Endovascular Alleviation of Deficits

To the Editor: I was most interested in the recent paper by Halbach and colleagues (Halbach VV, Higashida RT, Dowd CF, et al: The efficacy of endosaccular aneurysm occlusion in alleviating neurological deficits produced by mass effect. J Neurosurg 80:659–666, April, 1994) detailing their experience with endovascular techniques in the treatment of 26 patients presenting with mass effect due to intracranial aneurysm. Their results suggested that the majority of patients improved following endovascular endosaccular occlusion.

It was indicated in the Clinical Material and Methods section of the article that patients were selected from those treated over a 10-year period at their institution, as detailed in prior publications. One of those articles dealt specifically with balloon embolization of posterior circulation aneurysms.1 In that paper, five of the patients had presented with mass effect and underwent balloon occlusion of the aneurysm. Following treatment, complete occlusion was accomplished in three patients, and subtotal occlusion in two. One of the patients with subtotal occlusion is reported to have died from aneurysm rupture. The other had a stroke and died 5 months later from renal failure and sepsis; neither of these patients is included in the 1994 series. Of the three patients with complete aneurysm occlusion it appears that only one of them, a 61-year-old with a basilar tip aneurysm and an excellent outcome, is included in the 1994 series. Of the two others, one had a stroke but was described as a good result in 1989, and the other, also described as a good result, had her giant distal vertebral posterior inferior cerebellar artery aneurysm “resected” some time after balloon embolization.

I would assume that the authors excluded patients who died following their endovascular treatment, although this is not indicated in the Clinical Material and Methods section. I am interested in what happened to the other two patients who survived balloon embolization and why they were not selected for inclusion in this 1994 series.

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Response: We appreciate the interest of Dr. Lownie in our paper. The purpose of that paper was to establish what effect embolic devices, primarily balloons, had on aneurysms that initially presented with mass effect in cases in which preservation of the parent artery was accomplished. In reviewing the overall experience of patients treated at this institution, it became apparent that some patients with unruptured aneurysms would present with subjective complaints such as blurred vision, tingling, nausea, or pain, but appeared to have no objective neurological deficits. We therefore elected to include in this series only patients who had objective neurological signs, and whose response to treatment was based on the changes in these neurological deficits. This was stated as the first inclusion criterion in the Clinical Material and Methods section for this paper.

Dr. Lownie correctly noted from a prior publication1 that five patients presented with mass effect and were treated with endosaccular occlusion. Two patients expired following treatment, and, therefore, evaluation of the effect of treatment could not be undertaken. The indications, results, and complications of endovascular treatment of intracranial aneurysms have been previously reported by our group.1–8

One patient with progressive right-sided weakness was treated with two detachable balloons. Subsequently, she had complete resolution of her presenting symptoms and was reported as Case 15 in the mass effect paper. One patient, a 74-year-old woman presented with blurred vision and a basilar artery bifurcation aneurysm that was discovered and treated with preservation of the parent artery. She developed change in her vision 1 week after treatment, which resulted in a small superior quadrantanopsia, but improved and was able to drive. A follow-up arteriogram at 1 year revealed complete occlusion of the aneurysm. She did not have any objective neurological deficits at the time of presentation and was therefore excluded from the series on mass effects. The remaining patient, a 74-year-old woman, presented with severe nausea, vomiting, and weight loss presumably due to mass effect from a giant posterior inferior cerebellar artery aneurysm. She initially was treated with endosaccular occlusion but a follow-up arteriogram revealed persistent filling of the aneurysm. Her presenting nausea and vomiting resolved initially but recurred several months later, and a deconstructive procedure, balloon trapping of the aneurysm, was performed. Two months later she had surgical evaluation of her thrombosed aneurysm with subsequent relief of her nausea and vomiting. She was excluded from the mass effect series because the presenting nausea, vomiting, and weight loss were not objective neurological signs.

We have encountered a number of patients in our practice with unruptured intracranial aneurysms in whom the presenting symptoms are subjective, transient, or in many, unrelated to the aneurysm. To elucidate the effect that an endosaccular occlusive device has on an unruptured aneurysm we chose to include only patients with objective neurological findings. Our study has shown that in this population endosaccular occlusion can alleviate or improve symptoms in the vast majority of patients.
Melanotic Neuroectodermal Skull Tumor

To THE EDITOR: We read with great interest the article by Hoshino, et al. (Hoshino S, Takahashi H, Shimura T, Nakazawa S, Naito Z, Asano G: Melanotic neuroectodermal tumor of infancy in the skull associated with high serum levels of catecholamine. J Neurosurg 80:919–924, May, 1994). These authors described a very rare case of melanotic neuroectodermal tumor of the skull involving the left occipital and mastoid bone with intracranial involvement through a dural defect, and they offered a revision of the literature on melanotic neuroectodermal tumor of infancy (MNTI) with skull localization. We would like to add some comments to this interesting report to help increase knowledge about this rare and often curable entity.

In a recent paper,1 not cited in the review of the literature by Hoshino, et al., we described three cases of MNTI of the skull in infants who presented with a rapidly growing mass of the anterior fontanel in two and focal seizures in one. All three cases were interesting because of a huge intracranial component, which is very unusual in the MNTI of the skull.1

Hoshino, et al. stress the clinical importance of the relatively high mortality rate for patients with MNTI of the skull, although all of the cases were reported to be benign. They speculate that MNTI might secrete catecholamines, as was true in their case, and that the rapid fall in these levels after surgery may play a role in the sudden death of these patients. This is an interesting theory, but a more accurate evaluation of the literature reveals that most of the cases (7 of 8) that were complicated by operative mortality involved tumors with midline localization, in which surgery was complicated by extensive intraopera-

References


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