Journal of
Medical Case Reports

CASE REPORT

Open Access

Isolated superior mesenteric vein thrombosis in an adult with nephrotic syndrome due to minimal change disease: a case report

Abdulilah Ghazwan Dakak[1*], Mohammed Kamal Mahgoub[2], Tala Adnan Jalkhi[1], Mustafa Abdulrahman Mohammed[1], Abdulrhman Alkassar[1] and Maryam Amirrad[1]

Abstract

Background Nephrotic syndrome, a condition that induces a hypercoagulable state, poses a significant risk of thromboembolism, a potentially life-threatening complication. While venous thromboembolism in nephrotic syndrome typically manifests as pulmonary emboli, deep vein thrombosis, and renal vein thrombosis, mesenteric vein thrombosis is a rare occurrence. The rarity is further accentuated when it occurs in patients with minimal change disease, with only a handful of cases reported in the literature.

Case presentation The patient is a 25-year-old Syrian male, previously in good health, presenting with severe abdominal pain and vomiting that had persisted for 6 days. Investigations revealed hypoalbuminemia with nephrotic range proteinuria. Computed tomography scan of the abdomen and pelvis with contrast showed superior mesenteric vein thrombosis. Renal biopsy revealed minimal change disease. The patient was mainly treated with a therapeutic dose of enoxaparin, followed by apixaban. He also received oral prednisolone, a short course of cyclosporine, and furosemide. With the appropriate treatment, the patient's symptoms gradually improved and he was discharged. This successful management of a rare complication in a nephrotic syndrome patient demonstrates the potential for positive outcomes in such cases.

Conclusions This unique case underscores the importance of early recognition and appropriate management of thromboembolic events in nephrotic syndrome. By presenting this case, we aim to alert clinicians to the possibility of mesenteric venous thrombosis, a rare but potentially serious complication. Such reports are crucial in informing more effective clinical decision-making and improving outcomes for nephrotic syndrome patients.

Keywords Case report, Complications, Minimal change disease, Nephrotic syndrome, Venous thrombosis

Introduction

Nephrotic syndrome creates a hypercoagulable state that puts patients at risk of thromboembolism, one of the most serious and potentially fatal complications of the disease. The risk of thromboembolism in nephrotic syndrome mainly depends on the severity and the primary cause of the disease. The most significant risk of thromboembolism, specifically venous thromboembolism (VTE), is seen in membranous nephropathy [1, 2]. On the other hand, minimal change disease (MCD) showed one of the lowest incidence rates of VTE in nephrotic syndrome [3]. VTE in nephrotic syndrome mainly presents as pulmonary emboli, deep vein thrombosis, and renal vein thrombosis [1].

*Correspondence:
Abdulilah Ghazwan Dakak
201910662@ajmanuni.ac.ae
[1] College of Medicine, Ajman University, P.O. Box 346, Ajman, UAE
[2] Department of Medicine, Sheikh Khalifa Medical City, P.O. Box: 5166, Ajman, UAE

Nonetheless, mesenteric vein thrombosis (MVT) represents a rare complication in patients affected by this syndrome. The incidence of MVT in patients with MCD is even more uncommon, with rarely any cases reported in the literature. To our knowledge, only one case of isolated superior mesenteric vein thrombosis has previously been reported in a patient with minimal change disease [7]. We present a unique case of a previously healthy 25-year-old Syrian male presenting with isolated superior mesenteric vein thrombosis as the first presentation of nephrotic syndrome.

Case description

A 25-year-old Syrian male presented on 8 February 2024, with a chief complaint of severe, gradually worsening abdominal pain for 6 days. The pain was localized to the periumbilical region, characterized as stabbing pain, exacerbated by fatty meals, and graded as 9/10 on the severity scale. He reported four episodes of vomiting but denied fever, changes in weight, appetite, or bowel habits. He also noted occasional nocturia without urgency, frequency, or dysuria. On further questioning, he denied passing frothy urine. His review of systems was otherwise unremarkable. Past surgical history included nasal polypectomy and hemorrhoidectomy, while past medical history was unremarkable. He had no known allergies and was not taking any medications. Family history was significant for maternal ischemic heart disease managed with percutaneous coronary intervention (PCI), hypertension, and hyperthyroidism. He was a heavy smoker (seven pack years) and denied alcohol consumption.

The patient was alert and oriented and had an average build on admission. His heart rate was 84 beats per minute (bpm), blood pressure was 144/81 mmHg, respiratory rate was 18 breaths per minute, and oxygen saturation was 99% on room air. Physical examination revealed periorbital edema and bilateral lower limb edema. There was no jaundice, pallor, cyanosis, or palpable lymphadenopathy. Abdominal examination demonstrated mild tenderness in the periumbilical region, moderate ascites, and no guarding or rebound tenderness.

As presented in Table 1, laboratory investigations revealed a significantly low anion gap, accompanied by substantial proteinuria and hematuria on urine dipstick analysis. The 24 hour urine protein excretion was markedly elevated, indicating significant protein loss. The complete blood count (CBC) results were within normal limits. However, the erythrocyte sedimentation rate (ESR) was elevated, and liver function tests demonstrated low total protein, severely decreased albumin, and increased alkaline phosphatase levels. Renal function tests indicated a considerable elevation in creatinine, consistent with impaired kidney function. Coagulation studies showed a prolonged prothrombin time (PT), elevated international normalized ratio (INR), and increased D-dimer, suggesting a hypercoagulable state. Thrombophilia screening was mainly unremarkable, except for mildly reduced protein S levels, likely due to urinary loss. Serological analysis showed a mild reduction in complement component C3, while results for ANA, ANCA, dsDNA, and PLA2R antibodies were negative.

Table 1 Laboratory investigations

Test name	Test result	Ref. range
Hemoglobin	16.1 g/dL	M: 13.5–17.5 g/dL F: 12–16 g/dL
WBC	5.17×10^9/L	$4.5–11.0 \times 10^9$/L
Platelets	182×10^9/L	$150–400 \times 10^9$/L
ESR	32.0 mm/hour	M: 0–15 mm/hour F: 0–20 mm/hour
Total protein	40.0 g/L	66.0–87.0 g/L
Albumin	11.6 g/L	35.0–52.0 g/L
Alkaline phosphatase	155.0 U/L	40.0–129.0 U/L
Creatinine	4.88 mg/dL	0.7–1.3 mg/dL
PT	17.10 seconds	11.70–15.30 seconds
INR	1.29	0.8–1.1
D-Dimer	1.08 mg/L	0.0–0.5 mg/L
Anion gap	0.7 mmol/L	8–16 mmol/L
Protein urine dipstick	+ + + +	–
Blood urine dipstick	+	–
24 hour urine protein	> 15,000 mg	< 150 mg/day

An abdominal computed tomography (CT) scan with contrast enhancement was ordered, which revealed superior mesenteric vein thrombosis, leading to veno-occlusive mesenteric ischemia. This ischemia resulted in hypoperfusion of the jejunum, ileum, cecum, ascending colon, proximal transverse colon, diffuse mesenteric fat stranding, and moderate ascites (Fig. 1). Renal ultrasound showed bilateral increased cortical echogenicity, mild free fluid in the abdomen, and bilateral pleural effusion, indicative of nephrotic syndrome. There was no hydronephrosis.

A provisional diagnosis of nephrotic syndrome with secondary venous thrombosis was made, and inpatient management began with enoxaparin (70 mg, subcutaneous injection every 12 hours) and prednisolone (70 mg, oral, daily). An ultrasound-guided renal biopsy was performed to confirm the diagnosis. The patient was referred to a nephrologist, who initiated an empirical treatment regimen consisting of cyclosporine (100 mg, oral twice daily) and furosemide (40 mg, oral daily), on which he was discharged, along with apixaban (10 mg, oral, twice daily for 7 days, then reduced to 5 mg, twice daily for 3 months), prednisolone (70 mg, oral, tapered

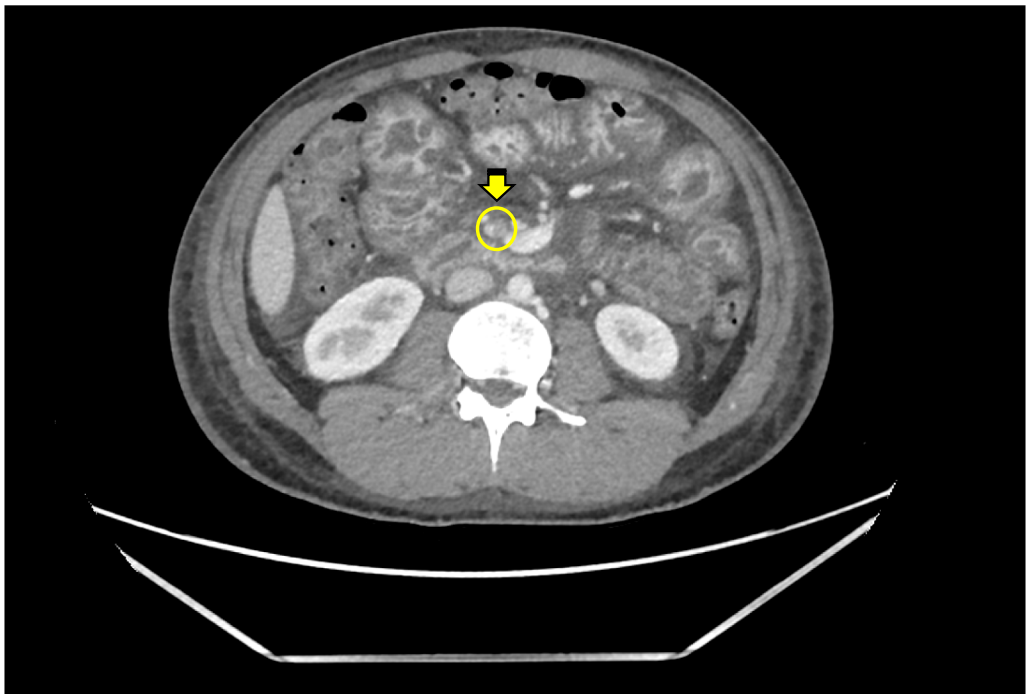

Fig. 1 Initial computed tomography (CT) study of the abdomen with contrast. A selected axial image showing superior mesenteric vein thrombosis (yellow arrow and circle)

over 6 weeks), atorvastatin (40 mg, oral, daily), alfacalcidol (1 mcg, oral, daily), calcium carbonate (500 mg, oral, daily), and pantoprazole (40 mg, oral, daily).

On 19 February 2024, the patient returned with worsening facial and periorbital edema, along with swelling in the lower limbs and newly developed scrotal edema. He reported poor compliance with the treatment regimen. These symptoms and the decline in his serum albumin levels were managed as a flare-up of his nephrotic syndrome, and he was started on steroid therapy. He was discharged on 20 February with perindopril (5 mg, oral, daily) being added to his treatment regimen and instructed to follow-up in 1 week to review renal biopsy results. On 24 February 2024, he revisited the emergency department due to a flare-up of his symptoms. However, he chose to leave against medical advice (LAMA) before he got further assessment and treatment.

The patient presented again on 29 February 2024, with severe right-sided abdominal pain and shortness of breath. He was not adherent to medications. Laboratory tests showed an elevated C-reactive protein (CRP) level of 110 mg/L (normal range: 0–5 mg/L) and a white blood cell count of 18×10^9/L (normal range: $4.0–11.0 \times 10^9$/L), suggesting inflammation. Chest X-ray confirmed a mild right-sided pleural effusion. Compared to the previous scan, a CT angiography of the abdomen revealed no evidence of recurrent superior mesenteric vein thrombosis; the portal vein, splenic vein, and superior mesenteric artery remained patent. However, there was submucosal edema, suboptimal mucosal enhancement in the cecum, and similar findings in the ascending and transverse colon. The jejunal and ileal loops demonstrated optimal enhancement. Mild free fluid in the abdomen and stranding in the mesenteric fat persisted. Additionally, a small perinephric collection was noted near the lower pole of the left kidney. There was no intraperitoneal air.

Initially, the patient had piperacillin/tazobactam based on clinical suspicion of infection, but cultures returned negative. On 1 March 2024, biopsy and electron microscopy findings showed subcapsular cortical tissue containing two glomeruli without crescent formation. The glomerular basement membranes were regularly thick and structurally normal, with no evidence of splicing or lamellation. Significant podocyte foot process effacement was noted, but no specific osmiophilic deposits or fibrils were observed. These findings were consistent with a diagnosis of primary podocytopathy of the minimal change type without indications of immune complex or complement-mediated glomerulonephritis. Following these results, the patient's condition gradually improved. He was discharged on his previous treatment regimen and chose to follow-up in his home country.

Discussion

Minimal change disease represents 70–90% of nephrotic syndrome cases in children over the age of 1 year establishing it as one of the leading causes of idiopathic nephrotic syndrome in this population [8, 9]. On the other hand, it is significantly less common in the older population, accounting for only 10–15% of primary nephrotic syndrome in adults [8, 9].

The pathophysiology of MCD needs to be better understood, with multiple theories in question. For several decades, MCD was thought to be a T-cell-mediated condition, releasing circulating factors that impair the glomerular filtration barrier. An alternative proposed mechanism for MCD involves elevated levels of certain cytokines. A "two-hit" theory has recently suggested that MCD may involve both CD80 (B7-1) induction and regulatory T-cell (Treg) dysfunction, potentially alongside impaired autoregulatory functions of the podocyte [10, 25]. Unlike T cells, the role of B cells in the pathogenesis of the disease has not been adequately studied. Despite that, B-cell biology has recently gained more attention after a monoclonal antibody targeting CD20 proteins on B cells demonstrated a promising therapeutic response in treating childhood and adult MCD [10, 26].

The mechanism behind the hypercoagulable state in nephrotic syndrome remains unclear. It could be related to an increased synthesis of prothrombotic factors, mainly fibrinogen and factor VIII, which can act as acute phase proteins in inflammation [3, 4, 24]. Another possible mechanism is the urinary loss of negatively charged anticoagulant proteins such as antithrombin and protein C and S, leading to reduced levels and impaired fibrinolytic activity [3, 4, 24]. In addition, the urinary loss of albumin and resultant hypoalbuminemia leads to increased hepatic synthesis of fibrinogen and other procoagulant factors [3, 4, 24]. However, the detailed pathophysiology of hypercoagulability in nephrotic syndrome is beyond the scope of our case.

Although nephrotic syndrome has long been recognized as a significant risk factor for thromboembolism, available data are based primarily on small studies and case reports, with no precise risk estimates. The primary cause of nephrotic syndrome notably influences the risk of thromboembolism, with the highest risk observed in membranous nephropathy (MN). One prospective study identified VTE in 36% of MN patients through screening with contrast-enhanced CT [3, 28]. A retrospective cohort study done on 298 consecutive nephrotic syndrome patients showed a significantly elevated risk (approximately 140-fold) for VTE within the first 6 months of diagnosis, with the ratio of proteinuria to serum albumin being the main predictor for VTE [12, 17]. This information is highly relevant to our patient, as he exhibited severe proteinuria with a 24 hours urine protein of more than 15 g/day and a critically low serum albumin level of 11.6 g/L, both of which put him at high risk for venous thromboembolism (VTE), consistent with the findings of the study mentioned above.

VTE complications in nephrotic syndrome mostly present as pulmonary emboli, deep vein thrombosis, and renal vein thrombosis [1]. Nonetheless, MVT represents a rare complication in nephrotic syndrome patients. Even more uncommon is the incidence of MVT in patients with MCD, with rarely any cases reported in the literature. Only one isolated superior mesenteric vein thrombosis case has previously been reported in a nephrotic syndrome patient with minimal change subtype [7]. However, multiple case studies have documented MVT and other thromboembolic events, as presented in Table 2.

MVT is a life-threatening condition that can be easily missed due to its nonspecific presentation. Patients may present with a wide range of symptoms, from being asymptomatic to exhibiting severe abdominal pain that may be out of proportion to physical findings. Additionally, common symptoms include anorexia, nausea, and vomiting [18]. In our patient, he rated his pain nine out of ten in severity, yet the abdominal examination revealed only mild tenderness. Although the exact pathophysiology of MVT in nephrotic syndrome is understudied, MVT commonly happens when inherited or acquired thrombophilia accompanies trauma, infection, inflammation, or surgery [18]. It may also arise from stasis caused by conditions such as heart failure or liver cirrhosis [18].

Treatment of MVT is mainly focused on preventing bowel infarction, limiting clot progression, and reducing recurrence risk. Depending on the patient's presentation and severity, management strategies can range from conservative measures such as bowel rest and anticoagulation to interventions such as thrombolysis and surgical bowel resection [18, 19]. In severe cases, interventional radiology provides a minimally invasive and precise treatment through different methods such as catheter-directed thrombolysis, thrombectomy, and focal administration of thrombolytic agents, eliminating the need for bowel resection in many critical patients [5, 18].

Anticoagulation is considered the mainstay therapy for MVT [18]. Unfractionated heparin (UFH) has long been the first-line treatment for VTE, including MVT. However, low-molecular-weight heparin (LMWH) has also been successfully used to treat reported cases of VTE in nephrotic syndrome patients due to its favorable pharmacokinetic profile, including more predictable anticoagulation effects, better bioavailability, and suitability for outpatient management, while minimizing

Table 2 A summary of case reports documenting MVT in patients with nephrotic syndrome

Ref.	Age	Sex	Onset	Clinical presentation	Thrombosis involvement	Creatinine	Proteinuria	Serum albumin	Renal biopsy
[6]	46	M	At diagnosis	Abdominal pain, abdominal fullness	Portal, splenic, and mesenteric veins	1.58 mg/dL	24-HUP: 24.15 g/day	22 g/L	MCD
[7]	25	M	At relapse	Abdominal distension, bloody diarrhea	Superior mesenteric vein	1.4 mg/dL	Unknown	15 g/L	MCD
[11]	31	M	At relapse	Abdominal pain, vomiting	Portal, splenic, and superior mesenteric veins	0.96 mg/dL	UPCR: 4353 mg/g	16 g/L	MCD
[12]	19	M	At relapse	Abdominal pain, bilateral peripheral edema, foamy urine	Superior mesenteric vein	1.03 mg/dL	24-HUP: 17.19 g/day	16 g/L	FSGS
[13]	19	M	At relapse	Abdominal distension, bilateral lower leg edema	Portal, superior mesenteric, and inferior mesenteric veins	0.75 mg/dL	24-HUP (at time of diagnosis): 6.68 mg/day	7 g/L	MPG
[14]	53	M	At diagnosis	Abdominal pain, bilateral lower leg edema, watery diarrhea	Portal and superior mesenteric veins	0.7 mg/dL	24-HUP: 5.29 g/day	21 g/L	MCD
[15]	12	M	At relapse	Abdominal pain and distension, facial puffiness, oliguria	Portal and superior mesenteric veins	0.93 mg/dL	24-HUP: 5.5 g/day	18 g/L	MPG
[15]	13	M	At relapse	Abdominal pain, vomiting, facial puffiness, oliguria	Portal and superior mesenteric veins	2.7 mg/dL	24-HUP: 3.5 g/day	17 g/L	FSGS
[16]	26	M	At relapse	Abdominal pain and distension, bilateral lower leg edema	Portal and superior mesenteric veins	1.2 mg/dL	SUP: 500 mg/dL	10 g/L	MCD
[29]	56	M	At relapse	Abdominal pain	Portal, splenic, and superior mesenteric veins	Unknown	24-HUP: 10.5/day	25 g/L	FSGS
[30]	41	M	At relapse	Abdominal pain, vomiting, oliguria	Portal, splenic, mesenteric veins	3.8 mg/dL	SUP: 250 mg/dL	17 g/L	MCD
[31]	52	M	At diagnosis	Abdominal pain and distension, oliguria	Portal and superior mesenteric veins	1.1 mg/dL	24-HUP: 3.467 g/day	17 g/L	Not done
[32]	18	M	At diagnosis	Abdominal distension, vomiting, diarrhea	Portal, splenic, and superior mesenteric veins	1.56 mg/dL	24-HUP: 15.28 g/day	14.6 g/L	MCD
Our case	25	M	At diagnosis	Abdominal pain, vomiting	Superior mesenteric vein	4.88 mg/dL	24-HUP: > 15 g/day	11.6 g/L	MCD

M male, *24-HUP* 24-hour urine protein, *SUP* spot urine protein, *UPCR* urine protein creatinine ratio, *MCD* minimal change disease, *FSGS* focal-segmental glomerulosclerosis, *MPG* mesangial proliferative glomerulonephritis

the risks associated with UFH such as heparin-induced thrombocytopenia and osteoporosis [14, 20, 21].

As previously mentioned, low serum albumin levels strongly predict VTE. Therefore, the prophylactic use of anticoagulants such as LMWH has been suggested by the KDIGO guidelines for patients with serum albumin levels below 20 g/L [27]. If low albumin levels persist beyond 3 months, warfarin with close monitoring of INR is considered [3, 22, 27]. In addition to LMWH, direct oral anticoagulants (DOACs) such as apixaban and rivaroxaban are emerging as possible alternatives for treating and preventing VTE in nephrotic syndrome [3]. Case reports, mainly focused on patients with hypoalbuminemia, have shown that DOACs can be very effective [3, 23]. Nonetheless, more extensive studies are

needed to establish their safety and efficacy in the long term.

Owing to a lack of solid evidence regarding the use of anticoagulants as a preventive measure against thromboembolic events in nephrotic syndrome patients, the decision to use them should be on a case-by-case basis. We should consider the risk factors and severity of the disease for each patient. We should also weigh the risk of bleeding against the risk of thrombosis before planning preventive anticoagulation to ensure the best patient outcomes [12, 24]. In our case, the use of LMWH proved successful in the acute treatment of our patient, as he showed significant improvement. We later switched him to apixaban as long-term anticoagulation. This approach allowed for more convenient outpatient care while preventing future thromboembolic events.

The long-term prognosis of VTE cases in patients with nephrotic syndrome depends on multiple factors, including early diagnosis, adequate anticoagulation, and progress of the underlying nephrotic syndrome [3]. Regular follow-up is crucial to monitor the patient's response to treatment, prevent recurrences, and make any necessary management adjustments.

Conclusions

The hypercoagulable state in nephrotic syndrome is a well-established risk factor for thromboembolic events, with VTE representing a significant threat to patients. The variability in reported thromboembolism rates among nephrotic syndrome patients, along with the limited data, highlights a gap in the literature that hinders the development of preventive strategies for such serious complications. In this context, our case of isolated MVT in a patient with minimal change nephrotic syndrome demonstrates that MVT, although rare, is a possible complication in these patients. Furthermore, this report highlights the importance of early recognition and appropriate management of thromboembolic events in nephrotic syndrome. By presenting this case, we aim to provide a reference for clinicians when considering MVT as a differential diagnosis in similar clinical settings, ultimately guiding effective diagnostic and management strategies.

Further studies are essential to understand the mechanisms underlying thromboembolic complications in MCD and to identify whether specific subgroups of MCD patients are at increased risk for thromboembolic complications. While case reports provide valuable insights into effective clinical decision-making and rare presentations, more extensive prospective studies are essential to guide more comprehensive, preventive, and therapeutic measures for nephrotic syndrome patients.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13256-025-05130-4.

Additional file 1

Acknowledgements
No specific individuals or organizations are acknowledged for this case report.

Author contributions
Abdulilah Ghazwan Dakak conducted the literature review, prepared the manuscript, including all figures and tables, contributed to the writing and revision of the manuscript, and approved the final version for submission. Dr. Mohammed Kamal obtained the patient's consent, secured ethical approval, and reviewed the final version of the manuscript for submission. Tala Jalkhi participated in the literature review, contributed to the writing and revision of the manuscript, and proofread the case report. Mustafa Abdulrahman Mohammed collected patient data and contributed to drafting the initial version of the manuscript. Abdulrahman AlKassar collected patient data and assisted in drafting the initial version of the manuscript. Dr. Maryam Amirrad supervised the manuscript preparation, revised the manuscript, proofread the final draft for grammatical accuracy and plagiarism, approved the final version for submission, and ensured compliance with the journal's guidelines and requirements.

Funding
This study did not receive any funding.

Availability of data and materials
All supporting data related to this case report are included within the manuscript.

Declarations

Ethical approval and consent to participate
The research committee at Sheikh Khalifa Medical City Ajman (SKMCA) granted ethical approval for this case report verbally.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The authors declare that they have no competing interests.

Received: 16 January 2025 Accepted: 7 February 2025
Published online: 01 April 2025

References

1. Zhang L, Zhang Z, Li S, Meinel FG, Nance JW, Zhou CS, *et al*. Pulmonary embolism and renal vein thrombosis in patients with nephrotic syndrome: prospective evaluation of prevalence and risk factors with CT. Radiology. 2014;273(3):897–906.
2. Medjeral-Thomas N, Ziaj S, Condon M, Galliford J, Levy J, Cairns T, *et al*. Retrospective analysis of a novel regimen for the prevention of venous thromboembolism in nephrotic syndrome. Clin J Am Soc Nephrol. 2014;9(3):478–83.
3. Parker K, Ragy O, Hamilton P, Jecko T, Durga K. Thromboembolism in nephrotic syndrome: controversies and uncertainties. Res Pract Thromb Haemost. 2023;7(6):102162–72.

4. Al-Azzawi H, Obi O, Safi J, Song M. Nephrotic syndrome-induced thromboembolism in adults. Int J Crit Illn Inj Sci. 2016;6(2):85.
5. Singal AK, Kamath PS, Tefferi A. Mesenteric venous thrombosis. Mayo Clin Proc. 2013;88(3):285–94. https://doi.org/10.1016/j.mayocp.2013.01.012.
6. Chang KC, Weng YM, Chen SY, Chang YC. Nephrotic syndrome complicated with acute mesenteric ischemia. Am J Emerg Med. 2011;29(2):243.e1-3.
7. Lin JL. Massive hemorrhagic necrosis of small intestine due to mesenteric vein thrombosis: an unusual complication of nephrotic syndrome. Nephron. 1992;61(2):230–1.
8. Waldman M, Crew RJ, Valeri A, Busch J, Stokes B, Markowitz G, *et al*. Adult minimal-change disease: clinical characteristics, treatment, and outcomes. Clin J Am Soc Nephrol. 2007;2(3):445–53.
9. Zamora G, Pearson-Shaver AL. Minimal Change Disease. PubMed. Treasure Island (FL): StatPearls Publishing; 2021. https://www.ncbi.nlm.nih.gov/books/NBK560639/. Accessed 5 Oct 2024.
10. Kim SH, Park SJ, Han KH, Kronbichler A, Saleem MA, Oh J, *et al*. Pathogenesis of minimal change nephrotic syndrome: an immunological concept. Korean J Pediatr. 2016;59(5):205–11.
11. Park BS, Park S, Jin K, Choi G, Park KM, Jo KM, Kim YW. Nephrotic syndrome complicated with portal, splenic, and superior mesenteric vein thrombosis. Kidney Res Clin Pract. 2014;33(3):161–4. https://doi.org/10.1016/j.krcp.2014.07.001.
12. Somaili M, Wang C, Almansour O, Alrefaei B. Superior mesenteric vein thrombosis in a patient with primary focal segmental glomerulosclerosis: a case report. Med Case Rep Study Protoc. 2021;2(12): e0199. https://doi.org/10.1097/md9.0000000000000199.
13. Luxmi SV, Yue S, Qing CX, Fang CF, Fedally S, *et al*. An unusual complication of mesenteric-portal axis thrombosis and septic shock in a patient with mesangial proliferative glomerulonephritis. Clin Med Rev Case Rep. 2021;8:375. https://doi.org/10.23937/2378-3656/1410375.
14. Wang YC, Chuang FR, Lee WC, Chen TC, Ko SF, Wang IK, Lee CH. Low-molecular-weight heparin successfully used to treat a nephrotic patient complicated by superior mesenteric vein thrombosis and portal vein thrombosis. Med Princ Pract. 2011;20(2):196–9. https://doi.org/10.1159/000319925.
15. Gera DN, Patel J, Patel K, Kute VB. Portal vein thrombosis: a rare complication of nephrotic syndrome. Ind J Nephrol. 2018;28(3):236–9. https://doi.org/10.4103/ijn.IJN_25_17.
16. Hsu CS, Chou YP, Fong TV, Yong CC, Lee CT, Chen JB. Portal vein and superior mesenteric vein thrombosis in nephrotic syndrome: a case report and review of literature. Acta Nephrol. 2007;21(3):224–8.
17. Mahmoodi BK, ten Kate MK, Waanders F, Veeger NJGM, Brouwer JLP, Vogt L, *et al*. High absolute risks and predictors of venous and arterial thromboembolic events in patients with nephrotic syndrome. Circulation. 2008;117(2):224–30.
18. Sulger, Elisabeth, et al. "Mesenteric Venous Thrombosis." *PubMed*, StatPearls Publishing, 2022, www.ncbi.nlm.nih.gov/books/NBK459184/. Accessed 8 Oct 2024.
19. Russell CE, Wadhera RK, Piazza G. Mesenteric venous thrombosis. Circulation. 2015;131(18):1599–603. https://doi.org/10.1161/CIRCULATIONAHA.114.012871.
20. Sun L, Xu C. Portal vein thrombosis as the first sign of nephrotic syndrome. Nat Rev Nephrol. 2008;4:342–5. https://doi.org/10.1038/ncpneph0810.
21. Weitz JI. Low-molecular-weight heparins. N Engl J Med. 1997;337(10):688–98. https://doi.org/10.1056/NEJM199709043371007.
22. Carvalho AC, Colman RW, Lees RS. Platelet function in hyperlipoproteinemia. N Engl J Med. 1974;290(8):434–8. https://doi.org/10.1056/NEJM197402212900805.
23. Kelddal S, Hvas AM, Grove EL, Birn H. Safety and effectiveness of direct oral anticoagulants in patients with nephrotic syndrome: a report of 21 cases. BMC Nephrol. 2022;23(1):305. https://doi.org/10.1186/s12882-022-02929-x.
24. Mirrakhimov AE, Ali AM, Barbaryan A, Prueksaritanond S, Hussain N. Primary nephrotic syndrome in adults as a risk factor for pulmonary embolism: an up-to-date review of the literature. Int J Nephrol. 2014;2014:1–9.
25. Shimada M, Araya C, Rivard C, Ishimoto T, Johnson RJ, Garin EH. Minimal change disease: a "two-hit" podocyte immune disorder? Pediatr Nephrol. 2011;26:645–9. https://doi.org/10.1007/s00467-010-1676-x.
26. Iijima K, Sako M, Nozu K, Mori R, Tuchida N, Kamei K, Miura K, Aya K, Nakanishi K, Ohtomo Y, Takahashi S, Tanaka R, Kaito H, Nakamura H, Ishikura K, Ito S, Ohashi Y, Rituximab for Childhood-onset Refractory Nephrotic Syndrome (RCRNS) Study Group. Rituximab for childhood-onset, complicated, frequently relapsing nephrotic syndrome or steroid-dependent nephrotic syndrome: a multicentre, double-blind, randomised, placebo-controlled trial. Lancet. 2014;384(9950):1273–81. https://doi.org/10.1016/S0140-6736(14)60541-9.
27. Rovin BH, Adler SG, Barratt J, Bridoux F, Burdge KA, Chan TM, Cook HT, Fervenza FC, Gibson KL, Glassock RJ, Jayne DR, Kidney Disease: Improving Global Outcomes (KDIGO) Glomerular Diseases Work Group. KDIGO 2021 clinical practice guideline for the management of glomerular diseases. Kidney Int. 2021;100(4S):S1–276. https://doi.org/10.1016/j.kint.2021.05.021.
28. Li SJ, Guo JZ, Zuo K, Zhang J, Wu Y, Zhou CS, Lu GM, Liu ZH. Thromboembolic complications in membranous nephropathy patients with nephrotic syndrome—a prospective study. Thromb Res. 2012;130(3):501–5. https://doi.org/10.1016/j.thromres.2012.04.015.
29. Dumortier J, Sicard A, Guillaud O, Valette PJ, Scoazec JY, Boillot O. Portal vein thrombosis and nephrotic syndrome after liver transplant. Exp Clin Transpl. 2019;17(3):418–20. https://doi.org/10.6002/ect.2016.0259.
30. Luca MD, Dugo M, Arduini R, Liessi G. Acute venous thrombosis of splenomesenteric portal axis: an unusual localization of thromboembolism in the nephrotic syndrome. Am J Nephrol. 1991;11(3):260–3. https://doi.org/10.1159/000168315.
31. Sun L, Xu C. Portal vein thrombosis as the first sign of nephrotic syndrome. Nat Clin Pract Nephrol. 2008;4(6):342–5. https://doi.org/10.1038/ncpneph0810.
32. Wang J, Fan Q, Chen Y, Dong X, Zhang Y, Feng J, Ma J, Wang L. A case report of minimal change nephrotic syndrome complicated with portal, splenic and superior mesenteric vein thrombosis. Clin Nephrol. 2012;77(6):505–9. https://doi.org/10.5414/cn107372.

Publisher's Note