MRI-based investigation on the outflow segment of human brain bridging veins

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Objective: Increased intracranial pressure (ICP) is responsible for inducing almost all nervous system diseases into serious progress until death. Recently volume-targeted therapeutic strategy against increased ICP, which release excessive intracranial liquid volume especially venous part, attracted more and more attention. Preclinical and clinical researches both proved drug-induced venous fluid draining or reabsorbing could alleviate even recover increased ICP. Our previous research found there existed a structurally and functionally special “outflow segment cuff” which locates at the juncture of superior sagittal sinus (SSS) and brain bridging veins in porcine model (Figure 1). Based on those findings we propose a further hypothesis at present study that there exists a similar structure in human being which might play a significant role to influence volume-induced ICP regulation in vivo.

Method: Forty patients that presented both increased ICP symptoms and more or less hydrocephalus were selected to undergo 2D time of flight (2D-TOF) venography and ten normal volunteers were selected as control group. Continuous ICP monitoring was not evaluated because of clinical research limitation (Most patients suffer from intracranial space-occupying lesion and ICP monitoring is not always necessary for them). All patients were underwent 2D-TOF venography with following parameters: repetition time/echo time, 50/4.9 milliseconds; flip angle, 45°; field of view, 250 ×250 mm; matrix, 256×256 pixels; section thickness, 1.5 mm. Normal MRI scanning was performed for selecting appropriate patients. Syngo fastview imaging system was used to process and analyze the targeted brain bridging venous section

Result: Most bridging venous profiles could be better visualized as well as SSS and vicinal cortical veins in 2D-TOF method in vivo in increased ICP patients. A short and narrow length, as previously described, was obviously shown resulted from MRI signal weakness even disappearance at the juncture of SSS and bridging veins (Figure 2). According to animal experimental findings we assumed this section of length with abnormal MRI signal should stand for similar bridging vein structure in human being. Such a special structure could be observed within a majority of increased ICP patients (32/40 cases), while only 1 case shown the existence of similar imaging structure.

Conclusion: Intracranial venous compartment occupies larger part of about 70-80％ blood volume inside inflexible cranial cavity. Our previous animal experiments have proven fluctuation of venous blood volume can regulate ICP effectively following volume-targeted rationale from different aspects of morphology, biomechanics and hemodynamics. At the same time the outflow segment cuff structure was evidenced as the possible key regulator. At present study the coincidence of animal model and human venography in vivo affords further evidences for the hypothesis that venous hemodynamics passively influence intracranial pressure environment by means of special venous structure. This narrow section formation which appears more at the patients with high ICP instead of normal pressure state indicates an important relationship between intracranial venous volume and ICP. We can postulate that the existence of such a narrow part deteriorates venous hypostasis by limiting blood draining out and further increase ICP. Meanwhile, the investigation based on MRI technology in vivo will provide a very useful and effective method for future research involved in venous blood volume-induced decreasing ICP therapy

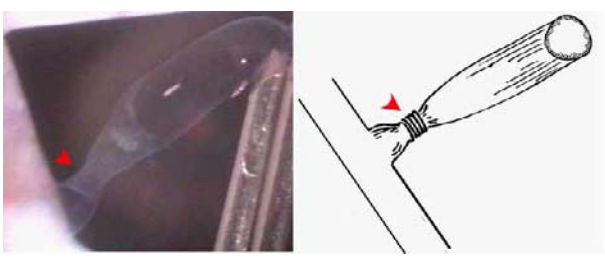

Figure 1: outflow segment cuff (red arrow)

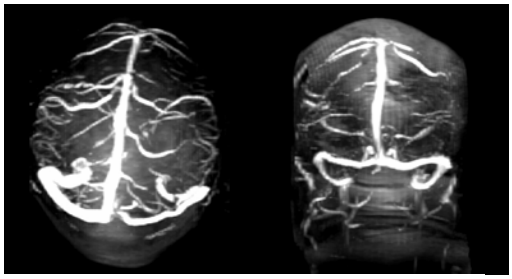

Figure 2: signal weakness between SSS and bridging veins