Intradural Spinal Lipoma in an Experimental Swine

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(With colour plate I)

The controversial metaplastic potential of the meninges\(^1\) has led to a number of varying accounts in medical literature about the relationship of neoplastic adipose tissue to the covering membranes of the central nervous system.

Reports of a supposedly congenital lipoma in the left cerebral hemisphere, of an extradural lipoma that compressed the lumbar spinal cord, and of a similar sacral tumor represented the compass of Feldman's\(^5\) information on such intracranial and intraspinal neoplasms in bovine animals. Feldman\(^6\) examined a lipoma from the brain of a slaughtered hog. Jackson\(^7\) cited a congenital lipoma in the meninges of a sheep. Willis\(^12\) mentioned lipomas related to the central nervous system in conjunction with various malformations in man and referred to some of these neoplasms in animals designated by Feldman\(^5\).

In their discourse on the classification of meningiomas, Bailey and Bucy\(^2\) referred to the lipomatous type with cells typical of adipose tissue. These authors stated that Virchow was one of the first to maintain that lipomas arise from cells of the human meninges. Brown's\(^8\) rather elaborate study of 130 cases of intraspinal meningiomas in man resulted in a somewhat modified presentation of the variants as compared with that of Bailey and Bucy\(^2\). In an apparently significant deviation from the trend at least partially established by the latter writers, Brown\(^8\) emphasized that only those lipomas characterized by meningothelial (arachnoidal) cells as an integral part of the neoplastic tissue were classed as lipomatous meningiomas. Neoplasms comprised only of fat cells were not included with this category but were regarded as pure lipomas despite their meningeal locations. Willis\(^12\) omitted the lipomatous form from an outline of the variants of meningiomas. In a slightly simplified version of the outline propounded by Brown\(^8\), Anderson\(^1\) regarded the lipomatous form as a variant of the metaplastic meningioma. Among the brief presentations on meningiomas in certain veterinary texts\(^4, 8, 10\), only Innes and Saunders\(^6\) explicitly mentioned the lipomatous type.

Stookey's\(^11\) report concerning the occurrence of extradural and intradural spinal lipomas in 19 people indicated pial derivation of the uncomplicated fatty neoplasms found beneath the dura mater. Apparently all of the intradural growths essentially contained well-differentiated adipose tissue, and intramedullary fibrous extensions from the pia mater often accompanied them. The account rendered by
EHNI and LOVE\textsuperscript{4} with regard to 49 cases of extradural and intradural spinal lipomas in man was largely compatible with the preceding analysis\textsuperscript{11}. The principal points stressed by these writers\textsuperscript{4} were as follows: composition of adult adipose tissue or of adult adipose tissue with excessive fibrous stroma in all but 1 of the 29 intradural lipomas reviewed; invariable separation of an intradural lipoma from the underlying neural tissue by a layer of fibrous tissue of pial derivation; envelopment of the free surfaces of the tumor with a pial sheath; probable nonmalignancy of the prominent pial extensions into the spinal cord; and the lack of relation of either form of lipoma to meningioma.

Clinical Anamnesis

The affected animal was one of a group of swine experimentally exposed to mixed neutron-gamma radiation from an atomic detonation at the Nevada Test Site during the spring of 1957\textsuperscript{9}. This individual was irradiated with a total, whole-body dose of approximately 75 rads and was subsequently transported along with other survivors of this study to Walter Reed Army Institute of Research, Washington, D.C., where the group was observed for 2 years. During July, 1959, these swine were transferred to this laboratory for continued study.

About 3 years after irradiation, the affected pig developed locomotor ataxia in the pelvic limbs. Within a month, the animal became paraplegic, the sensory reflexes being absent caudal from the anterior lumbar region. The paralysis was transitory, because after 2 months the swine was capable of assuming a standing position with assistance. Maintenance of the position was possible without support if the swine remained immobile. Any attempt to walk resulted in falling. During the 4th and final month of observation, further improvement was not seen.

Throughout the course of the clinical illness, there was conspicuous retention of alertness, normal appetite, a good state of nutrition, and a normal hemogram. Medication was administered in the form of multiple intramuscular injections of the vitamin-B complex for an interval of 7 days during the initial stage of the nervous malfunction. When it became obvious that the barrow's recovery was improbable, euthanasia was administered via intravenous nembutal and subsequent exsanguination.

Gross Pathology

External examination of the 318-kg cadaver (Necropsy No. 1953) revealed nothing of pathological significance. A few comparatively minor changes were observed in the internal organs. The brain and spinal cord were removed in toto and preserved in an aqueous solution of 10\% buffered formalin. The vertebral column and spinal cord were subjected to a relatively cursory examination, so the enlargement approximately at the middle of the thoracic cord was not regarded in its true light until after fixation. The spinal canal around the lesion was not measured, but no conspicuous expansion of the passage was observed.

A detailed macroscopic examination of the brain and spinal cord was conducted after fixation. The brain was normal. A subdural, fusiform enlargement approximately 7 cm long was near the middle of the thoracic cord on its dorsal
aspect. Observation of multiple transverse sections of the spinal cord revealed that a waxen, translucent material comprised the major part of the sharply delimited lesion. At the site of its greatest size, the lesion occupied an area roughly tantamount to one-third of the total area of the extraneous substance and spinal cord on cross section (Fig. 1). The mass had markedly encroached upon the dorsal funiculi and dorsal columns and, to a lesser degree, upon the lateral funiculi. The gray matter had undergone evident degeneration and distortion presumably because of continuing and gradually increasing pressure from the supposed neoplasm. The prosector further noticed that sections from the affected portion of the cord floated in the fixative solution, which fact led to the inference that the lesion was a lipoma.

**Histopathology**

Microscopic perusal of the brain revealed no significant aberration.

Upon examination of the spinal cord, minor extradural hemorrhage, partially involving adjacent epidural adipose tissue, was observed. The white matter generally appeared to be rarefied, as though some of the fibers and myelin sheaths had been destroyed. Scattered, moderate hemorrhages were observed in the gray matter. These hemorrhages were especially prevalent in the ventral columns. In sections containing the tumor, there were pronounced degenerative changes and loss of structural detail in the gray matter. These alterations were exemplified by neurons undergoing degeneration and necrosis, by apparent disruption and distortion of nerve fibers, and by numerous collections of amorphous, granular, acidophilic material assumed to have been derived from degenerative and necrotic nerve cells.

The bulk of the neoplasm was composed of well-developed «signet-ring» cells typical of fatty tissue. This aggregate of adipose tissue had slight to moderate amounts of fibrous stroma at various sites. The principal part of the neoplastic tissue was intimately associated with the pia mater, the growth lying dorsad to the thickened pia mater and within a thinner extension of this membrane over the free surfaces. Although the pia mater covering the tumor was in contact with the arachnoid at a few points, this appeared to be incidental rather than indicative of attachment through proliferative alteration of either component of the leptomeninges. (The pathologist could have proffered irrefutable evidence for no attachment of these membranes through transfer of hyperplastic or neoplastic tissue only with examination of perfect serial sections of the neoplasm and contiguous elements.)

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*Fig. 1.* Gross appearance of formalin-fixed spinal cord with intradural lipoma.

*Fig. 2.* Spinal cord with intradural lipoma. Weil’s iron hematoxylin stain for myelin sheaths, ×3.

*Fig. 3.* Spinal cord with intradural lipoma. Weigert’s iron hematoxylin and eosin stain, ×3.

*Fig. 4.* Normal spinal cord. Weil’s iron hematoxylin stain for myelin sheaths, ×3.

*Fig. 5.* Spinal cord with intradural lipoma. Heidenhain’s aniline blue stain for collagen and reticulum, ×5.
Fibrous ramifications from the thickened pia mater extended into the neural tissue below. The proliferating fibrous tissue was characterized in large part by immature cells containing the usual relatively pale, round, ovoid, and fusiform nuclei with small nucleoli. Mature fibroblasts and pronounced production of collagen were also observed in the pia mater and its branches. Occasionally the pial extensions formed an imposing mass of tissue with an invasive aspect, particularly beneath one end of the lipoma where the proliferating fibrous tissue comprised a roughly conical growth with the apex extending nearly to the central canal of the spinal cord. Invasion of the nervous tissue to this depth was not characteristic of the pial extensions generally, however; and apparently no criterion for malignancy—i.e., mitotic figures, extreme cellularity, or anaplasia—was in any of the fibrous tissue.

Pial collagenous tissue encroached in sundry areas and degrees upon the dorsal nerve roots, the dorsal funiculi, the lateral funiculi, and the dorsal columns of the gray matter. Enmeshed in the pia mater or among its branches were dorsal nerve roots, nerve fibers, glial tissue, degenerating neurons, a slight number of apparent irregular deposits of calcium salts (not typical of the concentrically laminated psammoma bodies in a meningioma), and a few individual adipose cells or small collections thereof. In addition, whorls of collagen were seen around a considerable number of vessels both within and beyond the periphery of the pial fibrous extensions. (The senior author has seen similar coils of fibrous tissue around vessels associated with chronic inflammatory reaction. Hence, one may assume that this condition can also accompany a degenerative and cicatricial process initiated by gradually increasing pressure instead of being restricted solely to neoplasms such as meningiomas.)

For histopathological evaluation of the lipoma and its apparent effects on the spinal cord, special stains were used in addition to the conventional stain of hematoxylin and eosin (Fig. 3). With Heidenhain’s aniline blue stain (Fig. 5) for collagen and reticulum and van Gieson’s stain for collagen, identification of an abundant amount of this material relative to the lesion was confirmed. Lipids in the lipoma and in degenerating myelin sheaths were stained positively in frozen sections with oil red O; and Sudan II was also used for a similar determination. Thionine marked the location of Nissl substance in neurons. Confirmation of the evident loss and distortion of myelin sheaths and nerve fibers was made with Weil’s myelin stain (Figs. 2 and 4). Less rewarding results were obtained by application of other special stains to affected parts of the cord.

Discussion

While controversy pertinent to the origin and metaplastic potential of the meninges is extant, this article does not presume to resolve the debate. If one be an advocate of the thesis that the meninges are of mesenchymal rather than of neuroectodermal origin, then the problem of metaplasia seems less insurmountable. Thus one can conceive of a lipoma arising as an uncomplicated neoplasm within the meninges and without the classical, neoplastic arachnoidal cells deemed essential to the identification of a lipomatous meningioma by Brown.
(Conversely, for whatever it may be worth, lipoblasts multiplying under pathological conditions may differentiate into other related types of mesenchymal tissues12.) Other possible sources of intradural lipomas would be aberrant fat cells in the pia mater or the inclusion of embryonal rests within the meninges at the time of formation of the neural tube11. Certainly the writers wish to emphasize that they consider the growth described in this article as one of probable congenital or spontaneous occurrence rather than as a sequel of exposure to ionizing radiation.

The pial thickening and the fibrous extensions from the pia mater into the spinal cord probably do not constitute a manifestation of malignancy. These alterations seemingly represent a chronic, proliferative fibrous replacement of degenerating nervous tissue resulting from neoplastic pressure, and the fibrous tissue incidentally encloses some neural elements that are not yet necrotic. This observation is essentially concurrent with one advanced by Ehni and Love13. Further agreement with these writers is achieved through the stipulation that destruction of the tissue in the cord by the fibrous extensions is questionable.

If pressure on the spinal cord by the mass of the tumor progresses without relief before destruction of the underlying nervous tissue occurs, neither the surgeon nor the pathologist can determine whether the pressure or the proliferating fibrous tissue has caused the necrosis. Should the lipomatous mass not recur after partial or complete surgical removal, then the continued growth of the pial extensions and their concomitant invasion and destruction of the spinal cord should result in their collective designation as evidence of a malignant neoplasm. Apparently such a case has not been observed. The postoperative clinical history of an occasional human patient with this type of lesion, moreover, has indicated a lack of malignancy in the pial fibrous tissue4.

**Summary**

A lesion designated as an intradural spinal lipoma was characterized by evident origin within the pia mater and by composition of well-differentiated adipose tissue with some fibrous stroma. Increased pial thickness and fibrous proliferation from the pia mater into the underlying neural tissue accompanied the neoplasm. No attachment of this growth to either the arachnoid or the dura mater could be discerned at any site examined. Since arachnoidal cells were not observed as components, the neoplastic tissue was not considered to be a variant of the meningioma.

**Zusammenfassung**

Eine als intradurales Spinallipom diagnostizierte Veränderung war durch ihren eindeutigen Ursprung innerhalb der Pia mater und ihre Zusammensetzung aus gut differenziertem Fettgewebe nebst geringem fibrösen Stroma gekennzeichnet. Das Neoplasma war von Piaverdickung und fibröser Proliferation von
der Pia mater aus in das unterliegende Nervengewebe begleitet. Keine der untersuchten Seiten zeigte ein Anhaften des Gewächses an die Arachnoides bzw. die Dura mater. Da keine arachnoidalen Zellkomponenten beobachtet wurden, wird das neoplastische Gewebe nicht als eine Variante des Meningioms betrachtet.

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References


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