Ultrastructure of the human posttraumatic syrinx


Departments of Surgery (Neurosurgery), Pharmacology, and Anatomy, University of Manitoba, Winnipeg, Manitoba, Canada

Although posttraumatic syringomyelia is a well-established clinicopathological entity, there is a paucity of information on the ultrastructural features of this condition. This study documents the light and electron microscopic features of posttraumatic syringes obtained from two patients who underwent surgical cordectomy. The syringes were lined largely by cell processes of astrocytes. Small regions near the caudal end were lined by flattened ependymal cells that lacked surface specializations. These were thought to represent remnants of the central canal ependyma. The ultrastructural appearance of the syrinx was similar to that of the communicating syringomyelia as well as the periventricular changes that accompany hydrocephalus. The authors conclude that the changes represent the nonspecific sequelae of a distensile force within the syrinx cavity.

KEY WORDS • syringomyelia • ultrastructure • spinal cord • histological study

POSTTRAUMATIC spinal cord cavitation was first recognized by Hallopeau in 1871, but the clinical correlates that accompany posttraumatic syringomyelia were established only recently. The pathogenesis of syrinx formation is unclear, although several theories have been proposed. Previous light microscopic descriptions of posttraumatic syringomyelia have indicated that the cavity was lined by gliosis or collagen with little or no ependymal component. Changes in the surrounding neural tissue such as enlarged perivascular spaces, perivascular collagen, and “schwannosis” have also been described. In view of the lack of information on the ultrastructural features of posttraumatic syringomyelia, this report presents features of the syrinx cavities and surrounding spinal cord in cordectomy specimens obtained from two patients with posttraumatic syringomyelia as observed on light microscopy, scanning electron microscopy, and transmission electron microscopy.

Clinical Material and Methods

Case Material

Case 1. This 29-year-old previously healthy man was rendered quadriplegic below the C-5 vertebral level following a diving accident in 1981. The patient presented 6 years later with a history of bilateral shoulder numbness, episodic hyperhidrosis, and hypertension. There were no new objective findings. Computerized tomography (CT) myelography demonstrated a syringomyelic cavity extending from the midcervical to the T-2 vertebral level. Selective cordectomy (2.5 cm length) was performed at the caudal end of the syrinx, producing a communication between the syrinx and the subarachnoid space. Postoperatively, the patient had considerable improvement in his autonomic symptoms. The cordectomy specimen containing the tip of the syrinx was used for this study.

Case 2. This 62-year-old man sustained injuries in a motor-vehicle accident in 1947 at the age of 21 years, resulting in quadriplegia below the C-6 vertebral level. He presented with a 15-year history of slowly progressive ascending neurological deficit culminating in respiratory distress and right facial paresis. A CT myelogram showed a syrinx extending from the cervico-medullary junction to the T-6 vertebral level. Selective cordectomy was performed at the caudal end of the radiologically defined syrinx. Postoperatively, the patient had considerable improvement in his symptoms. Part of the 2-cm long specimen containing the tip of the syrinx was used for this study.

Tissue Preparation

The excised spinal cord specimens were divided into sections 0.5 cm thick and fixed in 2.5% glutaraldehyde/2% paraformaldehyde in 1.0 mM phosphate buffer for 48 hours. Each section was subdivided and postfixed in buffered 2% osmium tetroxide for 2 hours and then dehydrated in graded ethanol solutions. For scanning electron microscopy, the specimens were critical-point dried in CO₂, split, sputter-coated with gold:palladium.
K. K. V. Reddy, M. R. Del Bigio, and G. R. Sutherland

(60:40), and examined with a JEOL JSM-35C scanning electron microscope. The remaining tissue samples were transferred into propylene oxide for Epon embedding. For light microscopic examination, the specimens were sectioned 1.0 μm thick and stained with methylene blue/azure II. For transmission electron microscopic examination with a Philips 201 unit, ultrathin sections were cut and stained with uranyl acetate and lead citrate.

Results

The cord specimen obtained from Case 1 revealed discoloration of the corticospinal tracts and a slit-like cavity in the center of the spinal cord (Fig. 1). Light microscopic examination demonstrated continuity between the syrinx and the ventral median fissure (Fig. 2 left). The loose connective tissue of the fissure blended into a network of glial cells lining the syrinx. The syrinx lining consisted predominantly of fibrillary processes that were filled with astroglial intermediate filaments (Fig. 2 right). Small areas of the syrinx were lined by flattened cells that had ependymal characteristics. Along the cavity lining were varicose fibers 0.25 μm in diameter, believed to be supraependymal axons. A few macrophage-like cells with ovoid bodies and multiple pseudopodia were also observed on the lining of the syrinx. The syrinx tapered caudally and ended blindly in a disorganized cluster of ependymal cells surrounded by reactive astroglial cells. Approximately 50 ependymal cells were observed in a single cross section. The cells did exhibit zonulae adherens junctions and occasional rosette formation, but lacked polarity and surface specialization. No patent central canal was identified.

In the cord specimen obtained from Case 2, the
Ultrastructure of posttraumatic syringomyelia

termination of the syrinx was a Y-shaped cavity lying just lateral to the center of the cord. In the specimen, no communication was observed between the syrinx and the ventral median fissure. The syrinx cavity was lined mainly by a meshwork of glial cells. There was, however, substantial coverage by smooth-surfaced cells with ultrastructural features of ependymal cells (Fig. 3). Within the wall of the syrinx were large arterioles, 25 to 60 μm in diameter, and venules with organized thrombus (Fig. 4). The syrinx tapered caudally and ended over a collection of disorganized ependymal cells. These cells lacked polarity and did not form a patent central canal. The latter features were similar to those in Case 1.

In both specimens, numerous blood vessels in the vicinity of the syrinx were surrounded by collagenous tissue (Fig. 5). In addition, there were some larger blood vessels, however, this was interpreted to be an artifact of fixation rather than pathological enlargement.

Discussion

Posttraumatic syringomyelia has been reported to occur in 1% to 1.8% of patients sustaining spinal cord injury. The early descriptions of light microscopic features of posttraumatic cord cavitation by Hallopeau and Holmes were followed by pathological descriptions of posttraumatic syringomyelia in numerous cases. Detailed ultrastructural studies are not available, however, with the exception of a single micrograph showing glial fibrils lining an early posttraumatic cavity.

Syringomyelic cavities, which when multiple may or may not intercommunicate, are usually not reported to be in continuity with the central canal. The cavities may extend cranially or caudally from the site of the injury. The lining of the cavity may have a “chamois leather-like” appearance on gross examination. Consistent with previous light microscopic observations, the lining in our cases consisted of fibrillary structures with ultrastructural characteristics of astroglial cell processes. Unlike most previous reports, however, a portion of the lining in both cases consisted of smooth cells that had cytoplasmic and nuclear characteristics resembling those of mammalian ependymal cells. Following trauma, the ependyma of the central canal in adult mammals is capable of proliferation, albeit localized and limited. Therefore, we believe that the

---

FIG. 3. Sections of spinal cord from Case 2. Left: Scanning electron micrograph showing a smooth area lining the syrinx. The cells, which are likely ependymal, are largely devoid of surface features except for some microvilli. Supraependymal axons are evident (arrow). Bar = 10 μm. Right: Transmission electron micrograph of tissue near the caudal end of the syrinx (S). Ependymal cells (E) lacking polarity and surface specializations are present along part of the wall of the syrinx. The remainder of the cavity is lined by the processes (arrow) of astroglial cells. Bar = 5 μm.
FIG. 4. Scanning electron micrograph showing the caudal end of the syrinx (asterisk) in Case 1. Two thrombosed blood vessels (arrows) lie in the wall of the syrinx. Bar = 50 μm.

FIG. 5. Transmission electron micrograph of a capillary adjacent to the syrinx. The endothelial cell layer is continuous (open arrow). Surrounding the endothelium is a layer of collagenous connective tissue (solid arrow). An erythrocyte (e) is seen in the center. Bar = 2 μm.

Ependymal cells in our two patients are remnants of preexistent ependymal lining of the central canal. Caudal to the syrinx cavities were clusters of disorganized ependymal cells, further supporting the concept that the central canal gave rise, in part, to the syrinx or that it was subsequently incorporated into the cavity and was not of mesothelial origin or the result of ependymal metaplasia as previously suggested. Blood vessels with organized thrombus were observed in the wall of the syrinx in our Case 2. This has been described previously in association with communicating syringomyelia, but we are uncertain of the significance. Collagen in the perivascular spaces of syringomyelic spinal cords is believed to reflect organization related to the initial trauma. The previously reported presence of collagen in the lining of posttraumatic syrinx has not been found in our cases.

The pathogenesis of posttraumatic syringomyelia is poorly understood. Proposed mechanisms for the formation of the syrinx cavity include: resorption of blood and necrotic tissue, lysosomal autodigestion, secretion of cyst fluid by the lining, and infarction. Passage of cerebrospinal fluid via enlarged perivascular spaces or direct communication with the subarachnoid space has also been suggested. The latter theory is supported by occasional radiologic and pathological demonstration of such direct communications. The findings in our Case 1 also support this theory, although causality cannot be established. Posttraumatic reactive ependymal proliferation causing segmental closure of the central canal with the resulting local distension leading to syrinx formation has also been proposed.

The findings of mural gliosis, patchy denudation, and flattening of the ependyma and the occasional presence of macrophages are similar to appearances within the ventricular wall in human and experimental hydrocephalus. Comparable findings have also been described in experimental feline and canine communicating syringomyelia. The similarities suggest that the pathological findings noted here are the nonspecific results of a distensile force, although the causation of this distensile force remains speculative.

Acknowledgments

The authors thank Roy Simpson, Department of Anatomy, for his help in preparing the micrographs, and are grateful to Eileen DeSouza and Emi Okamoto for preparing this manuscript.
Ultrastructure of posttraumatic syringomyelia

References


Manuscript received October 31, 1988.
Address reprint requests to: Garnette R. Sutherland, M.D., Departments of Surgery and Pharmacology, 61 Emily Street, Winnipeg, Manitoba R3E 1Y9.