Review Article

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Epidemiology and Management of Junctional Ectopic Tachycardia after Cardiac Surgery: A Review

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Abstract

Junctional ectopic tachycardia is a form of supraventricular tachyarrhythmia which is very common after cardiac surgery. It is more often detected in pediatric age group. The development of JET is because of multiple risk factors and triggers. The etiopathology is complex and diagnosis may be difficult. The association of JET creates hemodynamic alteration, delay extubation, ICU stay and hospital discharge. The treatment of JET is not fully established; many researches continue to explore better understanding and its management. Hence in this review authors described the different aspects epidemiology of JET after cardiac surgery in the recent literatures from different data base. The results from available studies suggest that younger age children, long bypass and cross clamp time, particular set of surgery, hyperthermia, use of inotrope and hypomagnesemia are the risk factor for inducing JET. Management of JET is complex; some occasions resistant to treatments and self-limiting. Hypothermia, judicious use of inotropes, amiodarone, dexmedetomidine, magnesium and radiofrequency ablation are most effective remedies for controlling JET.

Keywords: Junctional Ectopic Tachycardia; Cardiac Surgery; Epidemiology; Tetralogy of Fallot; Arterial Switch Operation; Dexmedetomidine; Amiodarone; Hypothermia; Magnesium.

Introduction

Junctional ectopic tachycardia (JET) is a form of supraventricular dysrhythmia occur must of the time after cardiac surgery. JET is more associated with pediatric cardiac surgery. The fast rate alters blood pressure and cardiac output leading to an increase in morbidity and mortality of the patients [1]. Some occasions may require advanced life supports like extracorporeal membrane oxygenation [2]. This increases the hospital stays duration and cost to health care. The etiology, diagnosis, epidemiology and treatment are still not fully established. Some recent developments have surfaced in this field. Hence the review article focuses all the aspects of JET in the recent years to have a better understanding and management of JET in post cardiac surgery patients.

Definition

Junctional ectopic tachycardia is a form of narrow complex supraventricular tachycardia with regular rate between 170-260/min with or without 'p' wave in ECG produced by abnormal automaticity originating from the compact atrioventricular (AV) node or the bundle of His leading to hypotension and low cardiac output [3].

Methodology

The review was conducted by literature search in Pub Med, Google scholar and Medline data base. We

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included all the clinical trial, observational case series and reports related to JET in last 15 years. The terms used for search are JET, cardiac surgery, pediatric, etiology, epidemiology, pathogenesis, treatment, risk factors, prevalence and diagnosis.

Etio-Pathogenesis

Junctional ectopic tachycardia is observed after heart surgery, congenitally and sporadically in adults with spontaneous origin. The etiology is multifactorial and still not fully established (Table 1). The congenital form was first described as a distinct category by Coumel et al. in 1976 [4], usually occurs in the first 6months of life as persistent sustained tachycardia. The clinical presentation may be dramatic, being associated with cardiomegaly or heart failure in up to 60% of cases. Secondary dilated cardiomyopathy, ventricular fibrillation and sudden cardiac death from JET have also been published [5,6].

Adults develop JET due to cardiac surgery near to conduction system and long duration of aortic cross clamp and cardiopulmonary bypass. Spontaneous developments are without any definite reason. The mechanisms like automaticity and trigger point activation are proved [7]. Pediatric age group show 2 types of JET, first a primary idiopathic disorder during infancy, configuring the so called "congenital" JET. Second type is "postoperative" variety, as a transient phenomenon immediately after cardiac surgery in children.

Pediatrics manifest JET because of injury to conduction system from cardiac surgery, fever, inflammation, electrolyte imbalance like hypomagnesaemia, hypokalemia and use of inotropic drugs [8]. The mechanism of production of JET are ectopic conduction focus at atrio-ventricular (AV) node to bundle of His junction, presence of ventricular atrial (VA) dissociation or association in conduction pathway and junctional acceleration. The high rise of inflammatory mediators like cytokines, histamines, eosinophil cation proteins, mast cells and basophil degranulation products during CPB are also implicated for the genesis of JET [9]. Pediatric cardiac surgeries like intra-cardiac repair in Tetralogy of Fallot, arterial switch operation, ventricular septal defect closure, single ventricle physiology, conotruncal defect and Sennig procedure have a high incidence of intractable JET in postoperative period [9,10].

Table 1:

Etiologies of JET
1. Pediatric heart surgery
2. Congenital
3. Adults with spontaneous origin
4. Fever,
5. Inflammation,
6. Electrolyte imbalance like hypomagnesaemia and hypokalemia
7. Inotropic and vasoactive drugs

Moak et al in a study determine that JET can be caused by prolonged ischemia and reperfusion injury to the myocytes and conduction system [11]. Reperfusion injury may originate from reactive oxygen species and free radical release in myocytes and mitochondria. Intracellular disruption of mitochondria and sarcoplasmic reticulum increases intracellular calcium levels, impairing diastolic relaxation and results in dysfunction of the conduction pathway [12].

These physiologic disturbances in turn can enhance automaticity or result in triggered activity from an ectopic focus within the AV node or bundle of His particularly in high catecholamine states. In contrast, Rekawek and colleagues reported that longer ischemic time is the best predictor of JET and suggested that JET is not related to surgery near the His bundle [13].

Diagnosis

The diagnosis is made by the typical ECG-appearance of small QRS-complexes at a rate of 170 to 260 per minute and AV-dissociation where the atrial rate is slower than the ventricular rate (Figure 1 and 2) [14]. The narrow complex tachycardia is in

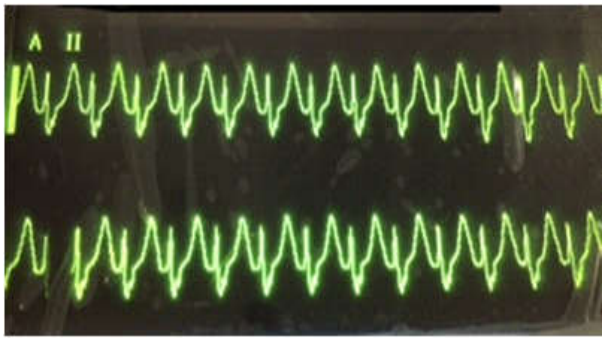

Fig. 1: ECG of a child with JET and heart rate of 170/min after intra cardiac repair for tetralogy of Fallot (TOF).

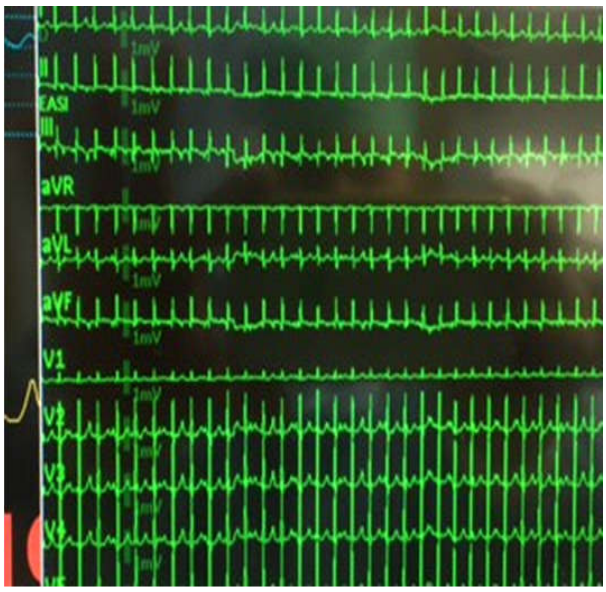

Fig. 2: ECG of a child with JET and heart rate of 250/min after ventricular septal defect closure.

the absence of bundle branch block.

Post-cardiac surgery JET is a focal supraventricular tachycardia caused by erratic automaticity arising from the compact AV node or the bundle of His. Significantly, there is an acceleration phase with an initial increase in heart rate at the beginning. The synchronization of atrio-ventricular conduction is lost. Either spontaneously or therapeutically, the heart rate peaks and then begins to slow down during the deceleration phase.

The 'P' wave is absent in the surface ECG in many occasions during the occurrence of JET [3]. If the p-wave is not visible on the rhythm-strip or 12-lead ECG, an ECG using atrial leads is helpful to diagnose. If there is still doubt whether tachyarrhythmia is due to JET, reentrant tachycardia, atrial flutter of fibrillation, ectopic atrial tachycardia or sinus tachycardia, adenosine should be administered and a rhythm-strip with atrial leads obtained [15]. The treatment of adenosine may covert JET to sinus rhythm with visible p wave. Application of atrial leads can help in diagnosis of JET. Whenever 'p' wave is present, it is inverted in deflection. Adenosine (doses of 0.1, 0.2, 0.3, 0.4 mg/kg, rapid bolus) will block the retrograde AV-conduction but not impair the ventricular rate [8]. The heart rate in adult for diagnosis of JET should be age related and compare to prior to development of the arrhythmia. Prenatal diagnosis of JET has been also reported, with the superior vena cava or ascending aorta Doppler echocardiography approach [4].

Epidemiology

Postoperative JET occurs in the range from 6% to 28.9% of all pediatric patients after surgery or repair of congenital heart defects [1,3]. The incidence is maximum in lower age group children in postoperative period [13].

Risk Factors

Studies have demonstrated high CPK-MB and troponin levels are associated with increased likelihood of postoperative JET [1]. Risk factors included high inotropic supports after surgery and extensive myocardial injury in terms of high CK-MB values and longer CPB duration [1,7,16]. A study by Moak et al, which aimed at identifying risk factors for the occurrence of postoperative(PO) JET, detected prolonged aortic cross-clamp time, longer CPB time, younger age, lower body weight and inotrope usage were associated with the occurrence of JET [10].

The surgical risk factors include resection of muscle bundles across right ventricular outflow tract and closure of ventricular septal defect in Tetralogy of Fallot (TOF) [4]. Junctional ectopic tachycardia often occurs in small children with corrective surgery including the closure of a VSD such as in AV-canals, TOF, single ventricle physiology and transposition of the great arteries with VSD [9]. It also occurs postoperatively in patients with univentricular hearts undergoing Fontan operation [17]. The relationship between circulating histamine and postoperative arrhythmias is documented [18]. Although it is not proven yet, it is highly likely that the placement of the suture lines in the area of the AV-node may lead to damage of the surrounding tissue including haemorrhage, oedema or foreign-body reaction inducing inflammation [19]. Junctional ectopic tachycardia occurred after 31months of surgery in a case report [20]. Studies have linked the presence of hypomagnesemia with JET [21,22]. Hoffman et al examined the perioperative risk factors associated with JET and identified younger age and dopamine use as risk factors for development of JET [23]. Mildh L et al in a largest retrospective study, included 1001 patients undergoing open cardiac surgery JET was diagnosed in 51 patients (5.0%) [24]. These patients had longer cardiopulmonary bypass time, higher body temperature and higher level of postoperative troponin-T. Ventricular septal defect closure was part of the surgery in 64.7% of these patients. Study by Sarubbi, et al relating familial occurrence of JET have shown incidence of 55.6% in their study, but exact gene responsible for this inheritance is not known [5]. Borgman and colleagues

Table 2: Risk factors of JET

Patient factors	Surgical factors	Other factors
1. High catecholamine states 2. Ectopic focus within av node or his bundle 3. Younger age 4. Lower body weight 5. Familial occurrence 6. Angiotensin converting enzyme (ace) deletion polymorphism	1. Longer CPB time 2. Prolonged aortic cross-clamp time 3. Ischemia and reperfusion injury 4. Surgery near the His bundle 5. High risk surgeries	1. High inotropes 2. Elevated cpk-mb and troponin levels 3. Hypomagnesemia 4. Hyperthermia 5. Milrinone

showed angiotensin converting enzyme (ACE) deletion polymorphism was found to be associated with more than two fold increase in the odds of developing JET undergoing congenital heart surgery [25]. These findings support the potential role of the renin-angiotensin-aldosterone system in the etiology of JET [25].

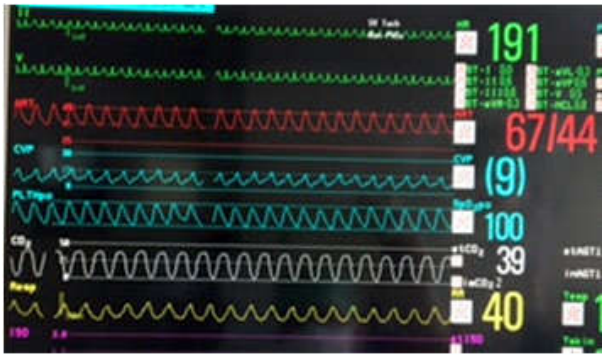

Fig. 3: Arterial BP, central venous pressure and JET with heart rate of 191/min after cardiac surgery.

Clinical Presentation

The person with JET present very fast heart rate, with or without hypotension, low cardiac output, impaired ventricular function and congestive heart failure(Figure 3) [5]. Occasionally hyperthermia is detected with these patients. Past history of tachycardia and syncope might be present.

Management

The successful management of JET is not well established till date. Studies suggest a multimodality approach for the treatment. Management can be categorized into non-pharmacological and pharmacological methods (Table 4).

Non Pharmacological Options

Temporary overdrive pacing is difficult with very higher rate, it may be tried if possible to capturethe cardiac cycle. Different techniques of temporary pacing exist that aim either to reduce the effective heart rate or to resynchronize atrial and ventricular contraction [26]. Four different strategies of external cardiac pacing in pediatric patients with postoperative JET are described. Atrial demand pacing(AAI), over drive pacing and Dual chamber (DDD, DVI) sequential pacing is relatively simple to implement, but requires a pacing rate higher than the patient's heart rate which negatively impairs diastolic filling. R-wave synchronized atrial pacing is a useful technique in pediatric patients with JET associated with very high heart rates [26].

Inducing hypothermia to 35^0C or less by air fan, air condition and cooling blanket corrects JET and preserves normal sinus rhythm. Treatment of hyperthermia with cold water blankets, antipyretics and sponging reverts back JET to sinus rhythm [27].

Pharmacological Options

Amiodarone has been reported to be effective in managing JET after pediatric heart surgery [13]. Amiodarone is a class III antiarrhythmic agent that prolongs phase 3 of action potential of myocardium, with both beta-blocker–like and potassium channel blocker like actions. Prophylactic administration of amiodarone through continuous infusion during rewarming phase was found to be effective in reducing incidence of JET by Imamura M [28].

He D et al in a large retrospective study of 1088 patients data confirmed that intra-operative usage of magnesium reduced the occurrence of postoperative JET in a larger number and more diverse group of patients with congenital heart disease [9]. Magnesium decreases inward calcium current via the calcium channels and stabilizes the membrane potential by facilitation of potassium entry into the cells. Magnesium increases the relative refractory period and decreases the vulnerable period, reducing the risk of reentry conduction.

Perioperatve dexmedetomidine therapy reduces and treats the development of JET [29]. Rajput RS and colleagues in a randomized clinical trial in 220

patients of TOF detected that perioperative dexmedetomidine therapy reduced incidence of PO JET to 9.09% compare to 20% in control, delay onset of JET and early normalization to sinus rhythm [29].

Beta blockers like metoprolol and esmolol are used to control ventricular rate. Ivabradine is a new drug that acts on SA node is successfully used to correct JET. The combination of amiodarone and ivabradine is proved to be more effective [30]. Amrousy DE et al in a study treated with amiodarone 5mg/kg bolus followed by continuous infusion 10-15 μg/kg/min for 3 days [13]. They detected that JET was reduced from 28.9 % in placebo group to 9.2 % in amiodarone group, and symptomatic JET from 11.5 % in placebo group to 3.1 % in amiodarone group [13]. Oka H et al detected landiolol was successful in treating postoperative JET [20].

Drago F et al in a retrospective study of 202 patients used catheter cryo-ablation to treat JET [31]. Cryoablation was safe and effective to abolish JET and the overall recurrence rate was 10.9% [31]. Meiltz A et al in a study of 49 patients proved radiofrequency ablation of reentrant pathway to treat permanent form of junctional reciprocating tachycardia in adults [32]. Digoxin may be tried after establishing a normal serum potassium level.

Table 3: Various treatment methods for JET

Pharmacological methods	Non-pharma methods
Amiodarone	Hypothermia
Dexmedetomidine	Radiofrequency ablation
Magnesium	Cryo-ablation
Landiolol	Sequential pacing
Ivabradine	

Prevention

The available treatments are in some occasions not fully effective to control JET. The prevention of modifiable risk factors are adjunctive to check the development of JET. The preventive measures are mentioned in table 3. Manrique AM et al in a randomized double blind study observed that magnesium administration during CPB prevented development of JET and hypomagnesemia compared to placebo group [33].

Table 4: Preventive measures of JET

Patient factor	CPB
1. Avoid hyperpyrexia	1. Wean patient from at 35^{0}C temperature
2. Correct hypomagnesemia, hypokalemia	2. Use of ultrafiltration during CPB to reduce inflammatory mediators and cytokines.
3. Judicious use of inotrope and vasoactive drugs	3. Minimize bypass and cross clamp time
4. Supplementation of magnesium during CPB	

Conclusion

To conclude JET is narrow complex supraventricular arrhythmia most commonly associated in children. Cardiac surgery is one of the major risk factor of JET. Junctional ectopic tachycardia causes hypotension, low cardiac output and increased morbidity as well as mortality. The diagnosis of JET should be confirmed after thorough 12 lead ECG study.

The risk factors for JET are younger age children, long bypass and cross clamp time, particular set of surgery, hyperthermia, use of inotrope and hypomagnesemia. Prevention and avoidance of the risk factors is best method of reducing the development of JET. Amiodarone, dexmedetomidine, magnesium and beta blockers are some of the effective treatments. Judicious use of inotropes, normalization of magnesium, potassium, and mild hypothermia reduces incidence of JET. In resistant cases cryo and radiofrequency ablation of aberrant conduction pathway establishes sinus rhythm from JET.

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