Neuronal and vascular changes in Syringomyelia: investigations using longitudinal MRI

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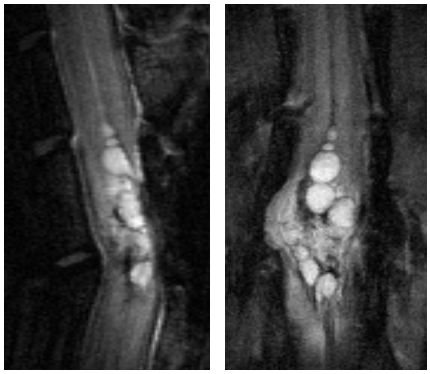
Introduction

Syringomyelia is a formation of fluid-filled cavities in spinal cord (SC) as a consequence of a number of reasons including trauma [1]. Clinical magnetic resonance imaging (MRI) is broadly used as a sensitive and specific method to detect this condition in human patients and visualize the extent of pathology [2]. To understand the origin and progression of syringomyelia, animal models were developed for mimicking the condition and computer models were implemented for performing numerical analysis. This paper characterizes the neuropathological stages of contused spinal cord before, during and after the formation of syringomyelia using longitudinal in vivo MRI. High resolution volumetric anatomical data depicting the spatial and temporal changes in syringomyelia should be highly useful in deepening the understanding of its formation, constructing numerical models and testing their predictions.

Materials and Methods

All MRI scans were performed on a 9.4 T INOVA Varian system (Varian Inc., Palo Alto, CA) with 31 cm horizontal bore magnet. The SC of Sprague Dawley rat was subject to a contusion-type injury at the vertebral T12 level. Anatomical MRI scans were performed on post-injury days 3, 14, 28, 72, 94 and 404. Data acquisition protocols included spin-echo (SE) and 3D gradient-echo (GE) sequences. The SE acquisitions included proton density (PD) and T2-weighted imaging. The acquisition parameters were T_R/T_E =2000 ms/ 12 ms or 26 ms, image matrix = 128x128 and averages=2. Field of view and slice thickness were 36 mm X 18 mm and 1 mm for the sagittal and coronal, and 18 mm X 18 mm and 2 mm for the axial images. Volumetric GE data were gathered for visualizing the arterial blood vessels using time-of-flight contrast, but without suppressing the background to serve as an anatomical reference. The parameters for these acquisitions were TR/TE = 45/4 ms and flip angle (FA) = 45°, and applied over a volume of 36x18x15 mm^3, where the former measure represents the dimension in the readout direction. The raw data were digitized at a sampling rate of 128x128x64, but later processed and interpolated to a matrix size of 256x256x128 pixels before the visualization and qualitative analysis. In addition, neurobehavioral tests were performed prior to the scans on these days.

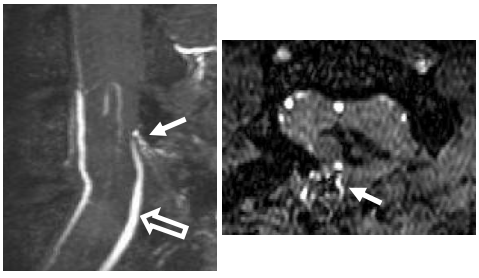
Figure 1. T2-weighted images visualizing the injured SC in sagittal and coronal views on post-injury day 94.



Results and Discussion

Response of the SC to the injury was revealed by the collected images. Pathological consequences included significant edema and, to a lesser degree, hemorrhage in the acute phase, followed by neuronal loss, tissue alterations and vascular changes in the late stages. The images from the post-injury day 14 indicated shrinkage of the injured tissue and occlusion of the central canal. Subsequently, the restricted flow of the cerebrospinal fluid initiated the formation of syringomyelia rostral to the occlusion. Concomitantly, the fluid-filled cavities enlarged with time (Fig. 1). Another important observation was angiogenic vessels, which projected from the posterior spinal artery to provide blood to the scar tissue deposited above the laminectomy Fig. 2. The neurologic deficits of the injured rat worsened with time. During the studies, the rat was experienced an early weight lost that was followed by a gradual weight gain, reaching to a range comparable to its same age but uninjured counterparts.

Figure 2. GE images visualizing the injured spinal cord in sagittal and axial views on post-injury day 28. Arrows point to vascular angiogenesis branching from posterior spinal artery to supply blood scar tissue. Open arrow points to enlarged posterior spinal artery in segments below the injury.



Conclusions

Contused rat SC develops syringomyelia with time. Longitudinal MRI visualizes the initiation and progression of syringes with fine details. The data indicates the inflammatory, but not the haemostatic, component be a prerequisite for syringomyelia. Abnormal flow of cerebrospinal fluid is likely to be a prevalent factor producing syringomyelia. Therefore, accurately measuring the cerebrospinal fluid flow and detecting any changes in the flow dynamics may be used as a bioimaging marker for the early detection of syringomyelia and take counter measures for its prevention. Feeding the high resolution anatomical data into the computer models should also allow testing current or future hypotheses formulated for explaining the precursorory events leading to syrigomyelia.

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References

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