Experimental Syringo-Hydromyelia Induced by Adhesive Arachnoiditis in Rabbit
Ultrastructural Changes by Cerebro-Spinal Fluid (CSF) Flow Disturbance and Syrinx Formation

INTRODUCTION: Much remains unknown about the pathogenesis of syringo-hydromyelia, although the presence of spinal arachnoiditis has been identified as a precursor to its onset. So far, there are many histological examination of syringomyelia in the literature, however, there has been very little experimental work on blood permeability in the spinal cord vessels and ultrastructural changes. The aim of the present study was to investigate the mechanism of onset of syringomyelia using an adhesive thoracic arachnoiditis model created by injection of Kaolin into the subarachnoid space. For the purposes of this study, we defined spinal cord cavities as matching either of two histopathological conditions. A cystic dilation of the central canal called hydromyelia and the other parenchymal cavity without ependymal lining called syringomyelia.

METHODS: 37 adult rabbits (2.5-3.0 kg) were used. As controls, 7 animals only underwent laminectomy of the 8th thoracic vertebra, and 30 animals had 0.1 ml of 30% Kaolin solution injected into the subarachnoid space (SAS). All these procedures were conducted under general anesthesia. The animals were sacrificed 4 months later, and changes of the spinal cord and the subarachnoid space were examined. Histopathological examination of the spinal cord and subarachnoid space was done by light microscopy (HE and LFB-PAS staining) and by scanning (SEM) and transmission electron microscopy (TEM). Changes of vascular permeability within the spinal cord were examined using Evans blue albumin (EBA) as the tracer and fluorescence microscopy (FM) for observation.

RESULTS: Of the 30 rabbits given Kaolin injection into the cerebrospinal fluid, and 23 showed complete circumferential obstruction of SAS. In the rabbits with partial obstruction, intramedullary changes and dilatation of the central canal were not observed (Fig.1B). However, among the 23 animals showing complete obstruction, dilatation of the central canal (hydromyelia) occurred in 19 (Fig.1C) and an intramedullary syrinx (syringomyelia) which is a syrinx with no connection to the central canal was observed in 9 (Fig.1D). In animals with complete obstruction, FM revealed intramedullary edema around the central canal an extending to the posterior column (Fig.2). SEM of hydromyelia revealed a marked reduction of villi on the ependymal cells, separation of the ependymal cells and cavitation of the subependymal layer (Fig.3). The dilated Virchow-Robin spaces below the Kaolin granulation indicate alterations of fluid exchange between the SAS and extracellular spaces and may even represent increased flow in these extracellular pathways to compensate for the CSF flow interference at the circumferential obstruction of the SAS (Fig.4). Syringomyelia revealed that nerve fibers and nerve cells were exposed on the surface of the syrinx (Fig.5A,B). Monocytes (macrophages) capable of passing through the vessel walls were observed around the syrinx wall (Fig.5C). The apoptotic ventral neurons were reduced in size and chromat condensation was seen in the nuclei (Fig.5D). Remodeling process was also seen in some axons with simultaneous degenerative changes in edematous areas (Fig.5E). Degenerative and necrotic tissue was removed by macrophages to leave a syrinx (Fig.5F).

DISCUSSION: The results of this study indicated that two types of cavities, or syrinxes, were present after adhesive arachnoiditis: hydromyelia, in which the central canal is dilated, and syringomyelia, in which there is no communication with the central canal. Disturbance of CSF flow associated with circumferential obstruction of the subarachnoid space appears to be closely related to the onset of hydromyelia. In contrast, the onset of syringomyelia seems to be closely linked to the breakdown of the blood-spinal cord barrier caused by disturbed intramedullary blood flow and the intramedullary edema that occurs as a result. In other words, intramedullary edema leads to necrosis of nerve tissue necrosis, and cavities are formed when the necrotic tissue is phagocytosed by macrophages.

SIGNIFICANCE: These findings suggest a cellular basis for unchanged or worsened clinical conditions of syringomyelic patients after surgical treatment.

REFERENCES: