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Pencil-shaped Softening of the Spinal Cord

Pathologic Study in 12 Autopsy Cases

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Summary. This report describes pathologic findings of pencil-shaped softening in 12 autopsy cases. Nine cases were associated with extradural metastasis of malignant tumor and three were observed in brain death. The cavity of the pencil-shaped softening containing necrotic debris and abundant macrophages was located mainly in the ventral part of the posterior column or the dorsal horn. It extended longitudinally over several segments of the cord, predominantly in an upward direction, and had a very clear demarcated margin from the compressed surrounding tissue. Reactive change of the surrounding tissue was very rare. Pencil-shaped softening was found most often in the level of the thoracic cord. The cavity was always continuous with an area of transverse necrosis or and area of patchy necrosis. In previous reports, the factor of circulatory disturbance was considered important for the pathogenesis of pencil-shaped softening. In addition, we believe that mechanical compression is also an important factor.

Key words: Pencil-shaped softening – Extradural metastasis – Brain death – Mechanical compression

Introduction

Pencil-shaped softening (PS) is an unusual cavitated lesion in the ventral part of the posterior column, or the posterior horn, which extends longitudinally over several segments of the spinal cord. PS is most frequently observed in compression myelopathy secondary to extradural metastasis of malignant tumor (Gruner and Lapresle 1962; Nagashima and Shimamine 1974), but is also seen in spinal cord trauma (Holmes 1915; Jellinger 1964), adhesive arachnoiditis (McLaurin et al. 1954), primary and metastatic intramedullary spinal cord tumor (Barnett et al. 1973; Jellinger et al. 1979; Hirose et al. 1980), and brain death (Schneider and Matakas 1971).

In Japan, the pathology of PS was first described in 1974 by Nagashima et al. and was later on confirmed by Makifuchi (1975), Hirose et al. (1980), and Inoue et al. (1982).

In its pathogenesis, circulatory disturbances must be considered, but mechanical factors may also play an important role.

In this paper, we present the histopathologic findings of PS in 12 autopsy cases and discuss the pathogenesis of PS.

Materials and Methods

At Nagoya University Hospital and Nagoya City University Hospital and related hospitals 372 spinal cords were examined between 1978 and 1982. Spinal cords were removed by anterior abdominal approach. Transverse sections were made at each segmental level of the spinal cord and were stained with hematoxylin-eosin (HE), Klüver-Barrera, Elastica van Gieson, and Bodian methods.

Compression myelopathy by extradural metastasis of malignant tumor was observed in 15 of 372 cases and six of these cases showed PS. The spinal cord from two "brain death" patients also had PS.

In the autopsy files of Montefiore Medical Center, Bronx, New York, three cases of PS were found in the spinal cord associated with extradural metastasis of malignant tumor and one case with "brain death". Altogether 12 cases were studied.

Results

Table 1 shows the summary of the pathologic findings of PS. In nine of 12 cases, transverse necrosis was seen with complete destruction of both gray and white matter (Fig. 1). The pia mater was intact (Fig. 2). In three cases without transverse necrosis, status spongiosus was seen in most areas of the involved white matter with loss of myelin and axonal swelling (Fig. 3).

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Table 1. Summary of pathological findings of pencil-shaped softening

Case no.	Age	Sex	Disease	Transverse necrosis	Status spongiosus or patchy necrosis	Pencil-shaped softening
1 (8490)	7	F	Neuroblastoma	C8	C7 Th1, 2	C7
2 (T8104)	56	M	Lung cancer	Below the level of Th9	Th3, 4, 5, 6, 7	Th2, 3, 4, 5, 6
3 (1641)	39	M	Plasmacytoma	Below the level of Th3	C6, 7, 8, Th1, 2	C6, 7, 8, Th1
4 (8563)	37	M	Renal cell carcinoma	(–)	Th8, 9, 10, 11	Th8, 11
5 (8646)	52	M	Gall bladder cancer	(–)	Th4, 5, 6	Th4, 5
6 (17312)[a]	22	F	Rhabdomyosarcoma	(–)	Th6, 10	Th6
7 (12101)[a]	67	M	Chronic lymphocytic leukemia	Th2		Thoracic
8 (8548)	33	F	Choriocarcinoma	Below the level of Th12	Th6, 7, 8, 9, 10, 11	Th6, 7, 8, 9
9 (15848)[a]	60	F	Hodgkin's lymphoma	Midthoracic	Midthoracic	Midthoracic
10 (8627)	11	M	Brain death, embryonal cell carcinoma	Below the level of Th9	Th7, 8	Th6, 7, 8
11 (8636)	37	F	Brain death, systemic lupus erythematous	C6, 7	C8–L1	Th1–12
12 (83-14)[a]	32	F	Brain death, toxoplasmosis	Above the level of C6	C7–Th6	C7–Th6

[a] Autopsy case of Montefiore Medical Center

Where the status spongiosus was most severe, it was continuous with patchy necrotic areas. Status spongiosus predominantly involved the lateral and posterior columns. In contrast, the gray matter was relatively intact, although degenerative changes were seen in some neurons. In nine of these 12 cases, serial sectioning revealed continuity of PS with areas of transverse necrosis or patchy necrosis. In three cases, detailed examination was not possible. PS extended upward or downward from the level of necrotic areas. In eight of 12 cases, the cavity of PS extended upward.

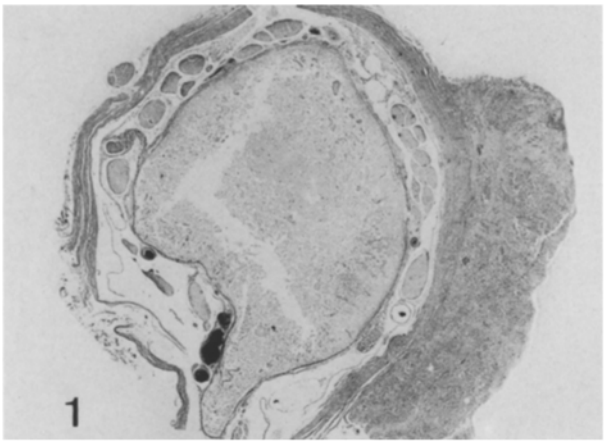
PS extended over one to 12 segments. All were in the thoracic cord and three involved the lower cervical cord in addition. No PS was observed in the lumbosacral region. PS had no communication with the central canal. The lesion was located in the ventral part of the posterior column or the dorsal horn in all cases (Fig. 4). In the dorsal column, PS was unilateral and compressed the opposite dorsal column. However, in some cases, a large cavity was noted in the center of the posterior columns. The cavity in the posterior horn was located mainly in the ventral part and was either confined within the posterior horn or extended to the posterior column and compressed the surrounding tissue. In cases 1, 3, 5, PS was noted in the deep part of the lateral column (Fig. 5) and in case 5, in the anterior column.

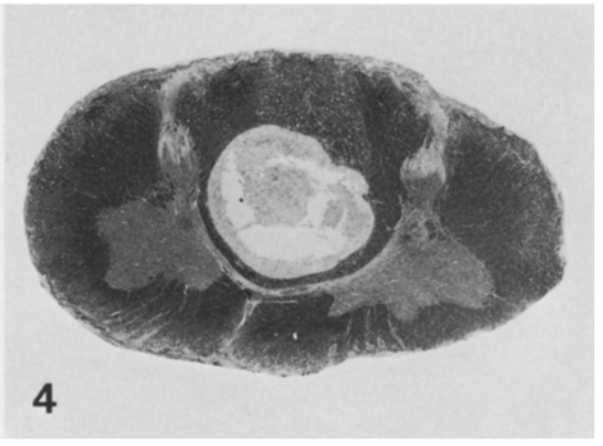
The well demarcated round to oval cavity was filled by necrotic debris and abundant macrophages. Reactive changes in the surrounding tissue to the cavity was conspicuously very minimal (Fig. 6). Reactive astrocytosis and macrophages were not present. Two cases (cases 8, 9) varied somewhat in that the necrotic areas were small, poorly defined, did not compress the surrounding area, and were associated with mild reactive astrocytosis.

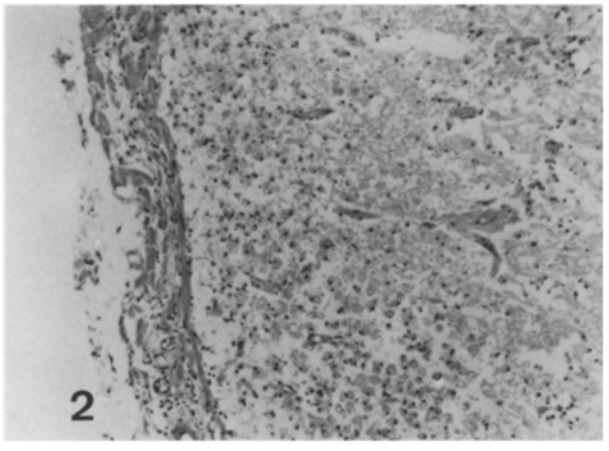
Occlusion or stenosis of the extradural vessels could not be found, although the vessels were surrounded by the tumor mass. Congestion of the veins in the subarachnoid space was noted in all cases, and mild fresh hemorrhage was noted within the cavity (cases 5, 7). Organized thrombus occluding small arteries and veins in the subarachnoid space was present in the level of transverse necrosis (case 2).

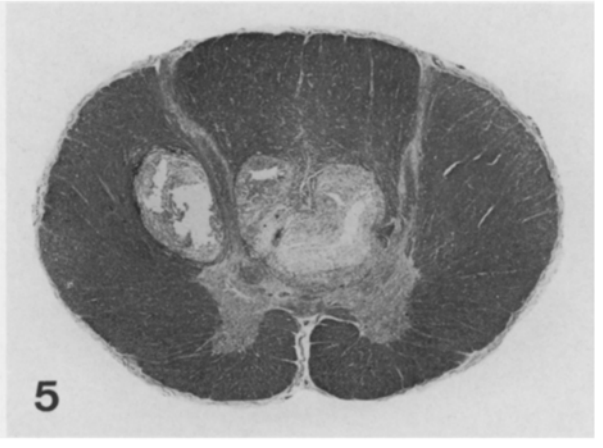
Tumor embolism of small veins was present in the areas of status spongiosus in the spinal parenchyma (case 5).

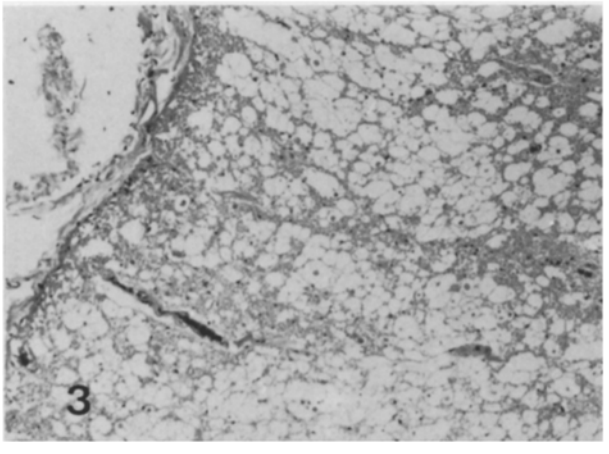
In case 10, which showed transverse necrosis below the level of Th10 segment, there was irregular necrosis of posterior half of the spinal cord in the level of Th9 segment. In the level of Th8 segment, a slit was found in the dorsal horn, through which the PS in the level of Th6 and Th7 segments was continuous with the necrotic areas. There was aggregation of degenerative ependymal cells within the necrotic debris of the cavity in Th7 segment (Figs. 7–11). Furthermore, in case 12, degenerative neurons were observed within the necrotic debris of the cavity, which was formed in the dorsal column.











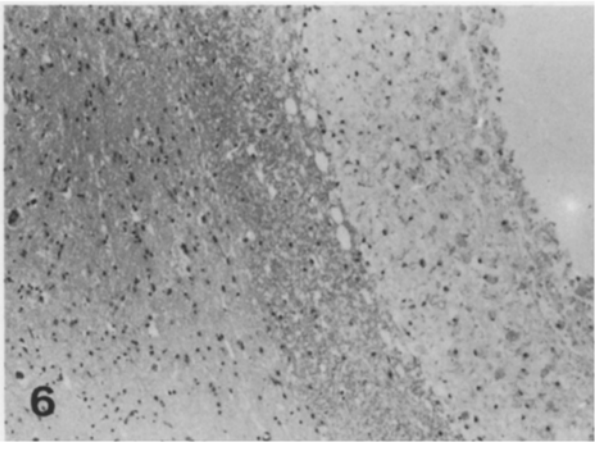


Fig. 1. Transverse necrosis and extradural metastatic tumor (case 2, Th12). HE, × 5

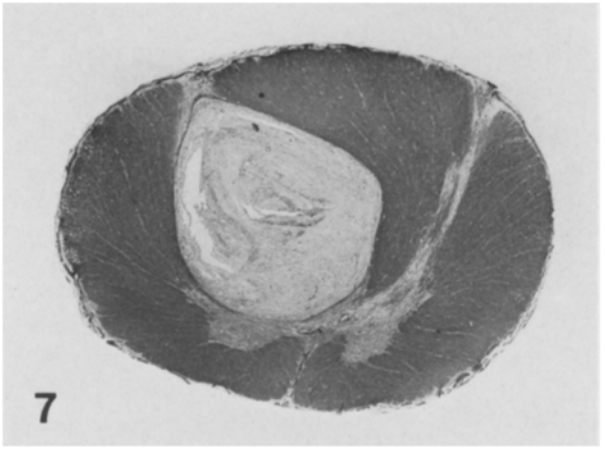
Fig. 2. Pia mater is well preserved at the level of transverse necrosis (case 2, Th12). HE, × 330

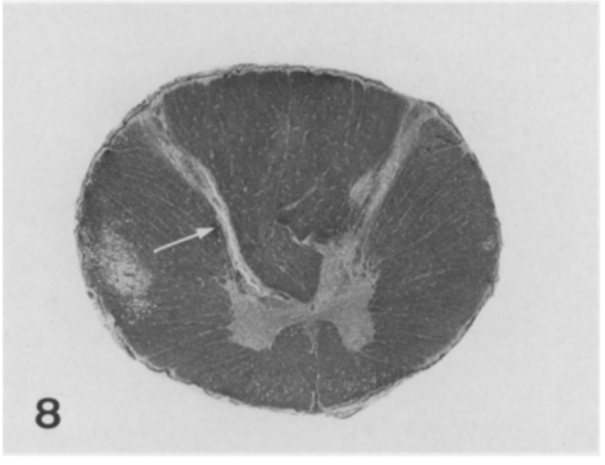
Fig. 3. Status spongiosus of white matter (case 5, Th7). HE, × 130

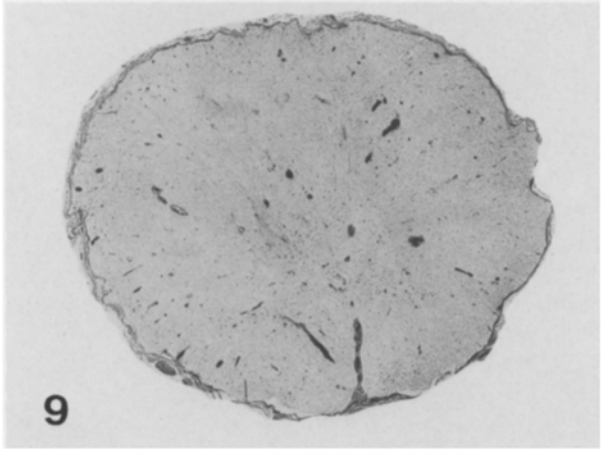
Fig. 4. Pencil-shaped softening of the ventral part of the posterior column (case 3, C6). KB, × 6.6

Fig. 5. Pencil-shaped softening of the posterior column and the lateral column (case 5, Th4). KB, × 8

Fig. 6. Surrounding tissue around the cavity does not show reactive change (case 3, C6). HE, × 130







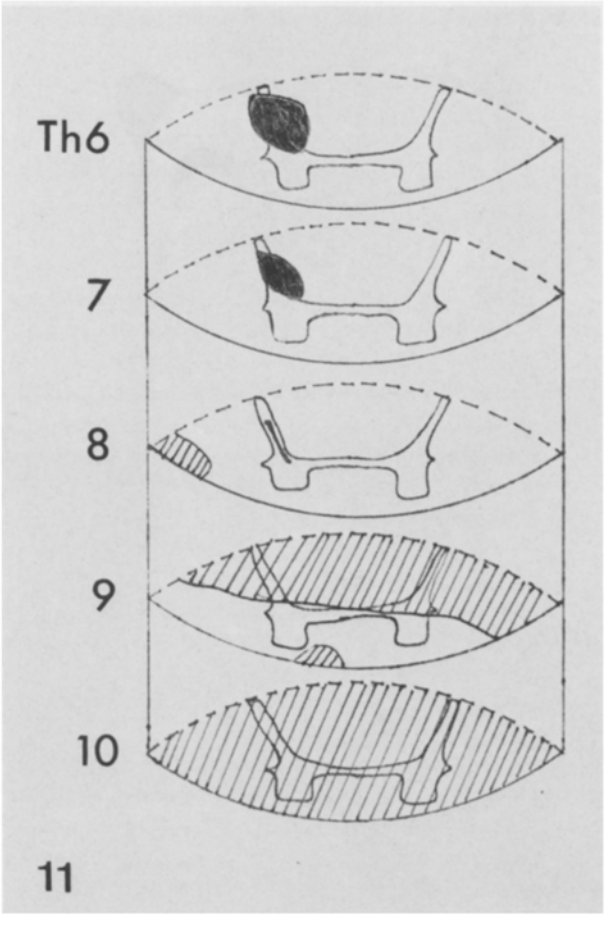


Fig. 7. Pencil-shaped softening of the posterior horn (case 10, Th6). KB, × 8

Fig. 8. Slit in the posterior horn (*arrow*) (case 10, Th8). KB, × 8

Fig. 9. Transverse necrosis (case 10, Th10). KB, × 8

Fig. 10. Degenerative ependymal cell (*arrow*) within the necrotic debris of the cavity (case 10, Th7). HE, × 110

Fig. 11. Schematic representation of the spinal lesions in case 10. The cavity is shown as a *black area* and the necrosis and status spongiosus as a *shaded area*

Discussion

In our series, PS was observed in the thoracic and lower cervical cord, and the cavity of PS was consistently located in the ventral part of the posterior column or the posterior horn. This finding confirms the reported predilection for this site (Jellinger 1964; Barnett and Rewcastle 1973; Nagashima and Shimamine 1974), which has been described as a watershed zone of arterial blood supply (Zülch 1954; Turnbull 1971) or venous drainage (Jellinger 1972).

Infarction from venous obstruction associated with hemorrhage was also reported as maximal in this area (Hughes 1978). Many previous reports (Zülch 1954; Gruner and Lapresle 1962; Jellinger 1964; Barnett et al. 1973; Nagashima and Shimamine 1974) stated that both the insufficiency of arterial supply and the failure of venous return may play an important role in the pathogenesis of PS.

In two cases of our series (cases 8, 9), the necrotic lesion in this area appears to be secondary to the circulatory disturbance. However, PS characterized by a clearly demarcated cavity, compression of surrounding tissue, lack of tissue reaction, and longitudinal extension of the cavity seems to be due to a second mechanism, most likely mechanical compression. McVeigh (1923) obtained canine and human spinal cords at autopsy and compressed them digitally with subsequent parenchymal destruction. The sections above and below the lesion for a distance of several centimeters showed destroyed parenchyma in the posterior central part of the cord.

In "toothpaste" artifact, the destroyed parenchymal tissue penetrates above and below the level of constriction by mechanical compression at the time of removal of the spinal cord. Areas of infarcts or tumors are especially prone to these changes (Hirano et al. 1980; Hirano 1981).

We confirmed the continuity between the cavity of PS and the necrotic areas by serial sectioning, particularly in cases 2, 10, and 11, where the cavity of PS was continuous with the necrotic areas through the slit in the dorsal horn. In case 10, there was aggregation of degenerative ependymal cell clusters within the cavity of PS in addition to the intact central canal of the segment. In case 12, degenerative neurons were observed within the necrotic debris of the cavity. These findings suggest that the necrotic tissue gets displaced upward and downward from the necrotic areas.

The expanding character of the cavity appears to be produced by the penetration of necrotic tissue or edema fluid. With an intact pial membrane, increased intrapial pressure may be caused by edema of spinal parenchyma, congestion of the spinal venous system, and mechanical compression by an extradural mass or at the time of removal of the cord. The initial pressure at the level of necrotic areas is reduced by the entrance of the necrotic tissue or the edema fluid into the segments of the cord above and below the lesion.

Certain anatomic features may explain the site of predilection for PS. The posterior column is composed of bundles of parallel fibers with no junctions between adjacent myelinated axons (Hirano 1981) and the ventral part of the posterior horn is rich in myelinated fibers (Carpenter 1976). These anatomic factors seem to permit the relatively easy penetration of necrotic tissue or edema fluid.

Thus, it could be stated that PS is a result of the combination of circulatory disturbance and mechanical compression.

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