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Perioperative Management of Junctional Ectopic Tachycardia [JET] during Cardiac Surgery. A Systematic Review

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Abstract

Perioperative junctional ectopic tachycardia [JET] is a potential life-threatening arrhythmia that mainly occurs after surgical correction of congenital heart defects. JET usually occurs intraoperatively or within the first 24 to 48 hours following cardiac surgery and is self-limiting that usually resolves within one week. The diagnosis of JET is established by the typical ECG appearance- as a narrow QRS-configuration at a rate of 170 to 260 bpm and AV-dissociation in patients with systolic and diastolic ventricular dysfunction. This arrhythmia with a high heart rate can lead to an acute deterioration of cardiac output and has a significant clinical impact on the postoperative course and intensive care stay. A variety of different therapeutic strategies have been utilized in postoperative JET. Treatment focus is to achieve as table sinus rhythm, decrease in the ventricular rate below 140-150/min and to improve the cardiac output. These various management strategies are based on specific institutional treatment protocols, including conventional supportive treatment, specific medical antiarrhythmic therapy, specific forms of pacing and surface cooling. Nowadays, the administration of high doses of amiodarone usually leads to adequate control of the rate and enables pacing. Rarely, a catheter ablation of the HIS-bundle is required in refractory cases. This systematic article reviews the literature about the management of perioperative JET over the past years and provides a specific treatment protocol.

Keywords: cardiac surgery, CPB, postoperative JET, hypothermia, amiodarone, Dexmedetomidine, cath. ablation, ECMO.

Introduction

JET is a narrow complex tachyarrhythmia arising from the atrioventricular node and His bundle area due to enhanced normal automaticity, and its differentiation from other arrhythmias especially atrioventricular nodal re-entranttachy cardia can be challenging. It is more common in children and can be seen as congenital or in postoperative settings. The diagnosis should be established based on both clinical and electrocardiogram (ECG) findings. It is associated with significant morbidity and mortality if not promptly diagnosed and treated. Controlled hypothermia, reducing the doses of vasoactive agents, correction of electrolytes abnormalities and myocardial edema, and medical treatment with amiodarone, beta blockers, dexmedetomidine usually leads to adequate control of the rate and enables pacing. Catheter ablation remains the mainstay of treatment in refractory cases with a high risk of atrioventricular block and recurrence.

Methods

Electronic searches for this systematic review included PubMed, Medline, research gate, google search up to March 2023. Selection criteria were case reports, case series studies, reviews and clinical guidelines of JET following cardiac surgery and corrective surgeries for congenital cardiac anomalies. Primary focus was on clinical presentation, pathophysiology, prevention and initial management, and management for postoperative refractory JET. This systematic review does not require any ethical clearance from the institutional committee.

Postoperative Junctional Ectopic Tachycardia

JET is most commonly encountered in children following congenital cardiac surgery with an incidence of 2-22% depending on the type of cardiac surgery performed, i.e. most frequently after TOF repair (21.9%), followed by AVSD (10.3%), VSD (3.7%), and with no occurrence after repair of common arterial trunk. The diagnostic criteria include as tachycardia with narrow QRS (wide QRS if there was bundle branch block), ventricular rate more than 170 beats/min, and atrioventricular dissociation with the ventricular rate faster than the atrial rate. ET usually occurs 24- 48 hours after CPB, whereas others have described its onset typically between 2–8 days after cardiopulmonary bypass (CPB. The occurrence of JET leads to a longer stay in the intensive care unit (ICU) and a prolonged inotropic support and mechanical ventilation. Mostly the JET rhythm subsequently converts to sinus or less frequently in the first-degree AV block. JET following paediatric cardiac surgery has been reported as a hemodynamically compromising tachyarrhythmia associated with a reported mortality up to 14%.

Etiopathology of Postoperative JET

The exact etiology is unknown; however, the development of JET is believed to result from the combination of the underlying congenital heart defect and the impact of the surgical procedure, with the proposed mechanism being a direct mechanical trauma or an indirect stretch injury to the conduction system. The global cardiac output may be reduced due to impaired ventricular filling that arises due to the loss of the atrial systolic contribution and the shortened diastolic filling time consequent upon the tachycardia. This can rapidly result in a life threatening low cardiac output state (LCOS), and increased central venous pressure, which result in multiorgan dysfunction especially if the patient was previously hemodynamically compromised. In addition, the reduced cardiac output stimulates the adrenergic system, which in turn further accelerates the

heart rate. It could have retrograded atrial conduction in 1:1 pattern or AV dissociation with variable conduction.

Perioperative risk factors for development of the JET

Peri-operative risk factors in the development of JET include younger age (Infants< 6 months old), long cardiopulmonary bypass and cross-clamp times, extensive myocardial ischemia or injury with increased CK-MB levels, transient AV block immediately post cardiopulmonary bypass, postoperative inotropic support, particularly dopamine, acidosis, electrolyte abnormalities, particularly hypomagnesaemia. The postoperative use of high doses of inotropic drugs is associated with an increased risk of JET due to the increase in cardiac systolic wall stress and heart rate and may be dose dependent. It has been observed that the independent predictors of JET, particularly in patients undergoing TOF repair are younger age, higher preoperative heart rate, cyanotic spells, non-use of β-blockers and low Mg^{2+} and $Ca^{2+}$with longer duration of CPB and aortic cross clamp time. JET after TOF corrective surgery resolves in 39.5% of patients responding to conventional measures.

Diagnosis

JET is most diagnosed as a tachycardia with rapid, regular ventricular rates of 170-260 beats per minute in lead II of ECG monitoring. The QRS complexes are usually narrow, but may be broad if a bundle branch block is present.[Figure.1]The differential diagnosis of JET includes supraventricular tachycardia, most commonly atrioventricular nodal re-entrant tachycardia (AVNRT). Adenosine bolus can be used to distinguish-it will terminate the AVNRT, whereas JET remains refractory due to its enhanced automaticity origin. The diagnosis can be conclusively proven by performing an invasive electrophysiological study. The electrophysiological hallmark of JET is that each QRS complex is preceded by a His bundle depolarization. Except when there is underlying conduction system disease, the His ventricular interval is normal.

Management of JET

The primary pathophysiologic mechanism of the JET has been postulated as the enhanced and abnormal automaticity. This is there as on for refractoriness of the JET to intravenous adenosine and direct current cardioversion or over drive pacing. A multipronged management approach has been utilized in postoperative JET to achieve a stable sinus rhythm, decrease in the ventricular rate below 140-150/min and to improve the cardiac output. The main focus of the treatment is to slow down the heart rate by adequate analgesia and sedation, correcting acidosis, electrolytes (magnesium and calcium and potassium), cooling the patient, and use of antiarrhythmic medications. Among the electrolytes, magnesium should be maintained >1.5mmol/L, potassium >4.0mmol/L and ionised calcium >1.2mmol/L. Medications used to treat JET include beta-adrenoceptor blockers (propranolol), calcium channel antagonists (verapamil), and anti-arrhythmics such as flecainide, amiodarone, and propafenone.

Occasionally pacing of the atrium at a rate higher than the JET may improve cardiac function by allowing atrial and ventricular synchrony. The most effective strategy used for reducing the rate of JET is a combination of modest hypothermia and intravenous amiodarone. Amiodarone is the safest and most effective agent. It can be administered as a bolus with 5 mg / kg IV over one hour and if postoperative JET persists, further infusion at 5µcg /kg/min is given till sinus rhythm is established or the heart rate slowed to an acceptable rate with stable hemodynamic. Once the rate has been reduced, AV synchrony can be achieved by pacing at a rate faster than the rate of the tachyarrhythmia. In addition, propranolol,

flecainide and propafenone are more commonly recommended for long term use due to the frequency of side effects associated with amiodarone. Ivabradine (0.1 *to 0.2 mg/kg/*) and iv sotalol(1 mg/kg over 1h)also appear to be an effective alternative to inadequate response to amiodarone therapy in children with post-operative JET. Procainamide, 7-10 mg/kg bolus over 30 minutes and then infusion at 40-60 mcg/kg/min has been used successfully to treat the JET.

In those who experience recurrent episodes of JET, an alternative to long term medical therapy is catheter ablation. In this procedure, the small area in which the cells initiating JET are found can be destroyed by heating or freezing the tissue. For those at risk of developing JET such as children undergoing heart surgery, treatment can also be given prophylactically.

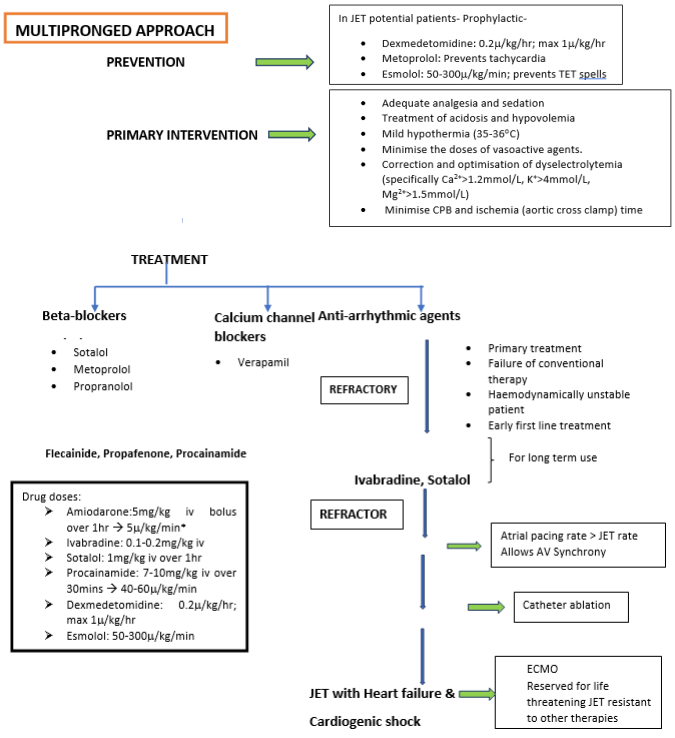
A meta-analysis of 9 studies found that sedation with dexmedetomidine reduces the risk of JET occurring post-operatively.

Dexmedetomidine infusion started at 0.2 mcg/kg/hr up to max of 1 mcg/kg/hr for extreme situations Preoperative B-blockers have been associated with significant reduction of postoperative JET. Some authors have reported that esmolol reduces the rate of JET, but also decreases the LV systolic functions, so only considered in refractory cases of JET.

Esmolol infusion at 50-300 mcg/kg/minis used to prevent the JET insusceptible patients after TOF repair, postoperative esmolol is associated with good cardiac output with minimal requirement for vasoactive support in most patients. Sotalol(1mg/kg over 1 h) with anti-arrhythmic properties similar to amiodarone and N-acetyl procainamide has been reported to be useful in the management of postoperative JET.

Mudery J et al., have reported that in patients with heart failure and cardiogenic shock secondary to congenital JET, extracorporeal membrane oxygenation (ECMO) provides time for selection of effective therapy as well as for cardiovascular support, but one should be aware of the fact that JET may have complicated the clinical course of the patient on ECLS. Therefore, ECLS is reserved for life threatening JET resistant to hypothermia and IV amiodarone. Both the in-hospital and total mortality of patients with JET has been reported as 7.1%, Similar results have been reported by Andreasen et al., with an ICU mortality of 13.5% and Mildh et al., with a total mortality rate of 7.8% in JET patients. On the bases of these above therapeutic approaches a systematic management protocol for postoperative JET management can be developed.[Figure.1]

Figure 1: Peri-Operative Management Protocol for Post-Cardiac Surgery Jet



* Infusion to be continued till sinus rhythm is achieved or heart rate is slowed to an acceptable rate with stable hemodynamic.

Conclusion

We conclude that the occurrence of JET remains an important complication during the initial postoperative period following cardiac surgery particularly TOF repair, increasing mechanical ventilation time, need for inotropic support, and prolonging the length of ICU and hospital stay, with both time to rate control and time to return to sinus rhythm having a crucial impact on the length of stay. Risk factors for development of JET are younger age at operation as well as longer ACC and CPB time and more stretch of the myocardium near the AV node. In susceptible potential high risk patients prophylaxis with preoperative beta-blockers or dexmedetomidine should be used against the JET. JET may be associated with increased in-hospital as well as total mortality. The prescribed protocol for the management of postoperative JET can be utilized for the prompt reversal to the sinus rhythm with acceptable rate and to avoid the morbidity and mortality as result of compromised hemodynamic and multiorgan failure.

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