Calcification of the ligamentum flavum of the cervical spine

Report of four cases

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Four cases of calcification of the cervical ligamentum flavum are reported, all in women over 60 years of age. Neurological findings were not significantly different from those of other cervical compressive diseases. Among radiological examinations, computerized tomography was the most valuable diagnostic tool. Calcification might have been induced by the degeneration or abnormal nutritional state of the ligamentum flavum. Endocrine abnormalities and inflammatory processes might also have been contributory factors.

KEY WORDS — spinal cord compression • ligamentum flavum • calcification • computerized tomography • cervical spine

SYMPTOMATIC calcification of the ligamentum flavum is rarely encountered in the cervical vertebrae. This report presents the clinical, neuroradiological, and histological manifestations in four cases of calcification of the ligamentum flavum of the cervical vertebrae.

Case Reports

Case 1

This 64-year-old woman was admitted in March, 1980, complaining primarily of pain in the posterior aspect of the neck. About December, 1979, she had developed persistent neck pain associated with a limitation of neck movement.

Neurological examination revealed limited neck movement, and hypalgesia of the extremities in a glove and stocking distribution. Plain radiographic examinations of the cervical vertebrae in the lateral projection disclosed a round shadow of calcification between the C-6 and C-7 laminae, concentrated mainly at C-6 (Fig. 1). In an oblique view, this shadow appeared as an oval mass projecting from behind and into the vertebral foramen between the C-6 and C-7 laminae. The space between the vertebral body and the calcification shadow was smaller when the patient bent backward than when she bent forward, suggesting that the area of calcification was mobile. Computerized tomography (CT) of the spine demonstrated an almost bi-symmetrical oval mass of high density projecting into the spinal canal at the C6–7 vertebral level (Fig. 2). Myelography revealed that the dorsal aspect of the spinal cord was compressed by the hypertrophied ligamentum flavum at C5–6, and by the calcified mass at C6–7.

In April, 1980, laminectomy of C-5, C-6, and C-7 was performed. The calcified masses were not adherent to the dura mater and were removed. The tissue obtained at surgery was studied histologically. Hematoxylin and eosin staining revealed a reddened amorphous substance surrounded by thickened ligamentum flavum (Fig. 3). The substance was so fragile that it tended to fall off in the staining process. The amorphous material was further studied using Kernectrol and McGee-Russel staining methods, and identified as calcified material. After operation, the sensory disturbance and limitation of neck movement resolved completely.
Case 2

This 62-year-old woman was admitted in August, 1981, complaining primarily of gait disturbance. In May, 1980, she had noted a dull feeling and weakness in both legs, but these symptoms gradually improved. In July, 1980, she had developed pain in the right knee joint, which was diagnosed as arthritis after aspiration of 10 cc synovial fluid. On June 18, 1981, after 2 days of persistent fever, she suddenly developed weakness and numbness of the lower extremities, which rapidly worsened.

On admission, neurological examination showed limitation of neck movement, spastic paraparesis, increased deep-tendon reflexes, and sensory disturbance below the T-9 vertebral level. Tomography of the cervical vertebrae showed evidence of slight spondylotic changes at C5-6, and oval shadows of calcification were observed at C5–6 and C6–7. Metrizamide CT myelography clearly demonstrated spinal cord deformity at C5–6 and C6–7, due to the pressure of a high-density mass projecting from the posterolateral aspect into the spinal canal.

On August 18, 1981, laminectomy involving the vertebral arches between C-5 and T-1 was performed. The calcified mass was slightly adherent to the dura mater and was separated without difficulty. Amorphous calcified material was identified histologically in the ligamentum flavum of C5–6 and C6–7. In addition, on one side of the ligamentum flavum at the level of C5–6 infiltration of chronic inflammatory cells was observed, mixed with calcified material.

Although there was no radiographic evidence of the presence of a mass between the C-7 and T-1 intervertebral arches, there was histological evidence of small deposits of calcium, scattered like islands in the ligamentum flavum at this level (Fig. 4). This finding probably represents an early phase of calcification of the ligamentum flavum. After the operation, the sensory disturbance almost disappeared and limitation of neck movement markedly improved.

Case 3

This 70-year-old woman complained of a gait disturbance of 4 months' duration and was hospitalized in July, 1981. Plain radiography demonstrated shadows of
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calcification at the C5–6 and C6–7 vertebral levels on the lateral view. Changes in position of the calcified area with changes in posture were also observed. Spinal CT showed high-density masses at C5–6 and C6–7. A myelogram revealed that the spinal cord was compressed in its ventral aspect by spondylotic changes at C4–5, and in its dorsal aspect by the hypertrophied ligamentum flavum at C3–4 and C4–5 and by calcified masses at C5–6 and C6–7. Laminectomy was performed without difficulty. The histological findings from the surgical specimens were the same as in Cases 1 and 2, although inflammation was absent.

Case 4

This 68-year-old woman complained of weakness of the right arm and both legs, and was admitted in July, 1981. Plain radiographic examination of the cervical vertebrae failed to demonstrate anything abnormal; however, spinal CT revealed small round high-density masses within the hypertrophied ligamentum flavum at C4–5 and C6–7. Close neurological follow-up review is under way at present. This patient has not yet undergone surgery.

Discussion

Hypertrophy and ossification are well known disorders of the ligamentum flavum of the thoracic and lumbar vertebrae. On the other hand, only a few reports are available describing patients with calcification of the ligamentum flavum of the cervical vertebrae. As far as we know, including the four cases reported here, only 10 such cases have been reported with descriptions of radiographic and histological findings. These 10 cases were analyzed clinically, and the following characteristics noted: 1) nine of the 10 patients were women; 2) all patients were over 60 years old; 3) the calcification was frequently observed in the lower cervical vertebrae; 4) associated disc disease was sometimes noted; and 5) concurrent ossification of the posterior longitudinal ligaments appeared only in the one male patient.

Some radiographic characteristics were common to these 10 cases. Plain radiography or tomography of the cervical vertebrae showed evidence of abnormal shadows of calcification on the posterior wall of the spinal canal. The space between the posterior aspects of the vertebral body and the abnormal shadow of calcification on the lateral view was reduced when the patient bent backward. Plain CT scans disclosed round or oval masses of high density projecting from the posterolateral side into the spinal canal. Myelography or metrizamide CT myelography demonstrated spinal cord compression from the posterolateral side; the cord was compressed not only by the calcified mass but also by hypertrophied ligamentum flavum at other levels. The calcified mass existed mainly on the rostral side of the intervertebral arches involved.

Although fibrocartilaginous or osteoid tissues were observed in some reported cases, the main histological characteristic was the calcium deposit. Calcium pyrophosphate dehydrate (CPPD) was identified chemically as the calcium component in three cases reported.

There has been some controversy concerning the cause of calcification of the ligamentum flavum. Ellman, et al., and Kawano, et al., have suggested that the calcification might be induced by CPPD deposits, as seen in cases of pseudo-gout. On the other hand, Jyotoku and Harada and Kida and Tabata have postulated that degeneration of the elastic fiber of the ligamentum flavum due to an abnormal nutritional condition might be a contributing factor causing calcification. However, the precise cause has not yet been established.

In our Case 2, an episode of arthritis of the knee was described clinically, and a non-acute phase of inflammation was identified histologically in a part of the diseased ligamentum flavum. In this case, therefore, pseudo-gout might have been related to the lesion. But in Cases 1, 3, and 4, there was no previous history of arthritis, and no positive serological study. It is quite possible that calcification may be induced by a combination of various factors. Since most patients are elderly females, it may be postulated that advanced age and some endocrine abnormalities are common causes.

On cervical myelography, hypertrophy of the liga-
mentum flavum was consistently observed in addition to the calcified lesion. Therefore, it may be reasonable to assume that hypertrophy of the ligamentum flavum predisposes to the process of calcification.

Although ossification of the ligamentum flavum is a well established finding, ossification and calcification may occasionally be confused. In ossification, which predominantly affects male patients, occasional involvement of the posterior longitudinal ligaments and the locations primarily in the thoracic and lumbar vertebrae is generally accepted. From the fact that the ossified mass does not move with postural change, it is suggested that the ossification is continuous with the lamina, as has been confirmed by surgery. In addition, ossified material sometimes adheres to the dura mater so firmly that laminectomy is achieved only with difficulty. Thus, calcification of the ligamentum flavum should be clearly distinguished from ossification.

References

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