Surgical resection of such a lesion or close observation with follow-up magnetic resonance imaging studies is recommended.

Detwiler, et al., are accurate in pointing out our finding that radiation-induced cavernous malformations represent a subset of these lesions with an aggressive behavior. In our series, there was an increased risk of clinically significant hemorrhage in the radiation-induced cavernous malformations compared with cavernous malformations in patients who had not received radiation treatment. This observation may lend support to the surgical treatment of lesions that have a more aggressive natural history.

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References


Medullary Compression and Hypertension

TO THE EDITOR: We read with great interest the recent article by Colón, et al. (Colón GP, Quint DJ, Dickenson LD, et al: Magnetic resonance evaluation of ventrolateral medullary compression in essential hypertension. J Neurosurg 88:226–231, February, 1998). In this report, the authors conducted a blinded prospective study in which magnetic resonance (MR) imaging was used to evaluate the role of lateral medullary compression in essential, or neurogenic, hypertension. Thirty patients with hypertension and 45 controls underwent thin-slice MR imaging of the posterior fossa and skull base for comparison by four blinded, independent reviewers. The authors concluded that although there was a tendency toward left vertebral artery dominance in the group with hypertension, no differences between the two groups were observed regarding brainstem compression or rotation.

As the authors point out, these results differ from other series in the literature. Morimoto and colleagues demonstrated a 74% rate of medullary compression detected by thin-slice MR imaging. Naraghi and colleagues demonstrated even higher rates of brainstem compression in neurogenic hypertensive patients at 83% and 91%, respectively. Despite these studies, we concur with the criticisms by Meaney, et al., concerning the sensitivity of MR imaging in which 3-mm slices are used. Medullary compression due to smaller vessels, such as the posterior or anterior internal carotid artery, may be missed by using this technology. Furthermore, as Colón, et al., maintain, medullary compression is a matter of degree, and conventional MR technology at present may be insufficiently sensitive to detect only marked brainstem compression.

In their paper, Colón, et al., suggest “perhaps only large associated deformities result in hypertension,” and that “subtle vascular contiguity or compression is not the culprit.” Based on our operative experience, we cannot agree with this hypothesis. We are reporting on 12 patients with severely uncontrolled, medically refractory neurogenic hypertension (without any other cranial nerve rhizopathies) treated with microvascular decompression of the left lateral medulla. All 12 patients had preoperative MR imaging of the brain, with special attention to the posterior fossa and skull base. Although all patients had clear intraoperative demonstration of medullary compression, only three (25%) had preoperative MR demonstration of medullary compression. The vertebral artery was the offending vessel in two of the patients, and a large posterior internal carotid artery was the offending vessel in the third. For the remaining nine patients, preoperative MR imaging demonstrated dolichoectatic basilar arteries in two patients, severe vertebralbasilar artery tortuosity and displacement in two patients, and was unremarkable in the remaining five patients. However, left medullary compression by one or more arteries was clearly seen intraoperatively in all 12 patients.

We are not surprised by findings such as those of Colón, et al., and Watters, et al., because we too were only able to show a 25% rate of medullary compression in a severely hypertensive (mean peak systolic pressures 212 mm Hg) population with intraoperative evidence of medullary compression. Our operative experience agrees with their conclusions that thin-slice MR imaging is not a good screening tool to identify patients with neurogenic hypertension. Symptomatic compression, however, is not limited to only the large brainstem deformities evident on thin-slice MR imaging. Furthermore, patients with subtle vascular compression detectable only by thin-slice MR imaging or surgical exploration may be the most promising candidates for improvement following microvascular decompression. Only with prospective investigation and rigorous outcome criteria will we be able to identify the population who can benefit most from microvascular decompression of the lateral medulla. We have recently embarked on such a study.

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References


RESPONSE: We greatly appreciate the comments of Jannetta, et al., regarding our manuscript. We believe that there is an association between ventrolateral medullary compression and hypertension and have seen this several times in our experience; we performed this study to clarify the diagnostic role of magnetic resonance imaging. We agree with Dr. Jannetta’s group, however, that thin-slice axial magnetic resonance imaging may not be the best screening tool for these patients, particularly when slices are obtained at 3-mm or larger intervals.

The role of brainstem compression in essential hypertension is an interesting and provocative idea presented by Dr. Jannetta. We agree that a well-designed prospective investigation and outcome criteria should be used to study his hypothesis so that patients with hypertension who might benefit from this procedure are diagnosed and treated appropriately.

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Arteriovenous Malformations After Radiosurgery

TO THE EDITOR: We read with interest the recent article by Schneider, et al. (Schneider BF, Eberhard DA, Steiner LE: Histopathology of arteriovenous malformations after gamma knife radiosurgery. J Neurosurg 87:352–357, September, 1997) regarding the histopathological changes in arteriovenous malformations (AVMs) following gamma knife radiosurgery. They demonstrated that the time interval following stereotactic radiosurgery was directly correlated with both the frequency and severity of histopathological changes in AVM vessels. This is to our knowledge the first series of AVMs evaluated from a histopathological standpoint after gamma knife radiosurgery, whereas previously there was only a single case report.

We previously published our series reporting pathological changes in resected AVMs following stereotactic radiosurgery; the majority of which were treated with He ions. We resected 33 AVMs in patients 1 to 10 years after radiosurgery failed to obliterate the lesion. Of these, 19 were available for pathological evaluation. We reached many of the same conclusions as Schneider, et al. However, our study also showed that there was a significant correlation between the extent of pathological radiation changes and dose of radiation received. Of interest to us is whether the authors of this paper can reach any conclusions regarding the optimal dose of radiation for AVMs based on their histopathological report. Furthermore, do they find any radiation dose threshold below which histopathological changes would not be expected to result in AVM obliteration? Also, based on their findings, do the authors have any indication of what should be the upper limit of treatment dose? At what point does the risk of radiation necrosis offset any increase in histopathological obliteration when treating AVMs? We would be interested in knowing whether the authors believe that the pathological changes observed following radiosurgery generally occur during the first 3 years, paralleling the time course of radiographic obliteration, or whether they think further pathological changes occur beyond the first 3 years. Finally, the authors do not comment regarding the treatment dose used in these AVMs with respect to AVM size. The two largest AVMs in this series, as measured by pretreatment size (Cases 3 and 6), were both associated with lower pathological changes (frequency × histological grade) compared with the other AVMs. Given the current debate regarding the optimal dose for these larger AVMs, we wonder whether pathological changes could serve as a guide for radiation dosage.

In summary, we believe that this article along with the previously published reports serves to justify further the use of radiosurgery in treating AVMs. As with brain tumors, a better understanding of pathological changes following radiosurgical treatment may allow us to refine treatment further.

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References


TO THE EDITOR: We read with great interest the article by Schneider, et al. (Schneider BF, Eberhard DA, Steiner LE: Histopathology of arteriovenous malformations after gamma knife radiosurgery. J Neurosurg 87:352–357, September, 1997). It is noteworthy how few publications report the histological analysis of arteriovenous malformations (AVMs) after radiosurgery in spite of the large number of patients treated during the last 20 years. In an earlier publication from Sheffield, England¹ we analyzed specimens removed from seven patients following various periods postradiosurgery. Using very similar histological and immunohistological techniques we demonstrated a similar time course of progressive vessel wall changes. Both analyses demonstrated the presence of α smooth muscle antigen in the proliferative cells within the intima. We showed that similar cells, probably myofibroblasts, also develop in the connective tissue stroma and postulated that their contraction may contribute to the eventual occlusion of the lumen.

The weakness we perceive in both studies is the possible confounding effect of hemorrhage in the histological picture. It would be of great interest to perform similar studies on an irradiated AVM that had never bled, either before or after radiosurgery, and that had been removed either at autopsy or surgery prior to its occlusion. With the growing number of AVMs being treated worldwide, the chances of such a case must be increasing.

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