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## 封面文章

脑小血管病(cerebral small vessel disease, CSVD)是指各种病因影响脑内小动脉、微血管及静脉系统所导致的一系列临床、影像及病理综合征。随着全球人口老龄化的加剧,CSVD已成为导致老年人血管性认知障碍、步态异常及卒中的主要原因,给公共卫生系统带来了沉重负担。在影像上CSVD常表现为脑白质高信号、腔隙、微出血及血管周围间隙扩大等多种标志物的共存。既往研究多聚焦于单一标志物的影响,往往忽视了这些病变对大脑产生的累积总负荷效应。CSVD本质上是一种同时累及皮层及皮层下结构的疾病。然而,关于累积的CSVD负荷如何具体影响关键皮层下核团(如丘脑)的精细结构与功能,以及大脑在结构受损后功能如何变化,目前尚缺乏系统性研究。

针对这一问题,我们创新性地结合高分辨率皮层下核团图谱与多模态磁共振成像技术,深入探索了120例CSVD患者在不同总负荷下的脑结构与功能变化。研究发现,随着CSVD总负荷的不断累积,大脑呈现出一种独特的“双模式改变”:一方面,关键的认知枢纽——丘脑及感觉运动皮层表现出显著结构萎缩,且在重度负荷阶段最为严重;另一方面,丘脑和左侧颞极的功能活动在重度阶段不仅未下降,反而异常升高。

这种“结构萎缩与功能代偿并存”的现象,不仅揭示了CSVD认知损害潜在的神经基础,更为临床早期识别和干预提供了新的影像学标志物。详见内文第36页。

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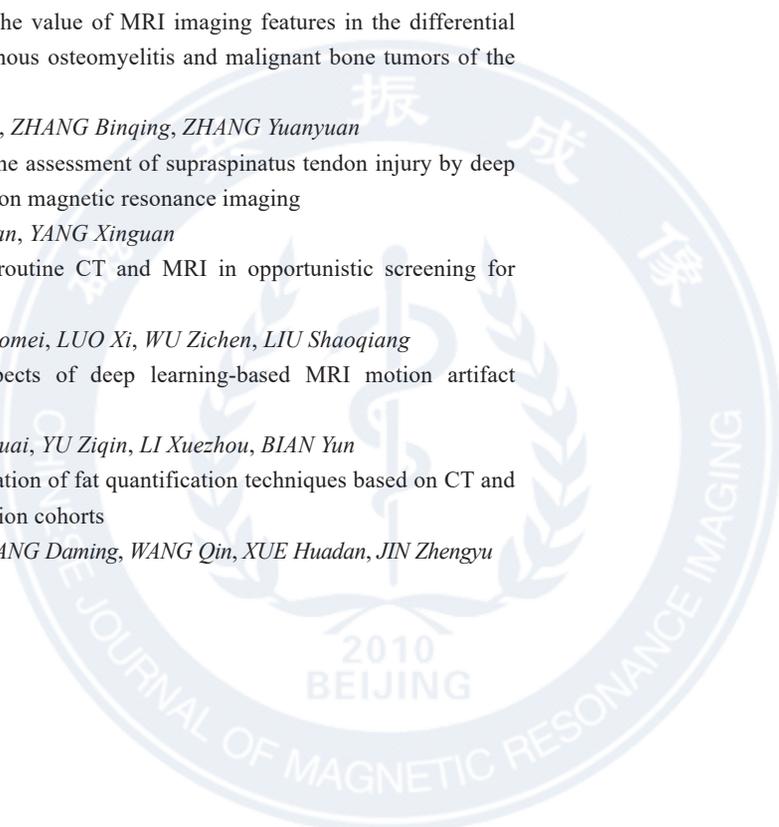
#### About the cover

Cerebral small vessel disease (CSVD) is a clinical, imaging, and pathological syndrome caused by various etiologies affecting the small arteries, arterioles, capillaries, and venules of the brain. With the accelerating global aging population, CSVD has become a primary cause of vascular cognitive impairment, gait abnormalities, and stroke in the elderly, imposing a significant burden on public health systems. Radiologically, CSVD typically manifests as the coexistence of multiple markers, including white matter hyperintensities, lacunes, cerebral microbleeds, and enlarged perivascular spaces. While previous studies have largely focused on the impact of individual markers, the cumulative "total score" effect of these lesions on the brain has often been overlooked. CSVD is intrinsically a disease that affects both cortical and subcortical structures. However, how the cumulative CSVD burden specifically impacts the fine-grained structure and function of critical subcortical hubs (such as the thalamus), and how brain function evolves following structural damage, remains poorly understood.

Regarding this issue, we innovatively combined the high-resolution Tian Subcortical Atlas with multimodal MRI techniques to systematically investigate brain structural and functional alterations in 120 CSVD patients across different total burden scores. The study revealed a striking "dual-pattern alteration": as the total CSVD burden accumulated, key cognitive hubs—the thalamic-somatomotor network—exhibited significant structural atrophy, with the greatest severity at the highest burden stage. Yet, remarkably, the local functional activity (ReHo) in the thalamus and left temporal pole did not decline but instead became abnormally hyperactive at severe stages.

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