**ARE SUBJECTS WITH SPONDYLOTIC CERVICAL CORD
ENCROACHMENT AT INCREASED RISK OF CERVICAL SPINAL CORD
INJURY AFTER MINOR TRAUMA?**

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Introduction

Degenerative changes in the cervical spine are part of the normal aging process and are almost omnipresent in older people [1]. They may lead to the development of clinical symptoms in some individuals if the discs and/or osteophytes impinge on neural structures such as the nerve root or spinal cord. Spondylotic cervical cord encroachment (SCCE) detected by imaging methods, mostly MRI, is a prerequisite for the clinical diagnosis of cervical spondylotic myelopathy (CSM). However, SCCE can also be asymptomatic with regard to myelopathy [1, 2].

CSM develops insidiously, but it has been reported to occur after trauma [3-6]. Some authors have suggested that individuals who have asymptomatic spondylotic cervical cord encroachment (ASCCE) on the cervical spinal cord are at increased risk of acute myelopathy if they experience minor trauma [7-8]. This has led some surgeons to recommend decompression surgery for the purpose of preventing this trauma-induced myelopathy in individuals presumed susceptible [9-10] and this topic became a matter of controversy [11].

The aim of our study was retrospectively to analyse all traumas of the cervical spine in our cohort of 199 individuals with ASCCE followed for the median period of 44 months, and investigate any relationship to clinical manifestation of myelopathy and type of cervical spinal cord compression.

Methods

Group

The study sample consisted of a cohort of 199 subjects (94 women and 105 men; median age 51 years, range 28–82 years) recruited consecutively between January 1993 and January 2005 and followed up to July 2007, who completed at least a 2-year follow-up. MRI examination of the cervical spine and spinal cord was performed in all patients who exhibited clinical signs and symptoms of cervical radiculopathy or moderate to severe chronic or intermittent axial cervical pain. All included patients had to meet the following inclusion criteria:

- MR signs of spondylogenic or discogenic compression of the cervical spinal cord with or without concomitant change in signal intensity from the cervical cord on T2/T1 images (see reference 12 for further details).
- Axial pain or clinical signs and/or symptoms of radiculopathy that could be controlled by conservative treatment.
- Absence of any current clinical signs and symptoms that could be possibly attributed to cervical cord involvement.

During the follow-up period (median 44 months) progression in symptomatic myelopathy was found in 45 patients (22.6%); the 25th percentile time to clinically-manifested myelopathy was 48.4 months.

Further details of the clinical, imaging and electrophysiological evaluation and algorithm of the study that centred upon risk factors predicting clinical manifestation of myelopathy have been published elsewhere [12]. The presence of any trauma, both before inclusion of a patient into the

study and during the follow-up period, was detected with a questionnaire administered at the end of the follow-up period, and focused on:

- Date and mechanism of trauma
- Short term sequelae (incl. loss of consciousness, fracture, weakness of extremities, disturbance of sensitivity or sphincters, therapy incl. surgery)
- Long-term sequelae (incl. disturbances of gait, loss of self-support, permanent disability, chronic pain.

Further, the patient case histories and a database containing complete relevant data on all patients included into the study were retrospectively analysed with respect to the occurrence and characteristics of any trauma. All traumatic episodes were subsequently classified as possibly relevant or irrelevant to cervical spinal cord injury. The following traumatic episodes were classified as possibly relevant:

Traumas of the head, spine, trunk and shoulder region, if at least one of the following characteristics was present:

- Any fall
- High-energy accident*
- Unconsciousness
- Fracture
- Necessity for surgical treatment
- Probable transient neurological deficit
- Permanent disability

*According to Advanced Trauma Life Support principles, a high-energy (vehicle) accident is defined as initial speed >64 km/h, major car-deformity, intrusion into passenger compartment >30 cm, extrication time from vehicle >20 min, falls >6 m, roll over, auto-pedestrian accidents, or motorcycle crash >32 km/h or with separation of rider and bike [13].

Traumatic episodes before the beginning of the follow-up period were correlated with the type of spinal cord compression (discogenic versus osteophytic) classified from MRI and CT scans, while traumatic episodes during the follow-up period were correlated with clinical manifestation of spondylotic cervical myelopathy.

Statistics: Univariate logistic regression models predicting probability of CSM were used for trauma as a potential risk factor, and the odds ratio was estimated with 95% confidence limits.

Results

During the follow-up period, 14 relevant traumatic events were recorded. None of them was of serious degree with fracture of the cervical spine and/or followed by immediate neurological deficit after the trauma. In terms of mechanism, there were 6 falls (all from height <6 meters), 4 sports injuries, 3 traffic accidents and one occupational injury. Among 45 patients (22.6%) who developed symptomatic myelopathy during the follow-up period, potentially relevant traumatic episodes were found in 3 patients: in two cases myelopathy became symptomatic 6 months before trauma and in one case manifestation developed 4 years after the trauma. There was no statistically significant association between traumatic events and subsequent development of symptomatic

myelopathy (odds ratio [OR] 0.935; 95% confidence interval [CI]: 0.247 – 3.535; $p = 0.921$).

We found 56 potentially relevant traumatic episodes before the beginning of the follow-up with possible relationships to the cervical disc herniation. In terms of mechanisms, 21 were falls (2 from height >6 meters), 12 sports injuries, 11 traffic accidents, 11 occupational injuries and 1 injury caused by a falling object.

The type of cervical spinal cord compression (herniation, osteophytes or both) was evaluated and classified as discogenic (due to disc herniation) in 50 patients (25.1%), osseous (due to osteophytes) in 67 patients (33.7%) and mixed (herniation + osteophytes) in 82 patients (41.2%) [12]. We found no statistical association between traumatic events and discogenic (OR: 1.281; 95% CI: 0.636 – 2.582; $p = 0.484$) or mixed type of compression (OR 1.767; 95% CI 0.879 – 3.549, $p = 0.106$).

Discussion

In a cohort of 199 patients with ASCCE who progressed into symptomatic myelopathy we detected only 3 traumas of the cervical spine in those who became symptomatic, but without direct chronological relationship. Furthermore, traumatic events before detection of cervical spinal cord encroachment showed no impact on the type of spinal cord compression.

Increased risk of cervical spinal cord injury in patients with ASCCE presumed sufficient to justify preventive surgical decompression is based on case reports, case series or retrospective cross-sectional studies [7-10]. Moreover, risks involved in surgery to the cervical spine in asymptomatic spinal cord encroachment have not been reported [11].

Some authors have reported increased risk of spinal cord injury in patients with cervical spondylosis. Regenbogen, et al [5], in a group 88 retrospectively analysed patients over the age of 40 with spinal cord injury resulting from trauma, found 25 cases with no bony or ligamentous injury but with signs of severe spondylosis as compared to 35 younger spinal cord injury patients (below 37 years of age) who showed severe bony or ligamentous injury in all but one case. Kang et al. [14], in a retrospective analysis of 288 spinal injury patients, found significantly lower sagittal diameter of the spinal canal and Pavlov canal/body ratio at both compressed and uncompressed levels in patients with complete and incomplete spinal injury compared to those with no nerve or spinal cord injury. Yoo, et al [15], among a series of 200 cases with cervical spondylosis or ossification of posterior longitudinal ligament, and symptomatic myelopathy, detected retrospectively minor trauma of the cervical spine in 63 cases, and deterioration of preexisting myelopathy or development of new myelopathy in 31 of them. Most of these cases (25) had narrow spinal canal (diameter < 10 mm).

Murphy et al. [11] addressed the question of whether patients with ASCCE are at increased risk of spinal cord injury after minor trauma and may thus warrant early decompression. They found none of the case-control or prospective cohort studies that are essential to drawing the firm conclusion that risk of spinal cord injury from minor trauma is increased in ASCCE.

Lauryssen et al. [10], combining data from several databases, estimated the "worst case scenario" risk of myelopathy in the ASCCE population at 1:2100. This low risk estimate is largely consistent with our own findings.

Furthermore, Murphy et al. found no relevant study reporting the outcome of surgery in asymptomatic patients with cervical cord spondylotic encroachment [11]. The reported frequency of serious complications or mortality in surgical series of patients with symptomatic CSM is generally well above 1%. As reported postsurgical complications generally relate to the surgery itself rather than to myelopathy, it is not likely that the complication rate would be substantially different in asymptomatic individuals as compared to symptomatic patients.

The major limitation of our study was its retrospective design, which could have had an influence on the exact timing of trauma in some individuals. However, significant traumatic episodes were found in only 3 cases with symptomatic myelopathy and the timing of their traumas correlated with records found in their case histories. The low frequency of traumas in our cohort may have resulted, at least in part, from our recommendation to all patients with diagnosed ASCCE that they avoid risky activities, such as certain sports (skiing, climbing etc.), walking on slippery surfaces, and so on. As we detected no severe trauma of the cervical spine in our cohort, we were not able to assess the hypothesis that stenosis of the cervical spinal canal (both degenerative and congenital) could worsen neurological sequelae of such a trauma.

We also investigated the possibility that disc herniation as a cause of spinal cord compression might more frequently be the result of preceding trauma, but found no such association.

In conclusion, the risk of spinal cord injury after minor cervical spine trauma in patients with asymptomatic spondylotic cervical cord encroachment appeared to be low in our cohort provided risky activities in these individuals are restricted. Implementation of preventive surgical decompression surgery into clinical practice in these individuals should be postponed until better designed studies show justification for giving it precedence over the conservative approach.

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