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 HUS[5] 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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E.J. Bardón Cancho[a,*], L. Butragueño Laiseca[b], O. Álvarez Blanco[a], A.J. Alcaraz Romero[b], A.B. Martínez López[a]

[a] *Servicio de Nefrología Pediátrica, Hospital Materno-Infantil, Hospital General Universitario Gregorio Marañón, Madrid, Spain*
[b] *Unidad de Cuidados Intensivos Pediátricos, Hospital Materno-Infantil, Hospital General Universitario Gregorio Marañón, Madrid, Spain*

* Corresponding author.
E-mail address: edubc15@hotmail.com (E.J. Bardón Cancho).

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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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extrasystoles, while the rhythm, HR and SBP remained unchanged. Thirty minutes after, a chaotic atrial rhythm was observed, with a HR of 135–145 bpm and a SBP of less than 70 mmHg, leading to injection of 10 mL of hydroxyethyl starch and assessment of the haemodynamic response: SBP below 60 mmHg, $SatO_2$ of 91%, no response to 100% O_2. The patient was given 50 mg of amiodarone (over 5 min) and 10 mg of furosemide, the external defibrillator was set up, the patient taken out of Trendelenburg, and a urinary catheter placed. Five minutes after administration of the bolus of amiodarone was completed, the ECG showed a sinus rhythm, the HR was 120–130 bpm, the SBP 80–85 mmHg, and the $SatO_2$ 98%. Subsequently, an amiodarone infusion of 150 mg/24 h was started. The decision was made to proceed with the surgery, which was completed 30 min later, after which the patient was moved under sedation to the paediatric intensive care unit (PICU). The procedure lasted a total of 4 h. Analgosedation was maintained for 20 h, and the patient was extubated without complications and discharged from the PICU 24 h later. He remained in the ward for 48 h without complications, after which he was discharged, pending a Holter monitoring. The ECG showed good contractility. The patient had a mild dilatation of the right chambers of the heart. Mild PE and TR.

Tetralogy of Fallot is the most common cyanotic congenital heart disease (5–10% of congenital heart diseases). It is managed by means of surgical repair, which consists of the closure of the ventricular septal defect and the widening of the right ventricular outflow tract. Surgical repair carries a mortality of less than 2% and a survival rate of 90% in the adult age group.[2]

In patients with repaired TOF, SVTs may appear in the immediate postoperative period; and while more than half of the patients develop supraventricular extrasystoles, the incidence of tachyarrhythmias is less than 10%. They are caused by haemodynamic and local factors resulting from surgery and extracorporeal circulation, and the most common type is junctional ectopic tachycardia.[3] They may also develop during puberty and after as a late complication, with an incidence of more than 30%. They are produced by reentry circuits around the atriotomy scar and by pressure or volume overload in the right chambers. The most common forms are atrioventricular nodal reentrant tachycardia, fibrillation and atrial flutter. Their development may increase the risk of sudden death.[4,5]

The anaesthesia of patients with repaired TOF in noncardiac surgery poses a challenge, as there are no large studies on surgeries with significant changes in volume, intracardiac pressure (thoracoscopy or laparoscopy) or surgical positioning (Trendelenburg or prone). Its management must prioritize the identification of patients at high risk of perioperative morbidity (Table 1) and preventing pressure overload (raised intrathoracic pressure, hypoxia or hypercapnia) and volume overload (fluid therapy or Trendelenburg) of the right chambers.[6]

The only risk factor in our patient was his age of less than 2 years, and he experienced no complications during the induction of anaesthesia or the difficult airway management, which leads us to believe that the sustained Trendelenburg position caused a volume overload in the right chambers that eventually resulted in atrial fibrillation and the drop in cardiac output. The symptoms responded to postural and pharmacological interventions without the need for defibrillation, which would have been the treatment of choice if the patient had not responded or his condition had worsened.

Table 1 Perioperative high-risk factors.

– Age < 2 years
– Urgent surgery
– Major surgery (intrathoracic, intra-abdominal or with considerable volume changes)
– Cardiovascular sequelae (arrhythmias, pulmonary hypertension, cyanosis or heart failure)

Table 2 Key points for the use of anaesthesia in patients with repaired tetralogy of Fallot.

– Identify high-risk patients
– Exhaustive monitoring and observation
– Approach any postural, surgical or pharmacological interventions or fluid therapy that may alter the haemodynamic condition of the patient with caution
– Keep in mind the potential for arrhythmias and their early treatment in case of haemodynamic compromise

We conclude with some key points for the management of any type of anaesthesia in patients with repaired TOF (Table 2).

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R. Eizaga Rebollar[a,*], M.V. García Palacios[b], J. Morales Guerrero[a], R. Gámiz Sánchez[a], L.M. Torres Morera[a]

[a] *Servicio de Anestesiología, Reanimación y Terapéutica del Dolor, Hospital Universitario Puerta del Mar, Cádiz, Spain*
[b] *Servicio de Medicina Preventiva y Salud Pública, Hospital Universitario Puerta del Mar, Cádiz, Spain*

* Corresponding author.
E-mail address: ramonchueizaga@yahoo.com
(R. Eizaga Rebollar).