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Subdural effusion protects the aging brain from harmful ventriculomegaly

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ABSTRACT

The human brain loses its volume and its function during aging. The solid part of the brain within the intracranial space, the brain parenchyma, decreases in volume with age; while the cerebrospinal fluid (CSF) volume increases. With progressive loss of brain parenchymal volume (BPV), CSF may shift from cerebral ventricles to the subdural space, forming subdural effusion (SDE), whose role in the brain aging process remains unclear.

We hypothesize that damages associated with ventriculomegaly can be lessened after formation of SDE. As the BPV decreases, the enlarged ventricular surface area causes dysfunction of its lining ependymal cells, followed by damages to the periventricular tissue. The periventricular nerve fibers are stretched by the enlarged ventricles. We hypothesize that after the formation of SDE, ventriculomegaly can be stopped or even reversed. By allowing the atrophic brain to reside in a smaller fraction of the intracranial volume, damages associated with ventriculomegaly can be alleviated.

If our hypothesis is correct, physicians should continue to maintain a conservative approach for uncomplicated SDE. For focal or global brain parenchymal loss caused by various pathologies, intracranial spacers can be employed to simulate the effect of SDE to protect the brain. For treatment of idiopathic normal pressure hydrocephalus, aggressive ventricular size reduction should be pursued. Finally, the protective effects of SDE have its limits. Extremely enlarged subdural volume can cause acute or chronic subdural hematoma, further damaging the brain.

Background

The human brain loses its volume and weight during aging, and its function also deteriorates. In addition to postmortem examination, noninvasive volume measurement (volumetry) of various intracranial compartments is possible with modern neuroimaging including computed tomography (CT) and magnetic resonance imaging (MRI). Even without overt change in cognition, the brain parenchymal volume (BPV) decreases and the cerebrospinal fluid volume (CSFV) increases with age, while the total intracranial volume (ICV) does not change [1]. The decrease in the BPV is predominantly reflected by expansion of the extraventricular CSF volume (EVV). Although the ventricular CSF volume (VV) also increases with age, it is much smaller than the EVV.

The process of brain atrophy with aging and relationships between these volumes can be illustrated by Figs. 1 and 2. Normally the EVV is composed of CSF within the subarachnoid space (SAS) located in fissures and sulci (Fig. 1, uppermost). With aging, the brain shrinks while fissures and sulci dilate (Fig. 1, middle right). When the arachnoid trabeculae in the SAS are intact, the brain is tethered to the inner surface of the skull, leading to increased VV, or ventriculomegaly (Fig. 2, upper). With tensioned arachnoid trabeculae disrupted after minor trauma or even with activities of daily living, the subdural space opens, allowing CSF to flow into it and redistributing the CSFV away from the ventricles (Fig. 2, lower).

The CSF-like fluid within the subdural space is called subdural effusion (SDE), also known as subdural hydroma or subdural hygroma [2,3]. The formation of SDE causes a significant increase of the EVV (Fig. 1, middle left). However, this process does not occur in an "all or none" fashion. Because there are numerous arachnoid trabeculae, their disruption can be fragmentary and can occur progressively. However, with serial neuroimaging

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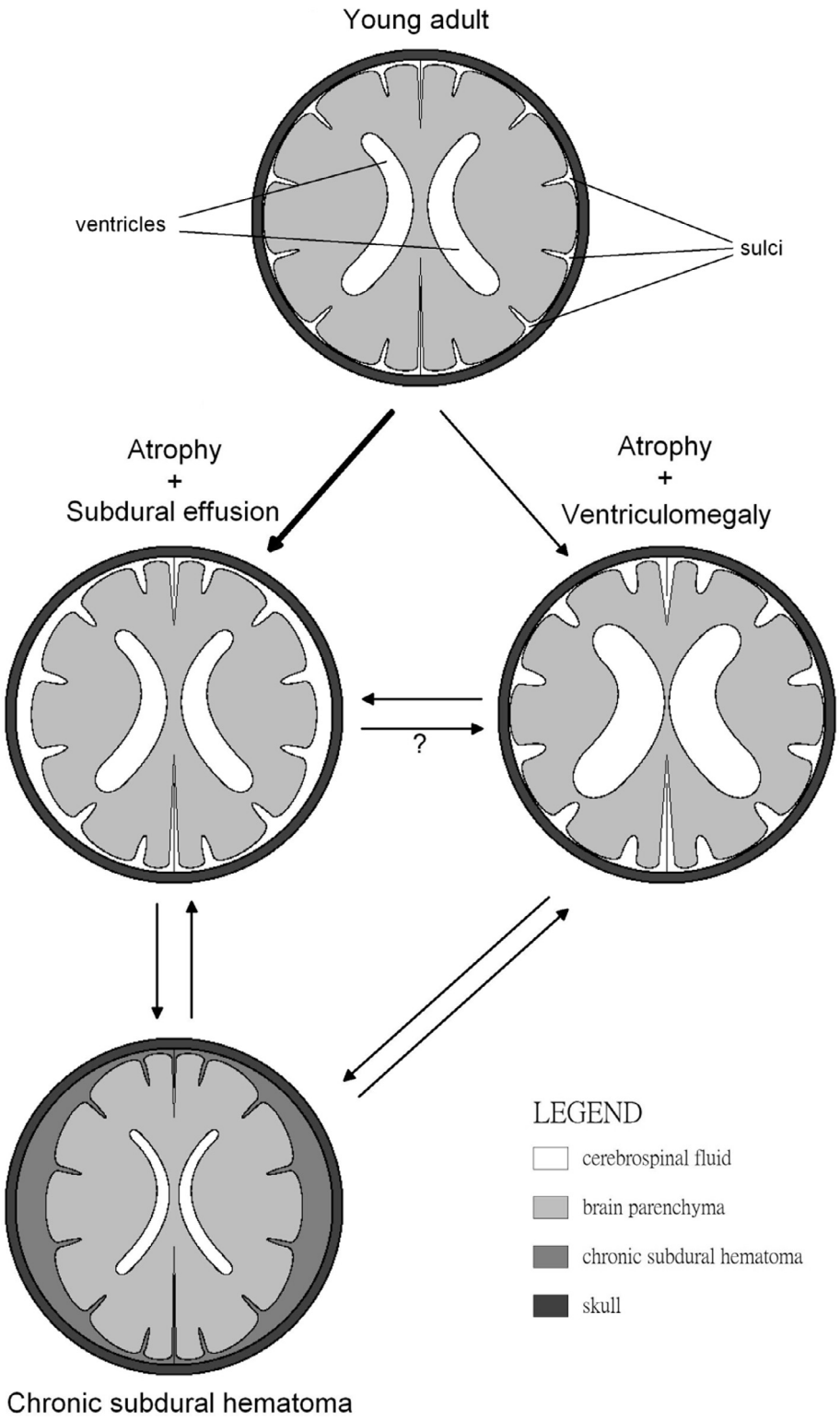

Fig. 1. Models of horizontal brain slices across lateral ventricles showing relationships between different conditions that accompany brain atrophy with aging.

follow-up, which can only be done intermittently, "de novo" formation of SDE in large amount is frequently observed in the elderly (thick arrow in Fig. 1). Moreover, severity of arachnoid trabeculae disruption also varies between subjects, resulting in subdural effusion accounts for a larger portion of the total CSFV in some, while ventricular CSF is more abundant in others. On the other hand, whether SDE may shift back to the ventricles remains unknown (denoted by the question mark in Fig. 1).

Despite its non-negligible volume, SDE was merely treated as a by-product of brain atrophy and therefore understudied. In contrast, chronic subdural hematoma (cSDH), another well-known complication of head injury in the aging population amenable to