Case Report
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첫 증상으로 흔하지 않은 원인에 의한 양하지 부종을 나타낸 당뇨병 환자 1예

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An Unusual Case of Bilateral Peripheral Edema in a Male with Undiagnosed Type 2 Diabetes Mellitus

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Abstract

The present article demonstrates an unusual case of bilateral lower extremity edema caused by neurogenic areflexic bladder as the first physical symptom of diabetes. A 52-year-old man presented to the emergency department because of massive edema of his lower limbs. The edema had been present for 2 weeks, was symmetrical, and was progressively covering the lower limbs up to the inguinal area, scrotal bag, and penis and was accompanied by dysuria and an interrupted urine stream. Laboratory findings revealed a serum glucose level of 657 mg/dL and glycated hemoglobin (HbA1c) level of 15.6%. Computed tomography (CT) of the abdomen and pelvis revealed marked enlargement of the bladder with bilateral hydronephrosis and hydroureter. In addition, CT demonstrated bilateral compression of the iliac veins caused by the enlarged bladder. This case highlights the importance of a broad differential diagnosis for patients with diabetes and extensive peripheral edema. Neurogenic bladder should be considered in the differential diagnosis, even in newly diagnosed diabetic patients.

Keywords: Diabetes complications, Edema, Neurogenic, Urinary bladder

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INTRODUCTION

Type 2 diabetes is a very common disease that typically has an asymptomatic phase between the onset of diabetic hyperglycemia and clinical diagnosis. This phase has been estimated to last at least 4~7 years, and 30~50% of type 2 diabetic patients remain undiagnosed, which can lead to the development of chronic complications of diabetes (the chief problems in diabetic care), leading to the inability to work, disability, and premature death [1].

One study detected an association between diabetes and autonomic neuropathy in 40% of patients with diabetes [2]. Herein, we report an unusual case of bilateral lower extremity edema caused by neurogenic areflexic bladder as the first physical symptom of diabetes.

CASE REPORT

A 52-year-old man presented to the emergency department complaining of progressive swelling of his lower limbs, inguinal area, scrotal bag, and penis for two weeks prior to presentation, painful urination, and an interrupted stream of urine. His medical history was unremarkable, and he was not on any medications. The patient stated he had not been to the hospital for more than 10 years because of his poor economic status. He had experienced 14 kg of unintentional weight loss over the past year, and his previous weight was 60 kg. He reported that he was eating three meals per day as usual. The patient did not smoke or consume alcohol. Upon admission to our hospital, physical examination revealed the following: height, 166 cm; weight, 46 kg; body mass index, 16.6 kg/m^2; body temperature, 36.6℃; blood pressure, 140/90 mm Hg; and heart rate, 86 beats/min and regular. Physical examination revealed prominent pitting edema of both lower extremities and swelling of the scrotum and penis. There was also a large palpable mass in the lower abdomen. His cardiovascular and lung examination were normal. Laboratory studies revealed the following: serum glucose 657 mg/dL, glycated hemoglobin (HbA1c) 15.6% without evidence of ketoacidosis, blood urea nitrogen 21 mg/dL, creatinine 1.0 mg/dL, total protein 6.6 mg/dL, serum albumin 3.6 g/dL, sodium 137 mEq/L, and potassium 5.0 mEq/L. The results of liver and thyroid function tests were within normal limits, and the albumin-to-creatinine ratio was 7.5 mg/g. The fasting C-peptide level was 0.98 ng/mL. The D-dimer was negative, and no deep vein thrombosis was detected on color Doppler ultrasonography. The electrocardiogram and chest radiograph did not indicate heart failure. A computed tomography (CT) of the abdomen and pelvis revealed marked enlargement of the bladder (14 × 10 × 16 cm) (Fig. 1A, asterisk) with bilateral hydronephrosis and hydroureter. In addition, CT demonstrated bilateral compression of the iliac veins by the enlarged bladder (Fig. 1B, arrows). A Foley catheter was inserted with an initial return of 1,400 mL of urine.

Because of the patient's glycemia, intense insulin therapy with human insulin analogues was initiated. The patient was diagnosed with the following diabetic complications: sensorimotor polyneuropathy, autonomic neuropathy of the gastrointestinal tract, and bilateral moderate diabetic retinopathy. After catheterization, elevation of the lower limbs was recommended for the edema, which resolved completely after 7 days. Therefore, there was no need to apply compression

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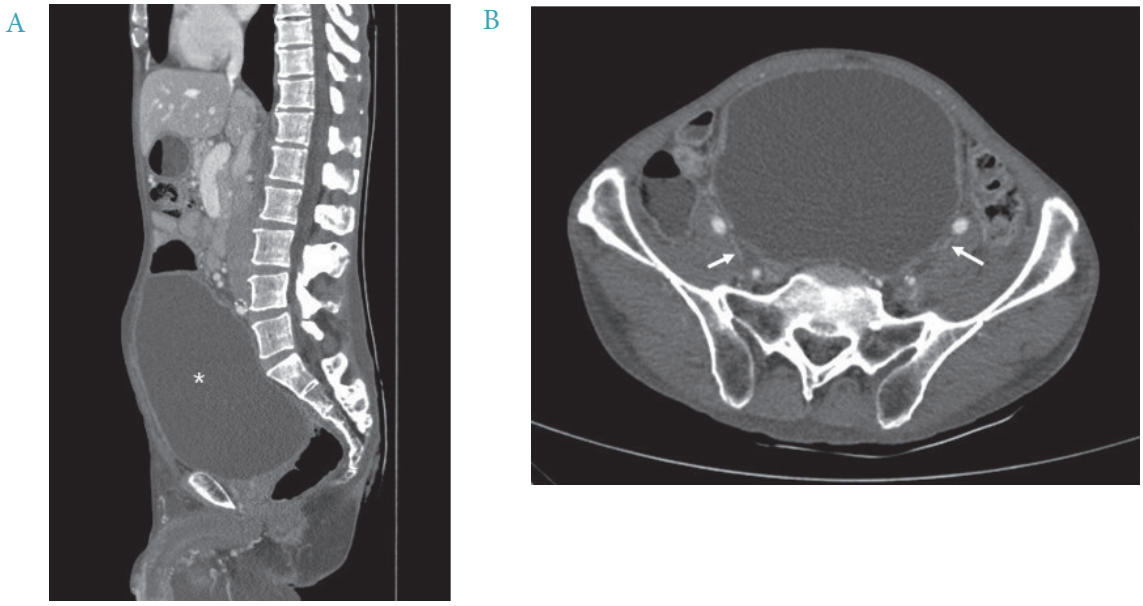

Fig. 1. Computed tomography of abdomen and pelvis showing massive enlarged bladder (*), sagittal view (1A) with compression of both external iliac veins (arrows), axial view (1B).

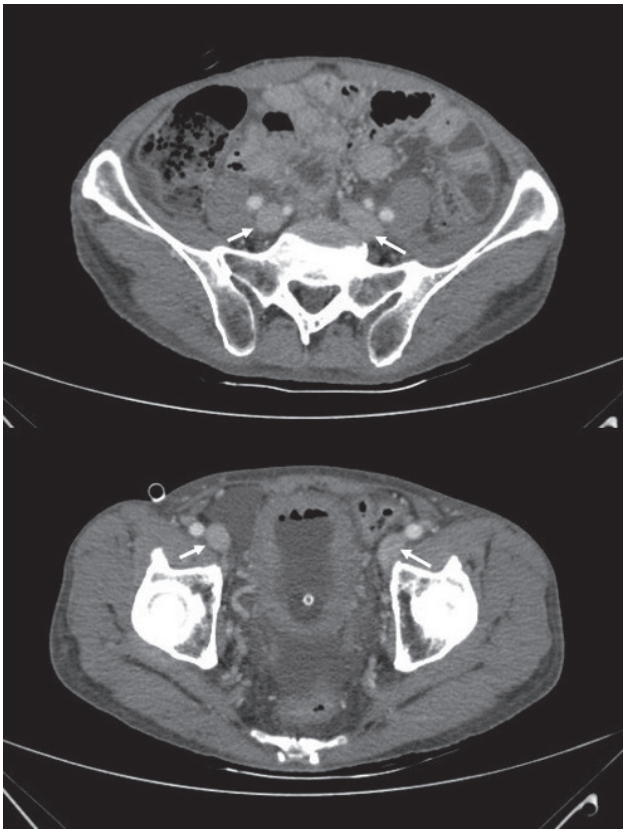

Fig. 2. Computed tomography examination of the abdomen and pelvis after 1 month, showing normalization of the bladder and resolution of both external iliac veins (arrows).

therapy or administer diuretic drugs. Bethanechol chloride (25 mg orally three times a day) was initiated, and subsequent abdominal and pelvic ultrasonography showed retention of approximately 300 mL of urine in the bladder after micturition. Upon further questioning, the patient did not have any sense of bladder fullness or urgency to void. The patient was discharged in optimal condition with the recommendation to continue clean intermittent bladder catheterization. Approximately one month after discharge, an abdominal CT demonstrated that the compressed external iliac vein returned to normal size (Fig. 2, arrows).

DISCUSSION

Bilateral lower extremity edema can be caused by a number of medical conditions, such as congestive heart failure, liver cirrhosis, nephrotic syndrome, lymphedema, and protein losing enteropathy, as well as obstructive pathologies, such as filariasis, hepatic vein

and inferior vena cava thrombosis, and inferior vena cava and external iliac vein compression [3]. In our case, heart failure (based on echocardiography), venous thrombosis (based on compression ultrasonography with Doppler imaging), and renal failure (based on normal serum creatinine and albumin concentrations) were excluded. Because the bilateral leg edema and dysuria had a similar time of onset, bilateral iliac vein compression by a distended bladder due to retention of urine was suspected in the present case.

Urinary bladder distension is a rarely reported cause of iliac vein compression. In 1960, iliac vein compression syndrome due to bladder distension was first reported by Carlsson and Garsten [4] in a neonate with posterior urethral valves. Since then, adult cases have been reported, chiefly in middle-aged and elderly men. The most common cause is benign prostatic hyperplasia followed by neurogenic bladder. Iliac vein compression syndrome secondary to bladder distension tends to present with acute onset bilateral edema that is not associated with thrombosis [5,6]. Less common causes of bladder distension that lead to venous compression include spinal cord injuries, stroke, and neurogenic bladder due to diabetes.

Diabetic cystopathy is caused by damage of the visceral afferent fibers in the bladder wall, and it is characterized by an increase in both post voiding residual volumes and bladder capacity accompanied with decreased bladder sensation and contraction [7]. The etiology of diabetic cystopathy is multifactorial but mainly recognized as a manifestation of diabetic neuropathy, which is thought to be present in patients with diabetes mellitus of long duration. Type 2 diabetes is an insidious illness with a long preclinical asymptomatic phase during which patients may be exposed to the ill effects of asymptomatic hyperglycemia for many years, as demonstrated by the present case. A substantial proportion of patients with type 2 diabetes have evidence of diabetic tissue damage at the time of diagnosis of diabetes.

Non-pharmacological treatment of diabetic cystopathy, such as diet, lifestyle modifications along with pelvic exercises and muscle training, are recommended. For those patients who present with detrusor overactivity, anticholinergic drugs may be an option, but pharmacotherapy has a limited role in the treatment of detrusor areflexia. Rarely, surgical intervention renders beneficial effects. Gene therapy and tissue engineering have opened new horizons with exciting promise [8].

In conclusion, this case highlights the importance of considering a broad differential diagnosis for patients with diabetes and extensive peripheral edema. Neurogenic bladder should be considered in the differential diagnosis, even in newly diagnosed diabetic patients.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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