Case Report

ANNALS OF MEDICINE & SURGERY

OPEN

Synchronized cardioversion resolving refractory supraventricular tachycardia in a neonate: a case report with comprehensive analysis

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Introduction and importance: Neonatal supraventricular tachycardia (SVT) poses unique challenges in diagnosis and management, with refractory cases requiring synchronized cardioversion being exceptionally rare. This case report explores the presentation and management of refractory SVT in a neonate, emphasizing the significance of sharing such clinical scenarios.
Case presentation: A 16-day-old neonate, born via emergency caesarean section, presented with respiratory distress, poor feeding, and vomiting. Initial diagnosis of SVT was made on the basis of electrocardiography (ECG) changes. Initial attempts with adenosine failed, leading to the recurrence of tachycardia. Despite amiodarone administration, the tachycardia persisted, prompting synchronized cardioversion. Post-cardioversion, the neonate was managed with oral medications, showing sustained improvement.
Clinical discussion: This case report highlights a neonate with refractory SVT, requiring synchronized cardioversion, presenting a rare and challenging scenario. The report addresses diagnostic challenges, treatment approaches, and potential mechanisms for refractory SVT, such as delayed presentation and resistance to adenosine. Emphasizing individualized care plans and vigilant monitoring, this report is a valuable resource for healthcare professionals, contributing to neonatal cardiology understanding and emphasizing the importance of early recognition and effective interventions. Ongoing follow-up and successful outcomes underscore the need for sustained management strategies.
Conclusion: This case report sheds light on the rarity of refractory SVT in neonates, emphasizing the complexities in diagnosis and management. Successful synchronized cardioversion and subsequent oral therapy highlight the need for a multifaceted approach in neonatal SVT cases. The implications for clinical practice underscore the importance of awareness and continued research in neonatal cardiology and emergency medicine.

Keywords: case report, supraventricular tachycardia (SVT), neonate, refractory tachycardia, synchronized cardioversion

Introduction

Neonatal arrhythmias, a relatively common occurrence, can manifest in neonates with either structurally normal hearts or those with structural heart disease. They can also result from a variety of cardiovascular, systemic, and metabolic conditions[1,2]. The reported incidence of neonatal arrhythmias ranges from 1 to 5%.Supraventricular tachycardia (SVT) represents a spectrum of cardiac arrhythmias distinguished by elevated heart rates arising from atrial or atrioventricular nodal regions, thus excluding ventricular origins[3]. It is a widely encountered non-benign tachyarrhythmia within both paediatric and adult cohorts. In neonates, SVT is particularly prevalent among non-benign tachycardia's, and reentry tachycardia facilitated by accessory conduction pathways stands out as the predominant subtype of SVT observed in neonatal and infant populations[3,4]. While SVT is often manageable with medical interventions, neonatal SVT

HIGHLIGHTS

- This case reports a rare, challenging refractory supraventricular tachycardia (SVT) in a neonate, presenting a unique clinical scenario.
- Neonatal SVT's nonspecific symptoms require a high suspicion and comprehensive diagnostic approach for accurate identification.
- Managing refractory SVT in neonates involves synchronized cardioversion after failed pharmacological interventions, highlighting complexities in neonatal cardiology.
- Refractory SVT prompts consideration of delivery challenges of adenosine, atrioventricular node resistance, and delayed medical evaluation, enhancing understanding of neonatal tachycardia.
- Early recognition, individualized care plans, and vigilant monitoring are crucial in managing neonatal SVT, offering insights for healthcare professionals in neonatal and paediatric cardiology and emergency medicine.

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Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

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Annals of Medicine & Surgery (2024) 86:1782–1788

Received 27 November 2023; Accepted 24 January 2024

Published online 5 February 2024

http://dx.doi.org/10.1097/MS9.0000000000001788

presenting as refractory tachycardia and necessitating synchronized cardioversion is a rare and unique occurrence[1,4].

SVT occurrences in newborns devoid of structural heart abnormalities are relatively infrequent, typically amounting to only a few instances annually in perinatal facilities. It exhibits diverse presentations, posing challenges for diagnosis and management. Symptoms may include poor feeding, irritability, respiratory distress, and vomiting, as observed in our patient[5]. This condition tends to manifest most dramatically during the initial days of life. Notably, SVTs in this specific demographic are generally associated with a favourable prognosis, characterized by rare fatal occurrences. Nevertheless, the initial therapeutic approach to SVTs in neonates and infants can pose substantial clinical challenges[6,7]. The hallmark of SVT is an abnormally fast heart rate, often exceeding 220 beats per minute. Prompt recognition and intervention are essential because untreated SVT can lead to cardiac compromise and hemodynamic instability[1,2].

While SVT can occur in neonates, the presentation of refractory SVT requiring cardioversion is less commonly reported. Typically, SVT in neonates can be managed through vagal manoeuvres, administration of anti-arrhythmic medications (e.g. adenosine), or electrical cardioversion[4]. However, this case presents a unique challenge in neonatal cardiology due to the refractory nature of the tachycardia and the necessity for synchronized cardioversion to restore normal sinus rhythm. The rarity of such a scenario in the neonatal population warrants a comprehensive case report to shed light on its distinctive features and the management strategies employed.

Rationale

The significance of this case report lies in several key aspects. Firstly, it presents a relatively uncommon occurrence of refractory SVT in a neonate with no underlying cardiac malformations or congenital heart diseases, differentiating it from the more common neonatal SVT cases that respond to medical interventions alone. Secondly, it highlights the clinical challenge posed by the management of refractory SVT in neonates, emphasizing the need for rapid and decisive action and contributing to the knowledge base in neonatal intensive care and paediatric cardiology. Thirdly, the case offers the potential for valuable insights into the pathophysiology and management of refractory SVT in neonates, ultimately aiding in the development of more effective treatment strategies. Lastly, it serves as a valuable educational resource for healthcare professionals in neonatal care, cardiology, and emergency medicine, providing guidance and insights for similar clinical scenarios. The work has been reported in line with the SCARE 2023 criteria[8].

Case presentation

A 16-day-old male, term, appropriate for gestational age, born via emergency lower caesarean section indication being the foetal distress without any immediate complications during and following delivery, weighing 3.5 kg at birth presented in the emergency department with complaints of fast breathing, poor feeding, and vomiting for 1 day. As per the documentations on birth history the initial hours and days of the newborn's life were uneventful, marked by routine adaptation and normal development. The baby demonstrated typical behaviours, including successful initiation of breathing, prompt breastfeeding, and stable weight maintenance. Apgar scores were favourable, reflecting the newborn's overall well-being. No notable events or complications occurred, and routine procedures were conducted without any issues. Upon admission, the baby was irritable and had respiratory distress (Downe's score 6–7/10) with tachypnea, audible grunting and severe sub costal retraction. Vital signs were recorded as RR 70/min, HR of 292 bpm with regular but weak, thready pulse, and capillary refill time (CRT) of 4–5 sec. On auscultation, the baby had bilateral equal air entry and normal vesicular breath sound. Heart sounds were difficult to differentiate due to high rate with suspected murmur. Abdominal examination revealed hepatosplenomegaly indicative of cardiac involvement with liver palpable 5–6 cm below right sub costal margin and palpable spleen tip. CNS examination yielded normal findings. Consequently, the baby was promptly admitted to NICU, where he was attached with cardiac monitor and oxygen, intravenous fluid, intravenous antibiotics, and other supportive measures were initiated. The baby's blood pressure was continuously monitored in NICU. Initially the baby was in shock. The plasma glucose level was within normal range. A 12-lead electrocardiography (ECG) confirmed narrow complex tachyarrhythmia with absent P-waves suggestive of SVT as shown in the Figure 1, which was reverted to normal sinus rhythm with rapid push of intravenous adenosine at a dose of 0.1 mg/kg. The cardiac monitor showed the HR reduced to 158 bpm. The baby was monitored in NICU, his respiratory distress resolved, so feeding initiated. Chest X-rays was normal. Echo done was suggestive of moderate MR, mild TR with dilated LA/LV and left ventricular ejection fraction (LVEF) of 60%. Investigations revealed negative septic markers, C reactive protein (CRP) negative. Ionized calcium, serum electrolytes, renal function test, complete blood count and liver function tests were normal. The infant had been receiving antibiotics, specifically injection ampicillin at a dose of 200 mg/kg/day in four divided doses and amikacin at 15 mg/kg once daily, as part of the treatment for suspected late onset neonatal sepsis. This regimen was initiated when the baby was transferred from district hospital to our facility. However, we discontinued the antibiotic administration upon receiving a sterile blood culture report and confirming negative results from other septic screening assessments.

On day 3 of admission, the baby experienced another episode of tachyarrhythmia with HR of 301 bpm as shown in the Figure 2, which did not respond to three subsequent doses of adenosine. Subsequently, three loading doses of intravenous amiodarone were administered at 5 mg/kg. Despite these interventions, the tachycardia persisted. Consequently, direct current synchronized cardioversion was performed with an initial dose of 0.5 J/kg, which failed to revert the HR. A second dose of 1 J/kg was administered, ultimately restoring the baby's normal sinus rhythm with heart rate of as shown in the Figure 3. Following cardioversion, the baby continued to receive amiodarone infusion, alongside the initiation of oral metoprolol. The amiodarone drip was slowly tapered and stopped, while oral amiodarone was introduced. Oral amiodarone and metoprolol was continued. The baby continued to maintain HR in the normal range, displayed good activity and resumed regular feeding. The neonate presented with hepatosplenomegaly due to SVT. No direct interventions were taken for hepatosplenomegaly. The neonate responded well, and hepatosplenomegaly resolved spontaneously with SVT treatment. Ongoing monitoring ensures cardiac stability. Discharge occurred on the seventh day with a prescription for oral amiodarone and metoprolol, later transitioning to oral

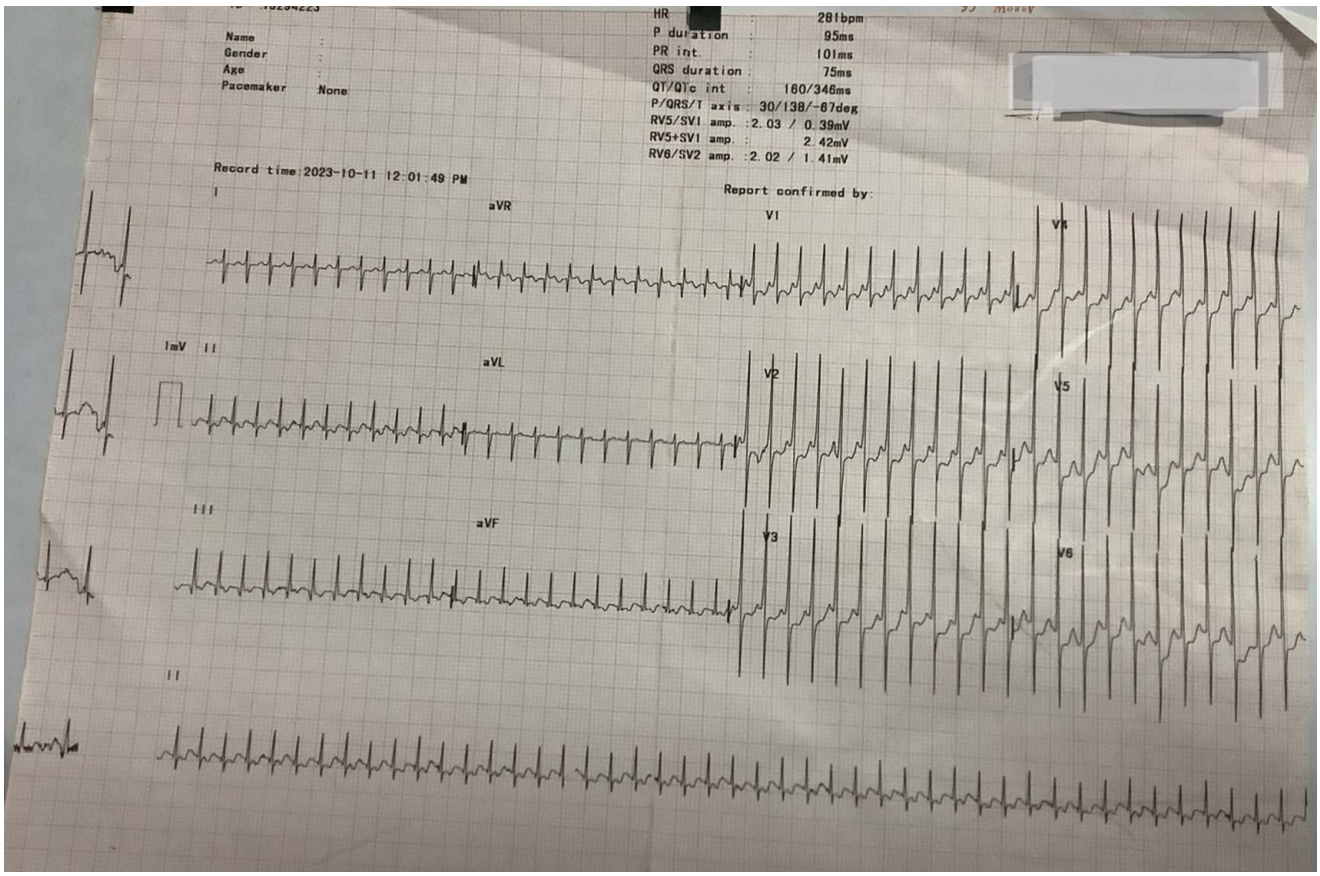

Figure 1. A 12-lead electrocardiography confirmed narrow complex tachyarrhythmia with absent P-waves suggestive of supraventricular tachycardia.

flecainide. Subsequent follow-ups indicated the baby's well-being, and ongoing monitoring is being conducted at our centre.

Discussions

SVT in neonates is relatively uncommon but critical condition that requires prompt diagnosis and management due to its potential life-threatening consequences. In this case report, we describe a 16-day-old male neonate who presented with refractory SVT, which was ultimately reverted to normal sinus rhythm through synchronized cardioversion. This case study discusses the clinical presentation, diagnostic evaluation, therapeutic interventions, and the management of this challenging case.

SVT in paediatrics is a type of arrhythmia that frequently needs immediate intervention. It occurs mostly in infants within their first year of life and involves a reentry mechanism in the A-V node. Babies with congenital heart disease, metabolic disorders, infections, or unknown causes in more than 50% of cases are commonly affected[1]. Despite the lower incidence of arrhythmias in neonates SVT is the most prevalent symptomatic tachyarrhythmia in neonates. The incidence of SVT in studies involving infants with cardiac anomalies falls within the range of 1–4 per 1000 infants, whereas among newborns without cardiac abnormalities, the occurrence rate is notably lower at 0.06 per 1000 infants[9,10].

Supraventricular tachyarrhythmia's are generally benign when the heart is healthy but can be life-threatening in the presence of heart disease or critical valve issues. Even in normal hearts, prolonged and treatment-resistant cases may cause heart failure and progress to cardiogenic shock warranting prompt treatment harmful[11]. In neonates, refractory supraventricular tachyarrhythmia's can pose a particularly challenging scenario, as their immature hearts may not respond well to conventional therapies, necessitating specialized care to manage these high-risk situations. Therefore this study adds upon the valuable insight in the management of the patients in similar scenarios. The neonate in this case presented with symptoms typical of SVT, such as fast breathing, poor feeding, and vomiting. The physical examination revealed respiratory distress, a high heart rate, and hepatosplenomegaly. These clinical findings are consistent with previous reports of neonates with SVT, emphasizing the importance of a high index of suspicion in neonates presenting with such symptoms[12].

Recognizing age-dependent variations in the clinical presentation of SVT is crucial. Delayed symptom recognition over several hours to days can result in significant hemodynamic compromise and the onset of heart failure symptoms in newborns. Neonates suffering from paroxysmal SVT typically present with heart rates ranging from 220 to 320 beats per minute, while older children exhibit heart rates within the range of 160–280 beats per min[7]. Newborns may display symptoms such as irritability, poor feeding, and tachypnea. The cardinal clinical indicators of SVT include tachycardia, which may be accompanied by hypotension, heart failure, pallor, or a reduced level of

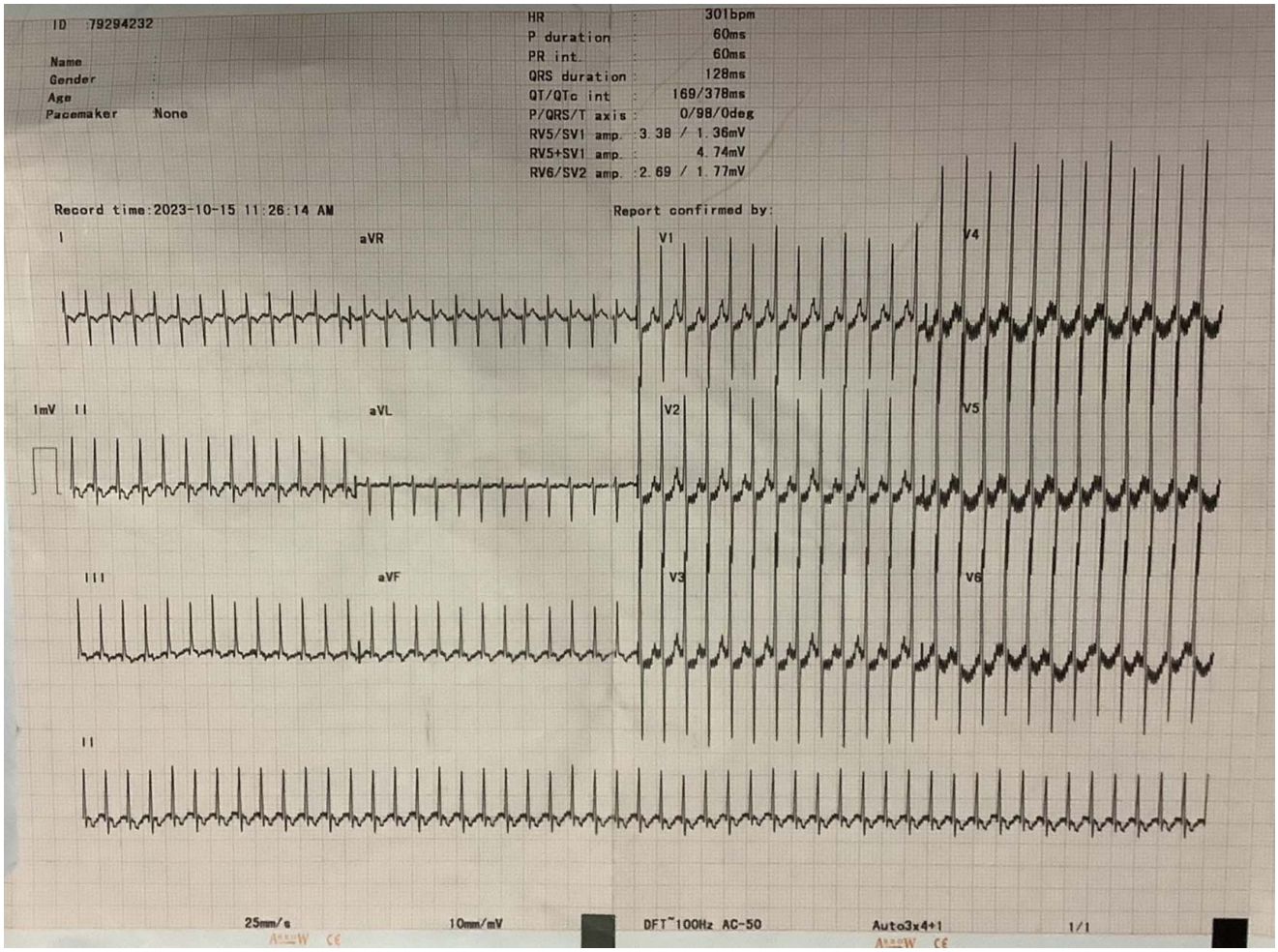

Figure 2. Electrocardiography showing tachyarrhythmia with heart rate of 301 bpm on day 3 of admission.

consciousness. Diagnosis is established when the heart rate remains consistently equal to or greater than 220 beats per min with a QRS duration less than 0.08 sec[4,12]. Failure to promptly recognize SVT can lead to significant hemodynamic compromise or the emergence of heart failure symptoms. Although SVT is frequently well-tolerated, in cases of prolonged tachycardia, it may be associated with noteworthy morbidity. Prolonged tachycardia's can manifest as refractory or treatment-resistant tachycardia's which are difficult and challenging to manage which may require electrical cardioversion as such in our case[3,4,11].

In a study by Lewis *et al.*[4] with SVT for a longer time in four infants had depressed myocardial function on echocardiogram as evident in our case where echocardiogram was suggestive of moderate MR, mild TR with dilated LA/LV and LVEF of 60% indicating reduced myocardial functions. These findings indicate that SVT may have led to the development of cardiomegaly and altered cardiac function. The presence of these indicators may contribute to refractory SVT in affected newborns. SVT can increase myocardial oxygen consumption and decrease cardiac output, potentially leading to the development of MR and TR, as observed in this neonate[13]. While many newborns may initially tolerate the early stages of SVT without significant complications, prolonged SVT lasting beyond 6–12 h can lead to the development of heart failure due to a reduction in stroke volume. Continuous monitoring and follow-up are recommended to assess and manage cardiac function, ensuring the neonate's well-being and preventing long-term complications[14,15].

Regardless of its origin, SVT typically presents in a consistent manner. Diagnosing SVT in neonates is notably challenging, primarily due to various contributing factors. First and foremost, neonates often present with symptoms that lack specificity, such as poor feeding, irritability, and respiratory distress, which complicates the differentiation of SVT from other potential conditions. Additionally, the absence of classic SVT symptoms such as palpitations or chest pain, intermittent and transient episodes, and limited access to diagnostic tools like ECG and echocardiography further challenge the diagnostic process. Moreover, the coexistence of SVT with other congenital heart diseases or arrhythmias, metabolic and systemic conditions can obscure the accurate diagnosis. Consequently, achieving a definitive diagnosis and effective management of SVT in neonates necessitates a comprehensive diagnostic approach and vigilant monitoring[4,14–16].

The diagnosis of SVT relies on a combination of clinical evaluation, history, physical examination, and ECG. Although

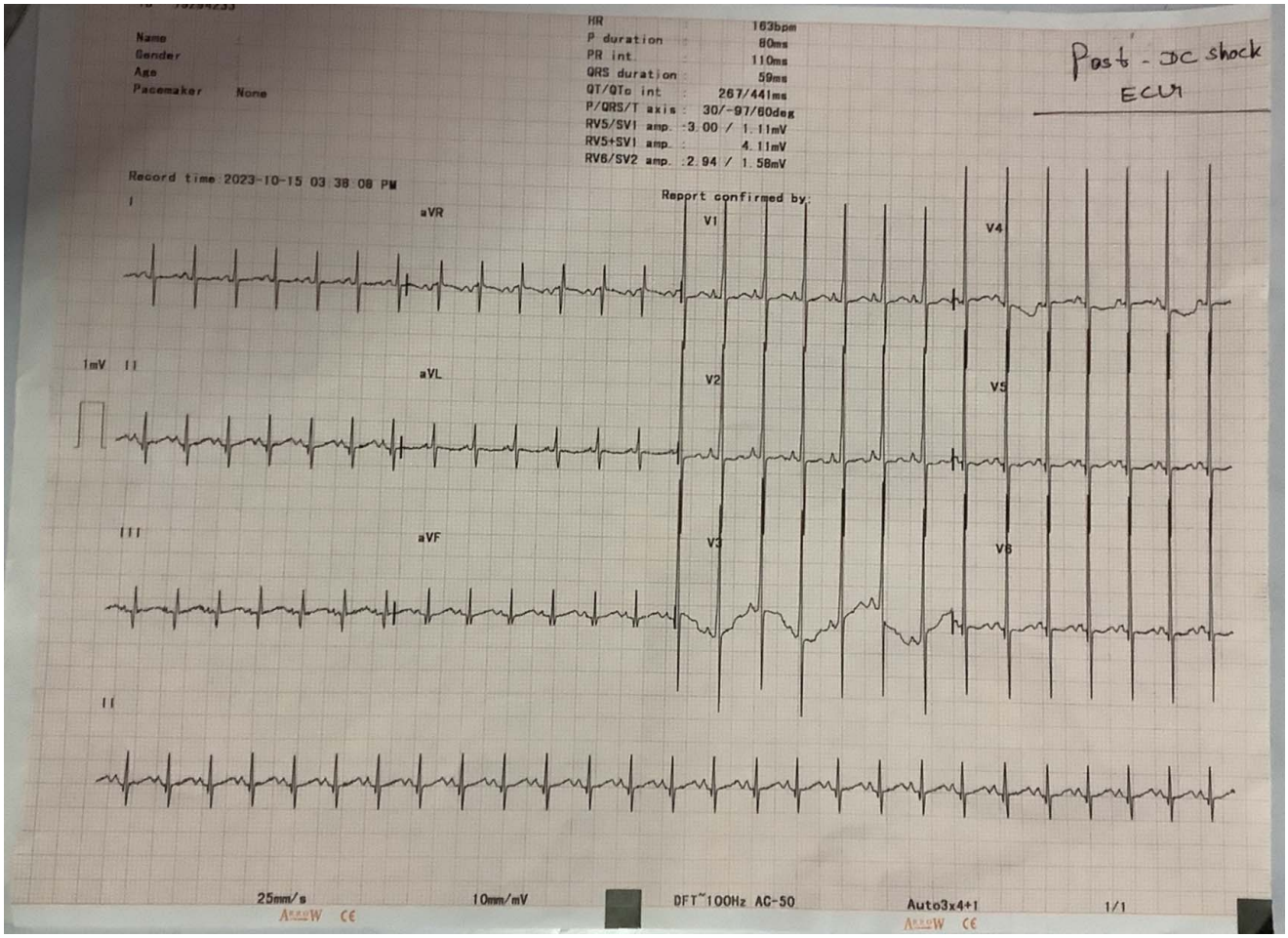

Figure 3. Normal sinus rhythm following DC (Direct Current) cardioversion.

tachycardia is a prominent feature in SVT, it can occasionally be challenging to distinguish from sinus tachycardia, which may be secondary to conditions like septicaemia or pneumonia. In such cases, certain key indicators, including the elevated heart rate, the presence of narrow QRS complexes on a 12-lead ECG, and the absence of septic markers in laboratory tests, play a pivotal role in confirming the diagnosis of SVT as supported by the above case with relevant ECG changes as shown in (Fig. 1)[17]. While the acute management of the SVT is crucial to prevent further consequences, clinical suspicion of refractory tachycardia's should be taken into account with prolonged and treatment-resistant SVTS. In the case described above, on the third day of admission, the neonate encountered another instance of tachyarrhythmia's that remained unresponsive to three subsequent adenosine doses. This condition was identified as refractory SVT, characterized by its resistance to conversion into sinus rhythm even after the administration of at least two doses of adenosine at or above the recommended levels by the American Heart Association (AHA)[4].

In the paediatric age group, SVT typically involves reentrant pathways, either accessory atrioventricular pathways or the atrioventricular node, or it may be automatic in nature. Vagal manoeuvres, like applying an ice water bag to the face to trigger the diving reflex, are an option for stable neonates. Carotid sinus massage is discouraged due to the risk of compromising cerebral circulation and airway compression, but it can be considered in older children. Managing arrhythmias with hemodynamic instability that do not respond to pharmacological cardioversion presents a considerable challenge, with the approach largely dependent on the patient's hemodynamic status[12,17]. Moreover, sustained SVT in children demands intervention due to the potential for hemodynamic deterioration. In infants and neonates, it can quickly escalate to a medical emergency, often leading to shock[17].

The neonate in the above case was swiftly returned to a normal sinus rhythm through the intravenous administration of adenosine at a dosage of 0.1 mg/kg. The cardiac monitor displayed a reduced heart rate of 158 bpm. Following this, the baby was placed under NICU observation, and as the respiratory distress abated, feeding was initiated. Following the recurrence of tachyarrhythmia in the neonate, three consecutive doses of adenosine were administered without success. In response to the persistent tachycardia, two loading doses of amiodarone were given. Despite these interventions, the tachycardia persisted, leading to the decision to perform two synchronized DC cardioversions due to its refractory nature. Ultimately, these cardioversions successfully restored the baby's normal sinus rhythm. The case typically highlights the consequence of a prolonged SVT

manifesting as refractory tachycardia reverted back by synchronized cardioversion which needed closed monitoring and rapid management.

In the majority of cases, SVT episodes in infants and neonates are typically resolved, either spontaneously or following the application of vagal manoeuvres. The effectiveness of vagal manoeuvres tends to be more pronounced in younger patients when contrasted with those aged 1 year and older[4]. Pharmacological intervention in SVT unless the patient is extremely sick often necessitates the use of adenosine, universally recognized for its efficacy during acute management. Adenosine acts by inducing a transient block of the atrioventricular node, thus interrupting the reentrant pathway. Notably, it possesses an exceptionally brief half-life of 10–15 sec and is highly effective in terminating SVT episodes in the majority of patients[17,18]. In the above case when the outcome remained ineffective with intravenous adenosine all the relevant investigations were performed to rule out any possibilities of secondary causes of arrhythmias which were unremarkable. In case of failure of this first-line drug, anti-arrhythmic drugs like class IA (procainamide or quinidine), class IC (flecainide), or class III (amiodarone or stall) drugs can be considered[3]. Amiodarone serves as a chronic anti-arrhythmic for neonates with challenging tachycardia's. It's chosen when initial control is difficult or other agents fail. Amiodarone, alone or with beta blockers, effectively manages neonatal tachycardia, proving well-tolerated, safe, and clinically effective within three months[19]. The choice to proceed with cardioversion hinges on the patient's hemodynamic stability and cardiovascular condition, particularly if SVT is unresponsive to anti-arrhythmic drugs reflecting its refractory nature. Subsequently, diligent monitoring is essential for timely identification and management of any recurrences[4].

Prior to commencing cardioversion, we employed adenosine as the primary pharmacological intervention for acute medical cardioversion, following the paediatric tachycardia with a pulse 2020 update. The initial intravenous (IV) dose administered was 0.1 mg/kg, with no discernible response observed within 2 min. Consequently, a second dose of 0.2 mg/kg was repeated, accompanied by continuous ECG monitoring. Unfortunately, adenosine proved ineffective in converting SVT to sinus rhythm.

In cases where SVT is refractory to adenosine, the next course of action involves second-line agents such as intravenous anti-arrhythmics, namely procainamide and amiodarone. Intravenous beta-blocker therapy stands as an alternative to intravenous anti-arrhythmic therapy for hemodynamically stable patients. In this instance, we opted for IV amiodarone, as IV procainamide was unavailable at the time. The administration of IV amiodarone took the form of a bolus infusion at a rate of 5 mg/kg over 20–60 min, as per recommended guidelines. In light of an inadequate response, the bolus was repeated, reaching a cumulative total of 15 mg/kg. Upon achieving arrhythmia control, the infant transitioned to oral maintenance therapy with metoprolol and amiodarone. Subsequently, the amiodarone tablet was substituted with flecainide when the latter became accessible. Flecainide is considered more effective and safer than amiodarone, earning it a recommendation as one of the first-line treatments for SVT in infants and children.

As described above due to the unresponsiveness to the anti-arrhythmic medications to their optimal doses and frequencies and considering the myocardial depression as evident by the echocardiography performed led to the decision of performing synchronize DC cardioversion which was done with an initial dose of 0.5 J/kg, which failed to revert the heart rate. A second dose of 1 J/kg was administered, ultimately restoring the baby's normal sinus rhythm.

The mechanisms behind refractory nature SVT and the reason behind the diminished response rate to pharmacological intervention particularly with anti-arrhythmic drugs like adenosine in neonates remains unclear, prompting consideration of several potential mechanisms. These include: (1) Challenges related to effectively delivering adenosine to infants and neonates, possibly stemming from slow infusion rates via small intravenous catheters, distant placement of intravenous access sites, or incomplete clearance of adenosine due to the saline flush process through the 3-way stopcock. (2) The possibility of relative resistance in the atrioventricular node to the effects of adenosine. (3) The likelihood that the neonates seeking medical evaluation may do so later in the course of their illness, possibly contributing to a reduced response to treatment attributable to lower cardiac output[4,5,17].

As defined in the literature by Gilljam *et al.*[5] refractory tachycardia has to be designated if time to control (conversion) exceeded 6 days or if more than two anti-arrhythmic drugs which is consistent with the above case. Predisposing factors for SVT's in neonates can include maternal medication use, structural heart defects, maternal history of arrhythmias, prematurity, electrolyte imbalances, and certain infections[20]. Pertaining to the above case the exact mechanism for such medical condition could not be evaluated as all the investigations for any such possible nature were unremarkable.

The refractory nature of the tachycardia in this case may be attributed to various factors. The initial presentation with symptoms like fast breathing, poor feeding, and vomiting suggested an underlying medical condition, potentially affecting cardiac function. Findings of hepatosplenomegaly and a cardiac murmur hinted at structural issues. Initial response to adenosine could be due to a transient mechanism, but the recurrence indicated a deeper cardiac problem. Negative septic markers ruled out infection. Cardiac abnormalities seen on echocardiography, including mitral and tricuspid regurgitation, dilated chambers, and reduced LVEF, pointed to possible structural or functional cardiac issues. Multiple adenosine doses and amiodarone failed, suggesting intrinsic electrical abnormalities or treatment-resistant mechanisms. Synchronized cardioversion was required to restore normal rhythm. Continued medication and tapering of amiodarone infusion maintained heart rate and improved the baby's condition, underlining the complexity of neonatal tachycardia diagnosis and management.

The long-term implications of oral amiodarone and metoprolol maintenance therapy necessitate vigilant monitoring for side effects and individualized regimen adjustments. Amiodarone, a Class III anti-arrhythmic, may induce hypothyroidism, pulmonary complications, and severe extravasation injuries. Metoprolol, a beta-blocker, may lead to bradycardia and hypotension. Essential aspects of monitoring include regular assessment of cardiac function, blood pressure, and potential complications such as liver, thyroid, and pulmonary dysfunction. The regimen selection is influenced by the patient's specific arrhythmia, medical history, and considerations for potential drug interactions. This comprehensive monitoring approach ensures the early detection of adverse effects and enables tailored management for optimal patient outcomes[20,21].

After successful cardioversion, the neonate was transitioned to oral amiodarone and metoprolol therapy. The gradual tapering and discontinuation of amiodarone infusion and the continuation of oral amiodarone and metoprolol allowed the maintenance of a normal heart rate and the resumption of regular activities and feeding. This comprehensive approach to therapy demonstrates the importance of ongoing management to prevent SVT recurrence.

Conclusion

This case report highlights the rare occurrence of refractory SVT in a neonate, emphasizing the need for early recognition and effective management. The case underscores the complexity of diagnosing SVT in neonates, necessitating comprehensive diagnostic approaches. Successful treatment through synchronized cardioversion is a key aspect, shedding light on the challenges of neonatal tachycardia. The report's implications for clinical practice stress the importance of vigilance among healthcare professionals in neonatal and paediatric cardiology, and it provides insights into the pathophysiology and treatment of refractory SVT. It serves as a valuable educational resource, contributing to the development of neonatal cardiology and emergency medicine.

Ethical approval

The study is exempt from ethical approval in our institution.

Consent

Written informed consent was obtained from the patient's parents/legal guardian for publication and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Source of funding

None.

Author contribution

N.K.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript, T.N.Y.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript, A.B.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript, N.P.: follow-up the patient, and final approval of the manuscript.

Conflicts of interest disclosure

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Research registration unique identifying number (UIN)

None.

Guarantor

Tek Nath Yogi.

Data availability statement

None.

Provenance and peer review

Not commissioned, externally peer-reviewed.

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