CASE REPORT

Severe Hypoalbuminaemia Associated with Extensive Deep Vein Thrombosis in an Eight-Year-Old Child in Maiduguri, Nigeria: A Case Report

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ABSTRACT

Background: Venous thromboembolism (VTE) is uncommon in the general paediatric population but is associated with significant morbidity. Although central venous catheters, malignancy, and trauma are well-established risk factors, hypoalbuminaemia is increasingly recognised as a potential prothrombotic condition that is often overlooked. **Case Summary:** We report the case of an 8-year-old girl who presented with a three-month history of recurrent fever, progressive generalised oedema, and recent weight loss. Her clinical condition initially manifested as anasarca and later progressed to painful localised swelling of the left lower limb. Doppler ultrasonography demonstrated an extensive thrombus extending from the internal iliac vein to the posterior tibial veins. A comprehensive diagnostic evaluation excluded common causes of paediatric thrombosis including nephrotic syndrome, malignancy, inherited thrombophilia, and infection. The only significant abnormality was profound hypoalbuminaemia (2.2 g/dL: reference: 3.5–5.0 g/dL). The patient was treated with low-molecular-weight heparin (LMWH) bridged to warfarin, in addition to nutritional rehabilitation. She showed progressive clinical improvement with complete clinical resolution by discharge. **Conclusion:** This case highlights the importance of considering hypoalbuminaemia as a potential provoking factor for paediatric VTE when common causes have been excluded. It underscores the possible pathophysiological link between low albumin levels and hypercoagulability.

Key words: Deep vein thrombosis, hypoalbuminaemia, venous thromboembolism, anticoagulation.

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Introduction

Venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT) and pulmonary embolism (PE), is a important cause of morbidity in hospitalised children, although it remains rare in the general paediatric population.[1] The reported incidence ranges from 0.14 to 0.9 per 100,000 children in the community, rising markedly to 8.6–57 per 100,000 among hospitalised paediatric patients. A consistent bimodal age distribution is observed, with peaks in infancy and in adolescence.[2] Importantly, over 90% of paediatric VTE cases occur in children with identifiable underlying risk factors. The most common provoking factors include central venous access devices (CVADs), malignancy, infection, trauma, and congenital heart disease. When these are excluded, clinicians must consider inherited thrombophilias or acquired hypercoagulable states.[2] Hypoalbuminaemia has been increasingly recognised as an independent risk factor for thrombosis, particularly in adults with nephrotic syndrome, cancer, and critical illness. In

children, this association is less frequently appreciated but is supported by a growing body of evidence. The prothrombotic state induced by low albumin is multifactorial, involving altered hepatic synthesis of coagulation factors, impaired fibrinolysis, and increased platelet aggregability.[3-6]

We present a case of extensive lower limb DVT in a child whom hypoalbuminaemia was the only identifiable risk factor following exhaustive investigation. This case offers an opportunity to review the role of albumin in haemostasis and to discuss current evidence guiding anticoagulation duration in paediatric VTE.

Case Presentation

An 8-year-old girl presented to our tertiary hospital with a three-month history of recurrent, low-grade fever. Each episode lasted 3–4 days and occurred at approximately weekly intervals. One month prior to presentation, she developed facial puffiness with diurnal variation, which progressed over five days to involve the entire body (anasarca). This generalised oedema later regressed and became localised to the left lower limb, which became progressively painful, swollen, and difficult to use. Her caregivers also noted darkening of the skin over the affected limb with areas of skin cracking and serous discharge. She had unintentional weight loss in the week preceding presentation.

There was no history of prolonged immobilisation, trauma, surgery, or oral contraceptive use. She was not fully immunised and had received various over-the-counter medications and herbal remedies without improvement. She initially sought care at a non-governmental organisation facility, where she received parenteral antibiotics (ceftriaxone and cloxacillin) and ibuprofen before being referred.

Her past medical history was unremarkable. She was the fourth of five children in a monogamous family, with no known family history of thrombotic disorders.

On examination, she appeared chronically ill with evidence of muscle wasting and facial fullness. She was pale, afebrile, and haemodynamically stable, with generalised lymphadenopathy. The left lower limb was markedly swollen from thigh to foot, with shiny, hyperpigmented skin, pitting oedema, and tenderness. The dorsalis pedis pulse was not palpable. The right foot had non-tender pitting oedema. Comparative limb measurements are shown in Table 1. Abdominal examination revealed shifting dullness consistent with ascites. Respiratory and cardiovascular examinations were unremarkable.

Table 1: Limb Circumference Measurements at Admission and Discharge

Measurement Point	Left Limb (Admission)	Right Limb (Admission)	Left Limb (Discharge) [change]	Right Limb (Discharge) [change]
Mid-thigh	33 cm	20 cm	26 cm [7]	25 cm[5]
Knee	29 cm	23 cm	23 cm [6]	23 cm[0]
10 cm below knee	23 cm	15.5 cm	15.5 cm [8.5]	15 cm[0.5]
Ankle	21 cm	14 cm	18 cm [3]	17 cm[3]

Key laboratory and imaging findings are summarised in Table 2. The most notable abnormality was a markedly reduced serum albumin of 2.2 g/dL (reference: 3.5–5.0 g/dL). Urinalysis was negative for proteinuria, effectively excluding nephrotic syndrome. Coagulation studies were normal (PT =13 seconds, APTT =33 seconds, INR 1.2). Infectious and malignant causes were considered and excluded with negative HIV serology, a negative Mantoux test, and unremarkable abdominal ultrasonography and echocardiography.

Table 2: Summary of Key Investigations

Investigation	Result	Reference Range / Interpretation
Haematology		
PCV	27%	36–46%
WBC	5.9 x 10^9/L	4.0–11.0 x 10^9/L
Platelets	150 x 10^9/L	150–450 x 10^9/L
Blood film	Dimorphic RBCs, predominantly microcytic hypochromic	Consistent with anaemia
Coagulation		
PT	13 seconds	11–13.5 seconds
APTT	33 seconds	25–35 seconds
INR	1.2	0.8–1.2
Biochemistry		
Serum Albumin	2.2 g/dL*	3.5–5.0 g/dL
Total Cholesterol	3.2 mmol/L	<5.2 mmol/L
Urinalysis		
Protein	Negative	Negative
Infection Screening		
HIV Antibody	Negative	Negative
Mantoux Test	0 mm	Negative
Imaging		
Doppler USS (Left Leg)	Echogenic thrombus, internal iliac to posterior tibial veins; reduced compressibility; absent colour flow	Extensive DVT
Abdominal USS	Unremarkable	No mass/nephropathy
Echocardiography	Normal	No source of embolism

***Profound hypoalbuminaemia.**

Doppler ultrasound of the left lower extremity revealed an extensive, echogenic thrombus extending from the left internal iliac vein distally through the femoral, popliteal, and posterior tibial veins. There was reduced compressibility, evidence of venous wall changes, and markedly reduced colour Doppler flow, consistent with extensive, likely chronic DVT.

The differential diagnoses were systematically considered. Nephrotic syndrome was excluded based on the absence of proteinuria. Inherited thrombophilia testing (including Factor V Leiden, protein C/S deficiency, antithrombin III, antiphospholipid antibodies) was deferred due to the acute thrombotic state and limited local availability, as results may be unreliable during active thrombosis. Malignancy was considered unlikely given unremarkable imaging and the absence of additional symptoms. No infectious focus was identified, despite empirical antibiotic therapy. Hypoalbuminaemia-associated hypercoagulability was therefore considered the most plausible explanation.

The patient was admitted and co-managed by the haematology and nutrition teams. Subcutaneous low-molecular weight heparin (LMWH Clexane®) was commenced on day 8 of admission and continued for one week. Oral warfarin was started at 2.5 mg daily, increased to 5 mg daily after five days. Low-molecular weight heparin was discontinued once the therapeutic INR (target 2.0–3.0) was achieved. The progression of INR response to Warfarin is shown in Table 3.

Table 3: INR Progression During Anticoagulation

Time Point	INR Value
Admission (baseline)	1.2
5 days after starting warfarin	1.3
10 days after starting warfarin	1.7
Target therapeutic range	2.0–3.0

Clinical improvement was noted within five days of initiating anticoagulation, with reduction in limb swelling. She also received empirical parenteral ceftriaxone and clindamycin for two weeks, followed by oral antibiotics for additional two weeks, although no definitive infectious source was identified. Nutritional rehabilitation with high-protein diet was commenced.

After an initial period of weight loss, she showed steady weight gain and clinical improvement. At discharge after 29 days, repeat limb measurements demonstrated near-complete resolution (Table 1). She was discharged on oral warfarin, with plans for follow-up Doppler ultrasonography and INR monitoring. Given the persistence of the underlying risk factor (hypoalbuminaemia of uncertain duration), a minimum of six months anticoagulation was planned, in line with recommendations for provoked VTE with ongoing risk factors.[7]

Discussion

This case highlights a diagnostically challenging presentation of extensive DVT in a child. The initial clinical picture—characterised by recurrent fever, anasarca, and weight loss—suggested an underlying systemic inflammatory, infectious, or malignant process. The later progression to unilateral limb swelling helped clarify the diagnosis of DVT, but the underlying aetiology remained uncertain. A striking feature in this case was the absence of the usual risk factors for VTE. The patient had no central venous access, no history of trauma or surgery, no evidence of malignancy, and no proteinuria to suggest nephrotic syndrome. This prompted consideration of hypoalbuminaemia as the most likely provoking factor. Hypoalbuminaemia is increasingly recognised as an independent predictor of VTE. A large meta-analysis involving over 2 million patients demonstrated a significant association between low

serum albumin and increased risk of thrombosis across diverse populations.[6] In paediatric oncology, reduced serum albumin has similarly been observed in children who developed VTE during treatment for acute lymphoblastic leukaemia compared to those who do not. The degree of hypoalbuminaemia also appears to correlate with thrombotic risk, with each 10 g/L decrease in serum albumin associated with a 2.13-fold increased risk of VTE.[2,4]

The underlying pathophysiological mechanisms are complex and multifactorial. First, low albumin leads to compensatory hepatic synthesis of large procoagulant proteins—such as fibrinogen and clotting factors V, VII, VIII, and XIII to maintain oncotic pressure. This increase is not matched by corresponding increase in natural anticoagulants like protein S, which may be lost in proteinuric states. Second, albumin plays a vital role in fibrinolysis by facilitating plasminogen binding to fibrin; therefore, low levels impair clot breakdown. Emerging evidence also suggests that clots formed in hypoalbuminaemic states have increased fibrin density and reduced susceptibility to fibrinolysis, partly mediated through a paraoxonase 1 (PON1)-dependent mechanism. Third, hypoalbuminaemia promotes platelet hyperaggregability, possibly through increased thromboxane A2 formation and reduced red cell deformability.[5,7,8]

In this patient, the exact cause of hypoalbuminaemia was not definitively established. Possible explanations include protein-losing enteropathy related to chronic subclinical infection or inflammation, or nutritional deficiency. The observed clinical improvement with nutritional rehabilitation suggests that nutritional factors may have contributed significantly.

The optimal duration of anticoagulation in paediatric VTE has been clarified by recent evidence. The Kids-DOTT (Duration of Therapy for Thrombosis in Children) multicentre randomised trial demonstrated that, in cases of provoked provoked VTE with clear and reversible risk factor, a 6-week course of anticoagulation is non-inferior to a 3-month course in terms of recurrent VTE and clinically significant bleeding at both 1-year and 2-year follow-up.[4,6]

These findings are reflected in the updated 2024 American Society of Hematology/International Society on Thrombosis and Haemostasis (ASH/ISTH) guidelines which suggest a shorter duration (6 weeks) of anticoagulation for selected children with provoked VTE. However, important exclusions include pulmonary embolism, recurrent VTE, persistent occlusive thrombus at 6 weeks, cancer-associated thrombosis, persistent antiphospholipid antibodies, major thrombophilia, and the presence of ongoing VTE risk factors.[9]

In our patient, the key challenge lay in determining whether hypoalbuminaemia represented a transient or persistent risk factor. As its underlying cause and chronicity could not be clearly established, the DVT was considered to be provoked by a potentially persistent risk factor. On this basis, a longer duration of anticoagulation - at least six - was chosen, with plans for reassessment based on repeat albumin levels and ongoing clinical evaluation. This approach aligns with current guideline recommendations, which emphasise individualised decision-making in the presence of ongoing risk factors.

We acknowledge several limitations in this report. A complete thrombophilia screen was not performed, although this is less critical given the presence of a likely acquired risk factor. The precise cause of the hypoalbuminaemia was not found, limiting confirmation of its reversibility. In addition, D-dimer testing which may be useful in selected low-probability cases was not performed due to resource constraints,[10]

Conclusions

This case underscores several important lessons for clinicians: The vast majority of cases of DVT cases have identifiable risk factors, and a systematic search is essential to guide management and inform strategies for secondary prevention. When common causes are excluded, hypoalbuminaemia should be considered as a potential contributor, as it may precipitate thrombosis through multiple mechanisms, including increased synthesis of procoagulant factors and impaired fibrinolysis.

Serum albumin measurement should therefore be included in the diagnostic evaluation of children presenting with apparently unprovoked or atypical paediatric VTE. The duration of anticoagulation should be guided by persistence of underlying risk factors, while a multidisciplinary approach to care remains crucial. Further research is needed to better define the causal relationship between hypoalbuminaemia and thrombosis in children and to establish evidence based thresholds for prophylactic anticoagulation in at-risk populations.

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