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CASE REPORT

Atrial Thrombus in a Preschooler with Nephrotic Syndrome: A Case Report

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Thrombosis,
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Abstract

Introduction: Nephrotic syndrome is characterized by proteinuria, hypoalbuminemia, edema, and hyperlipidemia, often associated with thromboembolism due to urinary loss of anticoagulant proteins. Intracardiac thrombi are rare but severe and require differentiation from cardiac tumors via echocardiography. Treatment includes anticoagulation, thrombolysis, or surgery. **Objective:** To report a case of atrial thrombus in a preschool child with nephrotic syndrome, highlighting its relevance for pediatricians and specialists. **Method:** A case report based on the review of medical records of a patient followed up at a pediatric cardiology university hospital. **Case Report:** A 3-year-old female patient with decompensated nephrotic syndrome. Computed tomography revealed a hypodense image in the right atrial appendage, suggesting a thrombus. Transthoracic echocardiography showed a hyperreflective mass measuring 1.7 x 3.2 cm, mobile, and not attached to the atrial walls or tricuspid valve, with no flow obstruction. The patient was asymptomatic from a cardiac perspective. Full anticoagulation with low molecular weight heparin was initiated. Cardiac magnetic resonance imaging confirmed the diagnosis. Serial echocardiograms were performed for follow-up. **Discussion:** Children with nephrotic syndrome have an increased risk of thrombosis due to the loss of anticoagulant proteins. Atrial thrombus, though rare, can lead to severe complications. Management requires continuous monitoring of renal and cardiac function, along with careful anticoagulation adjustments to prevent hemorrhagic complications. A multidisciplinary approach is essential to optimize treatment and reduce associated risks.

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INTRODUCTION

Nephrotic syndrome is characterized by proteinuria, hypoalbuminemia, edema, and hyperlipidemia, and is often associated with thromboembolic events due to urinary loss of anticoagulants.[1,2] The most severe complication is the formation of intracardiac thrombi, which carries high morbidity and mortality, although it is rare. Differentiating thrombi from cardiac tumors is essential and is primarily performed by echocardiography. Treatment may include anticoagulation, thrombolysis, or surgical removal.[2]

The aim of this study is to analyze this rare clinical condition and highlight its importance for specialists and pediatricians. This is a descriptive observational study based on the review of the medical record of a preschool patient treated at a university hospital, followed by a literature review in the PUBMED database using the terms thrombus, heart diseases, and nephrotic syndrome.

CASE DESCRIPTION

A 3-year-old female patient with a previous diagnosis of nephrotic syndrome was hospitalized due to disease decompensation. Abdominal and chest computed tomography revealed a hypodense image in the right atrial appendage/right atrium, compatible with a thrombus or a primary lesion. Echocardiography showed a mobile, non-adherent, hyperechogenic mass measuring 1.7 × 3.2 cm, not attached to the atrial walls or the tricuspid valve, with no obstruction to blood flow (Figure 1). The patient remained asymptomatic from a cardiological standpoint.

Given this scenario, full anticoagulation with low-molecular-weight heparin was initiated. Cardiac magnetic resonance imaging confirmed the diagnosis of an atrial thrombus (Figure 2). Serial echocardiograms were performed for follow-up, showing a progressive reduction of the thrombus.

The cardiac surgery and pediatric cardiology teams assessed that there was no indication for surgical removal, as the thrombus did not cause significant obstruction. The patient was monitored regularly, with anticoagulation adjustments as needed.

LITERATURE REVIEW

Nephrotic syndrome is defined by heavy proteinuria, hypoalbuminemia, edema, and hyperlipidemia. It may be caused by primary or secondary kidney diseases, such as infections or collagen vascular diseases. Treatment often involves corticosteroids and may include immunosuppressants in steroid-resistant cases. For edema control, diuretics are indicated, while angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) help reduce proteinuria and control blood pressure. Complications of

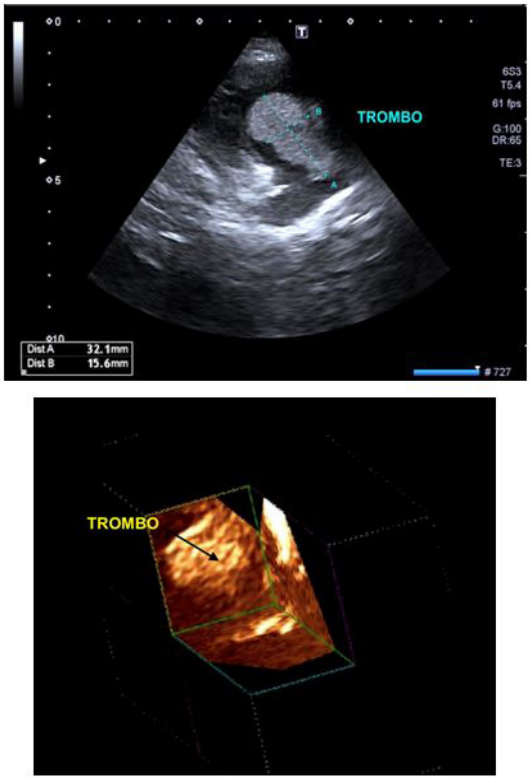


Figure 1. Two-dimensional (A) and three-dimensional (B) echocardiography of the patient. The thrombus can be identified by hyperechogenicity in the right atrium.

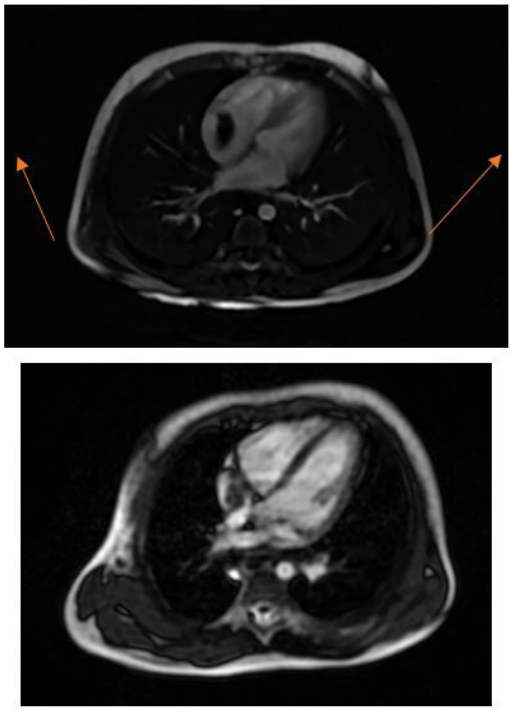


Figure 2. Cardiac magnetic resonance imaging of the patient. The thrombus (red arrow) can be identified by hypoechogenicity and lack of contrast enhancement.

nephrotic syndrome include infections, hypovolemia, kidney failure, and thromboembolic events, the latter being of particular concern.[1,2] The incidence of thrombosis in children with nephrotic syndrome is approximately 3%.[2] Factors such as hypoalbuminemia, hypercoagulability, immobilization, and prolonged corticosteroid use increase thrombotic risk.

Among thromboembolic events, intracardiac thrombus formation is rare but may lead to systemic or pulmonary embolism.[3] The differential diagnosis includes cardiac tumors, infectious vegetations, and mural thrombi. Echocardiography is the main diagnostic tool, complemented by cardiac magnetic resonance imaging and CT angiography for more detailed characterization.[3,4]

Primary cardiac tumors are rare in childhood, with an incidence below 0.3%. Rhabdomyoma is the most common in this age group, followed by myxomas, which are usually mobile and pedunculated. Cardiac hemangiomas may be associated with venous obstruction and arrhythmias. Differentiating thrombi from tumors is essential for defining appropriate treatment. Management of intracardiac thrombi depends on location, size, and embolization risk. Mobile thrombi or those larger than 2 cm usually require surgical removal, whereas smaller cases may be treated with anticoagulation or thrombolysis (Table 1).[5,6] The use of low-molecular-weight heparin is a common strategy, monitored by factor Xa activity. Anticoagulation should be carefully adjusted to avoid hemorrhagic complications. In some cases, thrombolysis may be preferred, especially when there are surgical contraindications. Prolonged anticoagulant therapy may be necessary in patients with persistent thrombotic risk.[7,8] Regular follow-up with imaging tests is essential to assess thrombus regression and prevent recurrences.[8] A multidisciplinary approach, including nephrologists, cardiologists, and hematologists, is essential to optimize treatment and reduce associated risks.Nephrotic syndrome can result in severe thromboembolic complications, including intracardiac thrombi. Early diagnosis and appropriate management with anticoagulation are essential to reduce morbidity and mortality. Differentiation between thrombi and cardiac tumors should be performed using appropriate imaging studies, guiding the best therapeutic strategy.[8,9]

In conclusion, the reported case highlights the importance of continuous monitoring of renal and cardiac function, together with careful adjustments in anticoagulant therapy, thereby reducing the risk of complications. In addition, it reinforces the relevance of knowledge and early diagnosis of atrial thrombus to enable timely therapeutic strategies, reducing patient morbidity and mortality. Finally, it underscores the essential role of a multidisciplinary approach in promoting safe and effective treatment, contributing to better clinical outcomes.

Table 1. Doses and medication adjustments for thrombosis management

Medication	Patient age	Dosage	Dose adjustment
t-PA (tissue plasminogen activator)	≤ 3 months	0.1 mg/kg/h initially	Fibrinogen > 100 mg/dL
	> 3 months	0.5 mg/kg/h initially	Fibrinogen > 100 mg/dL
Unfractionated heparin	≤ 1 months	100 IU/kg IV bolus	aPTT 60–85 s
	>1 months	75 IU/kg IV bolus	aPTT 60–85 s
	≤ 12 months	28 IU/kg/h	aPTT 60–85 s
	>12 months	20 IU/kg/h	aPTT 60–85 s
Low-molecular-weight heparin (LMWH)	≤ 2 months	1.5 mg/kg every 12 h (treatment)	Anti-Xa 0.35–0.75
		0.75 mg/kg every 12 h (prophylaxis)	Not required
	>2 months	1.0 mg/kg every 12 h (treatment)	Anti-Xa 0.35–0.75
		0.5 mg/kg every 12 h (prophylaxis)	Not required

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