Cite as: Napiórkowska W, Pluta NA, Mikołajczyk K, Żak M, Tkaczyk K, Będzichowska A: Scrotal swelling: a urological emergency or a symptom of systemic disease? A case report of nephrotic syndrome. Pediatr Med Rodz 2026; 22 (1): 58–61

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Received: 11.12.2025
Accepted: 11.02.2026
Published: 16.04.2026

Scrotal swelling: a urological emergency or a symptom of systemic disease? A case report of nephrotic syndrome

Obrzęk moszny: nagły stan urologiczny czy objaw choroby systemowej? Opis przypadku zespołu nerczycowego

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https://doi.org/10.15557/PiMR.2026.0009

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Abstract

This case report describes a diagnostically challenging presentation of nephrotic syndrome in a two-year-old boy initially suspected to have testicular torsion due to prominent scrotal oedema. The case demonstrates how the characteristic features of nephrotic syndrome – generalised oedema, proteinuria, and hypoalbuminaemia – may be initially obscured by focal symptoms mimicking surgical emergencies. The patient underwent scrotal exploration for suspected appendix testis torsion based on ultrasonography findings; however, postoperative persistence of oedema and development of ascites prompted recognition of the underlying nephrotic syndrome. Prompt initiation of corticosteroid therapy led to rapid resolution of proteinuria and clinical improvement. This report contributes to the existing literature by illustrating how nephrotic syndrome may present atypically, potentially delaying diagnosis when focal symptoms dominate the clinical picture.

Keywords: children, nephrotic syndrome, scrotal oedema

Streszczenie

W pracy przedstawiono opis przypadku dwuletniego chłopca z zespołem nerczycowym o nietypowej prezentacji klinicznej. Z powodu nasilonego obrzęku moszny początkowo podejrzewano skręt jądra. Typowe objawy zespołu nerczycowego – uogólniony obrzęk, białkomocz i hipoalbuminemia – zostały przesłonięte przez lokalne zmiany sugerujące ostry stan chirurgiczny. Na podstawie badania ultrasonograficznego rozpoznano skręt przydatku jądra i wykonano zabieg chirurgiczny. Po operacji obrzęk nie ustępował, a dodatkowo pojawiło się wodobrzusze. Te obserwacje doprowadziły do rozpoznania zespołu nerczycowego. Leczenie kortykosteroidami było skuteczne i doprowadziło do szybkiego ustąpienia białkomoczu i poprawy stanu klinicznego. Opis przypadku wzbogaca wiedzę na temat nietypowych postaci zespołu nerczycowego i pokazuje, jak dominujące objawy miejscowe mogą opóźnić prawidłowe rozpoznanie choroby ogólnoustrojowej.

Słowa kluczowe: dzieci, zespół nerczycowy, obrzęk moszny

INTRODUCTION

Nephrotic syndrome (NS) is one of the most common glomerular disorders in paediatric nephrology. It is characterised by urinary protein loss (determined in the first-morning urine or 24-hour urine collection): urine protein-to-creatinine ratio ≥2 mg protein/1 mg creatinine, or dipstick protein test result >3+; alternatively, proteinuria >50 mg/kg/day in 24-hour urine collection; and serum albumin reduced to <3.0 g/dL or oedema when serum albumin cannot be determined. These abnormalities may be accompanied by hyperlipidaemia[1]. In children aged 2–6 years, minimal change disease accounts for approximately 85–90% of cases and is associated with an excellent prognosis following corticosteroid therapy[2].

The typical presentation of NS in young children includes periorbital oedema that is most prominent in the morning, progressing to generalised oedema involving the lower extremities, ascites, and occasionally pleural effusions. However, the initial manifestation may vary significantly, and in rare instances, localised oedema may be the presenting feature, potentially obscuring the systemic nature of the disease. Acute scrotal swelling in paediatric patients represents a diagnostic challenge requiring prompt differentiation between surgical emergencies and medical conditions. Testicular torsion and torsion of the testicular appendages constitute true urological emergencies requiring immediate intervention to prevent testicular loss. Torsion of the appendix testis, a small embryological remnant (Müllerian or Wolffian duct), is particularly common in prepubescent boys and presents with acute scrotal pain and swelling. Doppler ultrasonography plays a crucial role in diagnosis, typically showing preserved or increased blood flow to the affected testis with a characteristic hyperechoic lesion[3,4]. Although scrotal oedema in NS has been described in the literature, it rarely presents as the dominant clinical feature leading to surgical intervention. This case illustrates the diagnostic complexity that may arise when systemic and localised pathologies coexist, emphasising the need for vigilant clinical assessment.

CASE PRESENTATION

A previously healthy two-year-old boy presented to the emergency department with progressive scrotal swelling of one day's duration. History revealed periorbital oedema for one week, initially attributed to a recent upper respiratory infection accompanied by fever. The past medical and family history were insignificant.

The primary care physician had documented severe proteinuria (>2,000 mg/dL) on urinalysis one day prior to presentation; however, this finding was not immediately correlated with the scrotal symptoms.

Physical examination revealed mild tachycardia (heart rate 135/min), while other vital signs were within normal limits, with a blood pressure of 92/53 mm Hg (<95th percentile for age, gender, and height). Marked bilateral periorbital oedema was present and was reported to be more pronounced in the mornings. Scrotal examination demonstrated significant right-sided swelling with overlying skin oedema. Mild lower-extremity oedema was also noted but was not initially emphasised in the clinical assessment.

Scrotal ultrasonography with Doppler imaging demonstrated peritesticular fluid accumulation and increased right testicular blood flow, interpreted as torsion of the appendix testis. However, upon surgical exploration, the appendix testis appeared intact, without features of torsion or tissue compromise. The testis was viable, with adequate perfusion. On the first postoperative day, the patient developed worsening generalised oedema involving the extremities, with persistent scrotal swelling. Ascites became evident, with abdominal distension and a demonstrable fluid wave. Body weight increased by approximately 3 kg compared with the day of admission. Daily diuresis was 500 mL. A 24-hour urine collection revealed proteinuria of 152 mg/kg/day, confirming nephrotic-range proteinuria.

The patient was transferred to paediatric nephrology department. Laboratory evaluation demonstrated severe hypoalbuminaemia (1.5 g/dL), hypercholesterolaemia (363 mg/dL), hypertriglyceridaemia (282 mg/dL), and mild metabolic acidosis (pH 7.324). Treatment was initiated with oral methylprednisolone 2 mg/kg/day (32 mg) and was later transitioned to prednisone 40 mg daily. Management included intravenous 20% albumin followed by furosemide, with the addition of hydrochlorothiazide

Parameter	Day 1	Day 2	Day 7	Day 10	Reference range
Urine studies					
Protein [mg/dL]	1,207	–	370	Trace	Negative
Specific gravity	1.026	–	1.015	1.018	1.015–1.025
Creatinine [mg/dL]	–	–	46.13	–	–
Serum chemistry					
Creatinine [mg/dL]	–	0.5	–	0.2	<0.4
Albumin [g/dL]	–	1.5	–	3.9	3.8–4.7
Total protein [g/dL]	–	3.6	–	6.2	5.9–7.2
Total cholesterol [mg/dL]	–	363	–	188	118–226
LDL cholesterol [mg/dL]	–	253	–	120	63–129
HDL cholesterol [mg/dL]	–	63	–	64	31–58
Triglycerides [mg/dL]	–	282	–	199	45–211
Potassium [mmol/L]	–	5.2	–	4.6	3.5–5.1
Other					
pH	–	7.324	–	7.36	7.35–7.45
CRP [mg/dL]	–	1.29	–	0.5	<1.00

Tab. 1. Sequential laboratory findings demonstrating nephrotic-range proteinuria, severe hypoalbuminaemia, marked hyperlipidaemia, and treatment response to corticosteroids

 DOI: 10.15557/PiMR.2026.0009

25 mg for enhanced diuresis. Prophylactic enoxaparin 9 mg was administered because of the hypercoagulable state. Omeprazole 10 mg was given for gastroprotection, and perioperative cefuroxime therapy was completed.

By day 7 of corticosteroid therapy, urinary protein decreased to 370 mg/dL, with only trace proteinuria by day 10. Weight decreased by 3 kg, with oedema resolution. The patient was discharged on tapering prednisone with a diagnosis of first-episode steroid-sensitive nephrotic syndrome, presumed minimal change disease. Outpatient follow-up included monitoring for relapse using home urine dipstick testing (Tab. 1).

DISCUSSION

This case illustrates the diagnostic complexity that may arise when acute scrotal swelling occurs in the context of an underlying systemic disease. The failure to initially correlate documented severe proteinuria with subsequent scrotal presentation represents a critical missed diagnostic opportunity. In a child with known marked proteinuria and periorbital oedema, NS should have been considered despite the presence of acute scrotal symptoms.

Scrotal oedema in paediatric patients can be a manifestation of both local conditions, often surgical emergencies, and systemic diseases. Although the distinction between these entities seems clear, certain signs and symptoms may be absent or omitted, complicating the clinical picture. The most prevalent causes of localised scrotal oedema in the paediatric population include testicular torsion, torsion of the testicular appendage, incarcerated inguinal hernia, and infectious processes[3,4]. In contrast, systemic causes of scrotal swelling include conditions such as NS, Schönlein–Henoch purpura, and Kawasaki disease[5,6] (Tab. 2).

Focusing on a complete physical examination may lead to an earlier suspicion of NS. In this case, the two-year-old patient presented with periorbital oedema and significant weight gain, both of which are critical signs that should prompt further investigation into systemic causes. Additionally, careful interpretation of laboratory findings, such as proteinuria and hypoalbuminaemia, is essential for improving the diagnostic process. Marked proteinuria and low serum albumin levels are hallmark indicators of NS, and recognising these abnormalities early can facilitate timely intervention[1,2].

Surgical causes	Systemic causes
Testicular torsion	Nephrotic syndrome
Appendix testis torsion	IgA vasculitis
Epididymitis/orchitis	Cardiac failure
Hydrocele	Hepatic disease
Incarcerated inguinal hernia	Renal failure
Trauma/haematoma	Kawasaki disease
Idiopathic scrotal oedema	Lymphoedema
Neoplasm	Systemic fluid overload

Tab. 2. Differential diagnosis of paediatric scrotal oedema

On the other hand, testicular torsion remains the critical diagnosis requiring exclusion, as intervention beyond 6–8 hours significantly increases risk of testicular loss[3,4]. Doppler ultrasonography demonstrates a sensitivity exceeding 90% for detecting absent or decreased testicular perfusion[7], although interpretation becomes challenging in cases of appendiceal torsion, where testicular perfusion is preserved or increased[8,9]. Increased Doppler blood flow could represent hyperaemic response to vascular changes associated with severe oedema.

Interdisciplinary collaboration among paediatricians, urologists, and nephrologists is essential to ensure timely and accurate diagnosis, which is critical for optimising patient outcomes and preventing complications. Early identification and intervention can significantly improve the prognosis for children with NS or other underlying systemic conditions, as demonstrated by the rapid resolution of symptoms following appropriate treatment in this case.

CONCLUSIONS

This case highlights several important lessons:

1. Data integration: Laboratory results must be actively integrated into clinical decision-making. The presence of known severe proteinuria should have prompted immediate consideration of NS.
2. Complete examination: Thorough assessment for signs of systemic disease (generalised oedema, weight gain) should be performed even when focal symptoms dominate.
3. Postoperative vigilance: Worsening oedema in the postoperative period, rather than improvement, should prompt immediate diagnostic reassessment.
4. Interdisciplinary communication: Early nephrology consultation in the presence of severe proteinuria might have prevented surgical intervention or ensured concurrent medical management.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.

Author contribution

Original concept of study: WN, NAP. Collection, recording and/or compilation of data: WN. Writing of manuscript: WN, NAP, KM, MŻ, MT, AB. Critical review of manuscript: MŻ, MT, AB. Final approval of manuscript: AB.

DOI: 10.15557/PiMR.2026.0009 PEDIATR MED RODZ Vol. 22 No. 1, p. 58–61

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 DOI: 10.15557/PiMR.2026.0009