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**Can MR help predict enlargement of  
posttraumatic spinal cord cysts?**

R M Quencer

*AJNR Am J Neuroradiol* 1998, 19 (1) 192

<http://www.ajnr.org/content/19/1/192.citation>

This information is current as  
of April 17, 2024.

it will not represent as valuable a tool as suggested by the authors. We look forward to the investigation of these threshold levels, and wish the authors luck in their search of the holy grail of cerebral ischemia.

WILLIAM P. DILLON  
Senior Editor

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## Can MR Help Predict Enlargement of Posttraumatic Spinal Cord Cysts?

Progressive enlargement of posttraumatic syrinx cavities occurs in a small percentage of patients who have suffered a severe spinal cord injury in the past. The exact mechanisms that cause this enlargement and why such enlargement is found in some patients but not in others remains uncertain. However, there are observations that could help explain the insidious progression of this posttraumatic complication, including the presence of scarring and adhesions within the subarachnoid space, alterations in cerebrospinal fluid flow dynamics, and fluid turbulence within the syrinx cavity itself. The article by Jinkins et al in this issue of the *AJNR* presents us with an MR finding not previously emphasized in patients with clinically pro-

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gressive posttraumatic syringomyelia. A significant extent of increased signal on T2-weighted images cephalic to the syringomyelia is suggested by the authors to be present in those patients who are worsening clinically and whose cysts are enlarging. That observation raises a number of questions worthy of further investigation.

The fact that this extensive abnormal signal disappeared after a cyst shunting procedure suggests that this is a situation analagous to the periventricular edema frequently observed in obstructive hydrocephalus. Clearly, if these signal changes were caused by neural tissue destruction alone, signal reversibility would not be seen after cyst peritoneal shunt placement. A possibility is that this pericyclic edema might,

in combination with other dynamic changes, hasten or potentiate cyst enlargement and therefore account for subsequent clinical deterioration. This interesting observation is limited, however, by the small number of patients who formed the basis of the report; only six patients were studied, three of whom were clinically unstable, and three of whom were clinically stable and had short-segment cysts with minimal or no abnormal signal adjacent to the syrinx.

A larger number of both clinically progressive and stable patients is required to determine the reproducibility of these findings and to determine whether the signal changes will serve as a useful sign for predicting cyst expansion. The extension over time of the hyperintense parenchymal signal in conjunction with enlargement of the syrinx would lend further weight to the postulate that the abnormal signal represents spinal cord edema. Were that the situation, one could argue for surgical treatment even in the absence of obvious progressive symptoms, or at a minimum one could recommend more frequent MR monitoring of the affected spinal cord. In any event, the wisdom of obtaining T2-weighted images when a well-documented posttraumatic syringomyelia is identified on the T1-weighted images is suggested by this article. Evaluation of all these MR features should deepen our understanding of the pathophysiology of progressive posttraumatic cyst enlargement.

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