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Case Report

Unilateral Painful Leg and Ankle Edema Secondary to Bladder Dysfunction in a Young Woman: A Case Report

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Abstract

Edema is generally a bilateral and painless condition. Unilateral and painful edema is a very rare pathology. External compression of the lower extremity veins is an uncommon yet significant cause of unilateral leg edema (ULE). Among various etiologies, compression exerted by the urinary bladder on these veins may result in ULE. This condition is more frequently associated with benign prostatic hyperplasia (BPH) in men; however, it can also occur in women under certain circumstances.

Case Presentation

A 30-year-old female patient presented to the internal medicine outpatient clinic with complaints of swelling and pain in her right leg and ankle, persisting for the past three months. She had a 14-year history of type 1 diabetes mellitus (T1DM) managed with intensive insulin therapy. Apart from T1DM, she had no other known chronic illnesses or regular medication use. Her family history was unremarkable for chronic diseases.

Investigations

Physical examination revealed pitting edema localized to the right leg and ankle, without accompanying erythema or increased warmth. Cardiovascular examination was unremarkable, with a recorded blood pressure of 100/60 mmHg. Doppler ultrasonography of the lower extremities and routine biochemical analyses did not reveal any pathological findings. However, abdominal ultrasonography identified a distended urinary bladder (vesical globe). Following bladder decompression via urinary catheterization, the patient's edema resolved completely within two days.

Conclusion

In cases presenting with unilateral — or even bilateral — lower extremity edema, clinicians should consider intra-abdominal and pelvic pathologies as potential underlying causes of venous compression. Particularly in female patients with diabetes mellitus, bladder dysfunction leading to urinary retention (vesical globe) should be recognized as a rare but treatable etiology of unilateral leg edema.

Key Words: unilateral leg edema; type 1 diabetes mellitus; bladder dysfunction; venous compression

Introduction

Bilateral leg and ankle edema often necessitates a broad differential diagnosis, typically encompassing conditions such as congestive heart failure, hepatic or renal dysfunction, medication-induced edema, chronic venous insufficiency, and bilateral deep vein thrombosis (DVT) or inferior vena cava thrombosis [1]. In comparison, unilateral leg edema (ULE) is more frequently associated with localized pathologies including DVT, cellulitis, erythema nodosum, ruptured popliteal (Baker's) cysts, or trauma-related injuries. Rarely, ULE can result from less common etiologies such as intravascular lipomas, lymphatic system anomalies, previous venous or lymphatic surgeries, lipedema, May-Thurner syndrome, and Klippel-Trenaunay syndrome [2-4].

Although leg edema typically presents without pain, when pain does occur, it often signifies an underlying inflammatory process, as observed in conditions like cellulitis, venous thrombosis, trauma, or a ruptured Baker's cyst [5].

In most instances, ULE arises from venous system involvement. However, external venous compression is an important, though less frequent, mechanism leading to edema. Various intra-abdominal and pelvic pathologies, including neoplasms, aortic aneurysms, retroperitoneal hematomas, and retroperitoneal fibrosis, have been implicated in such compressive syndromes. Additionally, gravid uterus-induced compression of the pelvic veins and inferior vena cava is a well-recognized cause of lower

extremity edema, particularly in the third trimester of pregnancy and in the supine position [6].

In exceptional cases, bladder distension has been documented as a contributing factor to venous compression of the lower extremities, resulting in ULE. This phenomenon is predominantly reported in male patients with benign prostatic hyperplasia (BPH) [7-9].

Identifying the exact cause of leg edema typically relies on a thorough patient history, comprehensive physical examination, and lower extremity venous doppler ultrasonography. Despite these diagnostic approaches, there are instances where standard laboratory investigations, including blood and urine tests, fail to elucidate an underlying etiology [10].

Case Presentation

A 30-year-old female patient with a 14-year history of type 1 diabetes mellitus (T1DM), managed with multiple daily insulin injections (insulin aspart thrice daily and insulin glargine once daily), presented to the internal medicine outpatient clinic. Her past medical history was otherwise unremarkable, with no chronic comorbidities or regular medication use apart from insulin therapy. There was no family history suggestive of chronic systemic diseases.

Approximately three months prior to admission, the patient began experiencing progressive swelling and discomfort in her right leg and ankle. Initially suspecting a rheumatologic origin, she sought consultation at a rheumatology clinic. However, laboratory investigations, including autoimmune markers, did not reveal any rheumatological pathology.

Subsequently, due to accompanying urinary symptoms such as dysuria and urinary hesitancy, she was referred to the urology department. A mild urinary tract infection was identified and treated with antibiotics and analgesics. Despite treatment, her lower limb edema and pelvic fullness persisted, prompting further evaluation at the gynecology and obstetrics clinic, which did not reveal any gynecological abnormalities. Ongoing complaints eventually led to her referral to the internal medicine clinic for a comprehensive assessment.

Investigations

On physical examination, unilateral pitting edema was noted on the right leg and ankle, without associated erythema or increased skin temperature. Cardiovascular examination was unremarkable, with a blood pressure reading of 100/60 mmHg. Limb circumference measurements demonstrated asymmetry: Ten cm above the patella measured 51 cm on the right versus 49 cm on the left; 10 cm below the patella, the right measured 43 cm compared to 35 cm on the left (Fig. 1). The patient's body mass index (BMI) was 27 kg/m², with no recent weight fluctuations.

Laboratory investigations showed elevated fasting blood glucose (215 mg/dL) and HbA1c (9%), indicating suboptimal glycemic control. Anemia was noted with hemoglobin of 9.7 g/dL, along with iron deficiency (serum iron 20 mcg/dL, ferritin 8 ng/mL). Renal function tests revealed mildly elevated urea (52 mg/dL) and creatinine (1.4 mg/dL). No other significant biochemical abnormalities were found.

Lower extremity venous doppler ultrasonography excluded deep vein thrombosis and showed no venous flow abnormalities. However, abdominal ultrasonography revealed a markedly distended urinary bladder (vesical globe) (Fig. 2) and bilateral grade I-II hydronephrosis (Fig. 3). Post-voiding ultrasound confirmed incomplete bladder emptying.

The patient was admitted for further evaluation and management. Bladder catheterization yielded 700 mL of retained urine, resulting in a gradual reduction of lower extremity edema. Complete resolution of edema was observed within 48 hours. Upon detailed questioning, it was discovered that the patient had been compensating for her voiding difficulty by manually applying pressure to her suprapubic area.

A final diagnosis of transient right-sided unilateral leg and ankle edema secondary to venous compression from bladder distension due to bladder dysfunction was established.

Discussion

This case illustrates a rare instance of painful ULE resulting from external venous compression by a distended urinary bladder. Imaging studies, particularly abdominal ultrasonography, played a pivotal role in establishing the diagnosis by revealing bladder overdistension compressing the right iliac vein.

The association between bladder distension and venous outflow obstruction has been recognized since the 1960s, with Carlsson and Garsten being among the first to describe this phenomenon [9]. In most documented cases, benign prostatic hyperplasia (BPH) in male patients is the primary underlying cause of bladder-induced venous compression [7-9]. However, in women, bladder dysfunction leading to urinary retention and subsequent venous compression is a much less frequently reported cause of lower limb edema.

This case underscores the clinical importance of a meticulous patient history and thorough physical examination. Over-reliance on advanced diagnostic tests without a fundamental bedside assessment may lead to unnecessary investigations and delayed diagnosis. Physicians should maintain a broad differential diagnosis when evaluating unilateral leg edema and consider uncommon etiologies, especially in patients with risk factors for autonomic neuropathy, such as long-standing diabetes mellitus.

Although bladder distension clearly accounted for the mechanical venous compression and resultant edema, the pathophysiological basis of the associated pain in this patient remains unclear, as bladder-induced venous compression typically manifests as painless edema.

Conclusion

External compression of the lower extremity venous system, though rare, represents an important and reversible cause of leg edema. Clinicians should remain vigilant for intra-abdominal and pelvic pathologies, including bladder dysfunction, as potential sources of venous outflow obstruction in patients presenting with unilateral or bilateral leg swelling. While benign prostatic hyperplasia is a common cause in men, bladder dysfunction-related urinary retention should be considered in female patients, particularly those with diabetes mellitus. Early recognition and prompt intervention can result in rapid symptom resolution and prevent unnecessary diagnostic procedures.

Conflict of interests

The author has declared that no competing interests exist.

Human Ethics

Patient consent was obtained for this case report.

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