Correspondence

Letters to the Editor

Diffuse Cerebral Angiomatosis

I read with interest the paper by Wagle et al. [1] in which the working diagnosis of diffuse capillary telangiectasis of the brain was based on the angiographic and MR findings. In the differential diagnosis of a case with diffuse cerebral angiomatosis, the syndromes of Divry–Van Bogaert [2] and Sneddon [3] should be considered.

Divry–Van Bogaert syndrome is manifested by noncalcified diffuse leptomeningeal angiomatosis and livedo reticularis. The syndrome may be sporadic or familial. The neurologic picture that appears around the third decade of life includes multifocal ischemic attacks, dementia, epileptic seizures, and pseudobulbar and extrapyramidal syndromes. Carotid angiography can show either extensive angiomatosis localized mainly in the distal territories of anterior and middle cerebral arteries [4] or distal, fine angiomatous circulation from tiny collateral vessels in distal arterial occlusions [3]. Postmortem examinations have shown leptomeningeal angiomatosis, multifocal infarcts, and parietal thickening of the leptomeningeal arteries with thrombosis and recanalization.

The clinical and radiologic findings in Sneddon syndrome are almost identical to those of Divry-Van Bogaert syndrome, so at present it is difficult to distinguish between them. The livedo usually precedes the first neurologic symptom but may appear afterward. In any case, if a diagnosis of Sneddon syndrome is to be considered, it would be worth performing angiography on the hand to see if the characteristic narrowing and dilatation in the common and proper palmar digital arteries are present.

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Neuroimaging and the Lesion of Multiple Sclerosis: An Addendum

In late November 1986, a second MR image was obtained of the patient previously described in the May/June 1987 issue of *AJNR* [1]. This procedure was performed on a much newer machine and resulted in an image that was technically far superior. The second MR image was compared with the double-dose delayed contrast-enhanced CT scan originally obtained in January 1984 (Fig. 1 on next page). The comparison dramatically confirmed and reinforced our original observation that few if any of the areas of impaired blood-brain barrier developed into areas of increased signal intensity that may represent plaques of multiple sclerosis. Close examination of the new MR image suggests that most, if not all, of the plaques located in the periventricular white matter did not enhance in January 1984 and thus probably antedate that particular exacerbation. Clinically, the patient has continued to maintain a completely stable condition.

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Subdural Hematoma Mimicking Epidural Hematoma

A recent article in *AJNR* [1] reported two cases of acute subdural hematoma "mimicking" an epidural hematoma. However, in both cases, the extraaxial hematomas clearly can be seen to cross nearby adjacent calvarial suture lines, suggesting a subdural location.

It is well established that an epidural hematoma rarely dissects past a calvarial suture [2–4]. Dissection usually is seen in association with a fracture and dural laceration involving a dural sinus (e.g., superior sagittal sinus, transverse sinus). In neither of the cases presented is the extraaxial collection adjacent to a significant dural sinus.

In addition, the hematoma on the right side of case 1 clearly shows a crescentic "tail," which is concave toward the brain. This is a reliable differential feature in favor of subdural hematoma. Ironically, this is a distinguishing feature that the authors describe in their discussion.

Acute subdural hematomas are seen most often in association with other injuries, especially cortical contusions. Both these patients had thrombocytopenia, and a hemorrhagic diathesis has been reported in patients with isolated acute subdural hematoma [5].