Complications of Percutaneous Laser Nucleolysis

To the Editor: In 1987, Choy, et al., reported their preliminary results of percutaneous laser nucleolysis for ruptured lumbar intervertebral disc unresponsive to standard conservative therapy. This procedure, which is performed under local anesthesia, involves placing a No. 18 needle into the affected disc space under fluoroscopic guidance. The tip of this needle is just inside the anulus fibrosis. A neodymium:yttrium-aluminum-garnet (Nd:YAG) laser is inserted through the needle until resistance is encountered, then the laser is activated in 40-W 0.4-second pulses. The fiber is advanced after each pulse for a distance of 1 cm. Nine of the 12 reported patients experienced pain relief during the 2-minute procedure, although five of these nine subsequently had open operations for recurrent sciatica. Choy, et al., were unaware of complications related to the laser treatment.

This letter reports my recent experience in caring for a patient after failed Nd:YAG laser nucleolysis. The patient had ruptured the L5–S1 disc in April, 1990, and had symptoms of left S-1 radiculopathy unresponsive to conservative treatment. In November, 1990, the neurological examination prior to the laser procedure revealed weakness of the left gastrocnemius-soleus muscle complex, depression of the left ankle-jerk reflex, limited range of motion of the lumbar spine due to paravertebral muscle spasm, and positive left Laségue’s test at 30°. Nonenhanced magnetic resonance (MR) imaging showed a subligamentous disc herniation at L5–S1 compressing the left S-1 nerve root a few millimeters caudal to the disc space. The patient underwent laser nucleolysis at another institution, at which approximately 2100 J was administered via an Nd:YAG laser to the L5–S1 interspace. The patient immediately reported increased left sciatica.

Repeat MR imaging 2 months later showed persistence of the left L5–S1 disc herniation. Gadolinium enhancement was noted around the S-1 nerve root, indicative of epidural scar and/or inflammation adjacent to the swollen left S-1 nerve root. At surgery, I found a carbonized, blue-discolored anulus and posterior longitudinal ligament adjacent to an encapsulated sequestrum of soft disc material. No evidence of a laser track was seen in the disc material obtained from the interspace. I presume that the laser energy had vaporized a core of soft disc material and proceeded to coagulate the adjacent anulus fibrosis and posterior longitudinal ligament. The S-1 nerve root was protected from further laser insult by the sequestered disc material which lay posterolateral to the carbonized anulus-posterior longitudinal ligament complex.

This patient was very fortunate in that the dura and nerve root were not injured. The caveat to all those surgeons potentially interested in laser discolysis is obvious.

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Reference

Variations in the A₁ Segment

To the Editor: In the recent paper by Mäurer, et al. (Mäurer J, Mäurer E, Perneczky A: Surgically verified variations in the A₁ segment of the anterior cerebral artery. Report of two cases. J Neurosurg 75:950-953, December, 1991), one of the variations presented is that of the A₁ segment of one anterior cerebral artery coursing between the optic nerves before joining with the other A₁ segment to form the anterior communicating artery. The authors cite several other reports in the literature in which a similar interoptic A₁ segment anomaly is described.

We know of at least 12 more reports of the same anomaly in humans.1-6,8,9,11,14-16 The anomalous anterior cerebral artery often supplies an azygous A₁ segment, and is sometimes associated with a duplicate A₁ segment that courses lateral to the optic nerve in the “normal” location. Frequently associated are other congenital vascular and/or nervous system anomalies.

More importantly, Mäurer, et al., as most other authors before them, did not discuss the ontogenetic basis of the anomaly. This is probably because the literature on human cerebrovascular ontogeny does not impart an immediately obvious explanation. However, Moffat,10 studying cerebrovascular ontogeny in the rat, appears to be the only writer to have understood the developmental basis of this human anomaly. Moffat’s report provides an excellent example of how comparative anatomy may elucidate the etiology of a cerebrovascular variation when the etiology is not clear from the human ontogeny alone, as we have recently described.7 This is possible because the development of the brain vasculature of other species generally proceeds...