Dynamic Chiropractic

Acoustic Neuroma

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The acoustic neuroma arises from the vestibular portion of the eighth cranial nerve in the internal auditory canal. Acoustic neuromas are also termed "acoustic schwannomas" and "neurilemmomas." Acoustic neuromas account for approximately five percent of intracranial tumors and 90 percent of cerebellopontine angle (CPA) tumors. The incidence for unilateral acoustic neuroma is highest between the ages of 30 to 60 years with male equalling female prevalence. Bilateral acoustic neuromas are possible and are characteristic of neurofibromatosis, an autosomal dominant condition which manifests in the teenage years or in the early twenties. The duration of illness due to acoustic neuroma ranges from two to ten years, or longer. Unresectable acoustic neuromas are often fatal, therefore any patient with a unilateral sensorineural hearing loss and any evidence of vestibular abnormality should be evaluated for the presence of a cerebellopontine angle tumor.

The acoustic neuroma produces symptoms initially by compressing the nerves in the narrow confines of the internal auditory canal. As the tumor enlarges it produces a funnel-shaped enlargement or erosion of the temporal bone and protrudes through the internal acoustic meatus to compress cranial nerves VIII, V, VII, and other brain stem and cerebellar structures. Cranial nerves VI, IX, X, XI, and XII are involved only in the late stages because of a massive tumor presence in the cerebellopontine angle region.

The earliest sign of an acoustic neuroma is the spontaneous appearance of episodic dizziness with problems in speech discrimination. Greater than 50 percent of patients complain of dizzy feelings described as unsteadiness, dysequilibrium, or imbalance. An acoustic neuroma patient can often describe a lateralizing feature such as a tendency to fall to one direction. Most patients with acoustic neuromas present with a progressive unilateral hearing loss which takes the form of a sensorineural hearing impairment. The hearing impairment may be so slight that the patient may only complain of either a difficulty in speech discrimination when listening to telephone conversations or tinnitus characterized as a high-pitched ringing. The auditory symptoms are due to compression of cochlear nerve by the expanding tumor. Additionally, another early sign of acoustic neuroma is a depressed or absent corneal reflex.

Late signs of acoustic neuromas include facial nerve palsy, sensory loss to the entire ipsilateral side of the face, gait unsteadiness, taste disturbance, and a diminished corneal reflex. Less common features include facial pain, spontaneous nystagmus, and lower cranial nerve palsies. Symptoms of increased intracranial pressure (headaches, papilledema, and occasional loss of consciousness) are only caused by very large acoustic neuromas. Nystagmus due to acoustic neuroma is coarse and slow upon gaze directed to the side of the lesion and is rapid and fine when gaze is directed away from the side of the lesion. The affected labyrinth fails to respond to caloric testing.

Tumors rarely cause intermittent or recurrent attacks of vertigo. Therefore, it is important to consider the presence of cerebellopontine angle tumors in all patients with persistent unilateral tinnitus and hearing loss and mild vestibular symptoms which persist between attacks.

References

Baloh RW: The dizzy patient: treatment options, in Hachinski, VC, Challenges in Neurology, F.A. Davis, Philadelphia, 1992.

Bonikowski FP: Differential diagnosis of dizziness in the elderly. Geriatrics, Vol. 38, No. 2, 1983.

Weiss HD: Dizziness, in Samuels, M.A., Manual of Neurological Therapeutics, Second Edition, Littlee, Brown, and Company, Boston, 1984.

Rowland LP: Merritt's Textbook of Neurology, 7th Edition, Lea and Febiger, Philadelphia, 1989.

Patten J: Neurological Differential Diagnosis, Springer-Verlag, New York, 1982.

Gundersen CH: Quick Reference to Clinical Neurology, J.B. Lippincott, Philadelphia, 1982.

DeJong RN: The Neurologic Examination, 4th Edition, Harper and Row, New York, 1979.

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