Enterovirus type 71: seek and ye shall find

We describe a patient with enterovirus encephalitis who presented with oral ulcers and a rash (Figs 1-3). Although the rash appeared to be non-specific, enterovirus was immediately suspected as the aetiological agent based on its hand, foot, and mouth (HFM), and body distribution. Enterovirus type 71 (EV71) was promptly identified by reverse transcription–polymerase chain reaction at multiple sites.

The previously healthy 12-month-old girl lives with her family in Shenzhen. She presented in August 2009 during the H1N1 influenza pandemic with a 2-day history of fever (maximum 38.4ºC) and rigors, and a 3-day history of ‘red spots’ on the abdominal wall, which spread to the limbs and soles and became vesicular. The rash faded as the child developed oral ulcers 1 day preceding her admission. She also had some watery stool and vomited once. Her mother had had a rash on her hands 20 days previously, some diarrhoea but no fever. The family had travelled to Henan in China the week before. The child received oral and intramuscular medications in Henan, and the family flew to Hong Kong the day before presentation.

At a district hospital in Hong Kong, the child was initially afebrile and haemodynamically stable; her Glasgow Coma Scale score was 15. She was treated for gastroenteritis with intravenous fluid. However, she spiked a fever of 39ºC and developed generalised tonic-clonic convulsion with upward rolling eyes and tongue biting, which was treated with rectal diazepam and lorazepam. Urgent computed tomographic scan of the brain showed no abnormal cerebral findings. Intravenous antibiotics (ampicillin 50 mg, 6 hourly; cefotaxime 50 mg/kg/dose, 6 hourly) and acyclovir (25 mg/kg/dose, 6 hourly) were started. She had three more episodes of seizures until phenytoin (loading dose; 20 mg/kg) was added. She was transferred to a paediatric intensive care unit (PICU) for management. Cerebrospinal fluid showed white cells 5 x 10⁶/L, red cells 820 x 10⁶/L, glucose 3.5 mmol/L (reference range, 2.2-3.9 mmol/L), and protein 0.26 g/L (reference range, 0.15-0.45 g/L). Weakness of the right upper limb became apparent. During her stay in the PICU, there was no evidence of cardiac involvement. Intravenous immunoglobulin infusion (1 g/kg) was given. Computed tomographic scan of the brain showed subtle hypodensity involving the left posterior temporal region with blurring of the grey-white matter differentiation. Cerebral magnetic resonance imaging (MRI) showed abnormal

FIG 1. A tiny papule with erythematous rim on one finger

FIG 2. A tiny papule on the ventral surface of the first metatarsal of the right big toe

FIG 3. Oral mucosal ulcers
Scattered vesicles with rim of erythema on the palms and soles, and vesicles or ulcers on the oral mucosa are typical of HFM disease.\(^{1, 9}\) In this patient, the rash involved the abdomen initially, spreading to the limbs, fingers, and feet. Vesicular rash can be confused with herpes and mycoplasma. Nevertheless, the typical HFM distribution may not always be present. The typical imaging finding may also aid diagnosis. Proper isolation and public health measures should be instituted in a timely fashion in enterovirus outbreaks.

Enterovirus type 71 was the major causative agent of HFM disease and encephalomyelitis. The clinical outcome is variable, with complete recovery being possible in some patients despite abnormal radiological findings.\(^{5, 10}\) Brainstem and cervical spinal cord involvement are characteristic findings of enteroviral encephalomyelitis. The radiological findings and outcome in this patient are consistent with those reported in the literature.\(^{10}\)

FIG 4. Axial T2-weighted magnetic resonance images showing hyperintensity (arrows) over (a) left thalamus and (b) brainstem, which demonstrate restricted diffusion on diffusion-weighted imaging (not shown) compatible with acute encephalitic changes

References