AJNR

Intralesional Injection of Absolute Alcohol into Vertebral Hemangiomas: A New Treatment Option?

Peter L. Munk and Tom R. Marotta

AJNR Am J Neuroradiol 1999, 20 (6) 959-960
http://www.ajnr.org/content/20/6/959

quantification of both diffusion and perfusion parameters will be essential for differentiating the reversible from the irreversibly ischemic tissue. Comparisons will have to be made between the MR techniques and xenon-CT, perfusion CT, and SPECT CBF analyses to determine the most cost-effective and efficacious methods for defining the salvageable brain. I am convinced that the risk-benefit ratio of aggressive therapy can be determined with such imaging techniques. Lastly, I believe that thrombolysis and other forms of image-guided therapy will be performed by MR or CT scanning and a battery of scans will be obtained before therapy to evaluate salvageable tissue, and after treatment to detect potential complications and determine early therapeutic efficacy.

RICHARD E. LATCHAW, M.D.

Member, Editorial Board

References

1. **PROACT II summary of initial results for participating clinical sites.** February 3, 1999; Abbott Laboratories; Abbott Park, Il
2. Hacke W, Zeumer H, Ferbert A, et al. **Intraarterial thrombolytic therapy improves outcome in patients with acute vertebrobasilar disease.** *Stroke* 1988;19:1216–1222
3. Zeumer H, Freitag HJ, Zanella F, et al. **Local intra-arterial fibrinolytic therapy in patients with stroke: urokinase versus recombinant tissue plasminogen activator (r-tpa).** *Neuroradiology* 1993;35:159–162
4. Endo S, Kuwayam N, Hirashima Y, et al. **Results of urgent thrombolysis in patients with major stroke and atherothrombotic occlusion of the cervical internal carotid artery.** *AJNR Am J Neuroradiol* 1998;19:1169–1175
5. Hacke W, Kaste M, Fieschi C, et al. **Intravenous thrombolysis with recombinant plasminogen activator for acute hemispheric stroke: the European Cooperative Acute Stroke Study (ECASS).** *JAMA* 1995;274:1017–1025
6. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. **Tissue plasminogen activator for acute ischemic stroke.** *N Engl J Med* 1995;333:1581–1587
7. Ueda T, Sakaki S, Yuh WTC, et al. **Outcome in acute stroke with successful intra-arterial thrombolysis and predictive value of initial SPECT.** *J Cereb Blood Flow Metab* 1999;19: 99–108
8. Levy EI, Scarrow AM, Kanal E, et al. **Reversible ischemia determined by xenon-enhanced CT after 90 minutes of complete basilar artery occlusion.** *AJNR AM J Neuroradiol* 1998;19:1943–1946
9. Hunter GJ, Hamberg LM, Ponzo JA, et al. **Assessment of cerebral perfusion and arterial anatomy in hyperacute stroke with three-dimensional functional CT: Early clinical results.** *AJNR Am J Neuroradiol* 1998;19:29–37
10. Sorensen AG, Copen WA, Ostergaard L, et al. **Hyperacute stroke: simultaneous measurement of relative cerebral blood volume, relative cerebral blood flow, and mean tissue transit time.** *Radiology* 1999;210:519–527
11. Maeda M, Yuh WTC, Ueda T, et al. **Severe occlusive carotid artery disease: hemodynamic assessment by MR perfusion imaging in symptomatic patients.** *AJNR Am* J *Neuroradiol* 1999; 20:43–51
12. Sorensen AG, Buonanno FS, Gonzalez RG, et al. **Hyperacute stroke: evaluation with combined multisection diffusion-weighted and hemodynamically weighted echo-planar MR imaging.** *Radiology* 1996;199:391–401
13. Kohno K, Hoehn-Berlage M, Mies G, et al. **Relationship between diffusion-weighted MR images, cerebral blood flow, and energy state in experimental brain infarction.** *Magn Reson Imaging* 1995;13:73–80
14. Bryan RN. **Diffusion-weighted imaging: to treat or not to treat? That is the question.** *AJNR Am J Neuroradiol* 1998;19: 396–397
15. Baird AE, Benfield A, Schlaug G, et al. **Enlargement of human cerebral ischemic lesion volumes measured by diffusion-weighted magnetic resonance imaging.** *Ann Neurol* 1997;41: 581–589

Intralesional Injection of Absolute Alcohol into Vertebral Hemangiomas: A New Treatment Option?

Hemangiomas of the vertebral bodies are commonly encountered, being found in 10–12% of patients in autopsy series (1). The vast majority of these are solitary, are discovered incidentally, and are seldom symptomatic. Symptoms, when they do occur, can vary from chronic, poorly defined pain to cord compression and paraplegia. These undesirable consequences are usually caused by compression fracturing, hematoma, epidural extension of a hemangioma, or bony expansion of the vertebral body (1, 2).

A variety of treatment options exists for addressing symptomatic hemangiomas. Surgery is well established, and has been proved effective. A surgical approach is technically challenging because of the markedly vascular nature of these congenital vascular malformations. The introduction of presurgical embolization has facilitated surgery significantly, and is now commonly performed (2). Scattered reports of treatment of symptomatic lesions with embolization alone exist; however, treating these lesions exclusively with embolization is generally inadequate in the majority of cases. Radiotherapy also has been used in treatment of these lesions, usually as a complement to surgery. Radiation therapy alone is often inadequate.

In 1994, Heiss et al reported two patients with spinal cord compression who improved appreciably after direct percutaneous injection of alcohol into vertebral body hemangiomas (3), and then they reported on seven patients 2 years later (4). In this issue of the *AJNR*, Goyal et al (page 1091) report their experience with 14 patients they treated with this technique, considerably expanding the reported number of patients in the literature. The authors found that 86% of their patients demonstrated appreciable clinical improvement in a manner analogous to those reported by Heiss et al.

Direct alcohol injection has several advantages. It is readily performed using conventional bone biopsy equipment and CT guidance without requiring the facilities of an operating room or the sophisticated equipment needed in an interventional angiography suite or radiotherapy unit. Only intrave-

nous sedation and analgesia are required. The procedure is less invasive than open surgery, and does not entail the risks of significant blood loss that may be encountered with invasive therapy. Embolization, although extremely helpful in preoperative treatment of these lesions, cannot directly reach or destroy the hemangioma because of the presence of an intervening capillary bed separating feeding arteries from the hemangioma (2). The risks of embolization to the spinal arterial supply, as well as surrounding normal tissue, is well recognized and a dreaded, if fortunately infrequent, complication of embolization (2). Radiation therapy carries the risk of possible toxicity to the spinal cord. Direct injection of alcohol, on the other hand, destroys the hemangioma without these problems.

Goyal et al, however, omit an important caveat. The authors encountered two significant complications. The first was penetration of the pleural space in treatment of a thoracic hemangioma, with complicating empyema. A less acute problem was also noted in that a treated vertebral body underwent collapse subsequent to ethanol ablation; interestingly, Heiss encountered the same problem (4). This is likely attributable to the osteonecrosis that can occur with injection of alcohol. Although data is inadequate to be certain, it may be that larger hemangiomas or hemangiomas treated with larger volumes of alcohol may be more prone to developing this complication, as osteonecrosis may be more likely to develop in those situations.

All patients, except one, were being treated for progressive neurologic dysfunction, and all patients had transient worsening of neurologic status. We presume this is related to alcohol-induced inflammatory changes, including swelling of the lesion. Steroids were administered empirically to all patients with possibly reduced inflammatory response. For those considering this treatment for their patients, neurologic worsening will have to be an acceptable and accepted event that will usually get better—although with increasing experience there may be instances when a neurologic improvement will not occur. Therefore, treating patients who present for reasons other than neurologic compromise may be difficult to justify.

Regarding technique, the injection of alcohol need not be ‘‘a blind procedure’’ as the authors have indicated. Without significantly changing the absolute nature of the alcohol, we have made alcohol radioopaque by mixing it in metrizamide powder when using it for other vascular lesions. This might allow for a more precise volume administration and reduce the ‘‘subjective assessment’’ of the rate of opacification.

The precise role for percutaneous alcohol ablation remains unclear. Unlike embolization, this technique allows actual destruction of the hemangioma. This presumably would make the risk of recurrence after treatment less likely, although in the current series recurrent (or more likely residual) hemangioma was noted in one instance. Because it appears that collapse of the vertebral body may be a significant complication of this procedure, its use as the sole treatment for symptomatic hemangiomas is, at the present time, questionable. It may, however, play an important role as an adjunct to surgery, allowing devascularization of the hemangioma without entailing the risks of angiography.

Another rapidly expanding technique is that of percutaneous vertebroplasty where image-guided injection of methyl methacrylate bone cement directly into the hemangioma is performed (5). Experience with this technique is increasing rapidly as injection of cement does have the advantage of providing structural support at the site of the ablated lesion. Long-term follow-up with this technique also is presently lacking. Cement, of course, creates a permanent incompressible cast of the hemangioma, which may be a problem in patients who have extensive expansion of the hemangioma with ballooning of the vertebral body or involvement of surrounding soft tissues or epidural space.

PETER L. MUNK, M.D.
TOM R. MAROTTA, M.D.

University of British Columbia, Vancouver, BC

References

1. Fox MW, Onofrio BM. **The natural history and management of symptomatic and asymptomatic vertebral hemangiomas.** *J Neurosurg* 1993;78:36–45
2. Smith TP. **Transarterial embolization of vertebral hemangioma.** *JVIR* 1993;4:681–685
3. Heiss JD, Doppman JL, Oldfield EH. **Brief report: relief of spinal cord compression from vertebral hemangioma by intralesional injection of absolute alcohol.** *N Engl Med* 1994;331: 508–511
4. Heiss JD, Doppman JL, Oldfield EH. **Treatment of vertebral hemangioma by intralesional injection of absolute alcohol (letter).** *N Engl Med* 1996;336:–1340
5. Galibert P, Deramond H. **La vertebroplasties acrylique percutanee comme traitment des angiomes vertebraux et des affections dolorigenes et fragisantes du rachis.** *Chirurgie* 1990;116: 326–335

Can You Get a Stiff Back from Lack of Spinal Stiffness?

Most neuroradiologists have a keen interest in spinal tumors or spinal vascular malformations, but have little curiosity for back pain, except if they happen to suffer from it. Unless back pain is associated with a definite medullopathy or radiculopathy caused by spinal stenosis or disk herniation, traditional neurologists and neurosurgeons share this lack of interest, and tend to refer afflicted pa-