Postoperative junctional ectopic tachycardia

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Purpose: To report the management of junctional ectopic tachycardia after cardiac surgery in an infant. Postoperatively, the patient suffered profound cardiac decompensation secondary to the accelerated rhythm and required extracorporeal membrane oxygenation (ECMO) for haemodynamic support.

Clinical features: A 14-day-old, 3.5 kg boy exhibited junctional ectopic tachycardia after cardiopulmonary bypass. Left atrial pressure was 25-28 mmHg. No impact on the tachycardia was seen after rapid overdrive atrial pacing or after 20 μg fentanyl iv, 45 μg digitalis, 100 mg magnesium or procainamide (loading dose 15 mg, then 30 $\text{mg}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Active cooling decreased the nasopharyngeal temperature to 35.2°C, when the heart rate decreased below 180 bpm with a left atrial pressure of 8-10 mmHg. Dopamine (2 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) and dobutamine (5 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) were added to improve the cardiac output. Sodium nitroprusside (0.25 to 1 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) maintained the systolic pressure < 100 mmHg. On arrival in ICU, heart rate increased to 200 bpm. The patient received cardiac massage for severe hypotension 75 min after surgery. Emergency ECMO was instituted for circulatory support. Procainamide, digoxin, dopamine, dobutamine, sodium nitroprusside and hypothermia were continued. Sinus rhythm resumed on the first postoperative day, but procainamide and induced hypothermia at 34°C were maintained for 36 hr after normalization of the rhythm to prevent recurrence of the tachycardia. Total duration of ECMO was three and a half days. Recovery was uneventful.

Conclusion: The use of ECMO, as a first line of defence, is suitable for the emergency support of patients with JET because of the ease of support of circulation and precise control of hypothermia.

Objectif : Décrire le traitement d'une tachycardie ectopique jonctionnelle (TEJ) survenue chez un enfant, après une chirurgie cardiaque, alors qu'il a souffert d'une profonde décompensation cardiaque secondaire au rythme accéléré et a eu besoin d'oxygénation extracorporelle (OEC) pour un soutien hémodynamique.

Aspects cliniques : Un garçon de 14 jours, pesant 3,5 kg, a présenté une tachycardie ectopique jonctionnelle à la suite d'une circulation extracorporelle. La pression auriculaire gauche était de 25-28 mmHg. Il n'y a pas eu d'impact sur la tachycardie à la suite de l'entraînement électrosystolique auriculaire rapide ou l'administration de 20 μg de fentanyl iv, 45 μg de digitale, 100 mg de magnésium ou de procainamide (dose d'attaque de 15 mg, suivie de 30 $\text{mg}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). Le refroidissement actif a diminué la température nasopharyngienne à 35,2°C, tandis que la fréquence cardiaque a diminué sous 180 bpm avec une pression auriculaire gauche de 8-10 mmHg. La dopamine (2 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) et la dobutamine (5 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) ont amélioré le débit cardiaque. Le nitroprussiate de sodium (0,25 à 1 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) a maintenu la pression systolique < 100 mmHg. À l'arrivée à l'USI, la fréquence cardiaque a augmenté à 200 bpm. Le patient a reçu un massage cardiaque pour une hypotension sévère, 75 min après la chirurgie. L'OEC d'urgence a été mise en place pour assurer la circulation. On a poursuivi l'administration de procainamide, de digoxine, de dopamine, de dobutamine, de nitroprussiate de sodium et on a maintenu l'hypothermie. Le rythme sinusal s'est rétabli le jour suivant l'intervention, mais on a maintenu la procainamide et l'hypothermie provoquée à 34°C pendant 36 h après le retour du rythme normal afin de prévenir une tachycardie récurrente. La durée totale de l'OEC a été de trois jours et demi. La récupération s'est déroulée sans incident.

Conclusion : L'utilisation de l'OEC, comme défense de première ligne, est appropriée au soutien d'urgence de patients qui présentent une TEJ parce qu'elle entretient aisément la circulation et le contrôle précis de l'hypothermie.

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POSTOPERATIVE junctional ectopic tachycardia (JET) is a life-threatening arrhythmia with highest incidence in neonates and infants undergoing complex cardiac surgical repairs.¹ Although JET is of special interest to the anaesthetist because of its possible occurrence in the immediate post-repair period, its anaesthetic implications and its unusual mode of treatment, it has not been described in the anaesthesia literature. We report a case where JET was treated with extracorporeal membrane oxygenation (ECMO) support.

Case report

A 14-day-old, 3.5 kg patient with D-transposition of great vessels, patent ductus arteriosus and bidirectional atrial shunting presented for an arterial switch procedure, using hypothermic cardiopulmonary bypass (CPB). Anaesthesia was induced with inhalation of sevoflurane (3 - 5%) and maintenance consisted of intermittent fentanyl (total dose 120 µg and isoflurane (0.5 - 0.75%), with vecuronium for neuromuscular blockade (initial dose of 0.1 mg/kg, followed by intermittent doses; total dose = 2.7 mg). Monitoring included an electrocardiogram, pulse oximetry, mass spectrometry, temperature, arterial and central venous pressure. Midazolam 1 mg *iv* was given before CPB. Isoflurane 0.5% was administered through the CPB pump. After discontinuation of CPB, the patient exhibited a regular tachycardia at 200 beats·min⁻¹ (bpm) with narrow QRS. Most QRS complexes were not associated with a visible P wave, but, intermittently, a P-QRS complex was seen, indicating AV association. Systolic blood pressure was 80-100 mmHg and right atrial pressure was 6-8 mmHg. The left atrial pressure was 25-28 mmHg, but decreased to 8-10 during periods of AV synchrony or when the heart rate decreased below 180 bpm. Potassium was 3.7 mEq/L and magnesium was 1.6 mg/dl. Arterial blood gas analysis revealed pH 7.53, PCO₂ 31 mmHg, PaO₂ 295 mmHg, base excess 4.1 (FIO₂ = 1.0). Mixed venous saturation measured by an oximetrix® right atrial catheter was 70 - 73%. Administration of *iv* fentanyl 20 µg had no effect on the tachycardia. Rapid overdrive atrial pacing was effective in capturing the atria, but did not result in resolution of the tachycardia after termination of pacing. The diagnosis of junctional ectopic tachycardia was made and *iv* digitalis 45 µg, magnesium 100 mg and procainamide (15 mg loading dose over 20 min, followed by an infusion at 30 µg·kg⁻¹·min⁻¹) were administered. Active cooling was initiated, first by the circulating water mattress, then by convecting atmospheric air at room temperature over the patient's head and arms using a Bair Hugger® (Augustine Medical, Inc, Eden Prairie, MN). The patient's nasopharyngeal temperature decreased

slowly from a postbypass of 37.8°C to 35.2°C. The heart rate decreased below 180 bpm with a corresponding left atrial pressure of 8-10 mmHg. Dopamine (2 µg·kg⁻¹·min⁻¹) and dobutamine (5 µg·kg⁻¹·min⁻¹) were added to improve the cardiac output. Sodium nitroprusside (0.25 to 1 µg·kg⁻¹·min) was necessary to maintain the systolic pressure below 100 mmHg.

In the intensive care unit, the HR was 180 bpm and the blood pressure 92/55 mmHg. Rectal temperature was 33.8°C. Shortly after arrival however, the heart rate increased to a mean of 200 bpm. Seventy-five minutes after the end of surgery, the patient developed severe hypotension with a systolic pressure of 30-40 mmHg and required cardiac massage. Emergency ECMO was instituted for circulatory support. Procainamide, digoxin, dopamine, dobutamine sodium nitroprusside and hypothermia were continued. An echocardiogram obtained nine hours after institution of ECMO showed a shortening fraction of 33%, indicating good left ventricular function. An atrial electrogram obtained using the pacing wire implanted at the time of surgery demonstrated atrio-ventricular (AV) dissociation with a ventricular rate of 160 bpm (Figure 1). Sinus rhythm resumed on the first postoperative day, but procainamide drip and induced hypothermia at 34°C (rectal temperature) were maintained for 36 hr after normalization of rhythm to prevent recurrence of the tachycardia. Total duration of ECMO was three and a half days. Recovery was uneventful and the patient was discharged in good condition on the 15th postoperative day.

Discussion

Our patient exhibited a regular tachycardia at 200 bpm after termination of cardiopulmonary bypass after an arterial switch procedure. The differential diagnosis included sinus tachycardia, primary atrial tachycardia, atrioventricular reentry tachycardias and junctional ectopic tachycardia.¹ Junctional ectopic tachycardia was the most probable diagnosis in view of the presentation of a narrow-QRS tachycardia, no visible P waves, AV dissociation and intermittent AV association. The atrial electrogram obtained at a later time documented the AV dissociation with the atrial rate being slower than the ventricular rate (Figure 1).

Junctional ectopic tachycardia is due to an abnormal automaticity of the AV node with AV dissociation. The morphology of the QRS is similar to that in sinus rhythm, although a wide QRS may be seen if there is an associated bundle branch block. The ventricular rate is commonly between 180 and 250 bpm. The atrial rate is usually slower at around 140 bpm, but the P waves are difficult to identify as they wander through the QRS.¹ One-to-one retrograde conduction may result in AV association.

FIGURE 1 Tracings obtained on ECMO nine hrs postoperatively. A: Lead II ECG. B: Atrial electrogram. C: Arterial pressure.

Two forms of JET, congenital and acquired (postoperative), are recognized. Congenital JET manifests early in infancy and carries a high mortality rate. Postoperative JET, occurring either immediately after repair or within the first 72 hours, is associated most frequently with correction of tetralogy of Fallot, atrial partition procedures for transposition of the great vessels, closure of ventricular septal defect and other complex congenital cardiac repairs.¹ Manipulation of the right atrium and ventricle in the vicinity of the bundle of His seems to be a factor in the development of JET.² Till and Rowland² found at autopsy in three patients who died after postoperative JET that there were haemorrhagic tracks invading the AV bundle. These tracks originated from stitches placed close to the conduction system.

Increased adrenergic tone contributes to the aggravation of postoperative JET. The tachycardia and the loss of "atrial kick" secondary to AV dissociation cause poor ventricular filling and low cardiac output.³ This in turn results in a reactive increase of adrenergic activity which perpetuates the vicious cycle by further increasing the heart rate. Thus JET has been described as "feeding upon itself".³ This cycle of events may compound the deleterious effects of surgery and cardiopulmonary bypass on cardiac function and lead to significant mortality in the absence of palliation.³

Treatment of postoperative JET is challenging. The general goals are to decrease adrenergic tone, maxi-

mize cardiac output, increase vagal tone, normalize serum electrolytes and induce hypothermia.^{3,4} Sympathomimetics and digitalis may be used as needed to maximize cardiac output. Any drug with vagolytic effect, such as pancuronium and meperidine, must be avoided.³ Similarly, hypotensive agents, such as sodium nitroprusside, and drugs causing histamine release, such as atracurium, should be avoided or used with extreme caution, because the resulting hypotension may induce a reactive increase in tachycardia. Potassium, calcium and magnesium levels need to be optimized to high normal values.³

The armamentarium of antiarrhythmics used to treat JET is limited and includes procainamide, digitalis, amiodarone and propafenone. Procainamide is frequently used but some believe it is ineffective and may have an adverse effect on myocardial contractility and vagal tone.³ Although digitalis decreases the heart rate only by a few beats, probably through a vagal effect,⁵ it is valuable to maximize the cardiac output without increasing the adrenergic tone. Amiodarone, a class III drug, induces a prolongation of the action potential of atrial, nodal and ventricular myocardial cells. It increases the repolarization and refractory periods and has non-competitive alpha and beta adrenergic effects.⁶ Amiodarone, by itself or in association with hypothermia, has been used successfully in the treatment of JET.⁷⁻⁸ Propafenone is a class Ic antiarrhythmic agent with local anaesthetic and beta-adrenergic blocking properties. Propafenone has also been reported to be useful in the setting of postoperative JET⁹ but it is not available in an intravenous form in all countries. Both amiodarone and propafenone have proarrhythmic effects (primarily torsades de pointe) and caution should be exercised in their use in pediatric patients.^{7,10} Of interest is that verapamil can induce preexcited atrial fibrillation and should be avoided.¹¹

Induced hypothermia to a temperature of 32 - 35°C reduces the rate of automatic depolarization of all cardiac cells and, although it does not convert JET, it lowers the heart rate to around 160 bpm.^{4,12,13} This promotes better diastolic filling and higher cardiac output and permits AV sequential pacing. Shivering must be prevented, usually through muscle paralysis. Rewarming is initiated after the patient's heart rate has been stable for 24 hours. Pfammatter *et al.* reported on six infants with postoperative JET who were treated with hypothermia while under sedation, paralysis and mechanical ventilation.¹³ Lowering the rectal temperature to 32 - 34°C was successful in decreasing the tachycardia from 219 ± 27 bpm to 165 ± 25 bpm within four hours. Cooling was maintained for a period of 24 to 88 hr, without severe metabolic acidosis or

electrolyte imbalance. Predominant sinus rhythm resumed after 5.5 days (range 3 to 10 days).

Keeping with these recommendations, magnesium was administered to our patient, along with a loading dose of digitalis. Low dose dopamine and dobutamine were started in an effort to improve the cardiac output. As expected, overdrive pacing was not successful in terminating the arrhythmia.¹ Hypothermia was instituted in the operating room but the elapsed time before the cardiac decompensation might have been insufficient to allow for the reduction of the rate of firing of the AV node. In conjunction with ECMO and procainamide, hypothermia at 34°C was effective in lowering the heart rate to 160 bpm within eight hours. The use of amiodarone was planned in case the above regimen was not successful.

In spite of what seemed to be adequate treatment, the patient sustained a cardiovascular collapse necessitating ECMO for support of the circulation. We believe that this circulatory collapse was caused by the rhythm disturbance, not by intrinsic myocardial dysfunction secondary to cardiectomy or cardiopulmonary bypass. The cardiac output in the operating room was adequate, as reflected by the high mixed venous oxygen saturation. The echocardiogram performed 9 hours after the patient was placed on ECMO showed excellent myocardial function. The elevated left atrial pressure may have been related to the lack of myocardial relaxation and poor ventricular and atrial compliance induced by the high heart rate. In addition, in AV dissociation, forceful atrial contractions during ventricular systole and against a closed mitral valve produce so-called "cannon waves" that increase the atrial pressure. Effectively, the left atrial pressure decreased to 8-10 mmHg when the heart rate decreased below 180 bpm or when there was AV association.

Junctional ectopic tachycardia is a life-threatening arrhythmia. In emergency situations, extraordinary measures may be needed. Braunstein *et al.* reported on an infant who developed postoperative JET unresponsive to medical treatment and who underwent emergency cryoablation of the His bundle with a subsequent permanent pacemaker.¹⁴ Newer techniques of radiofrequency ablation may be more successful in preserving AV conduction.¹⁵ However, as JET is a transient arrhythmia usually resolving within 72 hours, ECMO support is a viable alternative to ablation in life-threatening situations. ECMO is an established effective modality for circulatory support in patients with post-cardiac surgery heart failure.¹⁶ Although the risks of infection, bleeding and neurological damage¹⁶ preclude the use of ECMO as a first line of defense, ECMO is highly suitable for emergency support of patients with JET because of the ease

of support of the circulation and precise control of the hypothermia.

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