Peritumoral Brain Edema in Meningiomas: Relevance of White Matter Lesions in Centrum Semiovale

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Introduction

The incidence of peritumoral brain edema in meningiomas is 40-60% despite the extracerebral location and benign nature of the tumor. To date, several causative factors have been proposed but its pathogenesis remains to be elucidated. Recent reports have closely linked a cerebral-pial vascular supply associated with VEGF expression to peritumoral edema1). However, the edema can occur without pial vascular supply. Tatagiba and his colleagues showed that global cerebral perfusion may influence the qualitative grade of edema and implicated that the edema may develop due to an abnormal state of the brain itself2). Also, some reports suggested that tumor pressure ischemia may be a causative factor of edema.

We hypothesized that cerebral white matter hypoperfusion underlies the edema development and that it plays a main role when a pial supply is absent. As a potential index of underlying hypoperfusion we used white matter lesions (WMLs) of the centrum semiovale seen on T2-weighted MR images, because those WMLs are common in the elderly and are associated with reduced CBF in the white matter3).

Methods

51 patients with supratentorial meningiomas were studied. They consisted of 10 men and 41 women ranging in age from 15 to 78 years (average 57). After evaluating the presence of a pial vascular supply in selective angiography, edema, WMLs in the centrum semiovale and tumor volume were evaluated in MRI. For all patients MRI was performed on a 1.5- or 1-Tesla machine and involved axial T1-weighted, T2-weighted, and postcontrast T1-weighted images of three axes, using the spin-echo technique. TR/TE=570/12 and 5000/96 for T1-weighted and T2-weighted images, respectively. Other parameters were as follows: slice thickness=5mm, FOV=20cm, and 256*192 matrix. The edema was graded as: 0, none; 1, mild; 2, moderate; 3, severe. Based on a report of Miyazaki et al.4) WMLs were defined as hyperintense foci on T2-weighted images and did not have a matching hypointensity on T1-weighted images. WMLs were counted and classified into 4 grades as: 0, none; 1, fewer than scattered focal lesions; 2, more than ten scattered but not confluent focal lesions; 3, partially confluent lesions; 4, diffuse confluent lesions. Tumor volume was approximated by the equation *a*b*c/8: a, b, c indicate diameters in three axes. After dividing the patients into two groups according to the pattern of vascular supply, WMLs, age and tumor volume were correlated with the edema score. All parameters were analyzed either with Mann-Whiteney's U test or Spearman's test for rank correlation. A p value less than 0.05 indicated a statistically significant difference.

Results

Among 51 patients 28 had peritumoral edema (55%). Angiographically 10 patients (pial group) had a pial vascular supply and the other 41 (meningeal group) did not. The pial group was significantly greater than the meningeal group in the edema score ($p<0.05$) (Fig.1). The edema score correlated with the grade of WMLs ($r=0.60$, $p<0.01$) (Fig.2) and age ($r=0.47$, $p<0.01$) in the meningeal group, but not in the other. The edema score also correlated with tumor volume when the meningeal group was subdivided into three classes (<8cc, 8-64cc, and >64cc) ($r=0.41$, $p<0.05$).

Discussion

Our study showed that the severity of edema correlated with the grade of WMLs, age and tumor volume in the meningeal group but not in the pial group. However, meningiomas with a pial vascular supply had significantly higher grade of edema compared with those supplied only from meningeal side. This suggests that two distinct mechanisms are implicated in the edema development. One is related to a cerebral-pial vascular supply and the other is to cerebral white matter hypoperfusion.

WMLs are closely linked to age and associated with reduced CBF in the white matter3). The correlation of edema with WMLs and with age, thus, indicates that a cerebral status linked to WMLs, probably hypoperfusion, is a causative factor of edema. The pathological studies indicated that the white matter hypoperfusion associated with asymptomatic WML would be due to subclinical sclerosis of medullary arteries and arterioles. An increase in mass effect primarily reduces CBF. The result that the severity of edema also was related to tumor volume seems prove that pressure ischemia augments underlying hypoperfusion.

The histopathological studies revealed that WMLs on MR images were often associated with ischemic damage of brain parenchyma and perivenous edema. Signal abnormalities could start with episodes of perivascular edema caused by a temporary focal breakdown of the blood-brain barrier4). It is conceivable that a similar pathological change occurs in the brain tissue around a meningioma, due to ischemia caused by both sclerosis of medullary branches and tumor pressure. Then, edema should originate from the brain and represent a variety of pathological changes from true edema to necrosis.

In conclusion, this study suggests that two distinct mechanisms are implicated in the edema development. One is related to a cerebral-pial vascular supply and the other is to white matter hypoperfusion in combination with pressure ischemia. The understanding that subclinical abnormalities of the brain itself may underlie the development of edema is important because of unlikely complete recovery.

References

1. Yoshioka H, et al. Cancer 1999;85:936-944
2. Tatagiba M, et al. Neurosurgery 1991;28:400-404
3. Miyazawa N, et al. Stroke 1997;28:984-987
4. Fazekas F, et al. AJNR 1991;12:915-921