Could spinal canal compression be a cause of polyneuropathy?

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Abstract

Causality between spinal cord compression and polyneuropathy is difficult to define, especially under the circumstances that polyneuropathy can have many causes.

Seven patients with spinal cord compression and electrophysiological signs of polyneuropathy were treated surgically on decompression of their spinal canal stenosis in the time from April 2010 to January 2013. Median follow up time was 9 months (2-23 months).

Causes of polyneuropathy were: 1 patient with methotrexate-induced polyneuropathy, 1 endocrine-dysfunction-induced, 2 with diabetic-polyneuropathy, and 3 patients had unknown reasons. The localization of the spinal canal stenosis was also varying: 2 patients suffered of cervical spinal canal stenosis and 5 of lumbar. Decompressive surgery led to pain relieve in all patients initially. Surprisingly, also symptoms of polyneuropathy seemed to regress in all 7 patients for the first 5 months after surgery, and in 5 patients for the time of 9 months after surgery.

There are two points we would like to emphasize in this short report. Since 5/7 patients with polyneuropathy and spinal canal stenosis improved clinically after surgery, surgery has a place in the treatment of such a combined pathology. Since it seems to be a possible causality between polyneuropathy of unknown origin and spinal cord stenosis, decompression of the spinal canal could also be a therapeutic step in a specific kind of polyneuropathy. Which patients could possibly have a spinal canal stenosis induced polyneuropathy remains a subject of further studies.

Introduction

Causality between spinal cord compression and polyneuropathy (PNP) has not been identified yet, especially under the circumstances that polyneuropathy has multiple etiologies. Diagnosing polyneuropathy in a patient with spinal canal compression does not render such cases as non-operable because polyneuropathy could explain most of the symptoms. There are some cases, as shown in the present short report, where spinal canal decompression can lead to a regression of electrophysiological signs of polyneuropathy. The reasons and a clear explanation though, cannot be provided yet and has to be studied in future.

Materials and Methods

Patients

Seven patients were studied retrospectively. All patients suffered of polyneuropathy as proven electrophysiologically, as well as spinal cord stenosis proven on magnetic resonance imaging (MRI). All 7 were treated with surgical decompression of the spine in a 3-year period. Patients were followed up for 9-23 months (mean, 9 months). Table 1 illustrates the characteristics of the studied patients.

Clinical and neurophysiological follow-up

Clinical evaluation of the patients was determined the day before surgery, the day after surgery, before discharge and every 6 months or according to a clinical deterioration.

Every time, neurological examination was performed. All patients underwent conventional motor and sensory nerve conduction studies before surgery and 3 patients underwent an electrophysiological control at 4, 10 and 16 months after surgery.

We would not operate patients with spinal canal stenosis (SCS) when the clinical signs were clearly attributed to the polyneuropathy alone. In the 7 patients we performed surgery on the clinical signs could be an attribute to both entities therefore we decided to perform a decompressive surgery. We did not expect a clinical improvement of their polyneuropathic symptoms and missed the opportunity to perform a postsurgical electropysiological testing systematically in all patients.

Surgical technique

All patients underwent surgical decompression of the spinal canal, in two cervical cases with an anterior approach, in all other cases from posterior approaches.

Results

The median patient age was 71 years (range 47-78 years). Five were males and 2 females.

Case analysis

Six patients presented reduced walking ability over time caused by an atypical *claudicatio intermittens spinalis* (numbness or subjective weakening of legs always preceded a real leg pain). 1 patient presented a typical spinal claudication. For all patients these symptoms had developed over the course of several months, sometimes up to 3 or 4 years. Independently from the cause of peripheral PNP, all the patients referred initial symptoms on their feet and legs (the two cervical cases also on their hands and arms) including pins and needles, a sensation of burning, stabbing pains, more intense at night usually starting from feet and legs, and progressing to hands and arms and sometimes subjective muscle weakness more typical for polyneuropathy than SCS.

MRI demonstrated, in all cases, the *cul de sac* compression and ligamentum flavum hypertrophy. The authors collected 5 cases of lumbar SCS and 2 cases of cervical SCS.

Lumbar cases

Lumbar SCS was radiologically demonstrated at one level (lumbar discs 4-5) in 3 cases, at two levels (lumbar discs 3/4 and 4/5) in 2 cases. Among the patients with a lumbar SCS, in 4 cases bilateral symptoms were reported, while 1 patient reported pain just along the right leg. Surgical decompression was performed through a posterior approach. In all but 3 lumbar cases the decompressive surgery was followed by a fusion procedure (3x minimal invasive TLIF, 1x ACDF, 1x cervical dorsal fixation).

Cervical cases

Between the 2 cases of cervical SCS, 1 patient presented an SCS at 3 levels (cervical discs 3/4, 4/5 and 5/6), 1 at 2 levels (cervical

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Key words: Polyneuropathy; spinal canal stenosis; spinal canal decompression.

Conflict of interest: all authors declare no potential conflict of interest.

Received for publication: 20 October 2015.
Revision received: 16 February 2016.
Accepted for publication: 19 February 2016.

Clinics and Practice 2016; 6:816
doi:10.4081/cp.2016.816

discs 3/4 and 4/5), both with bilateral symptoms. Surgical decompression was performed through an anterior approach.

Outcome

During the follow up period 5 patients were still satisfied of the improvement demonstrated in their polyneuropathic symptoms (and not only the spinal canal stenosis symptoms), 1 patient presented a mild worsening of the symptoms circa 5 months after surgery, and 1 patient presented a severe worsening 18 months after the operation and was not able to walk anymore. Of the 3 patients who underwent postoperative electrophysiological nerve conduction examination, 2 patients were confirmed to be improved 10 and 16 months after surgery respectively; 1 patient presented a worsening of the known PNP. All three patients had no known reason for their polyneuropathy.

Discussion

There are numerous (known and unknown) etiologies responsible for PNP,[1] but to the best of the authors knowledge SCS has never been mentioned among them as coexisting factor or as a cause on its own. Nevertheless, spinal compressive syndromes in the context of hereditary hypertrophic neuropathies[2] and a chronic inflammatory demyelinating polyneuropathy have been described;[3] they were essentially due to nerve entrapment. Also a coexistence of diabetic amyotrophy with lumbar disc herniation and stenosis has been described.[4]

In everyday clinical practice, a polyneuropathy is more suggestive than an SCS if lumbar pain is lacking against an increased leg pain, even more during night and without a precise radicular distribution. Moreover, palsies can be associated to many nerve roots.[1,5-8] On the other hand, symptomatic SCS normally includes neurogenic claudication, with leg pain solved by sitting for few minutes to ease both the leg and often the low back pain. Symptoms usually develop over the course of several years in both diseases.[4,8,9-12] Despite the clinical signs it is difficult to distinguish between these diseases without the help of nerve conduction studies.

The pathophysiology behind a polyneuropathy is still debated and depends on the etiology. In general an ischemic injury, metabolic derangement (*i.e.*, by diabetic neuropathy) or autoimmune mechanisms (*i.e.*, vasculitis) can be a substrate for a polyneuropathy. Every polyneuropathy includes increased oxidative stress yielding advanced glycosylated end products, decreased nitric oxide and impaired endothelial function. Not only nerve cells are likely to be destroyed in an ischemic environment, but also repair mechanisms are defective.[4,5,6,8]

The pathogenesis of SCS is explained as an overlapping of factors, like degenerative changes in the intervertebral disc with a loss of height in the intervertebral disc space, bony transformations in the vertebral bodies (spondylosis) and in the facet joints, disruption of the articular surfaces, and thickening of the joint capsule, of the ligamentum flavum and of the ligamentum longitudinale posterius.[5,8]

In the patients described in this short report, the failure of conservative treatments and the present radiological evidence of SCS led to a decompressive surgical treatment in spite of coexisting clear signs of polyneuropathy.

In the cases reported in the present short report, there was a narrowing of the spinal canal diameter due to a spondyloarthrosis, to hypertrophy of the ligamentum flavum, to a disc bulging, or to a combination of all these factors, which is similar to that of entrapped peripheral nerves. In peripheral nerves a clinical significant improvement of polyneuropathic symptoms after microsurgical decompression has been demonstrated.[13] It should be of no surprise, therefore, that similar observations would be made in entrapped nerves of the cauda equina. The challenge is to establish, clear predictors for operative treatment of spinal canal stenosis in patients with typical signs of polyneuropathy. Even though it is extremely rare that these conditions determine clinical symptoms in the same patient at the same time, these two diseases can influence each other.[7] According to the results of the current study, polyneuropathic patients can highly benefit from surgical treatment of the SCS no matter what the cause of the polyneuropathy is. In some cases the SCS could be in a causal relationship with polyneuropathy and patients can benefit for a long term by spinal canal decompression.

In the cervical spine however, the explanation why patients had an improvement of polyneuropathy after SCS decompression seems to be more complex and should become subject of further analysis.

In literature, guidelines on treatments of these patients are still not stated. Some studies report a poor clinical outcome after discectomy or lumbar decompression by patients with diabetic PNP.[8-11] In these cases, metabolic involvements of a lumbar root can mimic a canal disease.

Pathological mechanism at the basis of this process still needs to be clarified. From previous studies[5,6,14] on patients with diabetic PNP associated with a compressive canal disease, it appears that nerves with metabolic derangement and reduced myelinization are oversensitive to injuries as discal prolapse or canal stenosis.[5,6,14]

Among the population described, postoperative improvement of the PNP suggested that peripheral pain could be emphasized from a radicular compression. In analogy to the double-crush model established by Dellon,[15] we like to postulate a similar model. According to this model, the compression of a nerve root of the cauda equina is one component in a multifactorial process of neuropathy. Surgically decompression can lead to clinical symptom release. According to our opinion, a mechanical impairment of the nerve had to be present as pain responsible cofactor.

Despite a clear initial improvement of the PNP-correlated symptoms in our report, a medium-term release of neuropathic pain is demonstrated in 5 patients, while in the remaining population (2 patients) a worsening was demonstrated over time. As described in literature for the diabetic PNP, clinical fol-

Table 1. Characteristics of the patients (N=7).

	N		N
Sex		Height	
Male	5	Cervical spine	2
Female	2	Lumbal spine	5
Age (years)		Levels included	
Median	71	1 level	3
Min	47	2 levels	3
Max	78	3 levels	1
Follow-up (months)		PNP clinical classification	
Median	9	Symmetric sensorial	4
Min	2	Symmetric senso-motorial	2
Max	23	Aymmetric sensorial	1
Side		PNP etiology	
One side	1	Diabetes	1
Bilateral	6	Endocrine disorders	1
		Chemotherapy	1
		Glucose intolerance	1
		Unknown	3

PNP, polyneuropathy.

low-up of patients with coexisting polyneuropathy and SCS after spinal canal decompression is variable[6,12] and about 20% of the patients relapse. Analgesics like narcotics, antiepileptic and tricyclic antidepressive drugs can help.[6,12,16]

Conclusions

It is possible that a specific kind of polyneuropathy is induced be a spinal canal stenosis. Although, it is not clear, which type of polyneuropathy can be induced or maintained by spinal canal stenosis it is of major interest to investigate further on that subject.

In order to further study the effect of surgery in such a patient collective prospective studies are needed which should not only focus on clinical improvement but more importantly on electrophysiological tests.

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