Differentiating Vascular Myelopathy from Transverse Myelitis

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Introduction

- Nearly 400 patients presented to the Johns Hopkins Transverse Myelitis Center (JHTMC) for Outpatient evaluation between July 2010 and June 2012. Patients with vascular myelopathies and inflammatory myelopathies were frequently presented to our center with similar clinical and neuroimaging features.
- Most patients who were later proven to have vascular myelopathies including stroke, arteriovenous fistulas (AVF), and arteriovenous malformations (AVM) were first treated as inflammatory TM. This prompted us to investigate how to better differentiate these two different diagnoses so that specific treatment could be optimized.
- Spinal angiography is the gold standard for diagnosing vascular problems in the spinal cord, but it is impractical to perform a spinal angiogram on every patient who presents with acute myelopathy since spinal angiograms are an expensive procedure and the technical resources and skillful neuroradiologists may not be readily available at all facilities.
- The main goal of our study was to evaluate clinical, neuroimaging and laboratory indicators that may help to differentiate inflammatory from vascular myelopathies.

Methods

- We performed a retrospective chart review on all patients that received clinical care at the JHTMC between July 2010 to June 2012 who were diagnosed and treated for transverse myelitis. Patients who had a comprehensive assessment during the acute phase of their illness including spinal cord MRI with and without gadolinium, cerebrospinal fluid (CSF), and who had a spinal angiogram at any point in their work-up were included to study the variables associated with a definitive diagnosis of inflammatory versus vascular myelopathy. We excluded those patients with identifiable myelopathies to focus on the presentation of idiopathic inflammatory and vascular myelopathies.
- We examined 49 different variables including clinical profile, CSF data, MRI data, vascular risk factors, and response to acute treatment to assess what factors may help to differentiate myelopathic syndrome with which the patients present.

Results

- We found 24 patients who fulfilled all criteria; 12 were ultimately diagnosed with inflammatory TM; 12 were later confirmed to have vascular etiologies. Of the 12 vascular, 3 were spinal cord strokes and 9 were AVF/AVM.
- CSF pleocytosis in the acute phase of presentation was the only significant indicator that helps to establish a diagnosis of inflammatory TM (p=0.0028), while increased age and longer symptom evolution are indicators favoring vascular myelopathy although these did not reach significance (Table 1).
- Patients with hyperacute presentations (time to nadir < 4 hours) were found to be angio-proven strokes and the majority of patients with a chronic presentation of symptom evolution were found to have AVF/AVMs (Figure 1). In contrast, the final diagnosis on the majority of patients who presented with an acute or subacute symptom evolution (4 hours to 21 days) was inflammatory, which is consistent with the AAN 2011 TM Guidelines.
- Other clinical indicators did not significantly associate with inflammatory myelitis versus vascular myelopathy, including 15 different MRI characteristics. This adds support to the challenge that practitioners face when presented with acute myelopathy patients and how to correctly diagnose and treat them.
- This study widely accounts for many variables when looking at the differences in presentation of inflammatory versus vascular myelopathic syndromes, but was limited to those patients who obtained spinal angiography. We plan to more widely collect data for all patients who present the JHTMC to see if the trends found continue and new ones emerge.
- In the future, we plan to use the knowledge gained from this pilot study and from future studies to develop a classification scale using a weighted set of criteria to include MRI, CSF, and clinical data to determine the likelihood of a diagnosis of inflammatory versus vascular myelopathies.

Conclusions/Future Directions

- We need to sufficiently powered studies that include additional variables that may explain the differences in vascular versus inflammatory myelopathy. These could include the number of lesions, the location of the lesions, and their characteristics. Future studies could also explore the role of hippocampal atrophy and cerebrospinal fluid (CSF) biomarkers in differentiating between these two conditions.

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