Journal of the Neurological Sciences 401 (2019) 79–80

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Letter to the Editor

Response to "Acute and subacute sensorineural hearing loss after radiosurgery for vestibular schwannomas: Avoiding what is avoidable?"

ARTICLE INFO

Keywords:
Vestibular schwannoma
Stereotactic radiosurgery
Acute sensorineural hearing loss
Sudden deafness

We thank Dr. Tuleasca and the colleagues' suggestion about our paper entitled "Acute sensorineural hearing loss in patients with vestibular schwannoma (VS) early after cyberknife radiosurgery" [1]. They mentioned their experience about adverse radiation events (ARE) during the first months after gamma knife radiosurgery based on their paper published on the proceedings of the 18th international meeting of the Leksell gamma knife society [2]. They reported that the incidence of ARE was 22% including vertigo, gait disturbance, exacerbation of pre-existing hearing loss, etc. The majority of their patients with acute sensorineural hearing loss following radiosurgery recovered after one-week steroid therapy. Thus, they expanded discussion on how to preserve hearing after radiosurgery.

Although their suggestion sounds valuable, unfortunately, it is an incoherent topic. Our 6 patients were the victims of VS who had "sudden" onset sensorineural hearing loss after radiosurgery. Definition was based on the criteria proposed by the National Institute on Deafness and other Communication Disorders in United States [3], namely a rapid decline (< 3 days) of mean sensorineural hearing loss > 30 dB in at least 3 contiguous frequencies. These criteria have already mentioned in the text [1], perhaps Dr. Tuleasca missed them. Those with mean hearing loss < 30 dB were likely due to temporary threshold shift from tissue reaction after radiosurgery, and were excluded from our cohort.

It is our hospital policy that every VS patient underwent an inner ear test battery comprising audiometry, caloric test, ocular vestibular-evoked myogenic potential (oVEMP) test and cervical vestibular-evoked myogenic potential (cVEMP) tests before and after stereotactic radiosurgery [4]. To determine the nerve origin of VS in those who received radiosurgery is difficult because tissue proof is lacking. Alternatively, the use of oVEMP and cVEMP tests help predict the tumor origin, since the cVEMP test evaluates the ipsilateral sacculo-collic reflex via the inferior vestibular nerve, whereas the oVEMP test runs through the superior vestibular nerve to assess the vestibulo-ocular reflex [5]. Restated, each patient underwent comprehensive inner ear function testing prior to radiosurgery as baseline data. Following radiosurgery, if the hearing level deteriorated > 30 dB in three contiguous frequencies, such cases were enrolled to this study. Those patients failed to fulfill this criteria i.e. temporary threshold shift (< 30 dB) were excluded since such cases have been substantially reported in the literature as Dr. Tuleasca said [2,6,7].

Noxious stimuli i.e. irradiation insult to the inner ear produce excessive reactive oxygen species and reactive nitrogen species that are directly responsible for oxidative intracellular damage, which disrupt cell components by oxidizing stable molecules. This process activates the programmed cell death pathway in hair cells within the cochlea, resulting in sensorineural hearing loss [8]. Temporal bone histopathological studies in patients with sudden sensorineural hearing loss have demonstrated various inner ear pathological findings such as degenerative changes in the organ of Corti and strial vascularis, degeneration in the spiral ligament and cochlear neurons, cochleosaccular atrophy, and fibrosis/ossification of the cochlea or semicircular canals [9]. Such various pathological features are not easily resolved by one-week steroid treatment. Notably, one reviewer suggested using the term" acute" hearing loss instead of "sudden" hearing loss to discriminate from "idiopathic" sudden deafness, which may be the reason why Dr. Tuleasca misinterpreted our cases as temporary threshold shift after radiosurgery like their cases.

The radiation dosage to the cochlea is, of course, one of the major predictors for hearing preservation after radiosurgery. For gamma knife radiosurgery, recommended dose is kept < 4.2 Gy in a single fraction [7]. For CyberKnife radiosurgery, Hayden Gephart et al. [10] suggested that controlling for differences in cochlear volume among subjects, each additional mm^3 of the cochlea receiving 10 to 16 Gy (single session equivalent dose of 6.6–10.1 Gy) significantly increased the odds of hearing loss by approximate 5%. In our cases, no cochlea irradiation dose was allowed > 8 Gy in three-session treatment. Therefore, possible risk of excessive volume and dose of cochlear irradiation can be neglected, and we postulated that potential radiation damage to the cochlear nerve may be responsible for the sudden onset hearing loss.

All our VS patients received corticosteroid (dexamethasone 8 mg) daily for 5 consecutive days after the start of radiosurgery [4]. Potential benefits of the corticosteroid for hearing impairment early after radiosurgery have been well documented [2,11], such as improving inflammation and edematous swelling of the inner ear endorgans; direct cellular effects of steroid on the cochlea to restore the hearing after damage; and reduced transient tumor compression on the nerve and vessels by inflammatory swelling. Opposed to this temporary threshold shift caused by tissue reaction after radiosurgery, our patients with

https://doi.org/10.1016/j.jns.2019.04.024
Received 12 April 2019; Accepted 15 April 2019
Available online 17 April 2019

"sudden" onset hearing loss after radiosurgery was administered with plasma expander (40% dextran, 1.0 L for 3 days) followed by oral antioxidants (*N*-acetyl-L-cysteine 600 mg, twice daily) for 3 months [1]. However, hearing outcome is not satisfactory. Only one patient had hearing improvement while hearing of the other 5 patients remained unchanged three months after treatment. Thus, failure to recovery of sudden onset hearing loss after radiosurgery is likely attributed to the severity and irreversibility of the cochlear nerve damaged by irradiation.

Conflict of interest

Nil

Financial disclosure

Nil

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