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The clinical outcome of lumbosacral plexopathy according to the extent and etiology of the injury

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Abstract

Background & Objective: Although the clinical manifestations and outcomes of lumbosacral plexopathy have been reported, the long-term outcomes are unclear. Therefore, we investigated clinical manifestations and long-term outcomes in patients with lumbosacral plexopathy in terms of the extent and etiology of the injury. *Methods:* We evaluated the clinical and electrodiagnostic data and outcomes of 23 patients with lumbosacral plexopathy in a retrospective longitudinal observational study. The enrolled subjects were divided into groups according to the etiology and extent of their injuries, and the clinical outcomes of each group 1 year after onset were investigated. Outcomes were classified as full recovery, able to walk, unable to walk, and follow-up loss. *Results:* The right lumbosacral plexus was involved in 11 patients, left lumbosacral plexus in 8, and both in 4. Among the 27 lumbosacral plexus lesions (4 patients had bilateral lesions), the upper lumbar plexus was involved in 6 cases, lower lumbosacral plexus in 12, and the entire lumbosacral plexus in 9. Thirteen cases arose from traumatic events, and the rest were non-traumatic. When the clinical outcomes of the groups were compared, non-traumatic cases had higher rates of full recovery than did traumatic cases. Those with lesions in the upper lumbar plexus had a higher rate of full recovery than the other groups.
Conclusions: Non-traumatic etiology and upper lumbar plexus injury were associated with better outcomes. These results will be useful when planning treatment strategies and will increase our understanding of the prognosis for lumbosacral plexopathy.

Keywords: Lumbosacral plexopathy, outcome, prognosis, etiology, recovery.

INTRODUCTION

Lumbosacral plexopathy is a neurological disorder of the lumbosacral plexus, which is subdivided into the upper and lower lumbosacral plexuses. The causes of lumbosacral plexopathy range from pelvic trauma or compression (i.e., a hematoma) to neoplastic or vascular diseases.[1] Colorectal and gynecological tumors, lymphomas, and sarcomas are common causes of lumbosacral plexopathy.[2]

Although several studies have investigated the natural history of lumbosacral plexopathy, the clinical findings and outcomes of lumbosacral plexopathy are unclear due to its heterogeneity and low incidence.[1-4] The long-term outcomes and clinical severity of the condition remain uncertain. We explored the clinical manifestations, electrodiagnostic findings, and long-term clinical outcomes of patients with lumbosacral plexopathy in terms of the extent and etiology of the injury.

METHODS

Study designs and subjects

This retrospective case study reviewed the medical records of 23 patients with lumbosacral plexopathy who presented at the Department of Rehabilitation Medicine of St. Vincent's Hospital between January 2011 and December 2017. All patients were diagnosed with lumbosacral plexopathy after careful clinical and electrophysiological evaluation within 60 days of symptom onset. The diagnosis for inclusion was based on clinical and electrophysiologic criteria, including 1) neurological symptoms (motor weakness and sensory symptoms) involving one or both lower limbs that were not caused by a lesion of the lumbosacral roots, spinal cord, or brain and 2) lumbosacral plexopathy confirmed by electrodiagnostic tests. The exclusion criteria

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were 1) any other peripheral nerve disease, 2) accompanying diseases that may cause neurological symptoms such as neurodegenerative diseases, and 3) pathology diagnosed more than 2 months after onset.[5] The patients were divided into groups according to the etiology and extent of their injuries, and the clinical outcomes of each group at 1 year after onset were investigated. This study was an observational study of clinical recovery and outcomes. The sample sizes of previous studies varied from 22 to 32.[6,7] We decided the sample size was over than twenty-two subjects.

Data acquisition

The demographic, clinical, and electrodiagnostic data were obtained from a review of medical records of all subjects. We use a Medelec Synergy platform (Oxford Instruments, UK) to collect electrophysiological data. All patients underwent both nerve conduction study (NCS) and needle electromyography (EMG). All NCSs were performed on the affected and unaffected sides and compared to eliminate individual variation resulting from anthropometric characteristics or aging. Changes in the electrophysiological parameters are related to the severity of the lesion. Under the best circumstances, the examiner can expect an average side-to-side amplitude difference of 15–20% on NCS. Therefore, when sensory nerve action potentials (SNAP) or compound motor action potentials (CMAP) are absent or the side-to-side amplitude difference exceeds 50%, the lesion is considered abnormal.[8] The CMAP amplitude is useful for determining axon loss. The anatomic extent of the nerve injury in each patient was confirmed using NCS and needle EMG.

Clinical outcomes were classified into four categories: "full recovery" included patients with complete recovery of their neurological symptoms; "able to walk" and "unable to walk" were based on the patient's ability to walk independently; and "lost to follow-up" referred to those patients with whom we lost contact.

RESULTS

Table 1 summarizes the histories and the results of the electrodiagnostic studies of 23 patients. The right lumbosacral plexus was involved in 11 patients, left lumbosacral plexus in 8, and both in 4. The 27 involved lumbosacral plexuses in 23 subjects (4 patients had bilateral lesions) included 6 upper, 12 lower, and 9 entire lumbosacral plexuses. The pathology resulted from trauma in 13 subjects and was non-traumatic in the remaining 10 subjects. For non-traumatic etiology, the proceeding causative condition was divided into three reasons; compressive mass such as benign tumor or hematoma, cancer, or idiopathic. These causative conditions of non-traumatic disease were related to co-morbidity such as cancer. On the other hand, we divided three causative factors for traumatic etiology; fall, motor-cycle traffic accident, pedestrian traffic accident (Table 1).

Regarding the clinical outcomes, of the trauma cases 23.09% had full recovery, 46.15% were able to walk, 15.38% were unable to walk, and 15.38% were lost to follow-up. The corresponding percentages in the non-traumatic cases were 40%, 40%, 20%, and 0%. The non-traumatic cases had a higher rate of full recovery than the traumatic cases had (Figure 1).

Table 1: Patient demographic and clinical characteristics

Characteristics	Number of patients
Age (years)	43.3±16.1
Male/Female	13(56.5) / 10(43.5)
Involved side.(Right/Left/Bilateral)	11(47.8) / 8(34.8) / 4(17.4)
Etiology (Traumatic)	13 (56.5)
Fall	6 (26.1)
Motor-cycle traffic accident	3 (13.0)
Pedestrian traffic accident	4 (17.4)
Etiology (Non-Traumatic)	10 (43.5)
Compressive cause (mass such as benign tumor or hematoma)	7 (30.4)
Post-Radiation therapy (cancer)	1 (4.3)
Idiopathic (not related with other disease)	2 (8.7)

Values are mean ± SD or n (%), unless otherwise indicated.

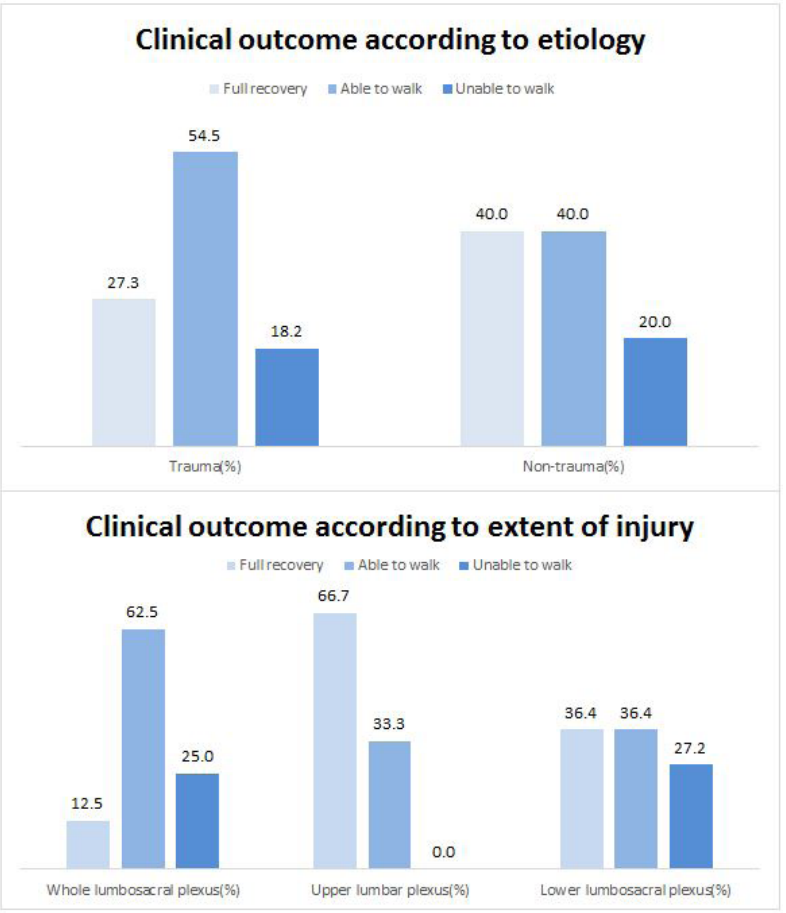

Figure 1. Clinical outcome of lumbosacral plexopathy according to etiology and the extent of the injury.

Considering the extent of the injury, 11.11% of cases with entire lumbosacral plexopathy showed full recovery, 55.56% were able to walk, 22.22% were unable to walk, and 11.11% were lost to follow-up. The corresponding percentages were 66.67%, 33.33%, 0%, and 0%, respectively, in upper lumbar plexopathy cases, and 33.33%, 33.33%, 25.00%, and 8.33% in lower lumbosacral plexopathy cases. Those with upper lumbar plexus lesions had a higher rate of full recovery than did the other groups (Figure 1).

The lumbosacral plexus nerve lesions identified by electrophysiology were heterogeneous. Table 2 shows the most commonly affected nerves (defined as >50%).

Table 2: Frequency of the nerve affectation and percent over 27 lumbosacral plexus

Nerve	Numbers of lesion	% of 27 lumbosacral plexus
LFCN	7	25.9
Peroenal nerve	21	77.8
Tibial nerve	20	74.1
Sural nerve	17	63.0
Sphaneous nerve	11	40.7
Femoral nerve	12	44.4
Obturator nerve	9	33.3
Superior gluteal nerve	14	51.9
Inferior gluteal nerve	12	44.4

Lateral femoral cutaneous nerve; LFCN

DISCUSSION

The long-term outcomes of lumbosacral plexopathy are unclear. Studies have shown a relationship between pelvic bone fracture and lumbosacral plexopathy; lumbosacral plexopathy can also result from compression by a benign tumor.[6,7,9] We found that upper lumbar plexus injury and non-traumatic etiology were associated with more favorable outcomes. These results demonstrate the merit of long-term follow-up of lumbosacral plexopathy according to the extent and etiology of the injury.

The clinical outcomes based on gait were better in the patients with upper lumbar plexus injuries. The leg extensor and hip muscles are important in gait and standing postures[10-12], and most of these muscles are innervated by peripheral nerves branching from the lower lumbosacral plexus. This suggests that the clinical outcomes with regard to gait are better when only the upper lumbar plexus is injured and the function of the lower lumbosacral plexus is preserved. The outcome was better in non-traumatic cases in this study. Generally, traumatic lumbosacral plexopathy has a poor prognosis, as it is typically related to severe trauma resulting in nerve disruption.[4] The risk of lumbosacral plexopathy can be predicted by evaluating pelvic fractures commonly associated with pelvic trauma.[13]

Lumbosacral plexopathies cannot be categorized as easily as can brachial plexopathies in terms of lesion localization. Compared with brachial plexopathies, there is considerably less epidemiological information about lumbosacral plexopathies, such as the etiology, prevalence, incidence, and male/female ratio. However, when examining which nerves were mainly injured, the most commonly affected nerves were the peroneal, tibial, sural, and superior gluteal nerves, all of which derive from the lower lumbosacral plexus. This suggests that cases involving only the upper lumbar plexus are rare compared with cases involving the lower lumbosacral plexus.

Our study has several limitations. First, because the study was retrospective, it was difficult to obtain accurate information on the clinical outcome and on variables that could affect that outcome. Second, the sample size was relatively small. However, criteria were strictly applied to exclude other peripheral neurodegenerative diseases and neurodegenerative diseases, so that better electrodiagnostic data could be collected for selected patients. Third, magnetic resonance imaging (MRI) was not obtained in many cases. Therefore, we did not include data from imaging studies. MRI is a valuable tool for evaluating the musculoskeletal system and soft tissues.[14] In addition, MRI is emerging as a radiological technique for evaluating the brachial and lumbosacral plexus.[14-17] However, MRI for lumbosacral plexopathy is not covered by the National Health Insurance Cooperation, so our study was limited to investigating clinical and electrodiagnostic data.

In conclusion, the subjects with non-traumatic lumbosacral plexopathy had better outcomes than did those with traumatic plexopathy. Furthermore, those with injuries limited to the upper lumbar plexus had better outcomes than those with injuries involving the lower lumbosacral plexus or entire lumbosacral plexus. These results will be useful when planning treatment strategies and will increase our understanding of the prognosis for lumbosacral plexopathy.

DISCLOSURES

The present study protocol was reviewed and approved by the Institutional Review Board of Catholic University College of Medicine (Registry No. VC18RESI0047). Informed consent was waived by the board.

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