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In Re: Basilar Artery Migraine and Reversible Imaging Abnormalities

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Internal Carotid Artery Narrowing in Children with Retropharyngeal Lymphadenitis and Abscess

We read with interest the article of Hudgins et al (1) describing, in 13 consecutive children, narrowing of the internal carotid artery (ICA) ipsilateral to retropharyngeal lymphadenitis and abscess, and suggesting that such narrowing is a common, benign, and, most likely, incidental imaging finding. We would like to draw the authors' attention to our article on this subject that was published in the March 1998 issue of the *AJNR* in which we emphasized the importance of the MR findings of this entity (2). It was a case report of carotid involvement by retropharyngeal abscess in a 4-year-old boy investigated by MR imaging. MR imaging showed not only lumen narrowing of the ICA but also enhancement of its wall. Although narrowing was greatest at the level of the abscess, the wall enhancement was seen all along the course of the ICA up to the cavernous segment. This finding, revealed only on contrast-enhanced T1-weighted images, is probably unidentifiable on contrast-enhanced CT owing to the contrast resolution of this technique. When this finding is present, however, it might indicate a more severe carotid involvement than suspected on the basis of CT images only and Hudgins et al might, therefore, have underestimated the degree of carotid involvement. Unfortunately, we lack data in the literature and actual histologic proof to define the exact nature of such wall abnormalities; as the authors suggest, this could have been from spasm or actual involvement of the wall of the vessel by the inflammatory process itself. This process could presumably be referred to as arteritis, because this term not only refers to an infection, but also to an inflammation of the arterial wall, whatever its severity. Intuitively, wall thickening with enhancement, as we found in our case, would be more readily expected in a true arteritis than in a spasm. Because arteritis can weaken the wall of a vessel, it could also cause perforation, leading to fatal hemorrhage or a pseudoaneurysm. We would like to emphasize another potential manifestation of carotid involvement by sepsis apart from arterial rupture—occlusion. Indeed, it has also been suggested that, in children, arteritis produced by direct extension of infection of the neck or throat might be the more important risk factor in addition to trauma for cervical occlusion (3). Although children almost always tolerate carotid occlusion well clinically, it reduces the capabilities of collateralization of the cerebral vasculature in adulthood. In their study, the authors did not exclude this second rare complication because the follow-up of the children included neurologic examination (all patients recovered with no neurologic deficits) and a head CT (normal in all patients) but no direct imaging of the carotids (eg, color Doppler sonography). Because those carotid complications are unusual, given the widespread and early use of antibiotics, we think further MR studies of more patients are needed before stating that intrinsic abnormalities of the ICA adjacent to an abscess are always benign. At this stage of knowledge, one question remains unanswered: should we perform an MR examination with gadolinium once CT shows carotid narrowing near a cervical abscess? Until the exact significance of these carotid imaging findings is known, we would still consider that lumen narrowing associated with enhancement is a sign of severity that should lead to aggressive treatment. In our case, the abscess was drained surgically the day after admission and the follow-up MR examination 1 week later showed normalization of the affected ICA.

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Reply

We would like to thank Ide and colleagues for their letter drawing our attention to their case report, *An Early Observation of Carotid Involvement by Retropharyngeal Abscess.* Their article was published after our paper had been accepted, but before it was revised, which likely explains the oversight on our part. We would like to comment on several of the points addressed by the authors.

To summarize, Ide et al appear concerned that we have underestimated the seriousness of internal carotid artery (ICA) narrowing in children with retropharyngeal abscess (RTPA), and that this may portend ICA occlusion of pseudoaneurysm that is clinically occult.

Although we did not appreciate the abnormal enhancement in the ICA wall to the level of the cavernous segment on CT that was enhanced on MR scans, we think it is unlikely that we have underestimated the degree of carotid involvement. Indeed, we pointed out that the degree of ICA narrowing was quite severe in several of our patients. It does appear, however, that if the perivascular inflammatory process (RTPA) is treated expeditiously with surgery, the vascular findings in our inves-

tigation, based on clinical follow-up, appeared to have no sequelae in the 13 patients we studied (1).

Ide et al suggest that clinical follow-up is not reliable to exclude ICA disease, because ''children almost always tolerate [ICA] occlusion well clinically.'' Based on clinical experience in large trauma and sickle cell populations treated at our pediatric hospital, complete ICA occlusion in childhood is commonly a devastating event resulting in dramatic neurologic impairment. We think it is unlikely that a complete ICA occlusion would be asymptomatic, especially in 13 sequential children.

We maintain that the only imaging required acutely is contrast-enhanced CT. Children with RTPA are seriously ill. Spiral CT is fast, requires no sedation in a child who already may have airway compromise, and, even if distal ICA enhancement is not appreciated on CT as it is on contrast-enhanced MR imaging, the treatment is still surgery or close observation and IV antibiotics. What additional information is gained with contrast-enhanced MR imaging? Of what significance is visualization of perivascular enhancement remote from the infection? Even in the authors' limited experience, the findings resolve after surgery. Furthermore, MR imaging might delay definitive treatment.

Should postoperative MR imaging be performed to rule out ICA occlusion or pseudoaneurysm? The authors are correct in stating that ICA occlusion/pseudoaneurysm are potential complications of cervical adenitis in children (2–4). The frequency of this complication, however, cannot be determined, as cases are sporadic and reported as single episodes. If the authors are aware of any literature suggesting that ICA occlusion/pseudoaneurysm is a frequent complication of RTPA, we would appreciate seeing the data. Although ICA injury is definitely a complication of penetrating neck trauma, we are unaware of any large series reporting vascular injury common (or even uncommon) after RTPA. Therefore, ICA injury following surgically treated RTPA appears to be rare, and to recommend postsurgical MR imaging because of a speculation of potential ICA injury is not based on enough evidence to change our practice. In our broad clinical experience dealing with RTPA, none of our surgeons or pediatric neuroradiologists have seen any child return with symptoms referable to ICA injury or occlusion.

In summary, ICA narrowing ipsilateral to RTPA is a dramatic imaging finding that, together with the clinical status of the patient, implies a serious inflammatory process that should be treated expeditiously. In light of the absence of compelling data that suggests otherwise, and based on our clinical experience, we maintain that neurologic sequelae are rare and follow-up imaging is not routinely recommended.

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In Re: Basilar Artery Migraine and Reversible Imaging Abnormalities

We read with interest the paper by Maytal et al, *Basilar Artery Migraine and Reversible Imaging Abnormalities* (1). The authors report a case of basilar artery migraine in a 17-year-old boy with transient CT and MR abnormalities after two migraine episodes. A repeat MR study 6 months after the last episode showed complete resolution of the lesions. The authors conclude that these transient abnormalities on brain images, similar to those shown in other neurologic conditions, are most likely related to cerebral vasogenic edema from abnormal vascular permeability, which causes reversible disruption of the blood-brain barrier.

In our opinion, Maytal et al's explanation seems unlikely. In fact, MR imaging of the patient revealed transitory cerebral abnormalities strictly involved the cerebral cortex. It is commonly known that vasogenic edema affects mainly the white matter, which was not involved in the reported case. Moreover, the lack of contrast enhancement argues against blood brain–barrier breakdown. Transient cortical abnormalities owing to blood brain–barrier breakdown have been reported in other conditions such as cyclosporine-related encephalopathy (2) and epileptic seizure (3). In these cases, contrast enhancement was always present. In addition, Jansen et al (2) suggest that the blood brain–barrier disruption in some cases can be detected only on contrast-enhanced images. This seems to be confirmed by Horowitz et al (3), who reported a case of complex partial seizures. The authors performed MR imaging with and without Gd-DTPA infusion. Noncontrast MR sequences were absolutely normal. Postcontrast MR imaging revealed bilateral symmetrical enhancement of the mesial cortex of the anterior temporal lobes. After adequate control of seizures with medication, repeat MR imaging yielded normal findings and the prior enhancement was no longer seen. The authors concluded that this transient seizure-induced enhancement was consistent with ictal or postictal hyperemia and breakdown of the blood-brain barrier. Therefore, transitory cortical abnormalities owing to reversible blood brain–barrier disruption should be unlikely when contrast enhancement is lacking. In the case by Maytal et al (1), a more likely explanation of

the cortical abnormalities should be a focal reversible cytotoxic edema owing to a transitory and reversible failure of the Na cellular pump arising from hypoxic ischemia secondary to the vasoconstriction during the migraine attack.

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Reply

We would like to thank Dr. Ambrosetto for his remarks regarding this case report. In our article, we concluded that the transient abnormalities revealed by brain imaging are most likely related to cerebral vasogenic edema owing to abnormal vascular permeability, which causes reversible disruption of the blood-brain barrier.

After reviewing the case, we agree with Dr. Ambrosetto, who pointed out that because the lesions revealed by MR imaging involved mostly the gray matter and did not enhance with contrast material, these most likely represent transient changes secondary to cytotoxic edema.

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