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Hypovitaminosis D and Myosteatosis of Paraspinal Muscles

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ABSTRACT

Myosteatosis is a pathological infiltration of fat in skeletal muscle reflecting an impairment in normal synthesis and elimination of triglyceride. Myosteatosis is associated with aging, type 2 diabetes, insulin resistance, and vitamin D deficiency. It is inversely related to loss of muscle strength and consequent disability. Lumbar paraspinal muscles are located on each side of the lumbar spine, attached to the vertebrae, providing necessary levels of trunk stability and trunk movement. We report a 65-year-old woman with vitamin D deficiency, type 2 diabetes, and laboratory findings of secondary hyperparathyroidism. Her physical examination indicated dominantly pronounced walking difficulties due to the weakness of the paraspinal and pelvic muscles and the instability of the lower part of the vertebral column. Characteristic radiological features of expressive myosteatosis of paraspinal muscles were determined by magnetic resonance. The treatment of vitamin D, calcium and physical therapy over the course of one year did not block her progressive mobility disorder.

Introduction

Myosteatosis is the infiltration of fat in skeletal muscle [1] and inversely associated with the loss of muscle strength or mobility disability [2]. It is associated with aging, insulin resistance, type 2 diabetes [1, 3], and vitamin D deficiency [4]. The exact mechanisms underlying the increase in myosteatosis with aging in humans are largely unknown [1]. Depending on the affected skeletal muscles and pain intensity, it could lead to significant functional disability and decrease in the quality of life. Myosteatosis causes walk-

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ing difficulties as it affects the functional ability of paraspinal and pelvic rim muscles.

Case Presentation

A 65-year-old white woman was admitted to our health center due to walking difficulties she experienced for years, but with a significant progression over the last 2-3 months, which lead to the use of the crutches, with the additional assistance for walking from another person.

The patient's history included frequent, sometimes daily, tetanic attacks since 10 to 18 years ago, osteoporosis treatment for the last 13 years (ibandronate acid therapy) and type 2 diabetes mellitus for the last 8 years (diabetic diet and metformin therapy). The patient was occasionally treated with vitamin D and calcium supplements as recommended by an endocrinologist.

Her physical examination indicated dominantly pronounced walking difficulties due to the weakness of the pelvic muscles and the instability of the lower part of the vertebral column, with the impossibility of the body flexion from the lying position and a weak femur flexion. The patient could not climb up the stairs. Neurologic tests did not indicate any signs of lesions in the central or peripheral nervous system. The patient was slightly adipose.

Laboratory findings indicated normal serum level of total calcium; 2.27 mmol/L (reference range [RR] 2.14 to 2.53 mmol/L), ionized calcium; 1.16 mmol/L (RR 1.13 to 1.32 mmol/L), inorganic phosphorus; 0.87 mmol/L (RR 0.79 to 1.42 mmol/L) and total magnesium; 0.79 mmol/L (RR 0.65 to 1.05 mmol/L). Serum level of 25(OH)D3 was low at 5.5 µg/L (RR 20.0 to 100.0 µg/L) and serum level of intact parathyroid hormone (iPTH) increased up to 102 ng/L (RR 12 to 88 ng/L). Her Vitamin B12 level reduced to 83.0 pmol/L (RR 138 to 652 pmol/L). Serum levels of cholesterol, triglycerides, urea, and creatinine were normal (3.8, 0.42, 7.7 mmol/L and 74 µmol/L, respectively). Serum C-reactive protein concentration was slightly elevated; 40.0 nmol/L (RR 0.76 to 28.5 nmol/L).

Spine Magnetic Resonance Imaging scans (MRI) indicated signs of myosteatosis of paravertebral muscles, but no signs of the bone lesion, spinal cord lesion, or any other nerve lesion. In addition to myosteatosis, thickening of subcutaneous adipose tissue and visceral adipose tissue were recorded. The most prominent MRI image of fatty infiltration of skeletal muscles was found in the lumbar region. Figures 1 and 2 present myosteatosis of paraspinal muscles with significant rarefication and atrophy of muscle fibers (myopenia) at L1/2 and L4/5 levels. We present an MRI scan of the L4/5 level of a woman of approximately the same age without myosteatosis in order to compare our patient's with normal MRI of the paraspinal musculature (Figure 3). Other than para-

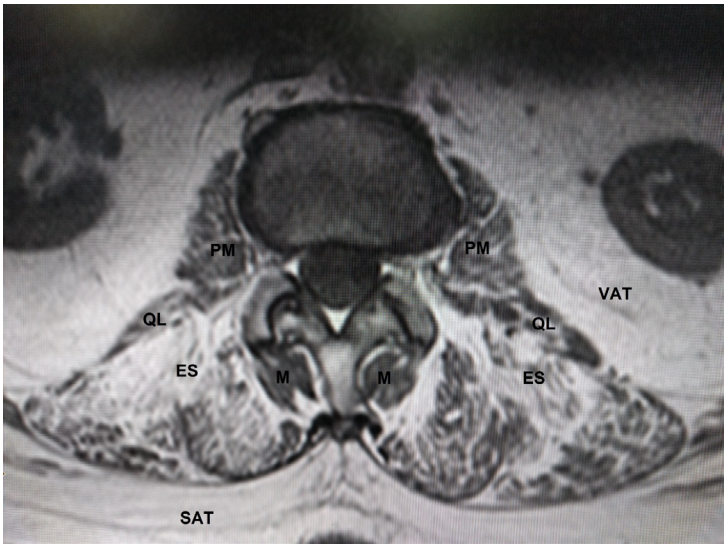

Figure 1. Axial MRI of the lumbar spine - T1 weighted image at the L1/2 level

The prominent paraspinal musculature atrophy and fatty replacement in a woman with disturbance gait. Apart from signs of myosteatosis, the quantity of the visceral and subcutaneous adipose tissue has extremely increased.

ES: Erector Spinae; PM: Psoas Major; M: Multifidus; QL: Quadratus Lumborum; SAT: Subcutaneous Adipose Tissue; VAT: Visceral Adipose Tissue

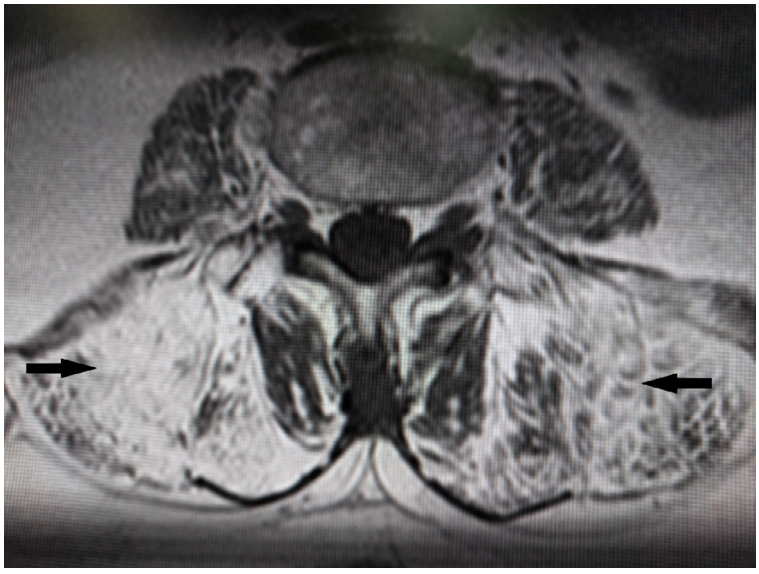

Figure 2. Axial MRI of the lumbar spine - T1 weighted image at the L4/5 level

The extremely fatty replacement of the erector spinae muscles (arrow)

spinal muscles, MRI myosteatosis signs were also found in iliopsoas with visible pathologically enlarged visceral and subcutaneous fat (Figures 1 and 2).

In consultation with the endocrinologist, the treatment continued with vitamins D and B12 replacements, calcium preparations, and physical therapy. After five months of intensive physical rehabilitation in a specialized rehabilitation institution, the patient was examined. She felt somewhat to be more active, but the objective examination did not reveal any significant changes in her mobility. Thirteen months after the initial hospitalization, the patient showed signs of a gradually progressively worsening mobility, including slow walking motions, more instability of the gait, weakened lateral flexion of the spine, almost complete loss of the trunk anteflexion and flexion of the leg in

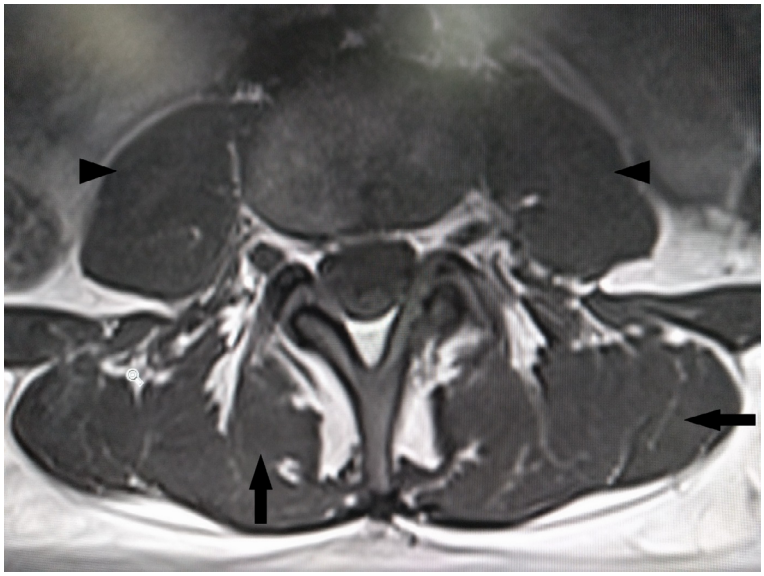

Figure 3. Axial MRI of the lumbar spine - T1 weighted image at the L4/5 level

The paraspinal (arrow) and psoas muscles (arrowhead) in normal healthy 65-year-old women. No signs of myosteatosis or pathological increase in visceral and subcutaneous adipose tissue.

the lying position, and very limited trunk rotation in the lying position.

Discussion

The factors leading to the myosteatosis are not well-defined yet [5]. Vitamin D plays a relevant role in muscle function [4]. We speculate that the primary disorder in our patient is a long-term vitamin D deficiency and consequently poor calcium absorption. Long-term hypocalcemia, which caused numerous tetanic convulsions, was the cause of secondary hyperparathyroidism [6]. Long-term secondary hyperparathyroidism is the possible cause of the secondary osteoporosis. The current normal serum level of total and ionized calcium, a reduced level of vitamin D3, and an increased level of iPTH in our patient suggest that condition.

Tagliafico et al. found that fatty degeneration of thigh muscles in the elderly people was associated with vitamin D deficiency and responsible for the impaired balance and gait [7]. Pfeifer et al. reached a similar conclusion that vitamin D deficiency would cause muscle weakness with muscle fiber atrophy, impairment of muscle quality, and increased intramuscular fat [4]. Benetti et al. proved the association of vitamin D and myosteatosis in an experiment where vitamin D reduced-diet induced myosteatosis of skeletal muscles in mice [8].

Lumbar paraspinal muscles are located on each side of the lumbar spine, providing the necessary levels of trunk stability and movement, and assisting in extension and lateral flexion of the spine [9]. Psoas muscle acts as a flexor for the lumbar spine, rotator of the femur, and as a hip flexor [10]. In our patient, progressive walking impairment, the instability of the lower part of the spine and loss of flexion of the femur, and flexion and rotation of the trunk in the lying position correspond to the weakness of the described musculature i.e. the pathological findings of myosteatosis and myopenia by magnetic resonance. The nature of the disease, of course, points to the likelihood that the other skeletal musculature, which was not in the focus of the diagnostic treatment, has been affected by the same process.

The course of the metabolic disorders in our patient corresponds to the findings of Miljković et al. who were the first to propose that myosteatosis, i.e. increase in inter-muscular fat, along with aging, may be an independent predictor associated with the development of type 2 diabetes [11]. Greater intramuscular adipose tissue was associated with higher mortality risk in both men and women [12]. In our patient, long-term metformin therapy could be the cause of the reduced resorption and low serum vitamin B12 level. Clinically, it is difficult to evaluate the presence of funicular myelosis as an additional cause of the gait impairment, especially as we could not find any MR signs of the possible funicular myelopathy.

In the case of our patient, we conclude that long-term vitamin D deficiency, with other accompanying factors as well as aging, is the underlying cause of the paravertebral musculature myosteatosis with the progressive mobility disability and consequential low quality of life.

Ethical Considerations

Compliance with ethical guidelines

All ethical principles were considered in this article. The participants were informed about the purpose of the research.

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Conflict of interest

The authors declare no conflict of interest.

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