Time and geographic clustering of adult patients with unilateral idiopathic sudden sensorineural hearing loss

Sapideh Gilani, Jennifer J. Shin

Department of Surgery, Division of Otolaryngology, University of California San Diego, United States

Department of Otolaryngology, Harvard Medical School, United States

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ABSTRACT

For the preponderance of patients with unilateral idiopathic sudden sensorineural hearing loss (SSNHL), the etiology is unknown. The pathogenesis of disease and a definitive mechanism for this abrupt hearing loss has not yet been elucidated. We hypothesize that there is a time-clustered presentation for idiopathic SSNHL; from an epidemiological standpoint, this suggests an environmental or infectious etiology. At a tertiary referral center in the northeast United States, we have observed that adult patients with SSNHL present in clusters with interspersed intervals of time with no presentations.

Introduction

Sudden sensorineural hearing loss (SSNHL) is defined as unilateral hearing loss which evolves in less than 72 h with a minimum 30 dB (dB) loss in 3 frequencies on pure-tone hearing evaluation [1]. While infrequent relative to other conditions (27 per 100,000 in the United States) [2], SSNHL remains a priority for identification and management [1], since patients may abruptly become deaf, and benefit only if treatment is provided within 14 days of onset of symptoms [3]. The etiology for this condition is not well defined, and a variety of causes have been suggested [4,5]. A recent systematic review concluded that the etiology may be idiopathic (71%), infectious (13%), primary otological (5%), trauma (4%), vascular or hematologic (3%), neoplastic (2%), and related to other causes (2%) [6]. Overall, most patients with SSNHL have no demonstrable reason for the sudden loss.

The diagnosis of idiopathic SSNHL is made based on clinical criteria, including audiometry, timeframe of symptomatology, and lack of the following diagnoses: bacterial infection, traumatic, vascular, hematologic, or neoplastic disease [1]. Physiological or laboratory diagnostics for SSNHL are limited, because the inner ear cannot be directly assessed at the time of an event; the inner ear space is just millimeters in size, and encased in the densest bone in the body, such that obtaining a directed biopsy or culture is problematic. In fact, opening that inner ear results in a potentially permanent deafness, introducing a risk of deteriorating the already present hearing problem in an irreversible way [7].

Viral etiologies have been implicated but not definitively isolated [4]. The cornerstone of proven therapies for SSNHL are oral and/or intratympanic corticosteroids, and these must be delivered rapidly [3]. The responsiveness to these agents and the short time course in which they are effective suggest the possible initiation of a causative inflammatory process [3]. Given the difficulties involved in directly accessing the inner ear, studies investigating a viral etiology of SSNHL have utilized serum viral titers [8], but systemic viral infection does not necessarily dictate that the virus entered the inner ear and was causative. Thus, additional approaches are needed to consider the possibility of a viral etiology.
Hypothesis

From an epidemiologic standpoint, time and geographical clustering could suggest an infectious etiology. Prior to the advent of modern microbial technology, epidemiologists relied on outbreak patterns to determine whether an infectious etiology might exist. In the classic account, John Snow demonstrated that cholera was an infectious disease by demonstrating time and geographical clustering of deaths from the disease [9]. The causative microbe was identified subsequently, and targeted scientific experiments were designed based on his work.

We hypothesize that there may be a time-clustered presentation of SSNHL in each locale. More specifically, there may be an elevated number of cases in the initial weeks (e.g. weeks 1–2), followed by a period of decreased incidence where no cases arise (e.g. during week 3–6). Then, if an infectious or environmental etiology resurges, we may see the case numbers rise again (e.g. weeks 7–8), followed by yet another period of waning incidence. To our knowledge, a time-clustered presentation of idiopathic SSNHL in specific locales has not been previously proposed.

Evaluation of the hypothesis

If SSNHL is virally triggered, then occurrences could be time and geographically clustered. If this hypothesis is correct, occurrences would map temporally as shown in Fig. 1 and geographically as shown in Fig. 2, with a resultant observed longitudinal pattern as seen in Fig. 3. Thus, in order to evaluate this hypothesis, we analyzed our hospital databases for clustering that we had noticed in our practices (Fig. 4).

As an initial foray into this query, after receiving an exemption from the Partners Institutional Review Board, we queried the Research Patient Data Registry (RPDR) at Partners Healthcare and searched for patients who, at the time of the visit, had a new diagnosis of unilateral SSNHL (ICD-9 code 388.2) and who also received treatment with intratympanic dexamethasone (CPT code 69801).

Among 152,646 patients seen in the otolaryngology clinic at Brigham and Women’s Hospital (BWH) from January 2009 to November 2014, the incidence of those with unilateral SSNHL who had their first treatment with intratympanic dexamethasone was determined (counts per 1000 patients seen). Visual inspection of these data over time suggests a potential time-clustering in the number of patients with SSNHL who were treated with their first injection of intratympanic dexamethasone (Fig. 4). Sample sizes are inherently limited by the infrequency of disease, but larger, coordinated studies could demonstrate these patterns in a more substantial way.

Other diseases in the respiratory tract have shown obvious seasonal variations [10], and some infectious etiologies have been previously studied for SSNHL [4,7,8,11]. Some authors have suggested that the annual incidence of idiopathic SSNHL may be increasing [2]. Future studies could also assess whether other infectious disease occurrences occur in a temporally related way to SSNHL. Since culturing the cochlea for infectious agents is currently infeasible, an alternative could be a study that would support the infectious or environmental etiology of SSNHL based on epidemiological criteria. For example, multi-institutional studies could be performed which mapped the neighborhoods of patients during their initial presentation of SSNHL (Fig. 2). These studies could demonstrate time clustering and geographical clustering suggestive of an infectious etiology. We thus propose this hypothesis as worthy of further study.

Conclusions

We hypothesize a time- and geographic-clustering of patients presenting with SSNHL. Such clustering would suggest a potential infectious etiology for this condition and merits further epidemiological investigation.
Fig. 2. Hypothetical involvement of each neighborhood with environmental or infectious factor for idiopathic sudden sensorineural hearing loss.

Fig. 3. Hypothetical number of patients with idiopathic sudden sensorineural hearing loss and month of presentation from a clinic servicing neighborhoods A-D.

Fig. 4. Idiopathic sudden sensorineural hearing loss patients treated with first intratympanic dexamethasone per 1000 at Brigham and Women's Hospital Otolaryngology clinic Jan 2009-November 2014.

Competing interests
None.

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Author contributions
Sapideh Gilani, M.D., concept, study design, data extraction, data analysis, manuscript drafting, final approval of manuscript for submission; Jennifer Shin, concept, study design, data extraction, data analysis, manuscript drafting, final approval of manuscript for submission.
Disclosures


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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.mehy.2018.11.010.

References