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Recurrent Supraventricular Tachycardia in a Child with Turner Syndrome and Confirmed Ebstein Anomaly: A Case Report

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Abstract: Turner syndrome is associated predominantly with left-sided cardiovascular anomalies, whereas Ebstein anomaly and recurrent supraventricular tachycardia are less characteristic findings. We describe a 9-year-old girl with a 45, X karyotype, horseshoe kidney, and recurrent symptomatic supraventricular tachycardia requiring repeated emergency treatment. Echocardiography demonstrated tricuspid valve dysplasia, regurgitation, and right atrial enlargement. Cardiac magnetic resonance angiography performed on September 6, 2022, followed by cardiac catheterization on September 8, confirmed Ebstein anomaly, according to the authors' supplementary clinical information. During hospitalization, a sustained episode with a heart rate of 205–215 beats/min and an initial blood pressure of 80/40 mmHg persisted despite vagal maneuvers and three doses of adenosine, and terminated after intravenous amiodarone (5 mg/kg). Electrophysiology referral was recommended because of recurrent tachycardia and difficulty achieving durable pharmacologic control. The case emphasizes the value of multimodality imaging, rhythm-specific assessment, and multidisciplinary management. Detailed magnetic resonance and catheterization measurements and a verified long-term follow-up were unavailable.

Keywords: Turner syndrome; Ebstein anomaly; supraventricular tachycardia; pediatric electrophysiology.

1. Introduction

Turner syndrome (TS), which results from complete or partial loss of one sex chromosome, is associated with multisystem comorbidity and a substantial burden of congenital cardiovascular disease. The most characteristic structural abnormalities are bicuspid aortic valve, coarctation of the aorta, and other left-sided or aortic abnormalities. International guidance recommends individualized cardiac imaging and lifelong surveillance [1].

By contrast, Ebstein anomaly, a congenital malformation of the tricuspid valve and right ventricle, is not a classic structural manifestation of TS. Ebstein anomaly may be associated with tricuspid regurgitation, enlargement of the right atrium, and supraventricular tachyarrhythmias, including accessory pathway-mediated reentrant tachycardia [2,3]. However, neither the presence of Ebstein anomaly nor the presence of TS alone determines the mechanism of a particular tachycardia.

Published cases have documented supraventricular tachycardia in Turner syndrome, although its pathophysiologic relationship to the chromosomal condition remains uncertain [3]. Pediatric electrophysiology management becomes more challenging when recurrent symptomatic tachycardia coexists with altered right-sided cardiac anatomy. Detailed imaging, ECG-based rhythm analysis, and, when appropriate, invasive electrophysiologic evaluation are therefore essential [4,5]. We report a child with cytogenetically confirmed TS, Ebstein anomaly confirmed by additional cardiovascular investigations, and recurrent supraventricular tachycardia requiring escalation of treatment.

2. Case Presentation

A 9-year-old girl weighing approximately 24 kg, with Turner syndrome confirmed by a 45,X karyotype and a horseshoe kidney, was admitted to a tertiary pediatric hospital on August 20, 2022, following recurrent episodes of palpitations and supraventricular tachycardia (SVT). The caregiver reported episodes beginning on July 16, 2022, accompanied by chest pain, pallor, nausea, and vomiting. Several previous emergency

department visits had required intravenous adenosine. An episode on August 19 prompted referral for specialist evaluation.

The cardiovascular history included congenital tricuspid valve abnormalities and regurgitation. Echocardiograms obtained in October 2021 and June 2022 had documented thickening or altered mobility of tricuspid leaflets, partial adherence of the septal leaflet, right-sided chamber enlargement, and moderate tricuspid regurgitation. Left ventricular ejection fraction was approximately 70%. The patient had received captopril (12.5 mg twice daily). Propranolol (20 mg/day) was subsequently prescribed for tachycardia but was discontinued following symptomatic hypotension.

At admission, the patient was alert, afebrile, and clinically stable, with a heart rate of 78 beats/min, respiratory rate of 20 breaths/min, blood pressure of 114/79 mmHg, and oxygen saturation of 96% on room air. Cardiac auscultation identified a grade 2/6 systolic murmur. Peripheral perfusion and the neurologic examination were unremarkable. The admission ECG demonstrated sinus rhythm; another ECG described right-axis deviation and features of right ventricular overload. Electrolytes and routine laboratory measurements did not suggest a major reversible metabolic trigger. Oral metoprolol succinate was initiated at 25 mg nightly, and captopril was discontinued.

Transthoracic echocardiography on August 23, 2022, showed mild right atrial enlargement, a patent foramen ovale, dysplasia and prolapse of the tricuspid valve leaflets, and mild tricuspid regurgitation. The report suggested the possibility of mild Ebstein anomaly but did not contain definitive quantitative measurements of apical leaflet displacement. The right ventricle was described as mildly enlarged with impaired systolic function; left ventricular findings were not significant. Intermittent palpitations persisted, including brief self-limited episodes on August 30 and 31.

On September 4, 2022, the patient developed a sustained tachycardia with a recorded heart rate of 205–215 beats/min and an initial blood pressure of 80/40 mmHg. A 12-lead ECG was interpreted clinically as SVT. Vagal maneuvers and three intravenous doses of adenosine failed to terminate the episode. Repeat blood pressure was 90/60 mmHg while tachycardia persisted. Under monitoring in the emergency treatment area, intravenous amiodarone was administered at 5 mg/kg over approximately 30 minutes. The tachycardia terminated, with the heart rate decreasing to approximately 105 beats/min; she subsequently remained clinically stable and returned to the pediatric ward after observation.

Repeat echocardiography on September 6, 2022, demonstrated a right atrial volume index of 51 mL/m², tricuspid leaflet abnormalities, moderate tricuspid regurgitation, a patent foramen ovale, and preserved right ventricular systolic function (tricuspid annular plane systolic excursion, 2.1 cm). Left ventricular ejection fraction was 70%. Although the echocardiographic interpretation continued to describe Ebstein anomaly as a possibility, the authors report that cardiac magnetic resonance angiography performed on the same day, September 6, 2022, established the diagnosis of Ebstein anomaly; cardiac catheterization performed two days later, on September 8, confirmed it. The original reports and quantitative imaging and catheterization findings were not available for independent review; accordingly, leaflet-displacement measurements, chamber volumetry, pressure data, and procedural details are not reported here.

Because of repeated symptomatic SVT and limited durable control with antiarrhythmic medication, cardiology and electrophysiology teams recommended specialized electrophysiologic assessment and consideration of catheter ablation. The treatment regimen was adjusted to include oral amiodarone and progressive reduction of metoprolol. Before oral amiodarone, liver and thyroid tests were obtained (aspartate aminotransferase 30.8 U/L, alanine aminotransferase 23 U/L, thyroid-stimulating hormone 3.2 μIU/mL, and free thyroxine 0.89 ng/dL). On September 9, the patient was discharged clinically stable with specialist follow-up and medication instructions.

A previously prepared conference abstract described later ablation and an asymptomatic course; however, an ablation report, the electrophysiologic mechanism, procedural date, and the duration of verified follow-up were not available in the records reviewed. Therefore, a definitive ablation outcome is not claimed in this report.

3. Discussion

This case is notable for the coexistence of three distinct clinical dimensions: a 45,X chromosomal diagnosis, right-sided congenital valve disease confirmed as Ebstein anomaly by multimodality evaluation, and recurrent symptomatic SVT. Cardiovascular surveillance in TS focuses primarily on aortic and left-sided lesions [1]. Ebstein anomaly should therefore be assessed on its own anatomic and physiologic merits; co-occurrence does not establish that TS caused the tricuspid abnormality.

Ebstein anomaly results from abnormal delamination and apical displacement of portions of the tricuspid valve, potentially leading to an atrialized right ventricular segment, regurgitation, and right atrial enlargement. Transthoracic echocardiography is the initial diagnostic modality, while cardiac magnetic resonance can better

delineate right ventricular anatomy, chamber size, and regurgitation, particularly when echocardiographic findings are equivocal [2]. This distinction mattered here: successive echocardiograms suggested Ebstein anomaly, while the authors report subsequent confirmation by cardiac magnetic resonance angiography and catheterization.

Ebstein anomaly is associated with a substantial burden of supraventricular arrhythmias. Accessory atrioventricular pathways are frequently located near the anatomically distorted tricuspid annulus, although atrioventricular nodal reentrant tachycardia and atrial tachycardias are also possible [3,5]. In this patient, the available ECG descriptions establish recurrent SVT but do not identify a specific circuit or demonstrate pre-excitation. A pathway-mediated mechanism must not be inferred solely from the structural diagnosis.

The September 4 episode had an important hemodynamic component, with an initial blood pressure of 80/40 mmHg. Failure of adenosine to terminate the rhythm does not, by itself, establish a non-AV-node-dependent arrhythmia, because effectiveness depends on mechanism, venous delivery, and timing. Amiodarone was followed by termination in this case; this observation describes the documented clinical response rather than recommending amiodarone as universally preferred therapy. Choice of acute treatment in pediatric SVT must depend on the ECG, the patient's perfusion and stability, and current resuscitation protocols.

Ablation is an established option for selected children with recurrent or poorly tolerated SVT. The 2016 PACES/HRS consensus emphasizes individualized decision-making based on symptoms, patient size, congenital anatomy, and specialized expertise [4]. Abnormal right-sided anatomy in Ebstein anomaly complicates mapping because the functional valve orifice and the true atrioventricular junction may not coincide. A 2024 pediatric case series described the utility of high-density open-window mapping for localizing accessory pathways in such patients [5]. These data support referral to an experienced pediatric electrophysiology team, but do not establish the mechanism or procedural outcome in our patient.

The management of Ebstein anomaly requires longitudinal assessment of the tricuspid valve, right ventricular size and function, interatrial communications, arrhythmia burden, and symptoms. The 2025 AATS expert consensus identifies cardiac magnetic resonance as a useful modality for comprehensive chamber and regurgitation assessment, and emphasizes individualized surgical and arrhythmia-related decisions [2]. Concurrent TS requires continued evaluation of aortic and other multisystem risks under TS-specific guidance [1].

The principal limitations are the absence of original magnetic resonance and catheterization reports, incomplete 12-lead ECG tracings during SVT and adenosine administration, no definitive intracardiac electrophysiologic mechanism, and no documented long-term follow-up or procedure report establishing ablation success. Confirmation of Ebstein anomaly and the dates of cardiac magnetic resonance and catheterization were supplied retrospectively by the lead author and should ideally be corroborated by source reports before publication. Patient consent and institutional publication requirements also require documentation.

4. Conclusion

Recurrent SVT in a child with TS and Ebstein anomaly illustrates why chromosomal, anatomic, and electrophysiologic diagnoses should be assessed separately and then integrated. In this case, echocardiography raised suspicion of Ebstein anomaly, with confirmation reported following cardiac magnetic resonance angiography on September 6, 2022, and cardiac catheterization on September 8. Recurrent tachycardia, including an episode unresponsive to vagal maneuvers and adenosine, prompted specialist electrophysiologic referral. Detailed anatomical and rhythm characterization and longitudinal follow-up remain essential for patient-specific treatment.

5. Declarations

Patient consent: Written consent for publication must be documented in accordance with institutional and journal policies before publication. The available records do not establish consent status.

Ethics approval: Institutional requirements for retrospective publication of a pediatric case must be confirmed.

Author contributions and competing interests: To be confirmed by all coauthors before final journal submission.

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