Could spinal canal compression be a cause of polyneuropathy?

Richard Bostelmann, Samis Zella, Hans Jakob Steiger, Athanasios K. Petridis
Department of Neurosurgery, Medical Faculty, University Hospital Duesseldorf, Germany

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.

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 electrophysiological 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 discs 3/4 and 4/5) and 1 at 1 level (cervical disc 4/5).
discs 3/4 and 4/5), both with bilateral symp-
toms. Surgical decompression was performed
through an anterior approach.

Outcome
During the follow up period 5 patients were
still satisfied of the improvement demonstrat-
ed 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 under-
went postoperative electrophysiological nerve
conduction examination, 2 patients were con-
firmed 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, 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 and a
chronic inflammatory demyelinating polyneu-
ropathy have been described; they were
essentially due to nerve entrapment. Also a
coexistence of diabetic amyotrophy with lum-
bar disc herniation and stenosis has been
described.4

In everyday clinical practice, a polyneuropa-
thy 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.5,6 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.7,8,9,10 Despite
the clinical signs it is difficult to distinguish
between these diseases without the help of
nerve conduction studies.

The pathophysiology behind a polyneuropa-
thy 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 pro-
ducts, decreased nitric oxide and impaired
endothelial function. Not only nerve cells are
likely to be destroyed in an ischemic environ-
ment, but also repair mechanisms are defec-
tive.11,12

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 pos-
terior.5,9

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 cli-
nical significant improvement of polyneuropath-
symptoms after microsurgical decompres-
sion has been demonstrated.13 It should be of
no surprise, therefore, that similar observa-
tions would be made in entrapped nerves of
the cauda equina. The challenge is to estab-
lish, 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 deter-
mine clinical symptoms in the same patient at
the same time, these two diseases can influ-
ce 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 polyneu-
ropathy and patients can benefit for a long
term by spinal canal decompression.
In the cervical spine however, the explana-
tion 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 stud-
ies report a poor clinical outcome after dissec-
tomy or lumbar decompression by patients
with diabetic PNP.4,11 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 previ-
ous studies5,6,14 on patients with diabetic PNP
associated with a compressive canal disease, it
appears that nerves with metabolic derange-
ment and reduced myelination are oversen-
vitive to injuries as discal prolapse or canal
stenosis.5,6,14

Among the population described, postopera-
tive improvement of the PNP suggested that
peripheral pain could be emphasized from a
radicular compression. In analogy to the dou-
ble-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 multi-
factorial process of neuropathy. Surgically
decompression can lead to clinical symptom
release. According to our opinion, a mechanici-
al 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 |                  |                  |
|---------------------|---|-----------------|-----------------|
| Sex                 |   | Male            | Female          |
|                     |   | 5               | 2               |
| Age (years)         |   | Median          | Min             | Max             |
|                     |   | 71              | 47              | 78              |
| Follow-up (months)  |   | Median          | Min             | Max             |
|                     |   | 9               | 2               | 23              |
| Side                |   | One side        | Bilateral       |
|                     |   | 1               | 6               |
| PNP clinical classif.
|                     |   | Symmetric senso-
|                     |   | rial            | Symmetric senso-
|                     |   | 4               | motorial         |
|                     |   | Symmetric sens-
|                     |   | 2               | motorial         |
|                     |   | Symmetric senso-
|                     |   | 1               | rial            |
|                     |   | Diabetes        | Endocrine disor-
|                     |   | 1               | ders             |
|                     |   | Chemotherapy    | 1               |
|                     |   | 1               | Glucose intolera-
|                     |   |                 | 1               |
|                     |   |                 | Unknown          |

N, patients’ number; PNP, polyneuropathy.
low-up of patients with coexisting polyneuropathy and SCS after spinal canal decompression is variable and about 20% of the patients relapse. Analgesics like narcotics, antiepileptic and tricyclic antidepressive drugs can help.

Conclusions

It is possible that a specific kind of polyneuropathy is induced by 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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