Painless motor radiculopathy of the cervical spine: clinical and radiological characteristics and long-term outcomes after operative decompression

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OBJECTIVE Various neurological diseases are known to cause progressive painless paresis of the upper limbs. In this study the authors describe the previously unspecified syndrome of compression-induced painless cervical radiculopathy with predominant motor deficit and muscular atrophy, and highlight the clinical and radiological characteristics and outcomes after surgery for this rare syndrome, along with its neurological differential diagnoses.

METHODS Medical records of 788 patients undergoing surgical decompression due to degenerative cervical spine diseases between 2005 and 2014 were assessed. Among those patients, 31 (3.9%, male to female ratio 4.8 to 1, mean age 60 years) presented with painless compressive cervical motor radiculopathy due to neuroforaminal stenosis without signs of myelopathy; long-term evaluation was available in 23 patients with 49 symptomatic foraminal stenoses. Clinical, imaging, and operative findings as well as the long-term course of paresis and quality of life were analyzed.

RESULTS Presenting symptoms (mean duration 13.3 months) included a defining progressive flaccid radicular paresis (median grade 3/5) without any history of radiating pain (100%) and a concomitant muscular atrophy (78%); 83% of the patients were smokers and 17% patients had diabetes. Imaging revealed a predominantly anterior nerve root compression at the neuroforaminal entrance in 98% of stenoses. Thirty stenoses (11 patients) were initially decompressed via an anterior surgical approach and 19 stenoses (12 patients) via a posterior surgical approach. Overall reoperation rate due to new or recurrent stenoses was 22%, with time to reoperation shorter in smokers (p = 0.033). Independently of the surgical procedure chosen, long-term follow-up (mean 3.9 years) revealed a stable or improved paresis in 87% of the patients (median grade 4/5) and an excellent general performance and quality of life.

CONCLUSIONS Painless cervical motor radiculopathy predominantly occurs due to focal compression of the anterior nerve root at the neuroforaminal entrance. Surgical decompression is effective in stabilizing or improving motor function with a resulting favorable long-term outcome.

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KEYWORDS cervical spine; motor radiculopathy; muscular atrophy; neuroforaminal stenosis; painless paresis

In cases of chronically progressive, painless, flaccid paresis of the upper limbs, a variety of neurological differential diagnoses have to be considered, ranging from motor neuron diseases (e.g., amyotrophic lateral sclerosis [ALS]) or spinal muscular atrophy (SMA) and autoimmune or demyelinating polyneuropathies (e.g., multifocal motor neuropathy [MMN]) or hereditary neuropathy with liability to pressure palsies [HNPP]) to atypical forms of chronic neuralgic shoulder amyotrophy, neuroinflammatory diseases (such as American Lyme neuroborreliosis [ALN]), and entrapment syndromes of the cervical plexus and/or peripheral nerves.
In this study we describe a rare form of cervical nerve root compression syndrome resulting from degenerative neuroforaminal stenosis with predominant radicular motor deficit and muscular atrophy, but completely without any accompanying pain. The aim of the study was a comprehensive characterization of affected patients with special regard to clinical and imaging findings, 10-year prevalence in our clinic, and surgical management, as well as long-term prognosis and quality of life after surgical decompression. The importance of thorough electrophysiological diagnostics and imaging for differentiation from the above-mentioned neurological disorders is highlighted.

Methods

The study was approved by the IRB of Ludwig-Maximilians University in Munich, Germany. A retrospective analysis of 788 patients who underwent anterior cervical discectomy and fusion (ACDF) or posterior cervical foraminotomy (PCF) due to degenerative cervical spine diseases at the Neurosurgical Clinic between January 2005 and December 2014 was performed to identify all adult patients (without exception) with painless radiculopathy and an accompanying motor deficit. Patients were excluded if their surgical indication comprised myelopathy, painful radiculopathy at any time, radiculopathy without motor deficits or atrophy, and/or radiculopathy with compromising sensory changes. To discriminate painless paresis from the above-mentioned noncompressive neurological diseases, a complete evaluation (including clinical, electrophysiological, and blood/CSF examinations) was performed by an experienced neurologist (T.N.W.). After identification of all matching patients, informed consent was obtained and the patients’ clinical characteristics, imaging findings, operative records, complications, and individual outcomes were analyzed.

Clinical Evaluation

Neurological examinations using the Medical Research Council (MRC) grading system for muscle strength were performed in all patients at hospital admission, shortly after surgery (mean 7.5 ± 2.1 days, range 4–12 days), and during the follow-up visits at our outpatient clinic; the mean last outpatient visit at our clinic was 1.4 ± 1.8 years (range 1–8 years) postoperatively.

Reference muscles for a specific nerve root compression were trapezius and supraspinatus/infraspinatus muscle for C-4, deltoid muscle for C-5, biceps and brachioradialis muscle for C-6, triceps muscle for C-7, hypothenar muscles for C-8, and interosseous muscles for C-8 and T-1.

Radiography-based diaphragmatic motility disturbance in combination with signs of isolated, electrophysiologic, chronic, and active denervation in the paravertebral muscles of C-3 were assumed to indicate a radicular deficit as a consequence of an ipsilateral neuroforaminal stenosis of C2–3 in 1 patient without additional motor deficit in the upper limbs.

Postoperative outcome of motor function was based on motor status according to MRC scale grade and was categorized as improved, stable, or worse in comparison with the preoperative status. The patients’ postoperative general clinical outcome was graded using Odom’s criteria.

The last long-term follow-up status was assessed with a standardized telephone interview. If patients reported any change of motor function and/or neurological status compared with the last outpatient visit at our clinic, a confirmative outpatient examination was performed between November 2015 and May 2016 with regard to residual symptoms and motor outcome. This outpatient examination included a questionnaire evaluation using the Patient Satisfaction Index (PSI), WHO/Eastern Cooperative Oncology Group (ECOG) Performance Status Scale (PSS), Karnofsky Performance Scale (KPS), Barthel Index (BI), and 36-Item Short Form Health Survey, version 2 (SF-36v2). The mean long-term follow-up period was 3.9 ± 3.2 years (range 1–10 years).

Electrophysiological Evaluation

Electrophysiological examinations included bilateral neurography of the median and ulnar nerve (distal motor latency, sensory nerve conduction velocity) and electromyography (EMG) of the upper limbs and paravertebral muscle groups.

Imaging Evaluation

The severity and distribution of neuroforaminal stenoses were assessed based on preoperative T2-weighted MR images or, if MRI was not possible due to ferromagnetic internal medical devices or claustrophobia, native or post-myelographic CT images were used. Grading of stenosis was conducted using a modified scoring system based on the MRI grading system by Kim et al., which has been validated for its reliability and high inter- and intraobserver agreement for assessing cervical neuroforaminal stenosis. The modified scoring system included a subdivision between predominantly ventral stenoses and predominantly dorsal/circular neuroforaminal stenoses (Fig. 1). Postoperative imaging was performed in all patients who had not improved in motor function after initial surgical decompression or who suffered from further motor deterioration during the follow-up visits.

Surgical Procedures

For initial decompression (index surgery), an anterior approach (ACDF) was chosen in cases involving a focal anterior compression of the ventral nerve root as a consequence of a mediolateral disk prolapse or an osseous stenosis at the entrance of the neural foramen (especially uncovertebral joint arthrosis). A dorsal approach (PCF) was preferred in cases with a sequester at the neuroforaminal entrance or an osseous long-distance foraminal stenosis reaching up to the extraforaminal space.

For ACDF, patients were positioned supine and a Smith–Robinson approach was used. The intervertebral disc, posterior longitudinal ligament, and osteophytes were resected with careful endplate preparation. Uncoforaminotomy was performed for the neuroforaminal decompression. Titanium or polyetheretherketone cages filled with autologous bone obtained from osteophytic resection were positioned in the intervertebral space under fluoroscopic control.
For PCF, a longitudinal midline incision was performed with exposure of the superior and inferior aspects of the laminae and involved facet joint. Then, a high-speed drill was used to create a keyhole foraminotomy with preservation of at least 50% of the facet joint to maintain cervical stability. The ligamentum flavum was resected and the dorsal aspect of the nerve root was decompressed circumferentially. In cases of sequestrated disc material, it was removed.

Statistical Analysis
All statistical analyses were performed using Sigma-Plot for Windows (version 11.0, Systat Software, Inc.). The patient population was described with summary statistics. Mean values are reported as means ± SDs. To investigate risk factors (sex, age at first clinical signs, duration and type of symptoms, grade of stenosis, age at first operation, and comorbidities) possibly associated with neurological status, reoperation rates, time to reoperation, and outcome parameters, Spearman rank order correlations, Pearson product moment correlations, logistic regression analyses (polytomous variables), and Fisher's exact tests (dichotomous variables) were used for univariate analysis. Statistical significance was determined by a probability value < 0.05.

Results
Clinical Characteristics
Altogether, 788 patients underwent operations due to degenerative diseases of the cervical spine, involving 499 patients initially undergoing ACDF and 289 patients undergoing PCF. Fourteen of 499 patients with initial ACDF and 17 of 289 patients with initial PCF presented with symptoms of a completely painless compressive cervical motor radiculopathy. In the 10-year period between 2005 and 2014, these 31 patients accounted for 3.9% (31/788) of all patients undergoing surgical decompression of cervical nerve roots due to degenerative disease.

Of these 31 patients, 23 (19 men and 4 women, male to female ratio of 4.8 to 1) with 49 total symptomatic neuroforaminal stenoses at index surgery were available for long-term follow-up interviews and questionnaires concerning quality of life and general performance evaluation; 8 patients were lost to follow-up, but there were no significant differences in baseline characteristics between the two groups. The mean age at first surgery was 59.9 ± 10.6 years (range 36–78 years). At the most recent follow-up, a total of 28 operations for decompression of 60 symptomatic stenoses had been performed with a mean of 1.2 ± 0.4 operations per patient (no patient underwent more than two operations).

In addition to a defining painless flaccid paresis without any history of previous or current radiating pain, presenting symptoms included a clinically visible muscular atrophy (78.3%) and concomitant, but negligible, sensory changes (radicular 13.0%, nonradicular/unspecific 26.1%). The mean age at first clinical signs was 58.8 ± 10.6 years (range 35–78 years) and mean duration of symptoms was 13.3 ± 10.2 months (range 1–38 months). Paresis always oc-
curred in a radicular manner according to the localization of neuroforaminal stenosis and was painless throughout for all affected levels in cases of multisegmental manifestation. Additionally, in all patients paresis was chronically progressive with a median MRC grade of 3/5 (range 0/5–4/5) at hospital admission. None of the patients had clinical signs of myelopathy. Nineteen (82.6%) of 23 patients had a history of smoking, 4 patients (17.4%) were suffering from diabetes mellitus, two patients (8.7%) from coronary/pe-

Electrophysiological Findings

Electrophysiological evaluation was available in 16 pa-

tients (69.6%). In all cases, there was no indication of neur-
ographical abnormalities, excluding a relevant contribu-
tion of entrapment syndromes of peripheral nerves. EMG

evaluation, however, consistently showed a reduced ampli-
ditude of the compound muscle action potentials (CMAPs)

Radiological and Surgical Characteristics of

Neuroforaminal Stenoses and Reoperation Rates

The most common locations of neuroforaminal steno-

ses were C4–5 (16 stenoses, 32.7%) and C5–6 (15 steno-

ses, 30.6%), followed by C2–3 (1 stenosis, 2.0%), C3–4 (6

stenoses, 12.2%), C6–7 (5 stenoses, 10.2%), and C7–T1 (6

stenoses, 12.2%). Most neuroforaminal stenoses were multisegmental (n = 41, 83.7%) and unilateral (n = 33, 67.3%).

Concerning morphology and severity, 39 (79.6%) of 49

total symptomatic foraminal stenoses were classified as

grade 2a, 9 stenoses as grade 1a (18.4%), and 1 stenosis

as grade 2 (2.0%); therefore, the vast majority of stenoses

(grade 2a and 1a = 98.0%) led to a focal ventral compression of the anterior nerve root. Significant cord compres-
sion or any signs of myelomalacia were not observed in

our patient population.

Altogether, 19 neuroforaminal stenoses (38.8%) in 12

patients were caused by a laterally sequestered disc prol-

apse at the neuroforaminal entrance alone or in combina-
tion with an osseous long-distance foraminal stenosis ex-
tending to the extraradicular space, and therefore ini-
tially decompressed via PCF. ACDF was performed in

11 patients to completely decompress 30 neuroforaminal

stenoses (61.2%) due to a focal anterior compression of the

ventral nerve root as a consequence of uncovertebral joint

arthrosis with osteophytes alone or in combination with a

mediolateral disc protrusion/prolapse. Examples of neuro-

foraminal stenoses are shown in Fig. 2.

There was no significant difference in clinical char-

acteristics between the ACDF and PCF cohorts. Surgical
details and complications are described in Table 1. Post-

operative complications included new sensory changes that were permanent in 1 patient after ACDF and tran-
sient (< 3 months) in 1 patient each after PCF and ACDF. Transient deterioration of preoperative paresis was noted in 2 patients after PCF and in 1 patient after index ACDF (without new deficits of preoperatively unaffected muscles), whereas there was no case of new permanent motor deficits postoperatively.

During the mean follow-up period of 3.9 years, 3

(25.0%) of 12 patients with index PCF and 2 (18.2%) of

11 patients with index ACDF had to undergo a second op-

eration for decompression of 11 total neuroforaminal

stenoses; the total reoperation rate was 21.7%. The same

level had to be reoperated ipsilaterally in 4.1% of all cases
due to primarily persistent painless paresis as a result of

residual stenosis, whereas in 6.1% reoperation was due
to recurrent painless paresis of initially affected muscle
groups as a result of secondarily relapsing stenosis. Ad-

ditionally, reoperation had to be performed for the same

level contralaterally in 2.0%, the adjacent segments in

6.1%, and the nonadjacent segments in 4.1% of all cases
due to newly emerged stenosis and painless paresis of ini-

tially unaffected muscle groups (no significant differences
between index ACDF or PCF). A focal compression of the

anterior nerve root at the neuroforaminal entrance was

found in the majority of these stenoses (grade 2a and 1a =

90.9%) without significant differences between the same,

adjacent, and nonadjacent segment. Overall mean time be-

tween index and second surgery was 5.9 ± 8.2 years (range

0.1–19.8 years) without significant differences between the

same, adjacent, and nonadjacent segment. Surgical vari-

tables in relation to the time to reoperation. An inverse rela-
tion was found for patients with a positive smoking history (p = 0.033, r = −0.907), indicating that

smokers required a reoperation sooner than patients with-

out a positive smoking history (regardless of the type of

index surgery). No significant correlations were found for

patient sex, age at first clinical signs, duration and type of

symptoms, grade of stenosis, age at first operation, and
other comorbidities (diabetes, obesity, and coronary/pe-
ripheral artery disease).

**Outcome Analysis and Risk Factors Affecting Long-Term Prognosis**

The change of MRC paresis grade between the preop-
erative status and the status at discharge (mean 7.5 ± 2.1
days postoperatively) and last follow-up (mean 3.9 ± 3.2
years postoperatively) is summarized in Table 2. Over-
all, the median MRC grade at the last follow-up was 4/5
(range 0/5–5/5), which therefore had improved by 1 grade
compared with the preoperative grade and the grade at
admission (both median of 3/5). The improvement of mo-
tor function between the latest follow-up examination and
the preoperative status reached significance (p = 0.046).

Compression of the C-6 nerve root appeared to be less
reversible than compression of other cervical nerve roots,
because the change of paresis grade between the preop-
erative status and the status at last follow-up was smaller
for the biceps muscle (median change 0/5) than for supra-
spinatus, infraspinatus, deltoid, triceps, and interosseous
muscles (each median improvement of 1/5).

The majority of patients presented with an im-
provement of general clinical outcome according to Odom’s
criteria, both at admission (69.6%: 1 patient with “excel-
lent,” 3 patients with “good,” and 12 patients with “fair”)
and at the last follow-up evaluation (91.3%: 2 patients with
“excellent,” 5 patients with “good,” and 14 patients with
“fair”).

The questionnaire evaluation revealed a high rate of sat-
isfaction regarding the surgical long-term outcome, with
13 patients (56.5%) giving a PSI score of I and 8 patients
(34.8%) of II (overall median PSI score = I, range I–IV).
Long-term performance in everyday life was excellent in
20 patients (87.0%) with a BI score of 100% indicating no

### Table 1. Surgical details and results of index surgery

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Overall (n = 23)</th>
<th>ACDF (n = 11)</th>
<th>PCF (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age at first surgery ± SD (yrs)</td>
<td>59.9 ± 10.6</td>
<td>60.7 ± 9.3</td>
<td>59.2 ± 12.0</td>
</tr>
<tr>
<td>Mean no. of index levels ± SD</td>
<td>1.8 ± 0.6</td>
<td>2.0 ± 0.6</td>
<td>1.6 ± 0.5</td>
</tr>
<tr>
<td>Surgical complications, no. (%)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Effect on the nerve root</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor deterioration</td>
<td>2 (8.7)</td>
<td>1 (9.1)</td>
<td>1 (8.3)</td>
</tr>
<tr>
<td>Sensory changes</td>
<td>3 (13.0)</td>
<td>2 (18.2)</td>
<td>1 (8.3)</td>
</tr>
<tr>
<td>CSF effusion, infection, extensive bleeding, and/or long-term instability</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Overall complication rate</td>
<td>5 (21.7)</td>
<td>3 (27.3)</td>
<td>2 (16.7)</td>
</tr>
<tr>
<td>Mean length of inpatient stay ± SD (days)</td>
<td>7.5 ± 2.1</td>
<td>6.8 ± 1.5</td>
<td>8.2 ± 2.4</td>
</tr>
<tr>
<td>Second surgery for restenosis during follow-up, no. (%)</td>
<td>5 (21.7)</td>
<td>2 (18.2)</td>
<td>3 (25.0)</td>
</tr>
<tr>
<td>Levels during second surgery, no. (%)</td>
<td>11 (47.8)</td>
<td>4 (36.4)</td>
<td>7 (58.3)</td>
</tr>
<tr>
<td>Index level (ipsilateral)</td>
<td>5/11 (45.5)</td>
<td>3/4 (75.0)</td>
<td>2/7 (28.6)</td>
</tr>
<tr>
<td>Index level (contralateral)</td>
<td>1/11 (9.1)</td>
<td>0/4 (0.0)</td>
<td>1/7 (14.3)</td>
</tr>
<tr>
<td>Adjacent level</td>
<td>3/11 (27.3)</td>
<td>1/4 (25.0)</td>
<td>2/7 (28.6)</td>
</tr>
<tr>
<td>Distant level</td>
<td>2/11 (18.2)</td>
<td>0/4 (0.0)</td>
<td>2/7 (28.6)</td>
</tr>
<tr>
<td>Average time to reoperation ± SD (mos)</td>
<td>71.1 ± 98.4</td>
<td>16.1 ± 2.1</td>
<td>108.8 ± 119.6</td>
</tr>
</tbody>
</table>

### Table 2. Change of MRC paresis grade during follow-up

<table>
<thead>
<tr>
<th>Change in MRC Grade</th>
<th>Preop vs Short-Term MRC Grade*</th>
<th>Preop vs Long-Term MRC Grade†</th>
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<tbody>
<tr>
<td></td>
<td>Overall (n = 23)</td>
<td>ACDF (n = 11)</td>
</tr>
<tr>
<td>Improved</td>
<td></td>
<td></td>
</tr>
<tr>
<td>+2</td>
<td>2 (8.7)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>+1</td>
<td>7 (30.4)</td>
<td>4 (36.4)</td>
</tr>
<tr>
<td>Stable</td>
<td>11 (47.8)</td>
<td>6 (54.5)</td>
</tr>
<tr>
<td>Deteriorated</td>
<td></td>
<td></td>
</tr>
<tr>
<td>−1</td>
<td>2 (8.7)</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>−2</td>
<td>1 (4.3)</td>
<td>1 (9.1)</td>
</tr>
</tbody>
</table>

Data given as number of patients (%). ACDF and PCF represent index surgery.
* Short term = mean 7.5 ± 2.1 days postoperatively.
† Long term = mean 3.9 ± 3.2 years postoperatively.
disability (overall median BI score = 100, range 85–100). Furthermore, 20 patients (87.0%) showed a WHO/ECOG PSS score ≤ 1 (overall median WHO/ECOG PSS score = 0, range 0–2) and all patients (100.0%) a KPS score ≥ 80 (overall median KPS score = 90, range 80–100), indicating the ability to carry on normal activity and to work with no need of special care. According to the SF-36v2, the mean physical health composite score in the long-term questionnaire evaluation was 49.9 ± 14.1 (range 21.5–68.8) and the mean mental health composite score was 40.8 ± 5.8 (range 31.8–47.8), indicating only a slightly worse physical or mental health than the average population. There was no significant difference in the above-mentioned data with regard to the performed type of index surgery.

Clinical and imaging factors (such as sex, age at first clinical signs, duration and type of symptoms, grade of stenosis, age at first operation, and comorbidities) were not significantly associated with long-term motor outcome, general performance, or quality of life in univariate analysis. However, the long-term motor outcome was significantly worse in patients who suffered from a transient deterioration of muscle strength immediately after index surgery (p = 0.034).

**Discussion**

In this paper we describe a rare clinical manifestation pattern of a cervical radiculopathy leading to painless, chronically progressive, flaccid paresis and concomitant muscular atrophy of the upper limbs as a consequence of compressive anterior neuroforaminal stenosis. In our series, approximately 4% of all patients with an indication for surgical decompression of the cervical nerve roots due to degenerative diseases were found to have this rare form of radiculopathy.

The vast majority of affected patients were male and suffered from diseases with effects on the vasculature, such as smoking or diabetes. In detail, both the proportion of smokers (82.6%) and diabetic patients (17.4%) was clearly higher than the proportion of smokers (29.7%) and diabetic patients (7.2%) in the total German population, underlining the potential disproportionately high influence of a disturbed microcirculation for this rare form of radiculopathy. The proportion of male individuals (82.6%; male to female ratio of 4.8 to 1) was significantly higher (p = 0.026) than in our overall study population (59.6%; male to female ratio of 1.5 to 1), which might be associated with the fact that smokers are predominantly male. According to our findings, smoking and diabetes have been previously identified as important risk factors for cervical radiculopathy, perioperative complication rates, and worse postoperative outcome.

In this cohort of painless paresis, we found C-5 and C-6 to be the most frequently affected nerve roots. Anatomically, the focal compression at the entrance of the neural foramen was most often attributable to uncovertebral joint arthrosis with or without soft-tissue pathologies and led to a predominant isolated lesion of the motor ventral nerve root. Because this focal anteriorly located pathology does not cause relevant compression of the sensory and nociceptive dorsal nerve root or spinal ganglion, isolated motor deficits can occur without concomitant pain or sensory deficits. Because this isolated ventral nerve root compression was overwhelmingly frequently identified in 98.0% of all cases and was strongly associated with the clinical syndrome of painless severe paresis of the respective nerve root, it can therefore be classified as a highly characteristic imaging pattern, making MRI and/or CT the diagnostic tool of choice for this rare clinical syndrome.

Surgical decompression of the foraminal stenosis—by ACDF or PCF—was shown to be very effective, even in cases of previously established muscle atrophy: 87% of all patients with a preoperatively progressive disease experienced a sustained stable or improved motor function postoperatively. This surgery-induced stoppage of further motor deterioration is essential for patients with compressive cervical motor radiculopathy who have preoperatively experienced chronically progressive paresis and its associated impairments of upper limb function and basic life skills. From this point of view, we assess the postoperative general clinical outcome of “fair” (or better) according to Odom’s criteria in 91.3% of the patients as a relevant success for the patients, which is also reflected by the reported high subjective satisfaction regarding treatment outcome. Moreover, an excellent general performance could be achieved in long-term follow-up evaluations after surgery. As a consequence, in cases of well-matched characteristic clinical syndrome and highly pathognomonic imaging, decompressive surgery offers an effective treatment, especially given the preoperative chronically progressive paresis and atrophy of functionally important muscles.

There are limitations in our study. First, due to its retrospective design, inclusion of patients without accompanying radicular pain depends on the accuracy of the documented patient’s history. We reconfirmed this detail in all included patients, but we cannot exclude that the number of patients affected by the described syndrome is actually higher as a consequence of documentation errors or false attribution of limb pain from other sources, such as as radicular. Second, only 23 (74.2%) of 31 patients were available for long-term follow-up, which might have influenced the results of postoperative outcome analysis and quality-of-life questionnaires.

Due to a variety of differential diagnoses, confirming the correct diagnosis in case of progressive painless paresis of the upper limbs can be challenging. Table 3 summarizes key features of painless compression-induced cervical motor radiculopathy and its clinically most challenging differential diagnoses. In case of clinical uncertainty, electrophysiology is the most important tool apart from the characteristic spinal MRI/CT changes that are typically found in compressive cervical motor radiculopathy. For example, both adult-onset spinal muscular atrophy type IV and ALS are neurodegenerative diseases with deletion of neurons that control voluntary muscles, leading electrophysiologically to a generalized lesion of the lower motor neuron without segmental distribution patterns. Especially in ALS, the lesion is usually extensive and can be found even in early stages in the form of a reduction of the CMAP amplitude as well as abnormal specific (e.g., unstable or large motor unit action potentials of increased duration and proportion of polyphasic components) and

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<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Cervical Motor Radiculopathy Due to Foraminal Stenosis</th>
<th>ALS</th>
<th>Spinal Muscular Atrophy Type IV</th>
<th>MMN</th>
<th>HNPP</th>
<th>Atypical Forms of Chronic Neuralgic Amyotrophy</th>
<th>ALN</th>
<th>Entrapment Syndromes of the Cervical Plexus/Peripheral Nerves</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Characteristics</strong></td>
<td>Early stages: variable lower &gt; upper motor neuron effects; later stages: upper (e.g., paresis, hyperreflexia, spasticity) &amp; lower (e.g., atrophy, hyporeflexia, flaccid paresis) motor neuron effects</td>
<td>Focal asymmetric flaccid muscle weakness and paralysis w/o pain, almost always proximal limb predominant</td>
<td>Focal asymmetric flaccid distal limb paresis and atrophy (upper &gt; lower) w/o pain</td>
<td>Recurrent painless attacks of focal numbness, flaccid muscle weakness and atrophy, often preceded by minor compression on nerve</td>
<td>Up to 15% of cases: painless flaccid paresis and atrophy of muscles around the shoulder girdle</td>
<td>Motor-dominated radicular symptoms may be present and mostly painless; typically subacute meningitis without or with associated facial palsy and erythema migrans rash</td>
<td>Progressive flaccid paresis and atrophy of muscle groups of circumscribed upper limbs according to the affected peripheral nerve, mostly in combination with sensory changes and pain</td>
<td></td>
</tr>
<tr>
<td><strong>Pathophysiology</strong></td>
<td>Degenerative neuroforaminal stenosis</td>
<td>Neurodegenerative disease of upper and lower motor neurons</td>
<td>Neurodegenerative disease of lower motor neurons due to SMN gene mutation</td>
<td>Immune-mediated</td>
<td>Demyelinating neuropathy due to PMP22 gene deletion</td>
<td>Unknown (immune-mediated?)</td>
<td>Neuroinflammation due to Borrelia burgdorferi infection</td>
<td>Chronic peripheral nerve entrapment/compression</td>
</tr>
<tr>
<td><strong>Electrophysiology</strong></td>
<td>EMG: reduced CMAP, patterns of chronic and active denervation in segmentally affected peripheral and paravertebral muscles</td>
<td>Generalized lesion of the lower motor neuron w/o segmental distribution patterns; EMG: reduced CMAP, neurogenic changes</td>
<td>Generalized and nonsegmental lesion of the lower motoneuron; EMG: neurogenic changes with reduced CMAP</td>
<td>Conduction block of motor axons, normal sensory nerve conduction</td>
<td>Increase in distal motor latencies (especially of median and peroneal nerve); decrease in motor and sensory nerve conduction</td>
<td>Unspecific, sometimes signs of denervation in EMG</td>
<td>Unspecific, sometimes signs of axonal polyradiculopathy</td>
<td>Distinct patterns of neurographical impairments in motor/sensory nerve conduction velocity + proximal/distal motor latency according to the different entrapment syndromes</td>
</tr>
<tr>
<td><strong>Imaging</strong></td>
<td>Focal compression of the anterior nerve root at the neuroforamens, mostly due to uncarthrosis ± soft-tissue pathologies</td>
<td>Hyperintensity of the corticospinal tract on T2-weighted MRI, cortical thinning &amp; gray matter volume loss especially of the precentral gyri</td>
<td>Nonspecific loss of muscle volume in upper and lower limb imaging</td>
<td>Increased signal intensity in the T2-weighted MRI of the brachial plexus in as many as 50% of patients</td>
<td>Sometimes nerve hypertrophy at entrapment sites in peripheral neuro-ultra-sonography</td>
<td>Sometimes loss of muscle bulk and diffuse areas of increased signal intensity within the muscle in T1-weighted spin-echo MRI</td>
<td>Unspecific, sometimes multilocal white matter lesions especially in periventricular location</td>
<td>Often signs of nerve entrapment in peripheral neuro-ultrasoundography or MRI; radiography for cervical rib</td>
</tr>
<tr>
<td><strong>First-line treatment</strong></td>
<td>Surgical decompression</td>
<td>Antiglutamatergic drugs (riluzole), symptomatic</td>
<td>Symptomatic</td>
<td>Intravenous immunoglobulin</td>
<td>Symptomatic, avoiding activities with risk for pressure palsies</td>
<td>Corticosteroids, physical therapy</td>
<td>Antibiotics, symptomatic</td>
<td>Conservative therapy, surgical decompression</td>
</tr>
<tr>
<td><strong>Prognosis</strong></td>
<td>Stable/improved in &gt;90% of the patients</td>
<td>Mostly slowly progressive, normal life span</td>
<td>Mostly slowly progressive</td>
<td>Mostly slowly progressive, marked recovery in days to weeks</td>
<td>Usually good with complete recovery</td>
<td>Usually complete recovery after antibiotic therapy</td>
<td>Usually good</td>
<td></td>
</tr>
</tbody>
</table>
unspecific (e.g., denervation activity such as spontaneous fibrillation potentials, positive sharp-waves, and fasciculation potentials) EMG findings. In contrast, compressive cervical motor radiculopathy leads to a strictly segmental lesion of the affected nerve root that can be seen in both the corresponding peripheral as well as paravertebral muscle groups in the form of a reduced CMAP amplitude and patterns of chronic and active denervation. In contrast, MMN and HNPP as autoimmune demyelinating polyneuropathies lead to changes of peripheral nerve conduction velocity: while the presence of a conduction block that selectively affects motor axons with sparing of sensory axons is strongly indicative for MMN, an increase of distal motor latencies in combination with a decrease of motor and sensory nerve conduction velocities can only be seen in HNPP. In both neuralgic shoulder amyotrophy as an idiopathic brachial plexus neuropathy, and ALN as a neuroinflammatory manifestation of the systemic infection with the spirochaete Borrelia burgdorferi sensu stricto, electrophysiology typically shows only unspecific changes. The clinical suspicion based on detailed patient questioning and neurological examination is mostly diagnostically more significant than electrophysiological examinations. Finally, entrapment syndromes of the cervical plexus (such as thoracic outlet syndrome) and peripheral nerves (such as carpal tunnel syndrome, supinator tunnel syndrome, or Guyon’s canal syndrome) show characteristic patterns of neurographical impairments of motor and sensory nerve conduction velocity including proximal and distal motor latency, which are highly discriminative from compression-induced cervical motor radiculopathy.

Conclusions

Painless compressive cervical motor radiculopathy is a rare but severe clinical manifestation pattern of a cervical neuroforaminal stenosis. It predominantly occurs in males with focal compression of the anterior nerve root and is associated with smoking and diabetes mellitus. A safe and effective treatment can be achieved with surgical decompression, as patients show a significant improvement of motor function and a convincing overall general performance in long-term evaluation.

References

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**Disclosures**
The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

**Author Contributions**
Conception and design: Siller, Witt, Zausinger. Acquisition of data: Siller, Kasem, Witt. Analysis and interpretation of data: Siller, Witt. Drafting the article: Siller. Critically revising the article: all authors. Approved the final version of the manuscript on behalf of all authors: Siller. Statistical analysis: Siller. Administrative/technical/material support: Siller. Study supervision: Siller, Zausinger.

**Supplemental Information**
Previous Presentations
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