Monkeypox, also in pediatric age

Viruela del mono, también en la edad pediátrica

Dear Editor,

We present the case of a girl aged 3 years brought in for observation of cutaneous lesions in the absence of fever, pruritus or other symptoms. The salient feature in the physical examination was the presence polymorphous cutaneous lesions localised to the buttocks, with umbilicated pustular, vesicular and papular elements and a maximum diameter of 3 mm (Figs. 1 and 2). There was no evidence of oral, perianal or genital lesions. There was also no significant lymph node or visceral organ enlargement, and the rest of the examination was normal. A few hours earlier, monkeypox virus (MPXV) infection had been confirmed in the father. The patient had no history of travel or contact with animals. Testing by of a vesicular fluid swab specimen turned out positive for MPXV (real-time polymerase chain reaction). Instructions were given to have the patient isolate at home with use of contact, airborne and droplet precautions until the full resolution of the cutaneous lesions (at 10 days from onset). At the 2-month follow-up, there were no complications and new lesions had not developed. Additional cases had not been detected in any contacts in the school.

This is the first paediatric case of MPXV infection reported in Spain (June 17, 2022).

Monkeypox is a viral zoonosis produced by MPXV, which has become the most important *Orthopoxvirus* species worldwide.[1] At present, numerous outbreaks have been identified in different countries, with more than 2000 cases reported in Spain as of July 2022. The severity of the infection depends on the presence of comorbidities and age, with a reported overall mortality of 15% and a higher risk in young children in endemic areas in sub-Saharan Africa.[2]

It produces symptoms similar to those observed in patients with smallpox, although less severe. The main mechanism of transmission in humans is through contact with mammals, chiefly rodents or primates, in endemic regions. Transmission from one person to another is also possible through air droplets, direct contact with cutaneous lesions, bodily fluids or objects used by infected individuals.[1] The most frequent mechanism of transmission in the current outbreak has been sexual contact.

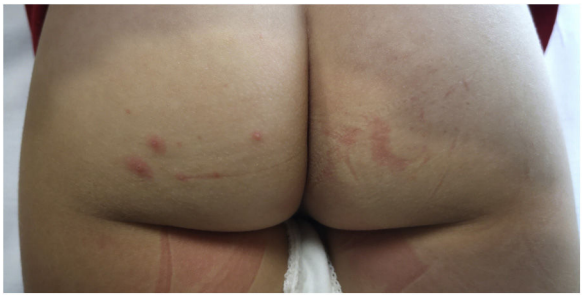

Figure 1 Cutaneous lesions in the left buttock.

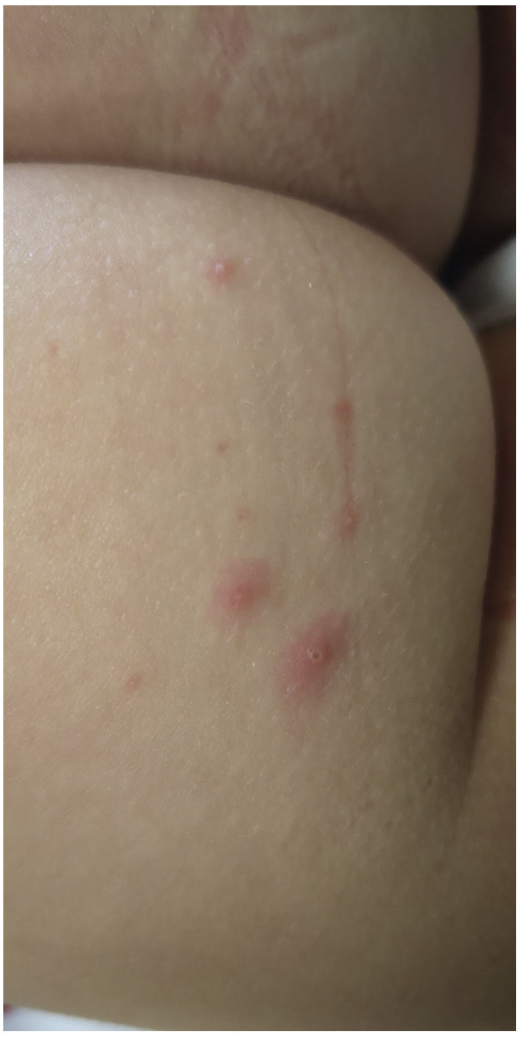

Figure 2 Detail of cutaneous lesions in Fig. 1.

Infection by MPXV tends to cause mild and self-limiting disease lasting 2–4 weeks that only requires symptomatic treatment. However, in some cases it may cause severe disease. Complications can include bacterial superinfection of the cutaneous lesions, bronchopneumonia, sepsis, encephalitis and keratitis. Isolation measures are indicated, with contact, airborne and droplet precaution, until the full resolution of the cutaneous lesions. Antiviral drugs, such as brincidofovir and tecovirimat, have been tried in humans, but there is not enough evidence yet to support the indication.[3] Post-exposure prophylaxis with intravenous immunoglobulin or smallpox vaccine is not approved in the paediatric age group, and should be reserved for compassionate use.[4] Whenever a case is confirmed, close contacts should be followed up, especially any sexual contacts, for 3 weeks.

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Necrotizing enterocolitis after intravenous immunoglobulin administration and exchange transfusion in a newborn with hemolytic disease due to anti-c☆

Enterocolitis necrotizante tras administración de inmunoglobulinas y exanguinotransfusión en anemia hemolítica por anti-c

Dear Editor:

Haemolytic disease of the foetus and newborn (HDFN) affects 3–80 per 100 000 patients each year. Although the most frequent cause of HDFN is Rhesus D (RhD) antigen alloimmunization, other red blood cell antigens can cause the disease, for instance, through anti-c isoimmunization. When the incompatibility is severe, it can cause foetal anaemia, hydrops fetalis and even intrauterine death. In neonates, phototherapy is the most widely used treatment in mild and moderate cases. In severe cases, intravenous immunoglobulin (IVIG) and exchange transfusion may be useful to prevent bilirubin encephalopathy.[1]

We present the case of a preterm newborn with HDFN caused by an uncommon type of antibodies (anti-c). The patient experienced an insidious course that required treatment with IVIG and exchange transfusion. She then developed necrotising enterocolitis (NEC).

The patient was a newborn girl admitted to our hospital due to jaundice with onset within 24 h of birth. She was the second child of a healthy mother aged 37 years that had received a diagnosis at 24 weeks of gestation of Rh isoimmunization due to anti-c antibodies. Foetal ultrasound examinations were carried out, which did not detect abnormalities in the foetus, with the exception of mild anaemia at 30 weeks of gestation.

The patient was born at 35^{+1} weeks of gestation. Her blood type was A Rh+ and the direct Coombs tests was positive, evincing the presence of anti-c antibodies. At 24 h post birth, the physical examination of the patient was normal with the exception of jaundice. Laboratory tests confirmed hyperbilirubinaemia (total bilirubin, 13.5 mg/dL; normal range, <8 mg/dL) in the absence of anaemia (haemoglobin, 16.7 g/dL), leading to initiation of intensive phototherapy for 14 days with a positive outcome. Bilirubin levels remained below the exchange range. Other tests were performed to rule out alternative diagnoses to hyperbilirubinaemia.

At 14 days post birth, the patient developed anaemia (haemoglobin, 7.7 g/dL; normal range, 12.5–20.5), leading to administration of IVIG (1 g/kg IV over 6 h). The patient remained haemodynamically stable before and during treatment. The anaemia progressed despite IVIG (haemoglobin, 6.7 g/dL 12 h after immunoglobulin administration), so the patient underwent partial exchange transfusion (80 cc/kg) delivered through a central venous catheter (left femoral vein). There were no adverse events associated with the transfusion. The haemoglobin concentration increased to 12.3 g/dL (Fig. 1). A few hours after the transfusion was completed, the patient exhibited general deterioration with abdominal distension and bilious vomiting. She required endotracheal intubation and haemodynamic support with dopamine. The findings of the abdominal radiograph were suggestive of NEC (Fig. 2). The patient underwent an urgent laparotomy that confirmed the diagnosis. This was followed by performance of an end ileostomy with removal of the distal ileum and ascending colon. After the surgery, the patient recovered gradually and was discharged at day 30.

Minor blood group incompatibility has been found to be responsible for 3%–5% of cases of haemolytic jaundice in neonates.[2] Antigen c is one of the most immunogenic antigens, following antigen D, with immunization usually developing antenatally and associated with a risk of moderate to severe HDFN.[3]

Haemolytic disease of the foetus and newborn can cause sustained anaemia, possibly with antenatal onset, causing tissue hypoperfusion and particularly affecting the gastroin-

☆ Previous presentation: This study was presented at the XXVIII Congress of the Sociedad Española de Neonatología in October 2021with the title ''Enterocolitis necrotizante tras la administración de inmunoglobulina intravenosa y transfusión de hematíes en pretérmino tardío con enfermedad hemolítica anti-c''.