

SUBSTANTIAL ATROPHY OF THE PSOAS MUSCLE AS LATE SEQUELA OF L2 OSTEOPOROTIC FRACTURE: A CASE REPORT

Zisis Ntontis¹, Constantinos Chaniotakis¹, Christos Koutserimpas², Nikolaos Achilleas Arkoudis³, Petros Kapsetakis¹, Eirini Pappa¹, Adamantios Alvanos¹, Kalliopi Alpantaki¹

¹ "Venizeleion" General Hospital of Heraklion, ² "251" Hellenic Air Force General Hospital of Athens, ³ "Attikon" University Hospital of Athens

INTRODUCTION-OBJECTIVE

Osteoporotic vertebral fractures are considered benign and heal after 8-12 weeks. Nevertheless, up to one third of patients will have persistent back pain, which may be complicated with neurological deficit or paraplegia. 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.

METHODS AND MATERIALS

A 76-year-old female suffered an osteoporotic burst fracture of the L2 vertebra 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. Initially, the patient was neurologically intact and was treated conservatively with thoracolumbar orthosis and pain medication. During the first 3 months, her rehabilitation was satisfactory. However, 9 months later, she complained of gait disturbance. Neurological examination showed profound weakness of the left hip flexors and lumbar spine MRI detected central spinal canal stenosis and considerable stenosis of the L2-L3 foramina bilaterally due to collapse and retropulsion of the lower end plate of the L2 vertebral body along with the L2-L3 disc bulge. Significant atrophy of the left psoas muscle was displayed, calculated with cross-sectional area measurements, while no activity of the muscle was detected by further needle electromyography.

RESULTS

Spinal stenosis at the L2-L3 level was considered to be the main cause of psoas denervation and the subsequent atrophy. The patient declined surgery and preferred physiotherapy, remaining pain free and managing her daily activities satisfyingly at the latest follow-up.

CONCLUSION

Osteoporotic vertebral fractures can cause late neurological sequelae with substantial disability and significant deterioration in the quality of life. We suggest that psoas muscle atrophy can be determinant clinical sign to diagnose neurological compromise resulting from these fractures, even if there is no other clinical indicators of spinal pathology.

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Figure 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.

Figure 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.



Figure 3 (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.



CONTACT

NTONTIS ZISIS MD, MSc
"Venizeleion" General Hospital of
Heraklion
ntontis1997@gmail.com