

In our patients, treatment with heparin was chosen because the adherence to oral administration is worse in children younger than 12 months. Pannus formation (a fibrous tissue overgrowth) was the reason for early reintervention in both patients. This complication is uncommon in the paediatric literature. Although it does not seem to be related to anticoagulant therapy, in both of our patients it developed while they were undergoing treatment with heparin, and did not recur when the patients were treated with a combination of a coumarin drug and ASA.

In short, MI in the paediatric age group continues to be a challenge. Mechanical prostheses have the disadvantage of requiring chronic anticoagulant therapy, which is difficult to adhere to, especially in infants.

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Typical haemolytic uraemic syndrome: Report of the first case due to *Escherichia coli* O26:H11 in Spain[☆]

Síndrome hemolítico urémico típico: comunicación del primer caso producido por *Escherichia coli* O26:H11 en España

Dear Editor:

Haemolytic uraemic syndrome (HUS), while rare, is one of the main causes of acute renal failure in the paediatric age group. It is characterised by the development of haemolytic anaemia, thrombocytopenia and acute renal failure.¹ Most cases present with diarrhoea (so-called typical HUS), with Shiga toxin (Stx)-producing *Escherichia coli* (*E. coli*), especially serotype O157:H7, being the most frequent aetiological agent. We are presenting a case caused by the O26:H11 strain of enterohaemorrhagic *E. coli*.

The patient was a girl aged 2 years presenting with fever, vomiting and watery stools with fresh blood of three days' duration, with asthenia and oliguria in the previous 48 h. The relevant findings of urgent laboratory testing were: haemoglobin, 9 g/dL; platelets, 67,000/ μ L; creatinine, 3.9 mg/dL; urea, 162 mg/dL; uric acid, 15.9 mg/dL; lactate dehydrogenase (LDH), 5397 IU/L; and 4–5 schistocytes



per high-power field in the peripheral blood smear. The patient was admitted to the paediatric intensive care unit for suspected HUS, and her persistent anuria required initiation of continuous venovenous haemodiafiltration, which was followed by peritoneal dialysis for a total of 21 days of blood purification. The complement levels were decreased at diagnosis (C3, 62.1 mg/dL; C4, 10.5 mg/dL) and had normalised three days later, including properdin (28.6 mg/dL) and CH100 (1.125 U/mL) values. The detection of anti-ADAMTS13 antibodies was positive (43.9 AU/mL), although ADAMTS13 activity was normal (64.5%). The investigation was completed with a stool culture that was negative for enterohaemorrhagic *E. coli*. Due to this result, the sample was analysed by PCR and found to be positive for verotoxigenic *E. coli*, and genetic characterisation and serotyping identified the isolate as strain O26:H11.

E. coli strain O26:H11 has been an emergent pathogen in Europe since it was first isolated in Germany in 1990.² It has been increasingly isolated in cases of HUS to eventually become the second most frequent cause (15–19%) following serotype O157:H7, even surpassing this serotype in some countries between 2008 and 2012. It can also produce two types of Stx, of which Stx2a is strongly associated with the development of HUS.³ Furthermore, there is evidence that children are more frequently infected by stx_{2a}- harbouring strains, and thus are more likely to develop HUS.²

The detection of enterohaemorrhagic *E. coli* O26:H11 by PCR in our patient constitutes the first time that this strain is found in a child with HUS in Spain. This serotype is associated with infection in younger children, and the form of HUS that it causes may have a similar severity and clinical course to the form produced by O157:H7. The patient was kept in isolation during the early weeks of hospitalisation, and is currently awaiting negative stool culture results to resume her schooling.

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Our patient did not receive antibiotic therapy, as it is not indicated for HUS; furthermore, antibiotic use has been associated with an increased risk of developing HUS in enteroinvasive diarrhoeas,⁴ and experimental studies have shown that it may induce the expression and release of Stx.

We have only found references to other two cases of diarrhoea-associated HUS⁵ with presence of anti-ADAMTS13 antibodies and preserved normal ADAMTS13 activity, as observed in our patient. The role of these antibodies in these patients has not been clearly established, so their presence alone would not indicate a specific intervention insofar as activity continues to be normal.

Our patient only required one red blood cell transfusion after her haemoglobin level had reached a minimum of 5.7 g/dL four days after admission; the platelet levels dropped to 64,000 in the first 24 h and then increased gradually from the fifth day of hospitalisation. The concentration of haptoglobin at diagnosis was less than 10 mg/dL and the LDH levels were reduced by half in the first three days, normalising two weeks later.

We should also note the absence of neurologic manifestations in our patient (save for mild confusion), the presence of which is variable in the early stages of HUS.

Our purpose in presenting this case is to emphasise, on one hand, that a high degree of suspicion is essential, as this disease can cause severe sequelae or even death in 5% to 10% of the cases, and on the other, that an exhaustive microbiological diagnosis is imperative, promptly submitting samples for the early identification of emerging strains so that appropriate preventive measures can be implemented. This requires the support of reference centres, which in our case was the Centro Nacional de Microbiología (Instituto de Salud Carlos III).

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Atrial fibrillation in a 22-month-old patient during cleft palate surgery[☆]



Fibrilación auricular en paciente de 22 meses durante palatoplastia

Dear Editor:

Supraventricular tachycardias (SVTs) that occur during non-cardiac paediatric surgery are usually sinus tachycardias, with a regular rhythm and secondary to pain, hypoxaemia, hypercapnia, hypovolaemia, hypothermia, or ion or acid-base imbalances. Non-sinus SVTs occur less frequently and may be associated with cardiac abnormalities.¹

We present the case of a boy aged 22 months, weighing 9 kg and classified as ASA II scheduled to undergo surgical repair of a congenital cleft palate. The salient aspects of his medical history included Pierre Robin sequence and tetralogy of Fallot (TOF), which was repaired at age 8 months

with a favourable outcome. ECG revealed a sinus rhythm of 150 bpm and a 90° axis. Echocardiography revealed good contractility. There was no evidence of cardiac or right ventricular outflow tract (RVOT) dilatation. The interventricular patch was intact. The patient had mild pulmonary stenosis (PS) and tricuspid regurgitation (TR).

The patient was premedicated with oral midazolam. Materials for the management of a difficult airway were prepared: flexible fibreoptic bronchoscope, Frova® introducer and supraglottic airway devices. Sevoflurane was chosen for anaesthesia induction, maintaining spontaneous breathing during the assessment of the airway by direct laryngoscopy. The patient was given atropine and propofol prior to intubation, which was performed by means of a Frova® introducer without complications. Sevoflurane and remifentanil were used for the maintenance of anaesthesia; lactated Ringer's solution for fluid therapy; and dexamethasone and magnesium sulphate as adjuvants. The monitoring values were the following: oxygen saturation (SatO_2), 99%; end-tidal carbon dioxide (EtCO_2), 40 mmHg; ECG, ectopic atrial rhythm (missing P wave) with regular QRS complexes; heart rate (HR), 110–120 bpm; systolic blood pressure (SBP), 75–80 mmHg; bispectral index (BIS), 45–50; body temperature, 36.5 °C. Surgery was initiated after placing the patient in a 30° Trendelenburg position. Two hours later, the ECG started to show isolated supraventricular

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