Lumbar epidural lipomatosis in a patient with Hodgkin lymphoma

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Abstract

Spinal epidural lipomatosis (SEL) is a pathological condition in which fat tissue is deposed in the spinal canal around the thecal sac in excess, causing neurological symptomatology (7, 9, 12). Fat tissue is present in our body almost everywhere. When the normal quantity is exceeded is created a pathological situation.

We present a 33 old man case with fat tissue excess deposited in lumbar spine canal. The pathological history of our patient includes a nodular form of stage IA Hodgkin lymphoma diagnosed in 2011 for what he went combined radio-chemotherapy. He also has ben diagnosed with grad I obesity. In last month he presented with rapidly progressive symptoms like lower back pain, paraparesis and sphincters dysfunctions.

Thoracic magnetic resonance imaging (MRI) establishes a lumbar lipomatosis located in L3-S5 spinal canal. Pathology results confirm the imagistic diagnostic. Under surgery was performed a median laminectomy with fat tissue excision with good results in patient symptomatology.

Cortisone chronic therapy may lead to SEL. However in our case, Hodgkin lymphoma therapy appeared to be related to cortisone therapy. Neurological symptomatically patients should be treated surgically.

Key words: Lumbar pain; Lipomatosis; Hodgkin disease; Radio-chemotherapy complications.

Introduction

Lumbar lipomatosis represents a pathological situation in which the fat tissue is deposed in excess in spinal canal (7, 8, 9). Fat tissue is present in our body almost everywhere. In spinal canal has a protective roll for spine and nerves (5, 9). When is in excess it creates neurological symptomatology. Lipomatosis is known to be a complication for chronic steroids therapy with neurological symptoms (8, 12). There were several other cases reported in which chronic steroids therapy lead to spinal lipomatosis (1, 10, 11, 15). Another case of spinal lipomatosis was in a patient with Cushing syndrome (1). We know that corticoids may influence the adipose tissue deposits. We present a similar case of a patient that was diagnosed with lumbar lipomatosis after he underwent radiotherapy with high doses of prednisone.

Case presentation

A 33 years old patient presented at our clinic with rapidly progressive symptoms like lower back pain, paraparesis and

sphincters dysfunctions during the last month. From his medical history we know that he suffer an appendicectomy in 2009 that continued with abdominal pain and in 2010 he was diagnosed with inguinal adenopathy. In 2011 he suffer a surgery intervention to remove the nodular formation. The patient was diagnosed with stage IA nodular form of Hodgkin lymphoma. The diagnosis was sustained by pathology result from the lesion. A PET-CT was made to evaluate the lesion extension. The result was negative for any other lymphoma extension after first session of chemotherapy. He underwent combined radio-chemotherapy after surgery. He underwent 4 sessions of chemotherapy with R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, prednisone) (17). The neurological symptoms starts after several months but in the last month they exacerbate rapidly affecting the motor functions and his quality of life. On the general exam we noticed that his body mass index (BMI) was 31 indicating grad I obesity. Neurological exam revealed motor and sensory dysfunction on L3-S5 dermatomes with external popliteal sciatic (EPS) paresis more evident on the left side and perineal paresis. He also had sphincter dysfunction with urinary problems. His clinical diagnosis was compressive myelopathy with cauda equine syndrome. The routinely laboratory and biochemical tests showed normally hematological parameters. The magnetic resonance imaging (MRI) of lumbar spine (Figure 1, Figure 2, Figure 3) whoever revealed high-intensity fat within in the lumbar spinal canal. After preoperative planning our team managed to operate.

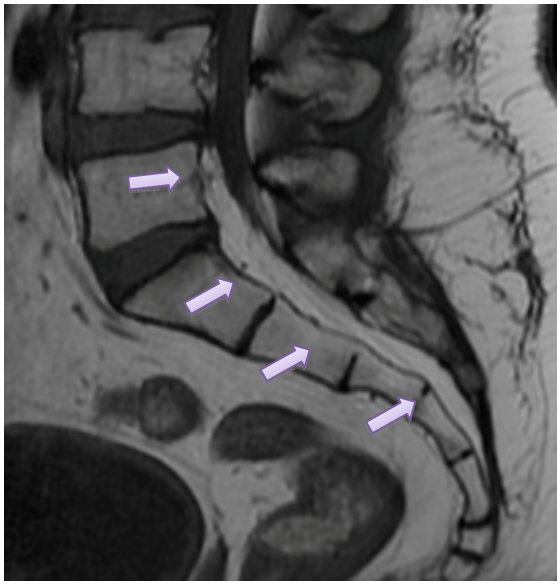

Figure 1 T1 MRI lumbar column sagittal incidence

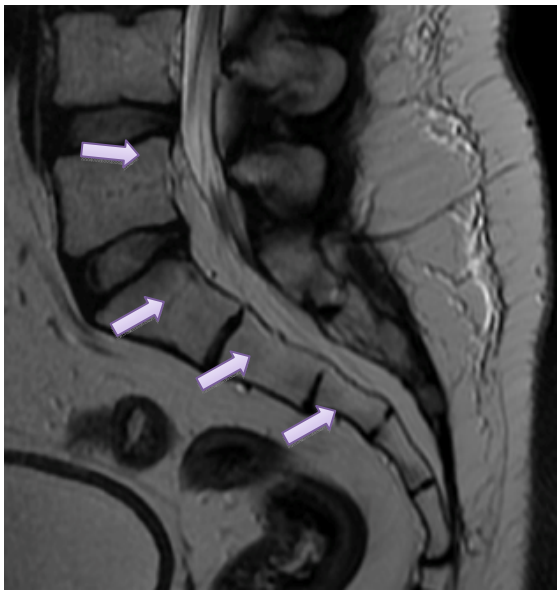

Figure 2 T2 MRI lumbar column sagittal incidence

A medial laminectomy L5and S1was performed with adipose tissue excision and nerve route decompression at the same levels. A tissue sample was taken for pathology. The result was positive for mature adipose tissue without lymphocyte infiltration (Figure 4).

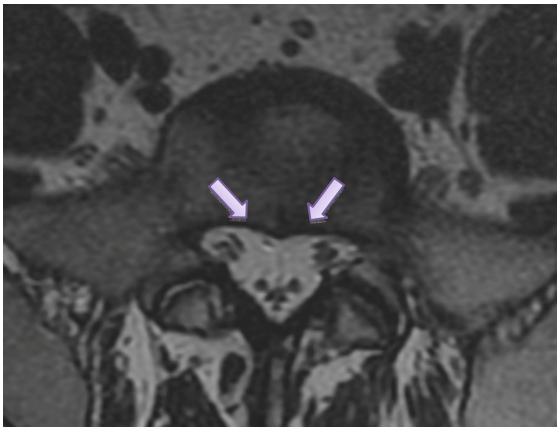

Figure 3 T2 MRI lumbar column axillar incidence

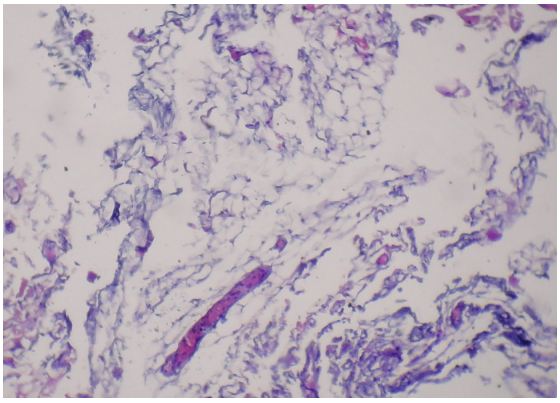

Figure 4 histological examinations demonstrate nodular proliferation of mature fat cells

Symptoms were improved after surgery but the patient also needs kinesiology for full recovery. The diagnosed was lumbosacral lipomatosis, grad I obesity, and stage IA nodular form of Hodgkin lymphoma.

Discussion

Spinal epidural lipomatosis (SEL) is a pathological condition in which fat tissue is deposed in the spinal canal around the thecal sac in excess, causing neurological symptomatology (7, 9, 12). Physiological the adipose tissue in spinal canal has a protective roll for spinal cord (5, 9). The adipose tissue thickness varies with spinal canal levels. In the cervical region the adipose tissue is almost inexistent. In thoracic region the distribution of epidural fat is patchy. In the lumbar region the epidural fat acquire it greatest volume (2, 9). The SEL etiology is unknown (12) but there are some risks factors causing the adipose tissue to gather in excess. Some of this includes Cushing disease, obesity, epidural steroid injection, hypothyroidism, pituitary prolactinoma, renal transplants, rheumatoid arthritis, exogenous corticosteroid therapy, spinal anesthesia and idiopathic (1, 3, 4, 7, 10, 11, 12, 13, 14). Off all this most common is exogenous corticosteroid therapy (11, 12, 16).

Spinal epidural lipomatosis could be found in patients with Cushing syndrome and morbid obesity (1, 13, 14). There were some other cases in the literature of spinal epidural lipomatosis due to corticoid therapy (8, 11, 16).

The best way to evaluate the adipose tissue in the spinal canal is by magnetic resonance imaging (MRI) (2, 7, 8, 9, 10). Borré (2) manage to evaluate the normal thickness of adipose tissue in the lumbar spinal canal and classify it in 4 grades. Grade 0 represents the normal value of adipose tissue in lumbar region. Lumbosacral epidural lipomatosis (LEL) grade III is symptomatic, grade II only in 14.5% symptomatic and no symptoms in grade I (2, 7). Although our patient had a grade II LEL (2), he had neurological symptoms that affect his quality of life. In our patient, signal intensity alterations were observed in lumbosacral level. The MRI reveals massive adipose tissue in lumbosacral with highest thickness of 0.997mm (Fig. 3). He suffers corticoid therapy after Hodgkin lymphoma diagnostic. One of the steps of the stage IA Hodgkin lymphoma protocol includes chemotherapy with R-CHOP (17). He underwent 4 sessions of chemotherapy in 4 moths period. PET-CT investigation did not confirm any dissemination after the first chemotherapy session. He underwent

radiotherapy with interferon (IF) 20Gy/ 10fr 2Gy/ fr on iliac ganglions. His neurological symptoms appear 5 months after chemotherapy with rapidly developing in the last month.

The patient was not on corticoid therapy on the time that symptomology increase and we did not find any connection between radiotherapy and spinal lipomatosis (3, 8, 11).

There are various methods of therapy (1, 11, 13, 14, 15, 16). However is a matter of debate, what treatment is best for the patient. There were good results only with conservative treatment. However the treatment should be determined according to each case (1, 3, 10, 13, 14, 15, 16). In our case the patient has rapidly neurological symptoms evolution that required surgical treatment with nerve routs decompression. The differential diagnostic included spinal tumor, dural lymphoma and angiolipoma, which needed also surgery treatment (4, 6). After the surgery recommendations included loose weight and neurological rehabilitation.

Conclusion

Cortisone chronic therapy may lead to SEL. However in our case, Hodgkin lymphoma therapy led to cortisone complications. We did not find any radiotherapy implication in the literature. However the patient suffer for grad I obesity. Neurological symptomatically patients should be treated surgically.

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