

Junctional Permanent Reciprocal Tachcardia (JPRT) in a Newborn: A Case Report

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ABSTRACT

Permanent Junctional Reciprocating Tachycardia (PJRT) is a rare form of Supraventricular Tachycardia (SVT). It generally presents in infants but can be difficult to diagnose. The characteristic EKG findings, response to Adenosine and persistence or frequent recurrences are helpful in making the diagnosis. It is usually difficult to manage with the initial and single medications used in SVT. The importance of electrophysiology and reading the ECG in making the diagnosis and differentiate from SVT.

Keywords: JPRT; SVT; Adenosine; Amiodarone; Esmolol; Flecainide ECG

INTRODUCTION

We present our case about a symptomatic newborn who was diagnosed With Permanent Junctional Reciprocal Tachycardia (PJRT) and treated early. PJRT is rarely diagnosed in the neonatal period. It is difficult to treat and is refractory to medical management. This is a case about a newborn who was symptomatic and recovered because was diagnosed early and was treated early. Early diagnosis helped improve his outcome. Supraventricular Tachycardia (SVT) is an abnormally fast heart rhythm in which the initiation or persistence of the arrhythmia requires electrical activity originating above the ventricles [1]. SVT is the most common form of cardiac tachyarrhythmia in infants and children [1-3].

The reported incidence varies widely from 1 in 250 to 1 in 25000 [1-3]. If the origin of the aberrant focus is in the sinoatrial region, it is called sinoatrial nodal re-entrant tachycardia [1].

If the arrhythmia originates in the atria, it could manifest as ectopic unifocal atrial tachycardia, multifocal atrial tachycardia or atrial flutter. If the substrate involves the Atrioventricular (AV) region, it manifests as AV Nodal Reentrant Tachycardia (AVNRT) [4], AV Reciprocating Tachycardia (AVRT) or as Junctional Ectopic Tachycardia (JET). In children, AVRT, is the most common form of SVT. While all of these tachyarrhythmias originate above the ventricle, the term SVT, by convention, usually refers to AVRT or AVNRT.

Permanent Junctional Reciprocating Tachycardia (PJRT) is a subtype of AVRT. It is also known as persistent junctional

reciprocating tachycardia. PJRT is a rare form of refractory and persistent SVT occurring predominantly in infants and children, accounting for 1% of SVT in this age group. PJRT is caused by AV re-entry using the AV node as the antegrade limb and a slowly conducting Accessory Pathway (AP) as the retrograde limb [2,5,6].

The diagnosis is rarely made in the neonatal period and the Electrocardiogram (EKG) during sinus rhythm is normal, without any manifestation of preexcitation. As the name implies, PJRT is incessant and if untreated can lead to cardiac decompensation and development of Tachycardia Induced Cardiomyopathy (TIC) [2,7,8]. TIC may resolve if early diagnosis and appropriate management is instituted.

PJRT is a subset of this type of concealed pathway causing AVRT. The AV node acts as the antegrade limb and a unique concealed AP acts as the retrograde limb. The retrograde conduction in the AP is characteristically slow and decremental, compared to the conduction velocity of normal myocardium, and is often similar to the normally slow antegrade conduction through the AV node. This similarity in conduction characteristics to the AV node thereby creates a stable reentrant circuit and results in narrow complex tachycardia. It is also the reason for the persistence and refractoriness of SVT, which can lead to left ventricular dilatation and TIC [2,7,9].

The heart rates in PJRT can range from 200 to 300 in infancy and later on decreases from 250 in early childhood to 120 in adults [7,10]. The guideline for Therapeutic Drug Monitoring (TDM)

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associated with VCM therapy aims to optimize efficacy and reduce toxicity and resistance. In particular, TDM is indispensable under special circumstances such as high-dose VCM administration, severe infection, renal dysfunction, obesity or low-weight, and burns, when distribution volume is difficult to predict [3]. It is, therefore, necessary to consider the infection site, disease severity, patient weight, renal function, and pathogen susceptibility to determine the appropriate dose of VCM required.

On the other hand, there were no studies focused on the pharmacokinetic indices of VCM in children with Central Diabetes Insipidus (CDI). CDI is characterized by deficient synthesis or secretion of antidiuretic hormone. Patients with untreated CDI typically present with polyuria, nocturia, and polydipsia, due to the initial elevation in serum sodium and osmolality. In children with CDI, the water balance tends to be negative, and the pharmacokinetic parameters can fluctuate significantly [4,5]. At first, we hypothesized that the clearance of VCM has been higher in children with CDI. However, we found different effects. In this study, we, therefore, analyzed the pharmacokinetics of VCM in five children with CDI.

Diagnostic EKG criteria:

The EKG criteria for the diagnosis of PJRT include (Figure 1),

- RP interval is longer than PR interval (because of the location and decremental conduction properties of the AP)
- 1:1 AV ratio (no dissociation)
- Inverted P wave are often visible in the inferior leads, II, III, and AVF
- Does not require a critically timed extra systole for initiation
- Not associated with frequent PACs
- PR interval is never prolonged
- The rates are typically slightly slower than typical SVT (can be confused with sinus tachycardia)
- Response to vagal maneuvers with gradual slowing of the tachycardia because of prolongation of both RP and PR intervals and eventual termination but with recurrence shortly afterwards
- The AV conduction is usually sensitive to adenosine with tachycardia terminating with AV or VA block but again recurring shortly afterwards

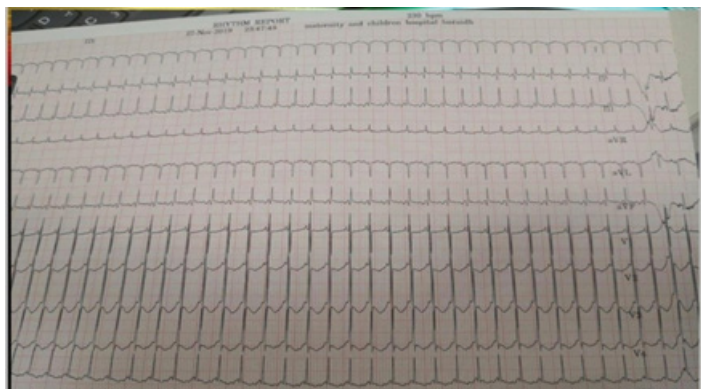


Figure 1: ECG shows narrow complex tachycardia with heart rate of 240 beats per min. The long RP interval and inverted P waves in inferior leads (lead I, lead II and aVF) is suggestive of paroxysmal junctional reciprocating tachycardia.

CASE REPORT

A newly born full-term baby was admitted to NICU due to respiratory distress and tachycardia. The baby was born after emergency cesarean section at 37 gestational weeks with a birth weight of 2700 gms. Apgar scores were 8 and 8 at the first and fifth minutes, respectively. The mother was in healthy condition at the moment of delivery and had no family history of hereditary or systemic diseases. Mother was referred to our hospital with concern of fetal tachycardia.

Upon physical examination, the patient was reactive, the anterior fontanel was normotensive, the muscle tone and neonatal reflexes were normal and body temperature was 36.7 C. Vital signs were as follow, heart rate was 230 beats/min, without murmurs and breathing sounds were clear with respiratory rate 70/min, capillary refill time was less than 2 sec, saturation was 94% and abdominal examination was normal.

NICU COURSE

Admitted to NICU, connected to ventilator due to increasing respiratory distress and 12 leads ECG was done Figure 1, impression of the NICU staff was supraventricular tachycardia and adenosine iv 2 doses given with no response and cardiologist consultation was done.

As seen by cardiologist commented as narrow complex regular long PR tachycardia with HR 240, Baby was hemodynamically stable, Echocardiography showed depressed LV function with mild to moderate pulmonary hypertension, mild Mitral regurgitation and large PDA with mainly left to right shunt, small ASD II with 4 mm diameter and bidirectional shunt. The cardiology team planned to start amiodarone 15 mic/kg/min for 4 hours followed by 5 mic/kg/min for next 20 hours with monitoring for hypotension and bradycardia.

Laboratory findings showed initially no abnormalities; in particular there was no leukocytosis, and inflammatory parameters were negative. In day 2 of life and due to persistent tachycardia rhythm another adenosine was given dose of Through umbilical venous catheter, urgent electrolytes requested which showed potassium 9.3 mmol/L. Hyperkalemia management protocols was followed and cardiologist consulted advised to optimize potassium level then give another dose of adenosine once potassium is corrected due to the effect of hyperkalemia on amiodarone (hyperkalemia may reverse the potent antiarrhythmic effects of amiodarone). In day 3 of life as per electrophysiology consultant which was consulted by telemedicine 12 leads ECG as shown in Figure 1, the showing narrow complex SVT with 1:1 relationship with long PR interval, inverted P wave in inferior leads which suggest re-entry tachycardia with high possibility of PJRT (permanent junctional reciprocal tachycardia). Echo was repeated and showed fair systolic function, mild mitral regurgitation, Tricuspid regurgitation with small PDA left to right shunt with mild pulmonary hypertension so amiodarone increased to 15 mic/kg/min and propranolol started 0.3 mg/kg/dose Q8h. In the same day esmolol infusion started at 50 mic/kg/min but patient developed hypotension which required inotropes. So, propranolol discontinued and started on flecainide 5 mg orally twice daily Amiodarone infusion changed to 10 mic/kg/min, as shown in Figure 2.

In day 5 of life (Figure 3)

- Started to taper inotropes and Increase esmolol dose to 100 mic/kg/min

- Flecainide was increased to 3 mg po TID
- Propranolol dose increased and *esmolol continued at same dose

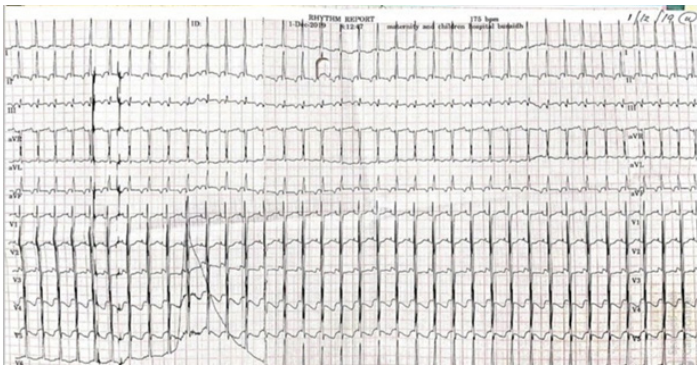


Figure 2: It shows the incessant nature of arrhythmia with no response after administration of multiple antiarrhythmic medications.

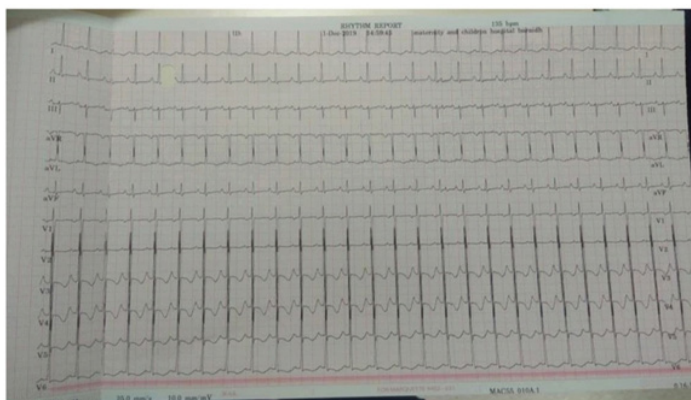


Figure 3: It shows patient reverting to normal sinus rhythm on day 4 of admission. Note the normal heart rate and normal upright P waves in inferior limb leads.

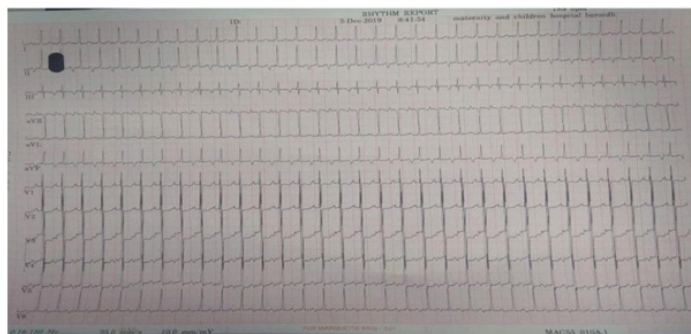


Figure 4: It shows recurrence of arrhythmia during the tapering of intravenous antiarrhythmic medications.

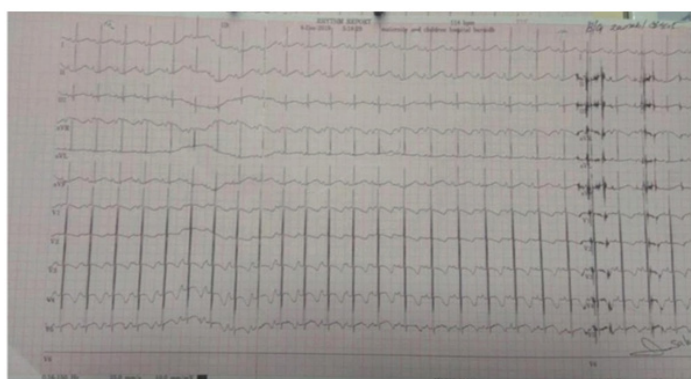


Figure 5: ECG in before discharge shows patient in normal sinus rhythm on oral antiarrhythmic medications.

- Amiodarone infusion tapered to 5 mic/kg/min and started amiodarone orally and to be stopped when 2nd dose given
- Flecainide same dose
- Patient extubated and connected to CPAP
- Antibiotics was discontinued

In day 7 of life the patient was disconnected from CPAP to nasal prong O₂. Esmolol infusion discontinued but resumed again due to recurrence of PJRT as shown in Figure 4.

In day 9 of life

Propranolol increased to 3 mg orally 3 times daily and esmolol discontinued and oral amiodarone discontinued as shown in Figure 5.

CONCLUSION

PJRT can be difficult to treat and respond to medical management our patient diagnosed early and responded to the medical treatment. This is a case about a newborn who was symptomatic and recovered because was diagnosed early and was treated early. Early diagnosis helped improve his outcome.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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