Haemorrhagic rash in infectious mononucleosis

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INTRODUCTION

Infectious mononucleosis (IM) is an acute, self-limiting disease typically caused by the Epstein–Barr virus (EBV). The incidence of dermatitis associated with IM is around 20% (Katz and Andiman, 1987). The rash is usually macular or morbilliform (Arnold et al, 1990). This article describes a 15-year-old boy with IM where the rash was severe, extensive and haemorrhagic and the main manifestation of the condition.

CASE REPORT

A previously healthy 15-year-old boy presented with a 1-week history of a sore throat and maculopapular rash for 3 days. He was seen by his general practitioner 2 days before admission and prescribed penicillin for suspected scarlet fever. The rash was present before antibiotics were commenced.

His symptoms gradually worsened and he was admitted to hospital. He had a mild pyrexia of 37.7°C, was lethargic and miserable but systemically well with a normal peripheral circulation and blood pressure. There was a widespread erythematous maculopapular rash all over the body which was confluent on the trunk. There were blisters on his scrotum but there was no joint swelling, hepatosplenomegaly or jaundice. His tongue was erythematous with papules and his throat mildly inflammed with cervical lymphadenopathy, but there was no mucocutaneous ulceration suggesting Stevens–Johnson syndrome.

The white cell count was 6.30 x 10^9/litre, the lymphocyte count was 2.10 x 10^9/litre and the platelet count was normal. His renal and liver function tests were normal, as was the clotting screen, with D-dimer and fibrinogen screen also negative. The erythrocyte sedimentation rate was 4 mm/hr and C-reactive protein 78 mg/litre. Reactive lymphocytes were seen on the blood film and the Paul Bunnell test was positive. A diagnosis of infectious mononucleosis was made and Epstein–Barr virus (EBV) serology was taken.

Over the next 24 hours his rash progressed and became haemorrhagic, especially on the trunk and limbs (*Figures 1* and *2*). Although systemically well meningococcal disease could not be excluded and after blood cultures were taken he was commenced on intravenous benzylpenicillin and cefotaxime. He also developed petechiae on his palate and a single pustule was seen on his tonsils. However, he remained systemically well and antibiotics were stopped at 48 hours when blood cultures and polymerase chain reaction (PCR) for meningococcus were negative. In order to reduce the progression of what appeared to be a generalized vasculitis he was given prednisolone 30 mg daily for 2 days, but this was discontinued since it did not appear to have any effect. He was discharged after 5 days in hospital.

The haemorrhagic rash persisted for 1 week, gradually faded over the next 2 weeks and had disappeared completely in 5 weeks. Rheumatological tests including immunoglobulins, complement and serology to leptospira, mycoplasma, cytomegalovirus and parvovirus were negative. The diagnosis of infectious mononucleosis was confirmed on the basis that EBV viral capsid antigen immunoglobulin M (EBVA IgM capture ELISA, Diasorin, Stillwater, Minnesota) was positive at presentation and remained positive 2 weeks later (*Figure 3*). His EBV nuclear antigen (EBNA IgG ELISA, Biotest (UK), Solihull) was negative, confirming recent infection.

DISCUSSION

Atypical exanthema have been described in IM. There may be purpuric or urticarial reactions (Kato et al 1993), and the administration of ampicillin commonly results in a copper-coloured papular pruritic eruption 5–10 days after the drug is given.

The diagnosis of IM is usually clinical. There is commonly a lymphocytosis with atypical lymphocytes on the blood film. In the acute stage of infection two antibodies are produced. First the heterophilic Paul Bunnell type which agglutinates various animal erythrocytes and second the EBV-specific antibodies. Both immunoglobulin (Ig) M and IgG antibodies to the viral capsid antigen (VCA) are present at the onset of systemic illness and peak after 2–3 weeks. The IgM antibody is short-lived while the IgG antibody persists. Antibodies to the nuclear antigen of EBV (EBNA) start to appear on the 3–4th week as the IgM antibodies fall and these remain positive for life.

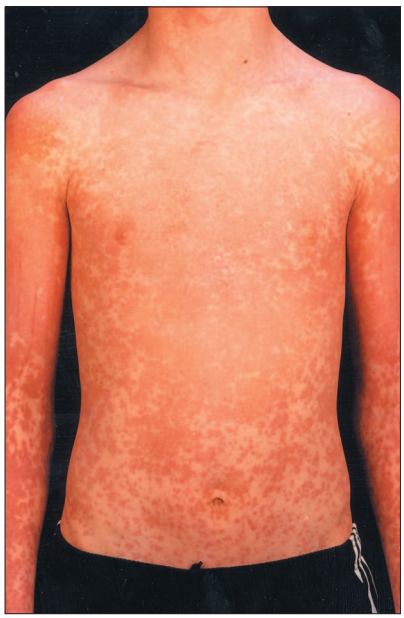

Figure 1. Widespread haemorrhagic rash.

In this case the most prominent feature of the condition was the haemorrhagic rash prompting treatment of

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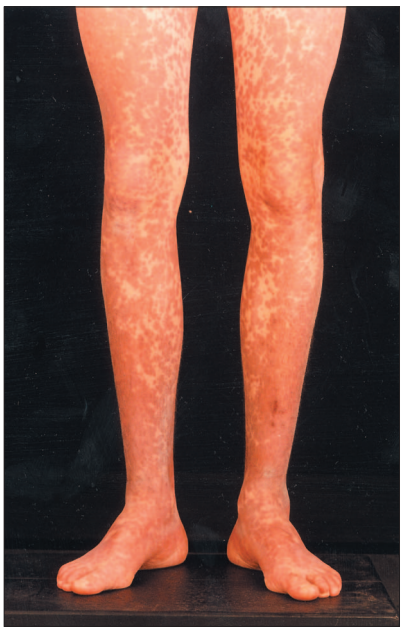

Figure 2. Haemorrhagic rash on trunk and limbs.

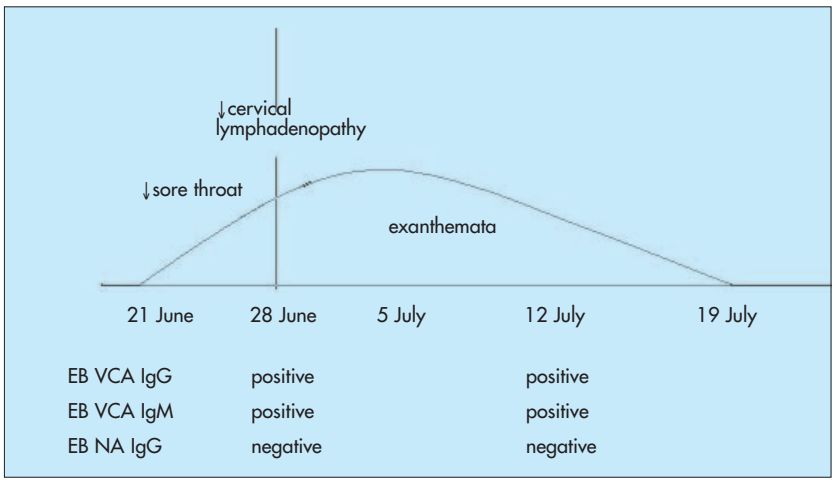

Figure 3. Clinical course and laboratory data.

more serious causes of such a rash before the diagnosis of IM was confirmed both clinically by the development of palatal petechiae and tonsillitis and later serologically. To the authors' knowledge no other case of a severe haemorrhagic rash with IM has been described. This patient broadens the clinical picture of this condition and raises IM as a potential diagnosis in a systemically well young adult with an extensive haemorrhagic rash. HM

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Katz BS, Andiman WA (1987) Infectious mononucleosis. In: Fitzpatrick TB, Eisen AZ, Wolff K, Freedberg IM, Austen KF, eds. *Dermatology in General Medicine.* McGraw-Hill, New York: 2372–5