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Lumbosacral Plexopathy Developing After Abdominal Surgery: A Case Report and Review of the Literature

Abdominal Cerrahi Girişim Sonrasında Ortaya Çıkan Lumbosakral Pleksopati: Bir Olgu Sunumu ve Literatürün Gözden Geçirilmesi

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ABSTRACT

Lumbosacral plexus lesions are rare as it is protected by the deep muscle layers in the retroperitoneal area and the wall of the pelvis. The lesions appear due to iatrogenic reasons after operations on the neighboring kidneys and internal genital organs. The lower extremities of cases that have undergone gynecological and/or retroperitoneal area operations should be neurologically evaluated in the postoperative period. However, since electrophysiological studies in the early period do not easily distinguish peripheral nerve lesions from plexus lesions, the cases should also be evaluated using the clinical findings.

KEY WORDS: Lumbosacral plexopathy, Complication, Surgery

ÖZ

Lumbosakral pleksus retroperitoneal bölgede ve pelvisin arka duvarında derin kas tabakaları arasında korunması nedeniyle lezyonları enderdir. Bu bölgenin patolojik lezyonları yanı sıra komşuluğunda yer alan böbrek ve iç genital organlara yönelik operasyonlar sonrasında da iatrojenik olarak ortaya çıkmaktadır. Jinekolojik ve/veya retroperitoneal bölgeye yönelik operasyon geçiren olguların postoperatif dönemde alt extremitelerin nörolojik değerlendirilmesi yapılmalıdır. Ancak erken dönemde elektrofizyolojik incelemeler periferik sinir lezyonları ile ayrımı kolaylıkla yapamayacağı için olgular klinik muayeneleri ile birlikte değerlendirilmelidir.

ANAHTAR SÖZCÜKLER: Lumbosakral pleksopati, Komplikasyon, Cerrahi

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INTRODUCTION

The lumbosacral plexus, composed of T12 – S4 roots, is protected among the deep muscle layers in the retroperitoneal area and the rear wall of the pelvis. Lumbosacral plexus lesions are therefore rare clinical occasions. Lumbosacral plexopathy (LSPP) develops as a result of trauma to the nerve roots, with tumors with a mass effect, aneurysm or hematoma, radiotherapy, and during toxic, metabolic, autoimmune, and viral diseases (4, 5, 7, 15, 17, 19). The lesions may also develop as a complication after operations in this area. Complications have been reported to be caused by certain factors, such as peroperative direct surgical damage, hematoma that may develop postoperatively or during retraction and its compression due to pressure, the exposure of nerve roots to ischemia, and scar development in the long-term (1, 3, 4, 11, 13, 14).

CASE REPORT

A 34-year-old male underwent splenectomy and left nephrectomy due to acute abdominal pain findings after a traffic accident 2 months ago. On the postoperative first day, he started to suffer from weakness of the left leg accompanied by pain and numbness, which were not present before the operation. The electrophysiological evaluation (EMG) on the day following the trauma revealed findings of a left peroneal nerve lesion. An operation on the peroneal nerve was planned but the patient presented at outpatient clinic with increasing weakness of the left leg. In the neurological examination, it was determined that his left patella was hypoactive, and his achilles was abolic. Thigh flexion and knee extension were 3/5, while thigh extension, knee flexion, foot dorsal flexion and toe dorsal flexion were 4/5. An atrophy leading to thinning of 3 cm in the left thigh compared to the right thigh was detected. Laboratory findings were normal. X-ray graphs revealed normal vertebral column and pelvic structure. In the magnetic resonance imaging (MRI) of the lumbosacral area, the spinal cord, vertebral column, and intervertebral disc were normal and no mass, disc, or lesion pressing the cord or nerve roots were detected. Furthermore, there were no pathological intensity changes in the paravertebral soft tissues within the range of the image (Figure1). Abdominal ultrasonograhy showed no lesion resembling a mass in the intraabdominal and/or retroperitoneal area(s) except for the traces of splenectomy and left nephrectomy (Figure 2). The neurological findings were not compatible with peroneal nerve lesions, and the control EMG revealed slow transmission on the left, compatible with the L4, L5 and S1 myotomes and partial denervation. No electrophysiological pathology of paraspinal muscles was detected. A diagnosis of LSPP was established in light of the clinical and EMG findings. A conservative treatment consisting of a non-steroid analgesic-anti-inflammatory drug (NSAAI) and B complex vitamin was used, and regression of the patient's pain was observed. However, no changes occurred in his neurological findings.

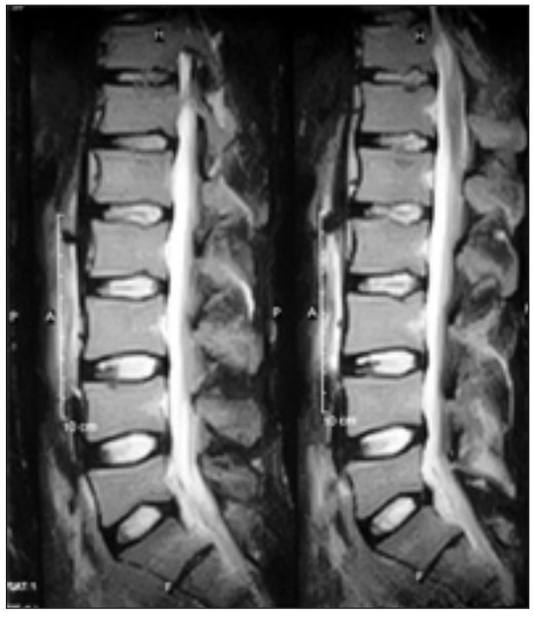

Figure 1: There were no pathological lesions in the MRI of the lumbosacral area

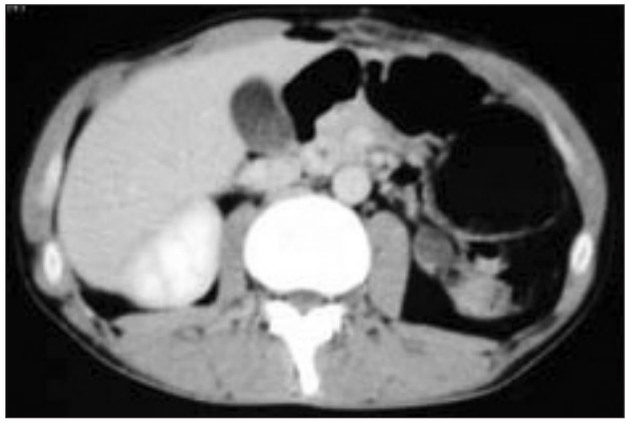

Figure 2: CT revealed no lesions except for the traces of splenectomy and left nephrectomy

DISCUSSION

The lumbar plexus consists of the T12 – L4 spinal nerve roots and the sacral plexus is composed of the L4 – S3 spinal nerve roots. Each nerve root passes under the transverse process located below it, turns lateral to the vertebral corpus and joins the lumbar plexus behind the iliopsoas muscle, and its branches exit between the iliopsoas and quadrotus lumborum muscles. Variations with the L4 nerve root joining sacral plexus have been observed. Sacral spinal nerve roots pass through sacral foramina and join the sacral plexus. The iliohypogastric (T12 – L1), ilioinguinal (L1 – L2), genitofemoral (L1 – L2), lateral femoral cutaneous nerves (L2 – L3); sensorial, femoral (L2 – L4) and obturator nerves (L2 – L4), which stem from the lumbar plexus, have both sensory and motor innervations. Sciatic (L5 – S3) and pudental nerves (S2 – S4) stemming from the sacral plexus have both sensory and motor branches. Furthermore, the sacral plexus provides the autonomic innervations of the bladder, genital organs and rectum. There are also variations in which the S4 root does not join in the sacral plexus. The branches stemming from the aorta and iliac arteries and their anastomoses present a rich vascularization.

In a lumbar plexus lesion, loss of strength in hip flexion, knee extension, and leg adduction occurs. Hypoesthesia compatible with L2-L3-L4 dermatomes is detected in the anterior and internal regions of the hip. In a sacral plexus lesion, on the other hand, loss of strength in hip extension, knee flexion, dorsal and plantar flexions of the foot are detected. There may also be urinary and/or stool incontinence. Sensory failure is associated with L5 and sacral dermatomes.

Lumbosacral plexopathies may be idiopathic as well as superimposed on the chronic course in metabolic diseases like diabetes and autoimmune diseases progressing with vasculitis (2, 5, 16, 17, 19). Plexopathy in subacute processes is caused by clinical conditions such as viral infections of herpes group, bacteria of the borrelia group, toxic agents such as alcohol and heroin, invasive tumors of the retroperitoneal and pelvic areas, radiotherapy for these tumors, and psoas muscle abscess (5, 8, 9, 15, 19). Acute plexopathy may also occur after surgery on the aorta, retroperitoneal area and pelvis and following lumbosacral and pelvic traumas (1, 3, 4, 7, 10 – 14).

Faye et al. retrospectively studied 10 cases receiving a diagnosis of LSPP after a traffic accident and concluded that the lumbar plexus was slightly affected and no associated findings were detected. However, sacral plexus pathologies were more common since they were accompanied by pelvis fractures (7). Kutsy et al. reported 22 cases with LSPP due to pelvic trauma. The probability of LSPP development in all sacral fractures has been reported as 2.03%. This rate is 0.7% for pelvic fractures (12).

LSPP has also been reported to develop as a surgical complication after surgery on anatomically neighboring structures like kidneys and internal genital organs (1, 3, 4, 10, 13).

Alsever reported lumbosacral plexopathy in 6 (0.2%) of 2500 cases who had undergone gynecological operations through the abdominal approach. The author has claimed that prolonged retraction, dissection, and direct neuronal damage during intervention for internal pelvis malignancies particularly result in such complications and has indicated that the femoral nerve as well as the lumbosacral plexus is often affected (7.25 – 11.6 %) (1).

Dhillon et al. have reported the risk of LSPP development as a complication of prolonged retraction and wide dissection in renal operations neighboring the psoas muscle (4). Similarly, Hefty et al. maintained that diabetic cases with renal transplantation suffered from ischemic damage to the lumbosacral plexus more easily and particularly those with transplantation of both kidneys developed vascular failure after the bilateral anastomosis of the iliac arteries and renal arteries (4, 10, 13).

Retraction during surgical intervention exposes neural structures to direct trauma and leads to circulatory disorders and hypoxia, and then to ischemia due to pressure and tension (1, 3, 4, 10, 13). Abscess or hematoma formation in the surgical area or in the psoas muscle has been reported to cause plexopathy through a mass effect (11, 13, 14). In addition to open surgery, hematoma may develop in the retroperitoneal area following minimally invasive surgery. For instance, Özçakar et al. have claimed that retroperitoneal hematoma, developing during the angiography process by femoral artery catheterization, causes lumbosacral plexopathy. Likewise, Klein et al. held retroperitoneal hematoma developing during a plexus block procedure through

the paraspinal region responsible for lumbosacral plexopathy (11, 14).

The most important tool for the diagnosis is electrophysiological evaluation. Biochemical, hematological, and radiological evaluations are essential in the differential diagnosis to focus on the etiology (2, 5, 16).

EMG indicates findings associated with the lumbosacral radicular root and related peripheral nerves, in which a lesion is determined. These include both sensorial and motor conduction latency and partial axonal lesion or denervation potentials. In EMG it is possible to detect varied and/or common findings that are not compatible with single radicular root involvement. The examination in the light of this information presents nonsegmentary widespread findings. The most noteworthy finding for differential diagnosis is that the paraspinal muscles are not involved (3, 12, 17). One can easily be mistaken and interpret EMG findings as a solitary peripheral nerve lesion if there is no partial axonal lesion or denervation potential in the early stages such as 2-3 weeks after the trauma. Thus, at least one more EMG must be performed 3 weeks after the first one. This will increase the chance of detecting partial lesions or denervation potentials and confirm plexopathy. Otherwise peripheral nerve surgery may be performed mistakenly. In our case, the first EMG findings were supportive of a peroneal nerve lesion. The second EMG ruled out this probability and confirmed the diagnosis of LSPP. Likewise, Faye et al. put forward that in LSPP cases, the results of early EMG studies may mimic peripheral nerve lesions; as a result, such cases should be evaluated in combination with clinical findings (7).

Our case did not have any symptoms related to either one of his lower extremities. In the radiological studies, the spinal column and cord were normal. However, the development of the symptoms of our patient following nephrectomy and splenectomy was suggestive of a surgical complication.

Although use of steroids, immunosuppressive chemotherapy and immunoglobulin in LSPP cases with idiopathic and vasculitic progression is recommended, no consensus as to the efficacy of these treatment modalities has been reached (5, 15, 16, 18 – 20). Treatment is usually symptomatic. Spontaneously resolving cases following idiopathic, autoimmune, toxic, and post-radiation factors, in which neural structures are protected, have been reported (2, 6).

Retraction, pressure, and ischemia should be avoided during surgical interventions and the dissection should be performed on the anatomical plane (1, 10,). Lesions such as hematoma and abscess detected in the surgical area during postoperative studies should be evacuated (1, 3, 4, 10, 13, 17). NSAAI, phenytoin, carbamazepine or antidepressant drugs can be used for sensorial complaints such as pain, paresthesia and numbness, or nerve root blockage can be performed for intractable cases.(1, 2, 6, 10, 17, 19, 20). The regeneration process should be followed with successive electromyography, especially in cases with incomplete nerve lesions, and a rehabilitation program should be planned (1, 3, 4, 7, 12, 17).

CONCLUSION

Lower extremity neurological evaluation must be performed following lumbosacral and pelvic traumas. Lumbosacral plexopathy should be considered when pain, paresis or numbness at lower extremities is observed after abdominal, retroperitoneal or pelvic operations. Since EMG, performed in early stages, can lead to misdiagnoses such as peripheral nerve lesions, the results should be correlated with neurological examination findings and a second EMG should be done five weeks after the trauma.

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