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Botulinum Toxin Type A Injection Combined With Cast Immobilization for Treating Recurrent Peroneal Spastic Flatfoot Without Bone Coalitions: A Case Report and Review of the Literature

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ABSTRACT

Peroneal spastic flatfoot is an uncommon condition. It often presents as a rigid and usually painful valgus deformity in the hindfoot with peroneal muscles spasms. Although tarsal coalition is an important cause, a few patients have not undergone bone coalitions. We describe a 27-year-old female who experienced recurrent peroneal spastic flatfoot after an injury. She was treated successfully with a combination of botulinum toxin type A and immobilization of the foot in a neutral position with a cast. After 3 years, the condition had not recurred, and she was pain free and walked normally, with no increase in muscle tone. This unique treatment could be of potential use to treat many patients with such conditions.

Peroneal spastic flatfoot, first described by Robert Jones in 1897, is rare condition characterized by a rigid and usually painful valgus deformity in the hindfoot with peroneal muscles spasms (1). It is often caused by intertarsal bars or bone anomalies that restrict tarsal joint motion. Although tarsal coalition is an established cause, other causes have been documented (2–5), including a few cases without bone coalition. To our knowledge, no more than 40 cases of peroneal spastic flatfoot without bone coalition have been reported in published studies (6–11). Furthermore, the condition has seldom been found in patients older than 20 years of age (1). These conditions can make the case complicated to treat because of its unclear cause and no documented treatment. We present the case of a 27-year-old female who experienced recurrent peroneal spastic flatfoot after an injury. She was successfully treated using a combination of botulinum toxin with cast immobilization.

Case Report

A 27-year-old female referred to our clinic reported pain and an everted deformity in her right hindfoot. Six weeks earlier, she had sprained her right later ankle while mountain climbing. At that time, she was examined at the emergency department of the local hospital. The findings from anteroposterior and oblique radiographs were normal and showed no evidence of bone coalitions (Fig. 1). Computed tomography and magnetic resonance imaging scans also showed no evidence of fractures or injury to the articular cartilage (Figs. 2 and 3). She was given an ankle support and advised to rest.

She continued to walk with full weightbearing and reported only a little pain on the lateral side of her ankle. When she came to our clinic, she described heavy pain and tenderness around the ankle and kept her foot everted (Fig. 4). The calcaneus had been pulled into a valgus deformity by the considerable peroneal muscle spasms, but the medial longitudinal arch was normal. She had no peritalar motion, and efforts to invert her foot to a more normal position were made difficult by obstinate spasm.

The muscle tone measured on the modified Ashworth scale (12) was grade 4. Interestingly, her foot would return to a normal position, without peroneal spasm, at rest or when non-weightbearing, or when she was asleep, as reported by her husband. The findings from electromyography and blood tests, including the erythrocyte sedimentation rate and the differential sheep-cell agglutination test, which can be used to reveal cases of early rheumatoid arthritis, were all negative. Given the negative test results and imaging examinations, we diagnosed her condition as peroneal spastic flatfoot from an uncertain cause.

Initial treatment consisted of a common peroneal nerve block, in which 3 mL of 2% lidocaine was injected 2 cm under the fibular head, between fibular head and fibular neck. Her foot returned to the normal position with increased motion of the subtalar joint (Fig. 5). The foot was immobilized in a neutral position in a below-the-knee plaster for 6 weeks. She was told not to bear weight on the treated

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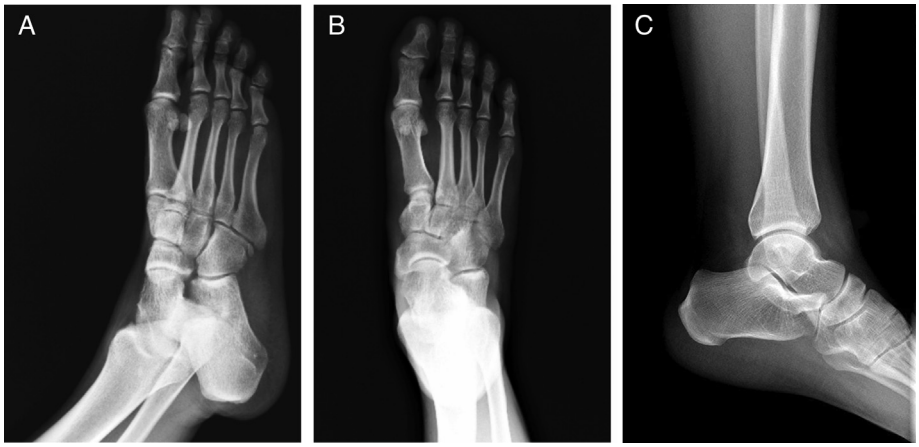

Fig. 1. (*A*) Oblique, (*B*) anteroposterior, and (*C*) lateral radiographs of a 27-year-old female with recurrent peroneal spastic flatfoot showed no signs of bone coalition.

foot after the cast was removed. Early in the immobilization period, she reported spasm several times, but the frequency declined with time. However, once the cast was removed, her symptoms recurred. The muscle tone was still grade 4, although her pain was less, and she still had a valgus deformity.

B-mode ultrasonography indicated that both the peroneus longus tendon and brevis muscle were in spasm. Under ultrasound guidance, we injected botulinum toxin type A (Botox®, Allergan Inc, Irvine, CA), 6 U/kg per muscle (100 U in 1 mL of 0.9% saline) into the mid-belly of each peroneal muscle. Her foot, especially the rearfoot, was immobilized in a neutral position with a cast for 6 weeks. She was instructed to keep her weight off the foot using crutches and was given a universal medial arch sole for her shoes. The arch sole was made of silica and fitted her shoe size. The arch was 1.5 cm high. During sleep, a T-strap was affixed to the foot in case the spasms recurred. During the next 2 months, she gradually increased weightbearing and afterward could walk with full weightbearing without crutches. No side effects from the botulinum injection were apparent after 3 months.

At every 3-month follow-up visit for 3 years, she was free of pain, free of spasm, her gait was normal, and her modified Ashworth scale score was grade 0, indicating no increase in muscle tone. Active and passive range of ankle movement was the same in both feet.

The patient and her family gave consent to have her case report submitted for publication.

Discussion

Robert Jones was the first to describe this particular type of flatfoot in 1897 (1). Not until 1930 did Malkin et al (13) begin a discussion on spasmodic flatfoot. For the most part, no separate forms of flatfoot were described. Instead, 2 forms of "rigid flatfoot" were vaguely described. One was "acute spasmodic valgus," and the other was "chronically or permanently stiff flatfoot."

Patients with peroneal spastic flatfoot present a diagnostic and treatment challenge. Bone coalitions, including talocalcanean bridge, calcaneonavicular bar, and cubonavicular coalition, are common causes (14–17). However, a few patients have had no bone coalitions. In 1939, Todd (18) reported 8 cases with acute spasmodic valgus, which formed 5.7% of his 141 cases of flatfoot. Harris and Beath (19) presented 2 cases with no bone coalitions, both caused by rheumatoid arthritis. Webster and Roberts (15) reported 21 patients with peroneal spasm or rigid flatfeet, 7 of whom had no bone abnormalities. They provided no details on the presumed cause.

Jack (20) described 7 cases of such condition. Of these, 1 had been caused by tuberculosis infection of the talus, 2 were secondary to osteoarthritis of the talus, and 4 had no specific cause but were thought to be related to trauma or chronic strain. Blockey (1) also described 8 feet without radiologic evidence of abnormality but, likewise, provided no detailed descriptions. Johnson (9) reported that a fibrosarcoma in the subtalar joint caused peroneal spastic flatfoot syndrome. Lowy (6) presented 4 cases without bone coalitions. In 2005, Kinoshita et al (11) found 2 cases with sinus tarsi syndrome. In 2007, a talar osteochondral lesion was reported to be the cause for a case of peroneal flatfoot. Most of the reported patients were adolescents younger than 20 years old. In contrast, our patient was 27-year-old female in whom peroneal flatfoot had developed 6 weeks after an injury.

In the review by Todd (18), some researchers proposed that focal sepsis was the cause of the condition and even nicknamed these feet, "tonsil feet." Todd himself did not agree with this proposal. He believed that certain feet succumbed to the effects of habitual overstrain because they were structurally and developmentally weak. However, he did not propose any detailed explanation.

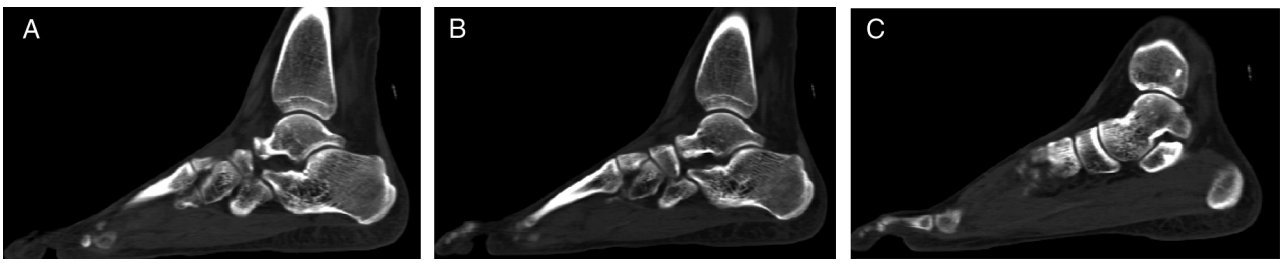

Fig. 2. (*A* to *C*) Computed tomography scans revealed no evidence of bone coalitions in the foot or ankle.