular mimicry between specific epitopes of mycobacterial and human Heat Shock Protein 65.5

BCG site changes are not included in the classic clinical criteria for KD published by the American Heart Association. However, they are mentioned among ‘other clinical findings’ that may be present in some cases.1 Awareness of this sign among Australasian paediatricians is likely to be low as BCG vaccination is only recommended for high-risk populations. An earlier report from Japan, where KD and BCG vaccination are both more common than in Australasia, found that 121 (43%) of 281 children who had KD had cutaneous reactions at their BCG site.6 Others have suggested that awareness of this sign may lead to earlier diagnosis and treatment of incomplete cases.7

Based on this case and previous reports, we advise paediatricians to examine patients with suspected KD for evidence of previous BCG immunisation, and to treat evidence of reactivation, inflammation or induration as highly suspicious.

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Dear Editor,

CHYLous ASCITeS FOLLOWING CHOLEDOChAL CYST EXCISION AND LADD’S PROCEDURE

Chylous ascites is a relatively uncommon clinical condition in children. It is, nonetheless, associated with significant morbidity. The key to the management of chylous ascites is the determination of its underlying condition. Common causes of chylous ascites in children include congenital lymphatic abnormalities, lymphatic obstruction and injury during operation, with two previous case reports suggesting a link between malrotation/Ladd’s procedure and the development of chylous ascites.1–3

In this paper, we present the first case of chylous ascites in a girl after excision of the choledochal cyst and Ladd’s procedure, and discuss the current management options. This case should further increase the awareness of this condition.

A 24-month-old girl was referred initially from the general practitioner because of progressive abdominal distension. Her antenatal and post-natal history was otherwise unremarkable. On physical examination, there was no stigma of liver disease. Abdomen was distended with a mass felt down to 10 cm below the right costal margin. Liver enzymes showed elevated alkaline phosphatase and alanine transaminase. Ultrasound of the abdomen showed a large cystic mass at portal region, suggestive of choledochal cyst. Subsequent magnetic resonance cholangiopancreatogram confirmed a type I choledochal cyst, measuring 10 cm in length and 5 cm in transverse diameter.

During laparotomy for excision of the choledochal cyst, malrotation was found incidentally. Ladd’s procedure was thus, performed together with choledochal cyst excision. The patient made an uneventful recovery with feeding resumed on day 4 post-operatively. On day 7 after the operation, progressive abdominal distension was noted. Urgent computed tomography (CT) was performed and showed evidence of ascites (Fig. 1). Abdominal aspirate sent for biochemical analysis confirmed chylous ascites (Fig. 2).

Our patient was initially managed conservatively with a pure medium-chain triglyceride diet but abdominal distension persisted after 1 week. Total parenteral nutrition was, therefore, commenced. The ascites eventually subsided after 4 weeks and the patient was discharged. Subsequent follow-up revealed no evidence of recurrence.

The incidence of chylous ascites has been reported to be 1 in 11 000 hospital admissions. In children, it may be compounded by respiratory complication. Most patients also present with malnutrition and hypoproteinemia. The diagnosis is usually

Fig. 1  An axial computed tomography image of the abdomen showing evidence of a significant amount of ascites.

Fig. 2  Photograph of the aspirated ascites.