Neurosurgical forum
Letters to the editor

Ventral Artery Compression

To The Editor: We read with interest the article by Sell, et al. (Sell JJ, Rael JR, Orrison WW: Rotational vertebrobasilar insufficiency as a component of thoracic outlet syndrome resulting in transient blindness. Case report. J Neurosurg 81:617–619, October, 1994). The patient appears to have been dramatically cured by an operation but the case report does not clearly define the pathology seen at operation.

The authors state: “At surgery, a tight anterior scalene muscle was discovered compressing the left vertebral artery and lower cord of the brachial plexus between the anterior and middle scalene muscles.” This is an anatomical impossibility. These cords are in the axilla. Presumably, what is meant is the lower “trunk” of the brachial plexus. Because the scalenus anticus originates from the anterior tubercles of the transverse processes, it is difficult to understand how that structure can compress the vertebral artery, which is running upward to the foramen transversarium of C-6 quite medially. The scalenus medius can compress the lower trunk of the plexus, but is less likely to compress the vertebral artery unless it is associated with a congenital band. The various congenital bands and variations of vestigial musculature have been well described, and we wonder if one of these abnormalities was the cause of this patient’s vascular compression syndrome.1–3 If this was indeed the case, the appropriate treatment is the division of the band of abnormal vestigial muscle, which was compressing the vertebral artery. If it was thought that scalenus anticus was somehow compressing the C-8 and T-1 spinal nerves and the origin of the lower trunk, then division of scalenus anticus is the appropriate treatment. What, however, is the logic of resecting the first rib? The first rib in no way compresses the vertebral artery.

We would be interested in the authors’ clarification of the operative findings to assist in the management of any future patients presenting with this syndrome.

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References

To The Editor: Severe vertigo may be the only symptom of neck torsion.1 In a recent case this vestibular activation (“lightheadedness”) was accompanied by “cerebral” blindness (Sell JJ, Rael JR, Orrison WW: Rotational vertebrobasilar insufficiency as a component of thoracic outlet syndrome resulting in transient blindness. Case report. J Neurosurg 81:617–619, October, 1994). It is therefore likely that this blindness was of labyrinthine origin mediated by reflex retinal artery constriction. Such reflex labyrinthine blindness has been well documented in cases of “cortical” blindness after minor head injury or kick on the neck.2

This explanation no doubt sounds far-fetched; however, it is not as implausible as postulating that a healthy 44-year-old woman without vascular disease had a muscle pressing on her vertebral artery that abolished blood flow to both visual cortices without inducing neurological symptoms.

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References

RESPONSE: We very much appreciate the comments of Drs. Kline and Hudson regarding our paper. Unfortunately, we must rely on the operative notes as the only record for the description of the curative surgical procedure in this case. It would appear that Drs. Kline and Hudson are entirely correct in their assumption that the term “trunk” would best be substituted for cord in the surgical description. It is entirely possible that vestigial musculature contributed to the compressive syndrome in this patient. The release of a unilateral vertebral artery compression relieved the dramatic symptoms in this patient. We hope that despite the concerns of Drs. Kline and Hudson regarding the surgical approach and description, this case can serve as a reminder of one of the significant clinical conditions associated with unilateral vertebral artery compression.

Regarding the interesting comments by Dr. Gordon, it would indeed appear to be fortunate that his diagnosis was not made in this case lest the patient continue to be plagued with her devastating symptoms.

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Stereotactic Biopsy for Nonpilocytic Astrocytomas

To The Editor: We read with interest the paper of Lunsford and coworkers (Lunsford LD, Somaza S, Kondziolka D, et al: Survival after stereotactic biopsy and irradiation of cerebral nonanaplastic nonpilocytic astrocytoma. J Neurosurg 82:523–529, April, 1995), which reports on survival after stereotactic biopsy and percutaneous radiotherapy of cerebral, nonpilocytic astrocy-
tomomas. The authors have analyzed the outcome of 35 consecutive patients with histologically verified nonpilocytic low-grade astrocytomas (according to the grading system of Burger et al.), who were treated with percutaneous radiotherapy (median dose 56 Gy; conventionally fractionated) between 1982 and 1992. There was no perioperative morbidity and mortality after stereotactic biopsy. The authors reported a 5-year survival rate of 88.4% (median follow up 62 months). Nine patients had died at the time of the study’s end: six of nine due to malignant transformation. It was concluded that early diagnosis and early radiation therapy are appropriate and effective treatment options.

The analysis of the efficacy and toxicity of radiotherapy alone for patients with low-grade astrocytomas is an important issue because the value of cytoreductive surgery is not well established, and the incidence of radiotherapy after surgery is usually high. However, the authors did not define the objective of this study clearly, although a prospective phase I–II study is a defined objective per se. The absence of any defined study protocol is irritating. The authors stated that all patients were treated consecutively, which does not say anything about an inherent selection bias in this one-institution study. Did the authors evaluate the efficacy of radiation therapy as an alternative to tumor resection? Did they select a specific patient population not suitable for surgery/radiosurgery/stereotactic radiotherapy or observation? We find it remarkable that 1) the number of patients treated (3.5 patients/year on average) was extremely low; 2) the incidence of a nonlobar tumor location was higher (13 of 35) than usually reported for patients suitable for surgery; 3) patients with brainstem tumors fared as well as those with lobar astrocytomas; and 4) no information was provided concerning the delineation of these tumors in either the computerized tomography or magnetic resonance studies. Taking into account the extremely favorable outcome for the patients presented here we assume there must have been specific selection criteria in this study, but this has not been addressed by the authors. It is disturbing that the authors do not make any effort to explain their outstanding treatment results: results that may be due to selection (the favorable natural course of the disease), therapy, or both.

The authors state that early diagnosis and early radiotherapy are most appropriate for patients with astrocytomas with moderate or no mass effects. However, the “best” treatment time has not been analyzed in this study. The duration of symptoms before therapy was not a prognostic factor in recent studies. The authors further maintained the absence of any side effects such as cognitive deficits, affective imbalances, or the like due to radiation treatment; however, no valid neuropsychological tests had been performed. A longitudinal neuropsychological investigation would be extremely helpful, particularly in consideration of the fact that 27 patients with a pretreatment performance status of 100% had been referred to external-beam radiation. Clearly, this is difficult to achieve, however, the objective of a phase I study is to test treatment toxicity.

This study does not define the appropriate patient population suitable for radiation therapy as initial treatment. We think that this should have been the most important objective of this study. The conclusion that patients with low-grade astrocytomas with no or moderate mass effect could benefit most from early diagnosis and early radiotherapy is not substantiated by the data. The authors did not discuss the possibility of a “wait-and-see attitude.” Modern alternative treatment modalities, such as stereotactic radiation therapy, interstitial radiosurgery, or radiotherapy, which do not exclude the option of additional percutaneous radiation therapy in the case of a progressive or malignant transformation of the disease, have not been mentioned.

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References

RESPONSE: The senior author is somewhat nonplussed to find himself irritating the readers from Freiburg. Although the senior author has been provocative on a number of occasions, he never intended to irritate the readers of the Journal of Neurosurgery. Dr. Kreth and colleagues raise several points relative to the unresolved nature of the proper role of stereotactic biopsy, cytoreductive surgery, radiation, and other alternative treatments in the management of nonanaplastic, nonpilocytic astrocytomas. After they review this letter, we are hopeful that they will go back and read the last paragraph of the abstract of our paper and the last two paragraphs in the Conclusions section on page 528. Their comments should be placed in the perspective of these words.

The correspondents may rest assured that the small number of patients in this series during a 10-year interval represents only a portion of the numbers of patients referred to us with glial neoplasms: some of whom underwent biopsy; some of whom underwent cytoreductive surgery; all of whom had radiation therapy; and many of whom had other treatment modalities as well. We have already reported our experience with the fallacious concept that image guidance alone (either computerized tomography or magnetic resonance imaging) provides a reliable histological diagnosis for glial neoplasms. In our experience almost one-third of patients will have an anaplastic glial neoplasm or another diagnostic entity altogether. Therefore, it is absolutely clear that empirical radiation therapy is contraindicated; however, a “wait-and-see attitude” is abhorrent in the senior author’s opinion. How can neurosurgeons continue to argue over this silliness?