CASE REPORT

Substantial Atrophy of the Psoas Muscle as Late Sequela of L2 Osteoporotic Fracture: a Case Report

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ABSTRACT

Osteoporotic vertebral fractures (OVFs) are considered benign and heal after 8-12 weeks. Nevertheless, up to one third of these patients will have persistent back pain, which may be complicated with neurologic deficit or paraplegia. A unique unusual case of delayed onset of neurological complication of an osteoporotic vertebral fracture (OVF) in an elderly patient is reported. The patient presented with paraparesis due to isolated substantial atrophy of the psoas muscle 12 months after the initial fracture. The patient was investigated with imaging and nerve conduction studies. We suggest that psoas muscle atrophy can be determinant clinical sign to diagnose neurological compromise resulting from OVF, even if there is no other clinical indicators of spinal pathology.

Keywords: vertebral fracture, psoas muscle atrophy, neurological complications, burst fracture.

INTRODUCTION

Most osteoporotic vertebral fractures (OVF) are considered benign and heal after 8-12 weeks. Usually, the recommended initial treatment consists of conser-

vative measures such as braces, analgesics, and antiosteoporotic medication (1). However, it is estimated that up to one third of these patients will have persistent back pain, which may be complicated with neurologic deficit or paraplegia. Delayed neurological deficit results from severe kyphotic deformity caused by collapse of

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pseudarthrotic fracture or by the retropulsion of posterior vertebral wall fragments (2). Symptoms and signs of delayed neural tissue compression may become apparent after a few days or up to 1.5 years from the injury (3, 4).

The precise incidence of neurocompression after OVF is not known but it is believed to be low. Fractures involving the middle column (burst fractures) have been suggested to have an increased risk of neurological complications because of retropulsed fragments into the spinal canal (4). There are several published series regarding burst OVF with associated neuronal compromise (5-10). Approximately 90% of OVFs complicated with paraplegia have been classified into burst type, while the average onset of paraplegia being six months after the fracture (9). Presenting symptoms may vary, including spinal and/or radicular pain, sensory disturbance, muscle weakness and bowel / urinary incontinence (2, 4).

Isolated nerve root pathology exhibited as isolated atrophy of the psoas muscle represents a particularly infrequent complication of such fractures. Currently, there is growing interest in the literature exploring the correlation between neuroimpingement disorders and paraspinal muscle morphology. In a recent meta-analysis, Cooley et al examined the relationship between paraspinal muscle morphology and neurocompressive conditions of the lumbar spine, concluding that localized injuries or pathologies resulting in nerve root or central canal stenosis may cause skeletal muscle denervation, muscle fiber atrophy and adipose tissue replacement (11).

In this context, we present an extremely rare case of delayed onset of unilateral hip flexor weakness due to substantial atrophy of psoas muscle, 12 months after an osteoporotic fracture of the L2 vertebral body. □

CASE PRESENTATION

A 76-year-old female suffered an osteoporotic burst fracture of the L2 vertebral body, with compression of the anterior column, involvement of the middle spinal column and retropulsion of bone fragments into the spinal canal, following minor injury. Her past medical history was significant for rheumatoid arthritis under hydroxychloroquine, hypertension and ischemic heart disease under clopidogrel and olmesartan and osteoporosis under denosumab.

FIGURE 1. (A) Lateral plain lumbar spine radiograph obtained at three months after the injury demonstrates an L2 vertebral body fracture (thick arrow) with associated loss of height. (B) Lateral plain lumbar spine radiograph obtained nine months later remains unchanged, displaying no further loss of body height (thick arrow) and no development of kyphosis

Initially, the patient was neurologically intact and was treated conservatively with thoracolumbar orthosis and pain medication. She was followed-up for three months at the outpatient spinal clinic. At the time, the patient was pain free, neurologically intact, while the vertebra had satisfactory sagittal alignment (Figure 1A).

The patient returned to the spinal clinic nine months later (12 months from the initial injury) complaining for gradual development of gait disturbance, which was getting worse during climbing the stairs. Upon neurological examination, profound weakness of the left hip flexors (2/5) was observed. However, no radicular or spinal pain, sensory impairment or bowel and bladder symptoms were present. No weakness was found in the right lower extremity muscle groups. The patellar reflex was normally and symmetrically released, while the Achilles and the adductor reflexes were slightly weaker, mainly on the left. Laboratory evaluation was within normal limits.

Plain lumbar spine radiographs obtained at the time showed no change since the patient's last appointment; no further kyphosis or vertebral body collapse was identified (Figure 1B). Plain radiograph of the hip joints revealed a mild narrowing of bilateral joint space. The patient subsequently underwent magnetic resonance imaging (MRI) of the lumbar spine. The lower end plate of

FIGURE 2. (A) Sagittal T2-weighted image displays the L2 vertebral fracture with resultant loss of body height and retropulsion (thick arrow). (B) Axial T2-weighted image in the L2-L3 vertebral disc level (as indicated by the white line seen in part A of the figure) demonstrates concomitant diffuse disc bulge, bilateral facet joint arthropathy, ligamentum flavum hypertrophy and central spinal canal stenosis. Cross-sectional area measurements of the psoas muscles (green lines bordering the perimeter of the psoas muscles) in the same level, show distinct atrophy of the left psoas muscle (white asterisk – area 2.447 cm²) when compared to the right psoas muscle (white dot – area 5.044 cm²). (C) T1-weighted coronal image also clearly demonstrates evident marked left-sided psoas muscle atrophy (black arrowheads) compared to the right psoas muscle (white arrowheads). (D) Sagittal T2-weighted image demonstrates left-sided foraminal stenosis (white arrow) in the L2-L3 level, which along with the aforementioned findings affects the ipsilateral exiting L2 nerve root, leading to left-sided psoas muscle atrophy.

the L2 vertebral body was collapsed and retro-pulsed and along with the L2-L3 disc bulge was causing central spinal canal stenosis and considerable stenosis of the L2-L3 foramina bilaterally. Additionally, significant atrophy of the left psoas muscle was displayed and further calculated with cross-sectional area (CSA) measurements. CSA measurements were conducted using the RadiAnt DICOM Viewer, Version 2020.2.3 software, by constructing polygons around the outer margins of both psoas muscles, excluding tissue external to muscle fascia. Lumbar spine MRI findings can be analytically seen in Figure 2.

The patient was further evaluated with nerve contacted studies. Needle electromyography (EMG) was unable to detect any activity of the left

psoas muscle, probably due to its significant atrophy. Innervation of multifidus muscle was normal.

Spinal stenosis at the L2-L3 level was considered to be the main cause of psoas denervation and the subsequent atrophy. The patient was offered surgical treatment (posterior decompression and instrumented fusion) anticipating some degree of neurological recovery. She declined surgery and preferred conservative treatment (physiotherapy). At the latest clinical follow-up, the patient remains pain free. The weakness of the left hip flexors shows only a slight improvement but the patient's management of daily activities is satisfactory. □

DISCUSSION

Atrophy and weakness of the psoas muscle have been reported as clinical manifestations of both lumbar spinal stenosis, secondary to neurogenic compromise of the L2–4 spinal roots, as well as hip osteoarthritis due to “disuse” atrophy of the muscle caused by restricted movement of the hip joint (12–14). However, this is the first case describing severe psoas muscle atrophy as late consequence of a vertebral fracture.

Psoas muscle receives multiple levels of innervation corresponding to the vertebral level of attachment from L1 to L4. The innervation of the psoas arises directly from the ventral rami, not from branches of the major nerves intended to the lower extremity. The branches from the L2 and L3 spinal nerves most likely have larger contribution to the innervation of the psoas, since they give longer and wider-diameter branches (15). This pattern is consistent with segmental muscle innervation model and suggestive of the dorsal paraspinal muscles (multifundus innervation pattern alike) (16).

Several conflicting reports have been published on the relationship between spinal pathologies and alterations in paraspinal muscle composition and morphology. Imaging studies in patients with unilateral back pain, using MRI, showed atrophy of multifidus, erector spinae, quadratus lumborum and psoas on the symptomatic side (17, 18).

In the present patient, the cross-sectional area of both psoas muscles was calculated. Significant muscle atrophy of the left psoas muscle was identified. Specifically, the CSA of the left psoas mus-

cle at the L2-L3 level measured 2.447 cm², while that of the right psoas muscle at the same level measured 5.044 cm², meaning that the CSA of the left psoas muscle was about 2.06-fold smaller than that of the right psoas muscle (5.044 cm²/2.447 cm²) (Figure 2B). Lumbar spine MRI additionally revealed lower end plate and disc retropulsion causing severe central spinal canal and bilateral foraminal stenosis in the L2-L3 level, as well as degenerative changes in multiple levels. It could be possible that the reported patient had evidence of degenerative spinal stenosis before sustaining the fracture. Preexisting canal stenosis has been proven to be an independent risk factor for developing spinal cord injury even with minor trauma (19). In some cases, preexisting stenosis is the culprit for the discrepancy between the severity of the neuronal injury and energy of the trauma, since acute spinal cord injury after minor trauma has been reported. It appears that the spinal cord may become more vulnerable against external forces when the degree of neuronal tissue compression exceeds a certain threshold (20).

The degree of psoas muscle atrophy was notably greater than that of the other paraspinal muscles such as the multifidus and erector muscle. In addition, multifidus showed normal activity in the EMG study, indicating intact innervation status. Asymmetrical alterations of the paraspinal muscles morphology have been previously described. Recent evidence failed to support any strong connection between low back pain and psoas muscle fat infiltration and atrophy, while it was found that multifidus muscle was negatively affected in these patients (21). Moreover, any correlation between spinal canal stenosis and altered muscle morphology was inconclusive (11).

Prediction of late neurological complications of OVf is not easy. However, some provoking risk factors have been described: (1) vertebral fracture involving both the anterior and the posterior vertebral body, i.e., burst fracture; (2) increased kyphotic deformity resulting from a wedge compression fracture (kyphotic angle measured at the affected spinal level more than 30 degrees); and (3) intravertebral cleft potentially suggesting ischaemic bone necrosis or pseudarthrosis (Kummell's disease). Pre-existing kyphosis may also facilitate fractured fragments to migrate into the spinal canal (4). Severe osteoporosis, advanced age, obesity and frailty are also deemed important

risk factors for conservative treatment failure in OVf (22).

The total three-month follow-up in the reported case may not have been sufficient, since neurological complications may arise several months later. Therefore, cautious observation for at least six months after an osteoporotic fracture has been previously recommended (4). The majority of elderly patients with OVf frequently attend general practitioner (GP) service for coexisting medical conditions. Increased level of suspicion by GPs for the occurrence of gradual or delayed onset of neurological complications in these patients may lead to prompt and efficient diagnosis and treatment (1).

Neurological deficit associated with OVf or progressive pseudarthrotic kyphosis may successfully respond to indirect postural reduction, cement augmentation and posterior segment transpedicle instrumentation. For burst fractures and rigid kyphotic deformity, corpectomy reconstructed with cages and anterior, or posterior or combined instrumentations may restore neurological function and maintain normal anatomy (2). Available data suggest that even long term denervated muscles exhibit remarkable developmental capability in active myogenesis. Functional electrical stimulation (FES) technique takes advantage of this capability to restore functional mass to a denervated muscle even within two years post-denervation (23). □

CONCLUSIONS

Osteoporosis and subsequent OVf is not an innocent disease with minor morbidity. Osteoporotic vertebral fractures can cause late neurological sequelae with substantial disability and significant deterioration in the quality of life. Clinical presentation with a variety of symptoms is not infrequent. The present report described a unique case in which delayed onset of neurological decline after OVf in an elderly patient appeared as a significant atrophy of psoas muscle and subsequent unilateral hip flexor weakness. Diagnosis and management of this situation is often challenging, with limited clinical data. □

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