

Inversed champagne bottle type muscle atrophy observed in acromegaly

Yoshiko Shibuta M.D.,^{#1#2}, Hiroyuki Nodera, M.D.^{#1#3}, Ryuji Kaji, M.D.^{#1}

#1. Department of Neurology, Institute of Health Biosciences, The University of Tokushima Graduate School, 3-18-5, Kuramoto, Tokushima 770-8503, Japan

#2. Department of Neurology, Tokushima National Hospital, National Hospital Organization, 1354 Shikiji, Kamojima, Yoshinogawa, Tokushima 776-8585 Japan

#3. Department of Neurology and Neuroscience, Weill Cornell Medical College, New York, USA

Received 26 November 2009; received in revised form 7 December 2009; accepted 22 December 2009

Abstract

We reported a 55-year-old man, who was diagnosed with acromegaly at age 23. He noted difficulty dorsiflexing his feet at age 25 and was diagnosed with lumbar spinal canal stenosis. His gait had gradually been worsened, to become wheelchair-bound at age 52. On neurological examination, there was inversed champagne bottle type muscle atrophy. Needle electromyography revealed active and chronic denervation in the distal and proximal limb muscles innervated by the L5 and S1 nerve roots. MRI of the lumbosacral spine demonstrated vertebral displacement with hypertrophy of yellow ligament. It is necessary for the painless progression of muscular atrophy in the presence of severe spinal canal stenosis to pay attention as acromegalic neurologic complications.

Key Words: painless progression, MRI, acromegalic neurologic complications, bottle type muscle atrophy, acromegaly,

Introduction

In acromegaly, several neuromuscular complications have been reported, including carpal tunnel syndrome, polyneuropathy, and myopathy. In rarer occasions, soft tissue hypertrophy and increased bone resorption can cause spinal canal stenosis, potentially complicating with radiculopathy and cauda equina syndrome [1-4]. However the recognition of such conditions in a patient with painless distal amyotrophy is not always straightforward and needs attention.

Case Report

A 55-year-old man presenting gigantism was diagnosed with acromegaly due to pituitary adenoma at age 23 and had Hardy operation. The levels of growth hormone have been elevated even after the surgery. He noted difficulty dorsiflexing his feet at age 25 and was

diagnosed with lumbar spinal canal stenosis, that was operated at age 32, with little improvement of the strength. A few years thereafter, he noted difficulty plantarflexing his feet. Diabetes was then identified and his gait had gradually been worsened, to become wheelchair-bound at age 52. He denied pain, numbness, or tingling sensation at any point. On neurological examination, there was inversed champagne bottle type muscle atrophy with complete foot drop (Figure 1a). There was moderate loss of light touch and pinprick sensation in the distal extremities and the deep tendon reflexes were absent in the lower extremities. Plantar responses were flexor. Nerve conduction studies in the upper extremities demonstrated prolonged distal latencies and low-amplitude compound muscle action potentials (CMAPs) in the bilateral median nerves. In the lower extremities, the CMAPs in the tibial and peroneal nerves were not evoked, but the sensory action potentials in the bilateral sural nerves were only moderately

decreased in amplitudes ($6\mu\text{V}$ on both sides using the needle recording electrodes, normal $>8\mu\text{V}$). Conduction velocities of the sural nerves were decreased ($31.5\text{-}37.5\text{m/s}$). Needle electromyography revealed active and chronic denervation in the distal and proximal limb muscles innervated by the L5 and S1 nerve roots.

1a



1b



Figure 1a. Inversed champagne bottle type muscle atrophy of the lower extremities.

Figure 1b. Sagittal T2 weighted MRI of the lumbosacral spine shows posterior protrusion of spinous processes and intervertebral discs, multiple hypertrophy of yellow ligament, and diffuse posterior vertebral scalloping.

Overall, these findings suggest severe polyradiculopathy with superimposed polyneuropathy due to acromegaly and diabetes.

Sagittal T1 and T2 weighted MRI of the lumbosacral spine demonstrated posterior protrusion of spinous processes and intervertebral discs, multiple hypertrophy of yellow ligament, and diffuse posterior vertebral scalloping, all compatible with spinal changes seen in acromegaly (Figure 1b) [1, 4-6].

Discussion

An association of spinal canal stenosis with acromegaly has been reported [1-4]. It is considered to be caused by widening of the interpediculate distance, osteoporosis, soft tissue hypertrophy, and intervertebral disc hypertrophy. The recognition of radiculopathy as a complication of spinal canal stenosis in acromegaly is often straightforward when symptoms such as paresthesias, radiating pain, and intermittent claudication are present. However, slowly progressive distal muscle weakness without such sensory symptoms can be falsely attributed to polyneuropathy due to acromegaly and diabetes without proper electrodiagnostic assessment. Then, the opportunity to receive spine surgery with potential good outcome may be missed. The painless progression of muscular atrophy in the presence of severe spinal canal stenosis may be explained by the partial resection of pituitary adenoma, causing only slow rate of progression of spinal deformity.

References

1. Cheng CL, Chow SP: Lumbar disc protrusion in an acromegalic patient. *Spine* 1990;15:50-51.
2. Epstein N, Whelan M, Benjamin V: Acromegaly and spinal stenosis. Case report. *J Neurosurg* 1982;56:145-147.
3. Mikawa Y, Watanabe R, Nishishita Y: Cervical myelopathy in acromegaly. Report of a case. *Spine* 1992;17:1542-1543.
4. Parikh M, Iyer K, Elias AN, Gwinup G: Spinal stenosis in acromegaly. *Spine* 1987;12:627-628.
5. Wakely SL: The posterior vertebral scalloping sign. *Radiology* 2006;239:607-609.
6. Woo CC: Postmyelographic cauda equina syndrome in an asymptomatic acquired spinal stenosis of a young acromegalic. *J Manipulative Physiol Ther* 1988;11:118-123.