Developmental Venous Anomalies (Venous Angiomas) on MRI Are More Common in Patients with Multiple Sclerosis

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Developmental venous anomalies (DVA), also known as venous angiomas (VA), are the most common intracranial vascular lesions detected by magnetic resonance imaging (MRI) or computed tomography (CT). They are characterized by a leash (caput medusae-like) of multiple venules converging on a single, or occasionally multiple, enlarged parenchymal or medullary vein. Most DVA are clinically asymptomatic and surgery is usually not necessary. Although the exact pathogenesis of DVA is not known, a congenital basis is most likely. The purpose of the present study was to describe the prevalence of DVA in patients with multiple sclerosis (MS).

Material and Methods.

700 adult patients with clinically definite or laboratory supported MS enrolled in three therapeutic trials were studied. Eligible patients had been in the relapsing-remitting or secondary progressive course of the disease for at least one year’s duration, and who had progressed by one point in their Extended Disability Status Scores (EDSS) (or 0.5 point between EDSS 6.0 and 6.5) over the preceding two years. There were no MRI inclusion criteria regarding minimum numbers of MS lesions or presence of gadolinium enhancing lesions.

All patients had brain MRIs performed according to a strict repositioning protocol using a variety of scanners (GE, Siemens, and Philips), operating at field strengths ranging from 0.5 T to 1.5 T. Either twenty-four 5mm or fifty 3mm thick slices, contiguous (or at most with a 0.5 mm gap), were obtained in the axial plane from foramen magnum to the vertex. In all cases, proton density/T2 weighted conventional spin echo (TR=2000-3000 msec, TE=20-30 msec and 80-100 msec) scans as well as pre- and post- gadolinium enhanced T1 weighted (TR = 500-600 msec, TE = 12-15 msec) scans were obtained. Enhanced scans were obtained 5 minutes after the intravenous administration of single dose of gadolinium (0.1 mmol/kg).

One radiologist (GJZ) identified DVAs from the scans. DVAs were defined as vascular lesions with multiple enlarged vessels converging on a single (sometimes multiple) dilated parenchymal vessel, best seen on post-gadolinium enhanced T1 weighted scans and always confirmed on its consistent presence on at least 1 follow-up scan. The DVAs were classified by location as supratentorial or infratentorial. Supratentorial DVA were further classified as juxtacortical (within gray matter or at gray-white junction), subcortical (between the juxtacortical and periventricular region) and periventricular (adjacent to the lateral, third, or fourth ventricles). The draining vein to which the caput medusae joins was classified as either a deep or superficial draining vein.

Results

 Eighty of 700 (12.6%) MS patients had a total of 97 DVA. 71 patients had single DVA and 9 patients had two or more DVA. Eighty-four DVA were supratentorial (figure 1) and 13 were infratentorial (figure 2). Forty-seven were on the left, 48 on the right, and 2 were midline. There were 42 juxtacortical, 29 subcortical, 13 periventricular, 4 pontine, and 9 cerebellar DVA. Of the 84 supratentorial DVA, 43 were located in frontal, 19 in parietal, 6 in temporal, and 16 in occipital regions (table 1). There were 47 superficial draining and 50 deep draining veins.

Discussion

DVAs were once considered rare vascular malformations. However, with the advent of CT and MRI, they are now known to be the most common of the cerebral vascular malformations. They are commonly identified as an incidental finding. In a study of 4069 consecutive brain autopsies, 105 DVA (2.5%) were found. In a study (1) of 4624 consecutive cranial MRI, DVA were identified in 61 patients (1.3%). In another study of 8200 craniospinal MRs, there were 100 patients (1.2%) with DVA, with DVA representing 50% of all vascular malformations. Our results indicate that DVA to be more common in patients with MS (12.6%).

The exact pathogenesis of DVA is unknown, with the 2 most popular theories being: (1) a primary dysplasia of capillaries and small transcerebral veins (2) a compensatory mechanism of an intrauterine accident resulting in thrombosis of normal venous pathways. Although the reason for the higher prevalence of DVA in MS patients is unknown, it is intriguing that there have been several case reports of cerebral venous thrombosis (2) occurring in patients with MS. Also of possible interest is the suggestion that a hypoxia-like metabolic injury may be a pathogenetic component of a subset of inflammatory MS brain lesions.

Table 1. Distribution of DVAs

<table>
<thead>
<tr>
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<th>Deep</th>
<th>Superficial</th>
<th>Left</th>
<th>Right</th>
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</thead>
<tbody>
<tr>
<td>Juxtacortical</td>
<td>10</td>
<td>32</td>
<td>14</td>
<td>27</td>
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<tr>
<td>Subcortical</td>
<td>21</td>
<td>8</td>
<td>16</td>
<td>13</td>
</tr>
<tr>
<td>Periventricular</td>
<td>12</td>
<td>1</td>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>Pontine</td>
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<td>1</td>
<td>2</td>
<td>0</td>
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<tr>
<td>Cerebellum</td>
<td>4</td>
<td>5</td>
<td>5</td>
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Reference
