THORACIC MYELOPATHY DUE TO ISOLATED OSSIFICATION OF THE LIGAMENTUM FLAVUM

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We report a 72-year-old patient with thoracic myelopathy due to isolated ossification of the ligamentum flavum at T9-T10. Severe paraparesis had developed before the lesion was identified when thinning of a segment of the lower thoracic spinal cord was suspected on a second MRI examination. The diagnosis was then established by CT.

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Myelopathy due to ossification of the ligamentum flavum (OLF) is extremely rare in caucasian patients. In Japan, ossification of spinal ligaments is a relatively common cause of stenosis of the spinal canal, often resulting in myelopathy. OLF is frequently found in association with ossification of the posterior longitudinal ligament (OPLL) and in diseases of the spinal column such as ankylosing spinal hyperostosis (ASH) and diffuse idiopathic skeletal hyperostosis (DISH) (Forestier and Rotes-Querol 1950; Resnick and Niwayama 1976; Tsue 1981; Yonenobu et al 1987; Yoshizawa et al 1988).

CASE REPORT

In August 1991, a retired female caucasian pottery teacher, aged 72, developed severe pain over the dorsum and sole of her right foot, aggravated by walking. She was treated by a chiropractor and relieved of pain. In November 1991, she became aware of a right-sided foot drop which caused her to fall frequently.

In February 1992, she sustained a fracture of her right ankle when she tripped on a step and after immobilisation of the right leg for four weeks, she noted numbness on the lateral side of her right leg, spreading from thigh to ankle. In July 1992, she consulted a neurologist because of persisting foot drop and increasing difficulty with walking. Plain radiography of her head and spine was performed as well as MRI of the brain and spinal cord, and further investigation was recommended to exclude a vascular disorder of the spinal cord. In October 1992, myelography failed to reveal any space-occupying lesion in the thoraco-lumbar canal; the investigation caused headache and vomiting and it was decided not to proceed with spinal cord angiography. Later that month, the patient was referred to us for a second opinion because of progressive difficulty with walking which had made her reliant on wheelchair transport.

In November 1992, further plain radiography of the thoracic and lumbar spine showed only anterior marginal osteophyte formation at T9-T10 with no evidence of generalised degenerative disease such as ASH or DISH. In the past, the patient had been involved in a rear-end collision in which her small car had been hit by a lorry and overturned. She remembered no major injuries but back pain had persisted for some time.

Further investigation was delayed by a fall in which she fractured her right wrist. In May 1993, MRI showed thinning of the thoracic spinal cord at the T9-T10 level (Fig. 1), and subsequent CT at that level revealed a symmetrical curvilinear ossific lesion in the capsular portion of the ligamentum flavum (Fig. 2).

During the weeks which preceded these MRI and CT examinations, the patient had become aware of weakness in her left leg and had become completely wheelchair-bound. She also had frequency and urgency of micturition.

There was severe muscle wasting of the right thigh and lower leg, the lower-limb reflexes were brisk with bilateral ankle clonus and both plantar responses were extensor. Muscle power in the right leg was moderately reduced with very weak extensors of the foot, and power in the left leg was also reduced. Pain sensation was diminished up to the level of the T11 dermatome on the right. Full blood examination including biochemical tests, urinalysis and CSF analysis showed no abnormality and no evidence of systemic disease. Decompression was recommended.

Operation. At operation on 8 July 1993, radiographic control was used to localise the affected level. The thoraco-
lumbar fascia was incised on both sides 5 mm from the midline, and the spinous processes were preserved. The ligamentum flavum was found to be ossified and greatly thickened, contributing to marked stenosis of the thoracic canal. The canal was entered using a diamond-tipped high-speed burr and decompression was completed using fine oblique pituitary punches. The dura was not adherent to the ossified ligamentum flavum. The upper border of the T10 lamina measured approximately 8 mm in thickness. At the conclusion of the procedure, the thoracolumbar fascia was reattached to the midline ligaments. Histopathological examination showed focal ossification in a hypertrophic ligamentum flavum (Fig. 3).

**Postoperative course.** After operation, the patient improved steadily with the help of intensive rehabilitation despite a further fall in which she refractured her right wrist. At latest review in February 1994, she had no pain in her legs and was able to walk unaided.

**DISCUSSION**

We could find only two papers reporting myelopathy due to thoracic OLF in non-asiatic patients. Johnsson et al (1983) reported a patient suffering from DISH, and in the other case the pathology was considered to be generalised degenerative disease of the spinal column (Omomola et al 1982). We could find no report of myelopathy due to isolated OLF in a caucasian patient.

The pathogenesis of OLF is poorly understood, but Okada et al (1991) claimed that hypertrophy of the ligamentum flavum preceded the development of OLF. Other authors consider that hypertrophy of the ligamentum results from mechanical factors (Dockerty and Love 1940; Ramsey 1966; Beamer, Garner and Shelden 1973). In Japanese patients, Otani et al (1988) reported 23 operative cases of thoracic disc herniation. Marginal osteophyte formation at the level of the involved disc space was noted in 20 of the patients (87%) and OLF at the same level as the affected disc in seven of the eight patients (88%) in whom CT had been performed. These authors postulated that localised, mechanical factors were probably responsible for the development of OLF. In our patient, the trauma of a traffic accident 20 years earlier may have produced a localised injury which set the stage for the development of OLF at the T9-T10 level.

Our report highlights the importance of continued efforts to establish the cause of progressive paraparesis and of certain cases of myelopathy when there is clinical deterioration. It may be necessary to repeat tests such as MRI and CT. Yoshizawa et al (1988) have recommended CT as the single most useful investigation for this rare problem. In our case it proved to be valuable only after repeated MRI had indicated the correct level in the thoracic spine; CT then provided a definitive diagnosis.

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**REFERENCES**


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