Acute Peroneal Neuropathy and Foot Drop in Two Adolescent Female Athletes with New-Onset Diabetes

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Introduction

The common peroneal nerve is derived from the sciatic nerve. It travels superficially along the lateral aspect of the knee near the fibular head where it bifurcates into the superficial and deep peroneal nerves. These nerves also provide sensation to the lateral lower leg and dorsal foot. The superficial and deep peroneal nerves innervate the muscles of the lateral lower leg and anterior lower leg compartments, respectively. Loss of the soft tissue and subcutaneous fat pad that surrounds and cushions the peroneal nerve near the fibular head leaves it susceptible to injury (1). This can affect the common peroneal nerve or either of its two branches, the deep or superficial peroneal nerves (2). Damage to the nerve by stretching or compression may result in loss of sensation and motor function, resulting in foot drop (3). Acute peroneal neuropathy in the context of rapid weight loss (also known as "slimmer's paralysis") has been reported in prisoners of war, extreme dieting, and after bariatric surgery (2,4). It also is more common in individuals who habitually cross their legs (2).

While seen less commonly than in adults, acute peroneal neuropathy also has been reported in children and adolescents (5). Etiologies in this age group include direct trauma, entrapment from bone tumors, compression from casting, and rapid weight loss (5,6). Causes of sudden and quick weight loss may be due to crash dieting and anorexia nervosa (6). There also are rare reports of slimmer's paralysis being caused by rapid weight loss from untreated type 1 diabetes (7,8). In this report, we present two active adolescent female athletes who presented with ankle pain or weakness that was ultimately due to acute peroneal nerve neuropathy associated with substantial and fast weight loss from undiagnosed type 1 diabetes.

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Patient 1

An 11-year and 8-month old female cheerleader presented to her local hospital with a gait abnormality and foot pain. Her mother had noted progressive changes in her daughter's gait over the preceding few weeks. Her foot would become painful with prolonged walking. She frequently sat cross-legged at her desk in school. Her review of systems was remarkable for polyuria, polydipsia, and a 20 lb weight loss. Neurologic examination was significant for 0/5 right dorsiflexion and eversion at the ankle. She had a steppage gait while walking. No other abnormalities were noted: 2+ reflexes, normal Babinski reflex, and no sensory deficits to pinprick or light touch. Foot, ankle, and leg X-rays were normal. Laboratory testing was consistent with new-onset type 1 diabetes, and she was started on insulin injections. She was referred to physical therapy where she received intermittent and inconsistent treatment. When she was seen in follow-up 2 months later, she had gained 11.7 lb, and the foot drop and other neurological deficits had resolved.

Patient 2

A 15-year and 6-month old female tennis player presented to a sports medicine specialist for left foot pain and weakness. She also had a gradual onset of pain starting a couple months prior. Pain was localized to the dorsal foot with palpation and activity, cold sensation, and tingling. Neurologic examination was normal except for deficits in the left lower leg and foot (Figure): 3/5 dorsiflexion, eversion, and inversion, as well as decreased sensation on the posterior and lateral ankle and dorsal foot. Electromyography revealed a left common peroneal neuropathy at the fibular head. Review of systems was remarkable for polyuria and polydipsia over the last 1 to 2 months, and a 35 lb weight loss. Testing revealed hyperglycemia. Additional studies confirmed the diagnosis of type 1 diabetes, and she was started on insulin injections. She was referred for physical therapy, but she was noncompliant. In follow-up 1 month later, she had gained 8.8 lb, and the foot drop and neurological deficits had resolved.

Clinical characteristics of the patients are found in the Table.

Discussion

With loss of the soft tissue and fat pad that protects the peroneal nerve at the lateral knee, compression and stretching of

Figure: Reduced range of motion with active dorsiflexion of the left foot compared to the right foot in patient 2.

the nerve may result in foot drop (1–3). Both of our patients quickly lost weight because of unrecognized and untreated type 1 diabetes. In young, otherwise-healthy individuals, it is uncommon to consider underlying medical conditions to cause orthopedic problems. Therefore, taking a complete history and review of systems to find unusual causes for musculoskeletal complaints is essential. Recent and sudden weight loss should be assessed in the evaluation of foot drop, especially if there is not another logical cause. The classic features of diabetes, including polyuria and polydipsia, are important for the primary care sports medicine specialist to consider when evaluating otherwise-healthy individuals who have lost weight.

Acute peroneal neuropathy is a rare and unexpected condition in new-onset type 1 diabetes. Neuropathy may occur after chronic and uncontrolled hyperglycemia, but it is extremely unusual to be present at diagnosis in children with new-onset type 1 diabetes. As seen in both of our patients, the neuropathy was transient and resolved with weight gain that occurred secondary to adequate glycemic control with insulin therapy. Diabetic neuropathy is thought to result from chronic, long-standing poor blood sugar control (9,10). Given the relatively short lifetime exposure to hyperglycemia and quick resolution of symptoms, the foot drop in our patients was not because of chronic diabetic neuropathy as has been previously reported (7,8). However, as seen in similar cases of acute peroneal neuropathy with sudden weight reduction, we propose that extreme weight loss over a short duration that may occur in new-onset type 1 diabetes is the cause of this peroneal neuropathy (2,6). Another condition that could account for these symptoms in the context of diabetes is diabetic lumbosacral radiculoplexus neuropathy (DLRPN). DLRPN involves severe lower extremity pain and weakness, but it is most commonly seen in adults with type 2 diabetes (11). In contrast to our patients who had prompt resolution with insulin treatment and resultant weight gain, the vast majority of patients with DLRPN have incomplete recovery with longstanding pain and weakness. While we cannot prove the sudden and dramatic weight loss caused the acute peroneal neuropathy in our cases, the resolution of the neuropathy closely correlated with weight gain.

Conclusions

In the assessment of acute peroneal neuropathy, it is important for the clinician to recognize rapid weight loss as a potential cause. This aspect of the history is particularly critical when assessing otherwise-healthy athletes, as an underlying organic etiology for the weight loss (such as undiagnosed type 1 diabetes) may be present. Recognition of this condition is important so that clinicians can counsel their patients, find appropriate treatment for the underlying problem, and avoid unnecessary testing and referrals.

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Table.
Clinical characteristics of female adolescents with new onset diabetes and unilateral foot drop.

	Patient 1	Patient 2
Age at diagnosis (yr)	11.6	15.5
Tanner stage	IV	V
HbA1c at diagnosis (%)	16.4	>14
Diabetes autoantibodies (+)	GAD, IA-2, Insulin	GAD, IA-2
Amount of weight loss (kg)	9.1	15.9
Weight at diagnosis (kg)	37.5	58.6
BMI at diagnosis ($kg \cdot m^2$, SDS)	16.5 (−0.56)	21.5 (+0.4)
HbA1c at follow-up (%)	8.9	8.4
Weight at follow-up (kg)	42.8	62.6
Change in weight (kg)	5.3	4
BMI at follow-up ($kg \cdot m^2$, SDS)	18.9 (+0.33)	22.1 (+0.54)

BMI, body mass index; GAD, glutamic acid decarboxylase antibody; IA-2, islet tyrosine phosphatase 2 antibody; SDS, standard deviation score.

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