Epidural spinal canal lipomatosis – diagnosis and treatment

Nadtwardówkowa lipomatoza kanału kręgowego – diagnostyka i leczenie

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Epidural lipomatosis of the spinal canal is a rare disorder involving the overgrowth of encapsulated adipose tissue in the epidural space within the spinal canal. It is a rare cause of neurogenic claudication and increasing loss of neurological symptoms.

The location of adipose tissue in the posterior space of the canal is considered a variant of the correct anatomical structure. Only its excess, causing neurological symptoms, is considered a pathological condition [1]. The aetiology of the disease remains unknown. Factors that may participate in the development of the disease are considered to be civilisation diseases such as type 2 diabetes and thyroid dysfunction or iatrogenic factors, e.g. chronic steroid therapy.

The symptoms of this disease include all the symptoms associated with the compression of nerve structures in the spinal canal or vertebral foramen, and thus the following: pain in the lower back, symptoms that intensify when walking and upright spine positions – neurogenic claudication, sciatica, burning and numbness in the leg, in severe cases, paresis and drooping feet, or loss of sphincter control.

The main tool of the diagnostic process in spinal epidural lipomatosis (SEL) is magnetic resonance imaging (MRI). Diagnosis includes thorough medical history, medical examination, and additional tests (RTG, CT). In the medical examination, special attention should be paid to the walking distance, examine the way of moving on the heels and toes, assess pelvic stability, muscular strength of the lower limbs, knee, and Achilles tendon reflexes.

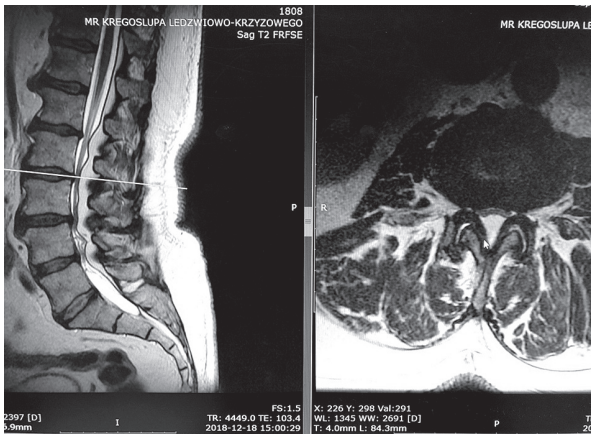

Figure 1. Image of the T2 MR at the L2/L3 intervertebral disc space in the cross-section

Treatment in most cases remains conservative. As a rule, this is symptomatic treatment and, unfortunately, rarely brings constant improvement. Surgical treatment is required in exceptional situations.

We present a case of a 74-year-old patient admitted to the Department of Neurosurgery to treat massive spinal stenosis at levels L2 to the level of the L4/5 intervertebral disc in the course of epidural lipomatosis of the spinal canal.

A few years before diagnosis, the patient reported increasing gait disturbances with neurogenic claudication. Before diagnosis, the patient was treated for 2 years for sciatica by a primary care physician. At the time of admission to the clinic, the patient had neurogenic claudication with a walking distance of up to 20 m, intensification of back pain: VAS 9/10 with a slight reduction of discomfort, with reduction of lordosis during forward leaning (6–7/10 VAS). The physical examination showed slight paresis of the lower limbs (Lovett grade 4) and Lasseque's signs. The subject examination showed hypertension, abnormal fasting glucose (patient treated for diabetes with oral medications, no current or past history of steroids), and overweight, without obesity (BMI 27.5 kg/m^2). No burden of spinal diseases and cancer was found.

The MR study described: "from the Th 12 level to the L5 level in the posterior part of the spinal canal, epidural thickening of the adipose tissue behind the dural sac is visible – the greatest intensity of lesions at L2-L4 levels – thickness of lesions up to 12 mm. These changes narrow the A-P dimension of spinal canal to 4 mm at the L3/L4 level" (Figures 1–3).

The criterion of functional instability was the lateral mobility of up to 2 mm between adjacent stems. A functional picture was taken in flexion and extension in the standing position, and no mobility of the segments was found in the assessment of the lateral radiograph. Due to increasing gait disturbance, the patient underwent surgery. A laminectomy was car-

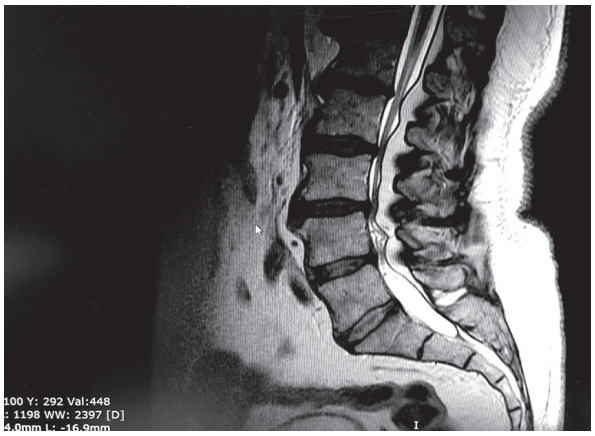

Figure 2. T2 image of the MR examination in the sagittal dimension

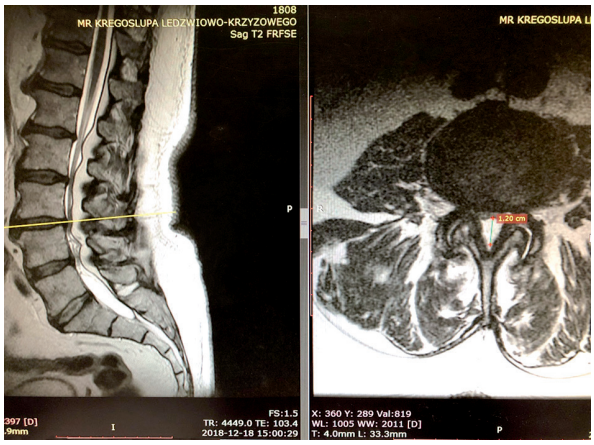

Figure 3. Image of the T2 MR at the L3/L4 intervertebral disc space in the cross-section

ried out at levels L4, L3, and partial L2 saving intervertebral joints (Figure 4). Then fat was removed to reveal a healthy dura mater. There were no perioperative and postoperative complications. The material taken for histopathological examination confirmed the diagnosis.

The patient was rehabilitated in the department and after discharge. Relief of pain was observed after three months. The determined VAS for back pain was 1–2/10. No root symptoms and paresis were found. Walking distance increased to 1000 m. At six months of follow-up imaging (functional X-ray) signs of spinal destabilisation (less than 2 mm movement between the adjacent vertebras) were not observed.

We present the case of a patient with epidural lipomatosis in the lumbar region. The patient was admitted to the Department of Neurological Claudication with a walking distance limited to 20 m. Epidural lipomatosis is a rare disease involving the overgrowth of encapsulated adipose tissue in the epidural space.

Adipose tissue hyperplasia in the course of SEL most often affects the thoracic spine. It is described that the location in the Th segment accounts for 50–60% of the disease. The next most common location is the epidural space of the lumbar spine. Current medical literature confirms existing SEL in the cervical spine [2].

The main tool of diagnostic process in SEL is MRI. Epidural lipomatosis can also be diagnosed by CT. In tomography, adipose tissue is hypodense (about 100 Hounsfield), which allows differentiation from the underlying disease states that cause neurological claudication. However, magnetic resonance imaging is recognised as the primary tool. In this study, non-depleted adipose tissue was hyperintensive in the T1 sequence and hypointensive in the T2 sequence. The diagnostic criterion is the thickness of the fatty tissue layer in the epidural space greater than 7 mm [3].

Based on the ratio of adipose tissue to the meningeal sac in the spinal canal, Borre *et al.* created a lipo-

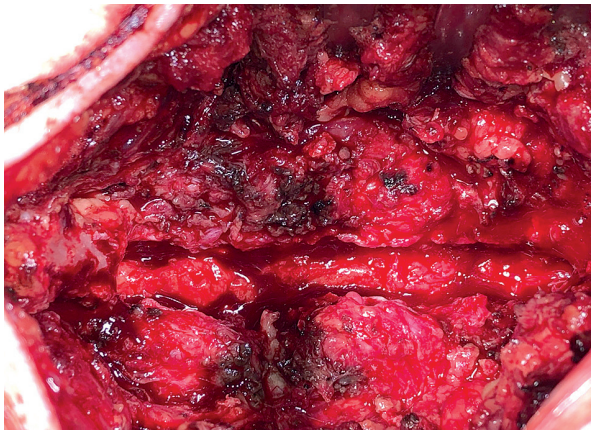

Figure 4. Intraoperative image after laminectomy. Fat tissue visible on the dural sack

matosis classification system at this location. Based on their classification, three stages of the disease are distinguished. In the first degree, the ratio of epidural fat to the diameter of the spinal canal is 41–50%, in the second it is 51–75%, while in the third it is greater than or equal to 75% [4]. We qualified our case to grade 3.

Symptoms of the disease are usually associated with compression of the nerve structures. Patients may complain of progressive back pain, neurological claudication, radicular limb pain, sensory disturbance, and paresis of the lower limbs – cauda equina syndrome. Cases of rapidly increasing paresis up to and including the lower extremities are also described. It should also be remembered that most SEL cases are asymptomatic, while grade 3 on the Borre scale causes symptoms in almost 100% of patients [4].

To date, the pathogenesis of lipomatosis remains unknown and is the subject of numerous scientific studies. There has been increasing talk in the last decade of the existence of risk factors that are considered as potential causes of SEL. These include the follow-

ing: extended steroids (27% to 75% of cases) [3], overweight and obesity with BMI > 27.5 kg/m^2 (79%) [5], Cushing's syndrome, and metabolic and endocrine disorders such as diabetes or hypothyroidism. It is reported that this disease is much more common in men (the ratio of men to women is 3 : 1).

Treatment of lipomatosis in many cases remains conservative. Weight loss is recognised as an effective method of SEL treatment. Surgery is recommended only when neurological symptoms increase, which involves decompression of nerve structures [6]. Interventional treatment requires laminectomy with removal of adipose tissue from the epidural space. In a few cases, reoperation is required due to the recurrence and re-growth of adipose tissue in the spinal canal [3].

Spinal stenosis can occur due to birth defect or acquired defect. Spinal canal lipomatosis remains a rare disease that is also rarely the cause of spinal canal stenosis and neurological claudication. Each case of back pain and increasing gait disturbance should be analysed individually. In the case of coexistence of obesity and chronic steroid therapy [7], lipomatosis should be considered as one of the potential causes of neurological claudication.

Conflict of interest

The authors declare no conflict of interest.

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