MRI might identify patients at risk of post-stroke thalamic pain

Researchers at the Technische Universität München in Munich, Germany have identified thalamic areas consistently involved in the generation of post-stroke pain. After a thalamic stroke, ~7% of patients develop severe, distressing neuropathic pain, typically accompanied by sensory disturbances, and characterized by thalamic lesions. “Our aim was to determine if the location of these lesions could be used to predict if a particular patient was at high risk of developing thalamic pain,” explains Till Sprenger, lead author of the study.

The research team compared structural imaging data from 10 patients with post-stroke thalamic pain and 10 pain-free control patients who had experienced a thalamic stroke >2 years previously. The application of nonlinear deformations to map each volumetric structural MRI image into standard stereotactic space facilitated voxel-by-voxel analysis of the thalamic volumes of interest. Odds ratios were also created for each individual voxel, to create a high-resolution risk map for post-stroke pain.

Control patients tended to have medial lesions; by contrast, nine of the patients with post-stroke thalamic pain had lesions affecting a region bordering the ventral posterior thalamic nucleus and pulvinar, coinciding with the ventrocaudalis portae nucleus (the remaining patient had a lesion adjacent to, but not including this area). “Lesions in this area produce exceedingly high odds of developing thalamic pain,” Sprenger notes.

Although the patients with thalamic pain typically had significantly larger lesions than controls, Sprenger and colleagues consider the precise lesion location to be the principal determinant of the risk of post-stroke pain. As Sprenger points out, specific pain and temperature relay neurons are indeed thought to be located in an area overlapping the ventrocaudalis portae nucleus.

Whether at-risk patients can be identified using these MRI features awaits investigation in prospective studies. The team also hope to clarify the pathophysiological mechanisms underlying thalamic pain. “The study of brain plasticity in these patients would be of great interest,” Sprenger says, “as reshaping of the functional network probably ultimately produces the pain.”

The pain-free interval after a thalamic lesion might also offer an important time window for preventive treatment. The authors speculate that drugs such as antidepressants, anticonvulsants or N-methyl-D-aspartate receptor antagonists could be tested in at-risk patients to see whether they can prevent the onset of symptoms. “Early interventions may be necessary and we believe that such strategies can now be tested,” Sprenger concludes.

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