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Antiarrhythmic management in postoperative pediatric patient for tetrallogy of Fallot correction

Manejo antiarrítmico en paciente pediátrico postoperado de corrección de tetralogía de Fallot

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Abstract

Arrhythmias are abnormalities in the rhythm of the electrical conduction of the heart that can occur in congenital and acquired heart disease. However, their occurrence is rare in the pediatric population. When they are present, they can induce high morbidity and mortality, especially when they occur in the 1st hours of the arrival of the child to the intensive care unit. Dedicated attention should be taken specifically to fluid management, temperature, serum electrolytes, analgesia and sedation, and strict monitoring of heart rate, blood pressure, left atrium and central venous pressure (CVP), oxygen saturation, lactate, hourly uresis, and fluid balance. The actual work is a review of antiarrhythmic management in a pediatric patient with congenital heart disease after the corrective surgery. This is the case of a 10-month-old male with tetralogy of Fallot (TOF) managed with amiodarone in loads and infusion, temperature correction, electrolytes, and use of pacemakers during his stay at the intensive care unit.

Keywords: *Arrhythmias. Junctional ectopic tachycardia. Pediatric cardiac surgery. Postoperative heart. Tetralogy of Fallot.*

Resumen

Las arritmias son anormalidades en el ritmo de la conducción eléctrica del corazón que puede presentarse en cardiopatías congénitas y adquiridas; sin embargo, son raras en la población pediátrica en general, cuando están presentes, pueden condicionar una elevada morbimortalidad, especialmente cuando ocurren en el postoperatorio del niño crítico postoperado de cardiopatía congénita. Se deben tener cuidados específicos como el manejo de líquidos, temperatura, electrolitos séricos, analgesia y sedación y un monitoreo estricto de ritmo cardiaco, presión arterial, presiones de atrio izquierdo y presión venosa central, saturación de oxígeno, lactato, diuresis horaria, balance hídrico estricto. Repaso del manejo antiarrítmico en el paciente pediátrico postoperado de corazón. Presentamos el caso de un paciente masculino de 10 meses de edad, quien se encuentra en el área de terapia intensiva cardiovascular pediátrica postoperado de Corrección de Tetralogía de Fallot. Se programa para corrección quirúrgica completa de la cardiopatía existente, se maneja con amiodarona en cargas e infusión, corrección de temperatura, electrolitos y uso de marcapasos. Se mantiene en la unidad de terapia intensiva cardiovascular hasta su resolución y signos vitales estables.

Palabras clave: *Arritmia postoperatoria. Cirugía cardiaca pediátrica. Cuidados postoperatorios. Taquicardia ectópica de unión. Tetralogía de Fallot.*

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Introduction

It is of great importance to have early detection of rhythm abnormalities in patients who have undergone heart surgery, as the identification and early treatment reduce morbidity and mortality. Junctional ectopic tachycardia (JET) typically occurs in the immediate postoperative period, especially in patients with congenital heart disease where there was extensive manipulation of the heart. Risk factors include prolonged aortic cross-clamping (ACC), ventricular septum surgery, significant traction and manipulation of the heart, need for inotropic support, hypomagnesemia, and early age[1-3]. Certain guidelines must be followed to obtain the best possible postoperative result and to improve outcomes in the short to long-term. Mortality is still high when patients are admitted to the cardiovascular intensive care unit (CVICU) with risk factors. Within the aforementioned guidelines, we can find potentially reversible risk factors, which we will outline during the case presentation and then summarize to emphasize the most important factors for the operative and postoperative period.

Case presentation

The patient is a 10-month-old male that was diagnosed at 4 months old. His mother denies the presence of a hypoxic crisis and reports cyanosis, fatigue, and diaphoresis. He was seen by pediatric cardiology and was started on beta-blockers (propranolol) with a dose of 1.5 mg/kg/day every 8 hours. Echocardiogram showed normal segmental anatomy.

The patient is admitted to the Pediatric Heart Center of the ABC Medical Center with the diagnosis of tetralogy of Fallot (TOF).

He is scheduled for a complete repair of TOF and ligation of the patent ductus arteriosus (PDA). Both are performed through a medial sternotomy with total cardiopulmonary bypass (CPB) at 32°C. Resection of the infundibular musculature is performed until the valve and the pulmonary annulus are visualized. A vertical incision is performed on the truncus of the pulmonary artery, where the rest of the infundibular muscle is resected, passing a 9 and 10 mm Hegar dilator through with no complications. The ventricular septal defect (VSD) is closed with an autologous treated pericardial patch, an autologous untreated pericardial patch is used to augment the right ventricular outflow tract (RVOT), and the atrial septal defect (ASD) is closed with an autologous untreated pericardial patch. The heart presents spontaneous rhythm while weaning off CPB, which appears to be originating from the junction with up to 160 bpm, managing to convert to sinus rhythm after 15 minutes by overstimulating the atria and ventricles with temporary pacing wires. There was a total of 2 hours and 4 minutes and 2 hours and 53 minutes of ACC and CPB, respectively. A right atria catheter, peritoneal dialysis catheter, pacing wires, and mediastinal Blake drain are all placed. Treatment with milrinone and esmolol is started, and 0.03 mcg/kg/minute of norepinephrine is started due to persistent hypotension that subsequently improves. Operatory findings include overriding dextrorotated aorta; 10 mm conotruncal VSD, 10 mm pulmonary artery with a 10 mm annulus; thin nondysplastic bicuspid pulmonary valve; narrow and obstructive infundibulum; pulmonary branches with a proper size; a PDA that greater resembles a ligamentum arteriosum; and a 7 mm ostium secundum ASD.

The intraoperative transesophageal echocardiogram showed adequate closure of the VSD with a residual VSD only seen by color Doppler, located on the inferior portion of the ventricular septum. No obstruction on the RVOT with a maximum flow speed of 1.75 m/seconds, with mild pulmonary regurgitation; mild tricuspid regurgitation; biventricular dysfunction; atrial filling pressures of 12 mmHg on both atria; and moderate hypertrophy of the right ventricle.

He was admitted to the CVICU of the ABC Medical Center. Monitored with an invasive arterial blood pressure catheter, right atrial pressure catheter, pulmonary artery pressure, left atrial pressure, continuous electrocardiogram, oxygen saturation, capnography, temperature, foley catheter diuresis, pulse pressure variation (PPV), and near-infrared spectroscopy (NIRS).

Cardiovascular—he is admitted while under the effects of continuous fentanyl and dexmedetomidine infusion, invasive mechanical ventilation with continuous monitorization. Electrocardiogram shows a 165 bpm, with an auricular electrogram that resembles JET (Fig. 1), currently under treatment with 500 mcg/kg/minute of esmolol. As soon as JET was identified the patient was given a loading dose of 5 mg/kg of amiodarone for 30 minutes without adequate response, repeating the dose and adding a continuous infusion of amiodarone that was gradually titrated to 20 mcg/kg/minute while the β-blocker was discontinued. Norepinephrine was titrated to 0.1 mcg/kg/minute with a shock dose of an intravenous (IV) corticosteroid and 3 mL/kg of a colloid fluid bolus was administered in order to maintain mean arterial pressures in the 50th percentile. Arterial and venous blood oxygen saturation were both above 95%, NIRS was between 65 and 75, PPV between 9 and 11, arterial and

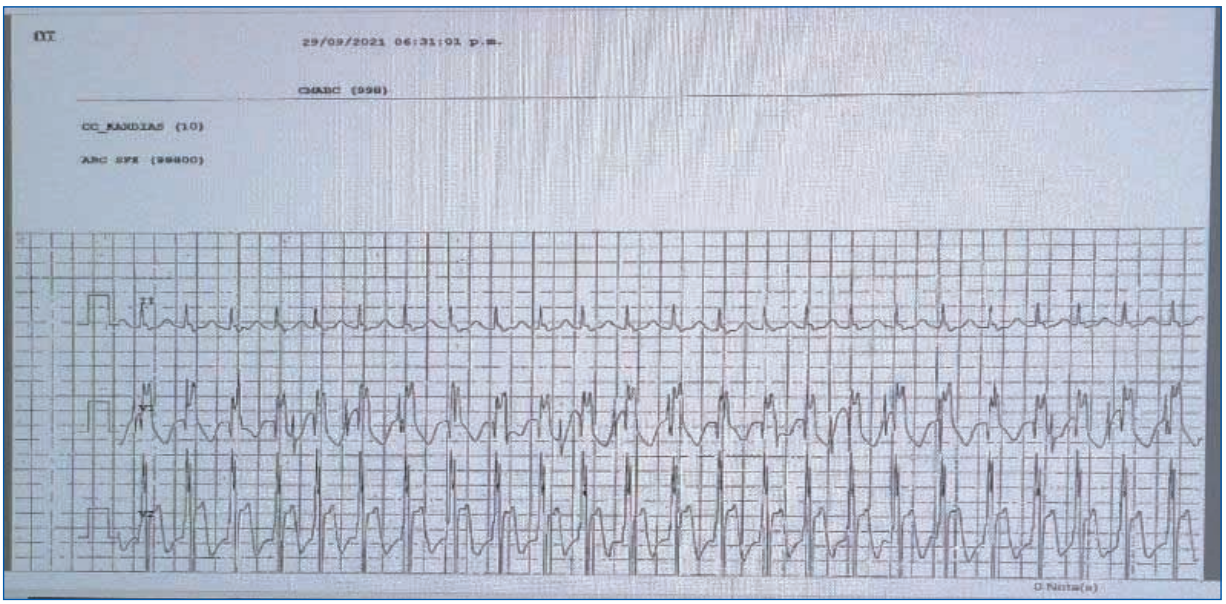

Figure 1. Identification of the junctional ectopic tachycardia rhythm by atriogram.

venous lactates both below 2 mmol/L, a venous reserve of 65%, hourly urine output between 0.5 and 1.0 mL/kg/hour, central venous pressure (CVP) between 6 and 12 mmHg. The patient remained in JET, for which he was submitted to therapeutic hypothermia (35°C) with a thermal mattress and dynamic management of physical means until the 5th postoperative day. Pacing wires were used on atrial mode and occasionally on atrioventricular (AV) mode with frequencies of 135-145 bpm for 5 days until they were disconnected and removed. Pressure differences between CVP and the left atrium were kept within 2-3 mmHg, and CVP's between 8 and 11 in order to maintain optimal perfusion pressure.

By the 5th postoperative day, the patient presented adequate contractility on both ventricles as shown by transthoracic echocardiogram, for which the patient was weaned off milrinone and amiodarone.

On days 7-10 the patient developed a first-grade AV block with isolated monomorphic supraventricular extrasystoles that had no hemodynamic repercussions. Dexmedetomidine and amiodarone were weaned off. On the 11 and 12th postoperative day milrinone and amiodarone were respectively discontinued. On the 13th postoperative day, the patient presented sinus tachycardia with up to 170 bpm, secondary to irritability and crying. Due to difficulty with the management of the tachycardia, the patient was started on 0.5 mg/kg/dose of propranolol which was discontinued after 72 hours.

Respiratory—airway was kept on pressure-controlled mechanical ventilation with a PEEP of 5 mmHg and a fraction of inspired oxygen of 60%, keeping the inspiration: expiration ratio 1:2, with an inspiratory pressure of 18 mmHg. This kept blood gasses in acid-base equilibrium and normoxemia. After dynamic management of ventilatory parameters, the patient was weaned off mechanical ventilation by day two and started on continuous positive airway pressure with up to 1 L/kg, being titrated through the course of 12 days after which were discontinued and replaced with nasal cannulas that were subsequently removed before being discharged home. The patient presented with pleural effusions that required drainage with a chest tube that was removed 48 hours after placement.

Genitourinary—the patient was kept on 25% of the daily basal requirements by Holliday Segar for the 1st day, increasing to 35, 50, and 75% on the 2nd, 3rd, and 4th day, respectively. Peritoneal dialysis was performed in order to clear inflammatory mediators during the first 24 hours, and in order to improve fluid balance up to the third postoperative day, when the catheter was removed. Furosemide was administered through continuous infusion and was titrated to a maximum dose of 220 mcg/kg/hour and was gradually weaned off until it was administered through IV boluses and later orally at a dose of 1 mg/kg/dose every 8 hours.

Gastrointestinal—the patient was kept on NPO during the first 24 hours, and once hemodynamically stable by day 2 was started on TPN, with diet progression as tolerated by day 3, advancing until daily calorie and protein needs were met according to patient weight and

height. TPN was discontinued by day 11, PO diet was maintained until discharged home.

Metabolic—electrolytes were managed on a continuous basis, aiming to maintain ionic calcium above 1.2 mmol/L, magnesium above 2.3 mg/dL, potassium above 4 mEq/L, base deficit no lower than −5, and pH within normal ranges. Thyroid hormone replacement was done through the monitorization of free T3.

Infectious disease—the patient was started on prophylactic cefalotin for three days, after which the patient began presenting fever with no elevation of inflammatory markers. An extended β-lactamase-producing *Escherichia coli* was identified on a urine culture, with 50,000 colony-forming units. Antibiotic treatment was advanced to vancomycin until the antibiogram showed sensitivity to cefepime, which was started and administered until the concluding 10 days of treatment with serial negative cultures. The patient also tested positive on a respiratory viral panel for rhino enterovirus, for which the patient needed increased requirements for high-flow oxygen. These requirements were evaluated with blood gases and the clinical status of the patient.

Neurologic—the patient was kept on sedation and analgesia with IV boluses as needed with a continuous infusion of up to 4 mcg/kg/hour of fentanyl and up to 1 mcg/kg/hour of dexmedetomidine, and 10 mg/kg of acetaminophen every 6 hours. They were titrated according to the patient's needs and were gradually weaned off until they were discontinued altogether. Fentanyl was alternated with sufentanil and morphine either in continuous infusion or in boluses depending on the dose-response relationship. Clonidine and ketamine were also used in boluses as needed and for invasive procedures or catheter removals.

Discussion

The management of JET still represents a great challenge for critical care physicians that take care of patients with congenital heart disease in the immediate postoperative period. Given the magnitude of the mortality generated both in the short and the long-term, there are several key considerations that should be taken into account, such as the risk factors associated with management, since this is the most common postoperative arrhythmia in the immediate postoperative period. This arrhythmia results from the increased automaticity in the Has-Purkinje complex which gradually increases the heart rate and creates a dissociation between the atria and ventricles. According to Mery et al. JET arises from insults that occur during the surgical period and is a transitory process that we have seen starts within a few hours after surgery and can last up to 1 week afterward[2].

The case presented highlights the need for early detection of JET, which is often identified when heart rates increase to 170 bpm with the possibility of further increasing up to 250 bpm. Treatment is necessary when heart rates reach up to 180 bpm and show signs of low cardiac output. JET is usually a narrow complex tachycardia but can widen in the presence of a bundle branch block and can resemble a ventricular tachycardia, making the diagnosis more difficult. An irregular heart rate can appear when both the JET and sinus rhythm have similar depolarization rates, as there are two competing mechanisms trying to control the heart rate. When JET accelerates it takes control of the heart rate, and the beats become regular again. Agreeing with Cassalet et al. JET originates downstream from the AV node and can occasionally cause retrograde conduction. When patients have absent P waves and auricular electrogram can be obtained with the pacing wires placed during surgery, and a JET can be confirmed when a QRS precedes the formation of a P wave[3].

As JET is an autonomous arrhythmia, we must keep in mind that they can "heat up and cool down." These can be influenced by body temperature, endogenous and exogenous catecholamines, and the sedatives used (similar to a sinus rhythm)[3]. Because of this, the continuous monitorization of all of these parameters and especially of the CVP can provide clear evidence of JET, through the demonstration of cannon A waves caused by atrial contraction against a closed tricuspid valve. In the normal heart, atrial contraction accounts for 10-15% of the ventricular filling and hence it accounts for the same amount of the systolic volume[4]. We can commonly find some degree of diastolic dysfunction in the postoperative period, which creates a need for higher ventricular filling pressures in order to overcome the dysfunction and puts further emphasis on the need for adequate atrial contraction. This is particularly dangerous for a patient that is already struggling with low cardiac output and then develops JET.

The optimization of surgical technique, anesthesia, and cardiopulmonary bypass could possibly aid in the prevention of the development of JET[1,2,4]. We agree with Mery et al. and Entenmann et al. in that the use of magnesium sulfate, amiodarone, and dexmedetomidine during the operative period can decrease the postoperative incidence of JET[2,5,6]. We have administered amiodarone as our preferred antiarrhythmic in continuous infusions and less so in boluses, as the later has

a greater risk for hypotension. Patients sometimes require atrial or AV stimulation with pacing wires to reinstate the synchronicity with frequencies above the JET and in the event of drug-induced bradycardia.

Conclusion

We consider the early detection of arrhythmias in the postoperative period of patients who have undergone congenital heart disease surgery to be of utmost importance in order to promptly treat and reduce morbidity and mortality. Treatment should always seek to both decrease the frequency of JET and try to establish AV synchronicity, if necessary with pacing wires. These goals can be achieved by maintaining normal electrolyte values, such as magnesium above 2 mg/dL, ionic calcium above 1.2 mmol/L, and potassium above 3.5-4.0 mEq/L. Additionally, the administration of exogenous inotropes should be limited, and therapeutic hypothermia to 35°C can be induced. Adequate sedation should also be provided in order to minimize the endogenous sympathetic impulse, and antiarrhythmics should be used carefully.

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Conflicts of interest

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Ethical disclosures

Protection of humans and animals. The authors declare that no experiments on humans or animals have been performed for this research.

Confidentiality of data. The authors declare that they have followed their center's protocols on the publication of patient data.

Right to privacy and informed consent. Right to privacy and informed consent. The authors have obtained approval from the Ethics Committee for the analysis and publication of routinely obtained clinical data. The informed consent of the patients was not required because this was a retrospective observational study.

Use of artificial intelligence to generate texts. The authors declare that they have not used any type of generative artificial intelligence in the writing of this manuscript or for the creation of figures, graphs, tables, or their corresponding captions or legends.

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