

Calcified and ossified spinal leptomeninges producing cord compression.

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Calcified and ossified plaques within the spinal arachnoid are usually an incidental finding at necropsy examination and rarely produce neurological symptoms. In this report two male patients aged 70 and 40 years, having spinal cord compression due to calcium and osseous deposits in the arachnoid are described. The first patient fell on his back and noticed a progressive paraplegia. The relationship of spinal cord compression to a previous spinal subarachnoid hemorrhage and spinal arachnoid calcification is stressed. The second case presented with a history of a progressive paraplegia after a previous myelogram. The symptoms in both instances had considerably improved after surgical intervention.

วีระ กasantikul. ไขสันหลังถูกกดจากหินปูนและกระดูกงอกที่เชื่อมไขสันหลังจนเกิดอาการ. จุฬาลงกรณ์-
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หินปูนและกระดูกงอกที่เชื่อมไขสันหลัง มักพบโดยบังเอิญจากการตรวจศพ โดยที่ผู้ป่วยมักไม่มีอาการ ในที่นี้ได้รายงานผู้ป่วยชาย 2 ราย อายุ 70 ปี และ 40 ปี มีอาการของไขสันหลังถูกกดทับจากสาเหตุดังกล่าว ผู้ป่วยรายแรกมีประวัติหกล้มหลังกระดูกแตก และมีอาการอ่อนแรงของขาทั้งสองข้าง อาการดังกล่าวเชื่อว่าสืบเนื่องมาจากเลือดออกในช่องใต้ชั้นเชื่อมไขสันหลัง และมีหินปูนมาเกาะ ผู้ป่วยรายที่สอง มีประวัติอ่อนแรงของขาทั้งสองข้าง หลังการฉีดสีตรวจไขสันหลัง ผู้ป่วยทั้งสองรายมีอาการดีขึ้นมาก หลังการผ่าตัด

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Calcified and ossified plaques within the spinal arachnoid membranes have long been discovered at post-mortem examination on persons who have shown no neurological deficits.⁽¹⁾ The true incidence of arachnoid plaques is not known because spinal cords are not usually removed at routine necropsy examinations. According to Knoblich and Olsen,⁽²⁾ the incidence of arachnoid plaques has ranged from 6.3 percent to 76 percent. The authors also found that 43.2% of spinal cords had arachnoid calcification in 217 unselected necropsy patients. Compression of the spinal cord with clinical symptoms due to arachnoid plaques is extremely rare. Indeed some authors^(2,3) have disputed whether this is ever the cause of such neurological symptoms. Two cases are presented here not only for rarities but also in support of the conviction that spinal arachnoid plaques may result in pressure upon the cord and a consequent loss of functions. The relationship of such lesion to post traumatic spinal subarachnoid hemorrhage in one example is noted.

Case Report

Case 1.

A 70-year-old man was hospitalized because of progressive weakness of both lower extremities. One month previously he fell on his back and sustained a severe injury. Nevertheless, the patient could walk with crutch. Nine days later, he noticed a weakness and numbness in his left leg. The symptoms rapidly increased and the weakness appeared in the right leg. He also developed precipitancy of urine and constipation.

Neurological examination revealed a spastic paraparesis of both lower extremities, involving the left leg more than the right. There was decreased proprioception and hypalgesia below T₁₀. The plantar reflexes showed bilateral extensor response, and the abdominal reflexes were absent.

Routine laboratory data were unremarkable. The serum calcium was 8.6 mg/100 ml, and phosphate 3.8 mg/100 ml. The cerebrospinal fluid (CSF) was xanthochromic. The cisternal myelogram demonstrated a total block at T₉ and T₁₀ (Fig 1).

Laminectomy was carried out from T₇ through T₁₁ and disclosed a dense thickened adhesive arachnoid tissue which was located between the T₉ and T₁₀. It became apparent that the lesion causing the subarachnoid block was a calcified mass. The offending lesion was removed and the spinal block relieved. The postoperative course

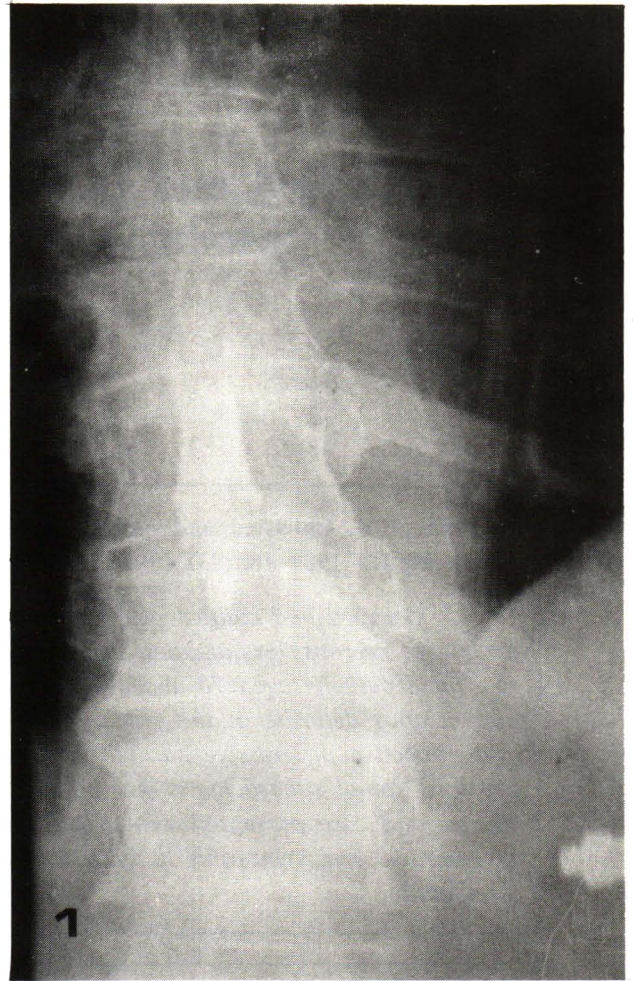


Figure 1 Case 1. Myelogram showing complete obliteration of the spinal subarachnoid at the T₁₀ vertebra.

was entirely uneventful. His walking, sensation, urination and bowel function had improved when seen 1 year after surgery.

Specimens of decalcified tissue in this case as well as in the other (case 2) were embedded in paraffin and stained with hematoxylin and eosin (H & E), and Gomori's iron preparations.

Microscopically, the plaques consisted of fibroblasts and numerous capillaries which were severely calcified and interspersed by occasional islands of bone (Fig 2). Numerous red cells and hemosiderin-laden macrophages were noted. Iron was demonstrated within the plaque.

The diagnosis was calcification and ossification of spinal arachnoid with subarachnoid hemorrhage involving T₉-10.

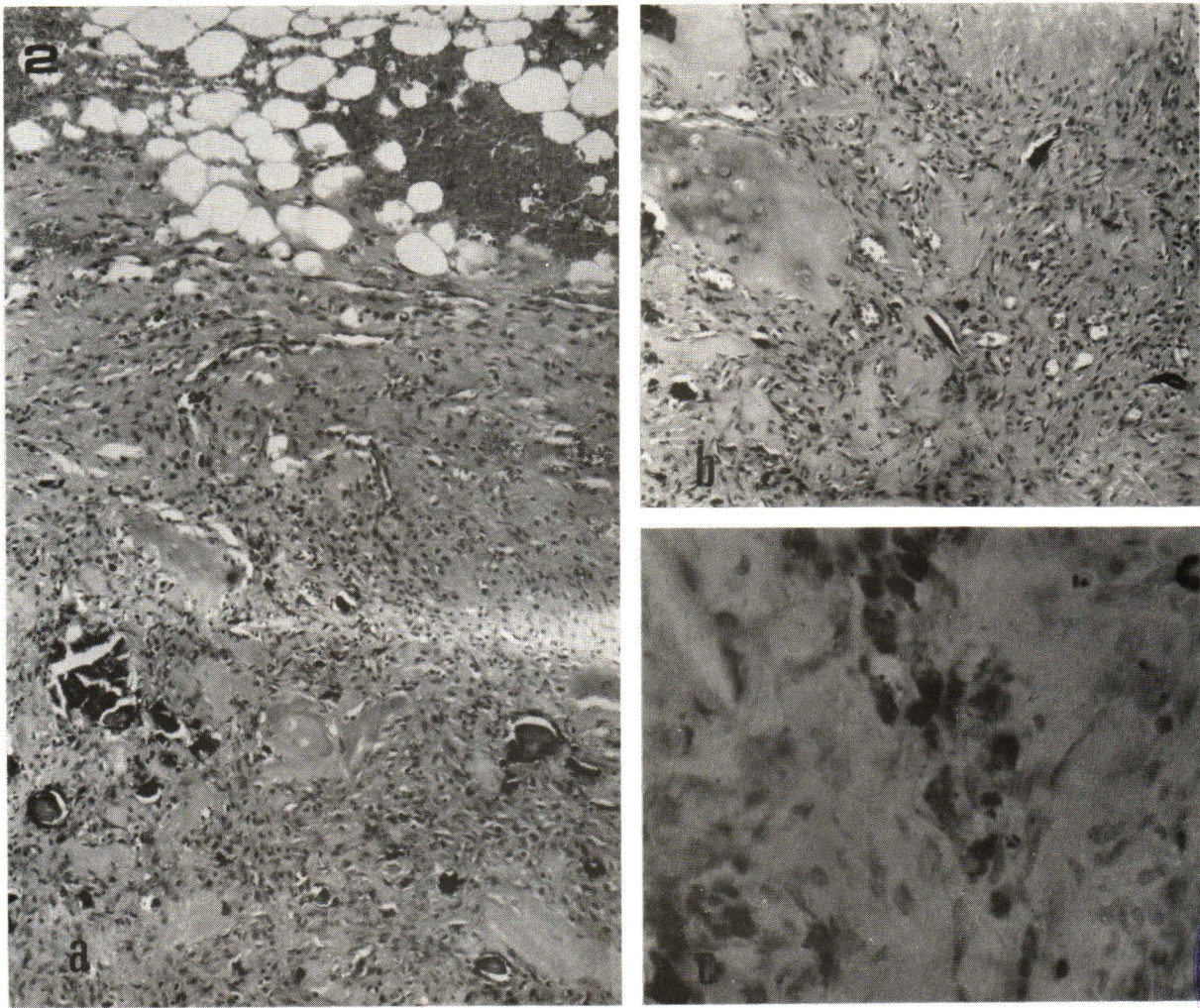


Figure 2 Microscopic features of the lesion in case 1.

- A. Diffuse calcification and proliferation of fibroblasts and numerous capillaries. Note epidural fat with recent hemorrhage. (H & E \times 100)
- B. Island of bone within the lesion. (H & E \times 200)
- C. Deposition of iron and hemosiderin-laden macrophages. (Gomori's iron stain \times 400)

Case 2.

Five years before hospitalization, a 40-year-old man had noted left back and side pain which was aggravated by bending at his waist. He received medical treatment from several general practitioners without clinical improvement. One year later he experienced numbness on the left side below the umbilical level, radiating to the left thigh. A myelogram was performed at another hospital and he was told that he "had a complete block" No surgery was performed. Gradually the patient began to notice a progressive weakness of the right leg. Since then he had difficulty in walking and became almost totally paralyzed. Additionally he noted burning dysuria, frequency, nocturia, constipation and sexual impotency. No

history was elicited of any accident. Neurological examination revealed weakness of both legs, involving the right more than the left. There were hypalgesia, analgesia and loss of temperature sensation below the left T₁₀. Vibration and joint sensation were intact. The sphincter tone was loose. The cremasteric and abdominal reflexes were depressed. No ankle clonus or Babinski's signs were elicited.

Routine laboratory data were within normal limits. The cisternal myelogram and lumbar myelogram demonstrated an irregular filling defect of myodil at the T₈₋₉ with complete block at the T₁₀ (Fig. 3).

At laminectomy, the arachnoid membrane was thickened and opaque with calcification surrounding the right posterior spinal cord at the

level of T₉₋₁₀. The cord appeared normal. The patient did well 6 months after surgery. Sensory and motor functions partially recovered, but the problem of urinary incontinence remained.

Histologically, the removed tissue consisted of fragments of bone and dense fibrous tissue (Fig. 4). There was no evidence of neoplasm or inflammation.

Figure 3 Case 2. Myelogram demonstrating an irregular filling defect of Myodil with a block at the T₁₀ vertebra.

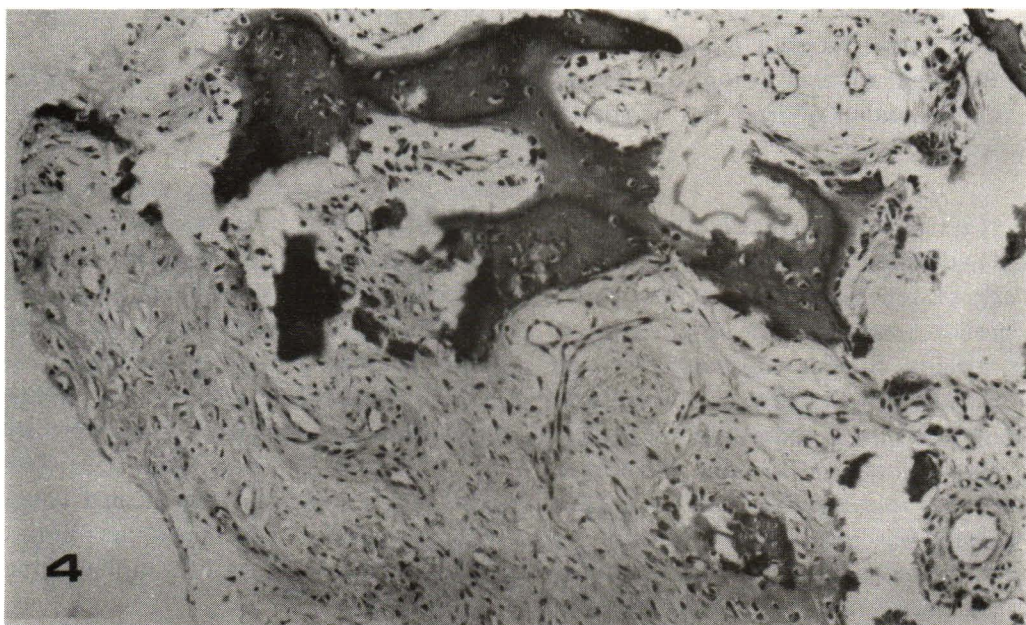
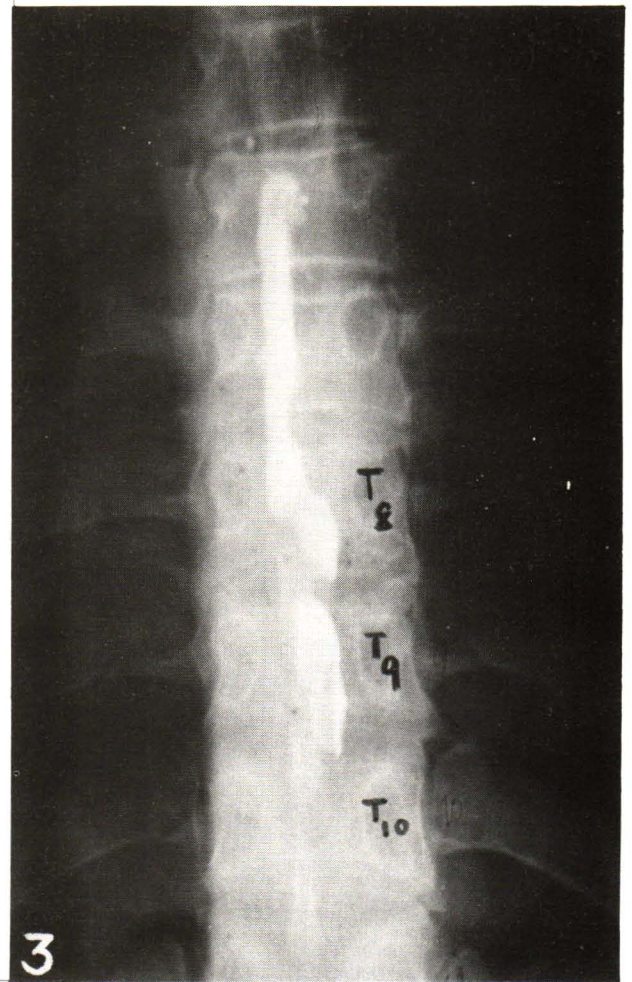


Figure 4 Photomicrograph of the excised arachnoid (case 2) exhibiting bone formation, fibroblasts and many capillaries. (H & E × 100)

Discussion

Calcification and ossification within the spinal arachnoid membrane are often clinically insignificant, and their discovery is usually by chance at autopsy.⁽¹⁾ However exceptions do occur. In 1931 Puusepp⁽⁴⁾ first described four cases all presenting with progressive paraparesis and all of whom underwent laminectomy as in our cases. Three were found to have well circumscribed osseous lesions attached to the dura mater while the last had bony deposits in the wall of a subdural cyst. Several authors have subsequently reported similar examples.⁽⁵⁻¹⁰⁾ For instance, Wise and Smith⁽¹⁰⁾ have reported two cases of spinal arachnoiditis ossificans. The first patient had a history of an unusually long spinal anesthesia 31 years prior to his presentation, while the second had a spontaneous spinal subarachnoid hemorrhage 20 years previously that had left him paraparetic. The cases reported here had localized calcification and osseous deposits in the arachnoid which were responsible for the neurological manifestations. Our examples therefore resembled in all respects the reported symptomatic cases found at surgery.

It should be noted that Knoblich and Olsen⁽²⁾ have suggested that the calcified and ossified of the spinal arachnoid are extremely common findings and are often falsely accused of being the cause of the patient's symptoms. This statement appears incorrect as our patients improved clinically after laminectomy. Furthermore, of the eight sympto-

matic cases in Slager's review, who had been treated by surgical intervention, six improved postoperatively.⁽¹⁾ These data strongly suggest that in these cases the clinical manifestations of spinal cord compression were due to calcification and ossification within the spinal arachnoid membrane and may be alleviated by their surgical removal.

Considerable controversy exists regarding the etiology of these lesions. As in all rare conditions, theories concerning the mode of occurrence have been offered by several authors.^(1,4,5) For the plaques which appear as an incidental finding, they include local trophic disturbances, aging process and degenerative ossification in relation to foci of hyperplastic meningotheial cells.⁽¹⁾ Various investigators have attributed the cause of symptomatic plaques to four factors, including previous trauma, myelography, spinal anesthesia, and a previous subarachnoid hemorrhage with its associated vascular anomaly^(4,5,8,10) There was obviously a relationship between a previous traumatic subarachnoid hemorrhage and the arachnoid calcification in one of our examples (case 1). The presence of hemosiderin-laden macrophages with iron deposits as well as a clinical history of trauma supported this concept. Although the etiology in or case 2 is not clearly established, it is reasonable to assume that a previous myelogram could have been a precipitating factor as suggested by Miles and Bhandari.⁽⁸⁾ Clinical history of a progressive paraparesis of the lower extremities following a previous myelogram tended to support this view.

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