



Computed Tomography and Magnetic Resonance Imaging Findings in Idiopathic Sudden Sensorineural Hearing Loss



Joseph W. Clyde, B.S.¹; Vijay A. Patel, M.D.¹; Sangam Kanekar, M.D.¹; Huseyin Isildak, M.D.¹

¹Department of Surgery, Division of Otolaryngology – Head and Neck Surgery, The Pennsylvania State University, College of Medicine, Hershey, PA, USA

Introduction

Sudden sensorineural hearing loss (SSNHL) has been described as a hearing loss of ≥ 30 dB over 3 sequential frequencies in a standard pure-tone audiogram over < 72 hours¹. SSNHL can be classified audiometrically into two varieties: sensory (cochlear) and neural (retrocochlear). Sensory SNHL may be caused by abnormalities of the bony labyrinth or membranous labyrinth whereas lesions of the internal auditory canal or cerebellopontine angle may cause neural SNHL². Hearing loss is a very common initial presenting symptom with a broad associated differential of possible etiologies³. Some of the most common entities include: presbycusis (age-related hearing loss), noise-induced hearing loss, Ménière's disease, medications, and infections⁴. Other etiologies include: vascular anomalies, autoimmune disorders and neoplastic diseases. Furthermore, dozens of genetic loci have also been identified and linked to hearing loss.

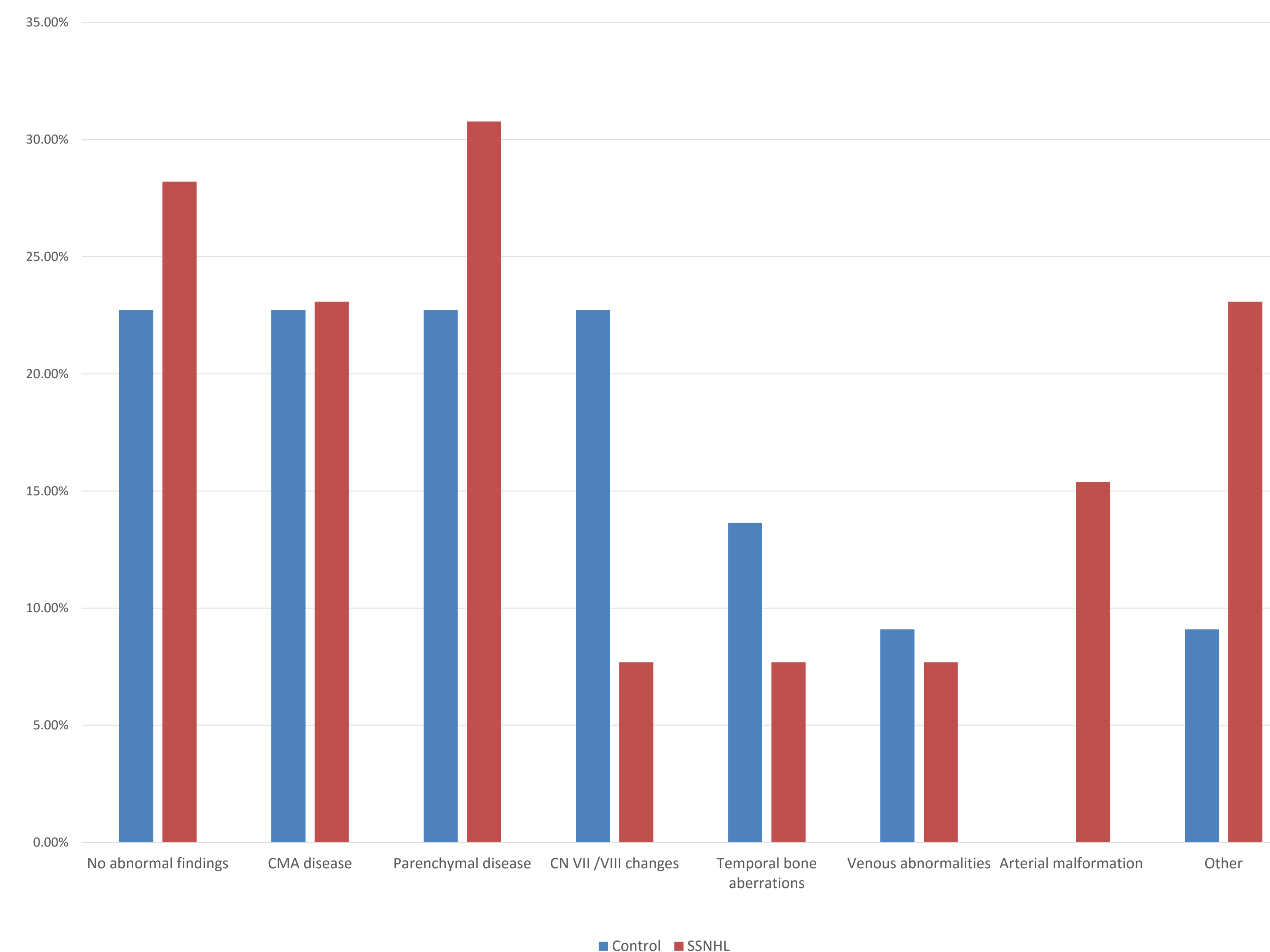
Background

The incidence of SSNHL is reported to be 5 to 20 per 100,000 persons; however rates have been reported as high as 160 per 100,000 per year with approximately 4,000 new cases reported yearly in the United States⁵. The prevalence is highest for those who are white, male, elderly, less educated, and those who have a history of diabetes mellitus, hypertension, and/or a greater than a 20 pack-year history of smoking. The predominant form of SSNHL is unilateral in location (95%), and 90% of cases are idiopathic in nature⁶. Generally, imaging in patients with SNHL is of low yield given the fact that the most common observed abnormality occurs at the level of the hair cells within the organ of Corti⁷. Positive MRI findings have been identified in 57% of patients with SSNHL with only 7% suggesting a possible etiology^{8,9}. Recently, studies have focused on the use of the MRI to exclude retrocochlear etiologies. In this study we attempt to identify MRI findings which might be related to SSNHL.

Methods

We performed a retrospective case-controlled chart review including patients between January 2006 – January 2015. We reviewed 39 patients with a documented diagnosis of idiopathic sudden SNHL that underwent 1.5 or 3 Tesla MRI with and without contrast of the brain; 22 of these patients also had a CT of the temporal bones without contrast. Patients also had otologic, vestibular, and audiometric examinations. The control group consisted of 22 patients without a diagnosis of sudden SNHL who also underwent MRI with and without contrast of the brain; 6 of these patients also had CT of the temporal bones without contrast. Patients without an imaging study, younger than 18 years of age, and/or a diagnosis of Ménière's disease were excluded.

Figure 1. MRI Findings



Results

MRI

- Findings in patients with idiopathic SSNHL include: 9 patients (23.1%) with chronic microangiopathic disease, 12 patients (30.8%) with cerebral or brainstem parenchymal disease, 6 patients (15.4%) with arterial malformations, 3 patients (7.7%) with facial and/or vestibulocochlear nerve changes, 3 patients (7.7%) with temporal bone aberrations, and 3 patients (7.7%) with venous abnormalities, and 8 patients (23.1) with “other” abnormal findings.
- A Fisher’s exact test revealed no statistical difference between the MRI findings in patients with SSNHL and the control group. However, the increased incidence of arterial malformations seen on MRI of the SSNHL group is approaching significance (P = 0.079).

CT

- Twenty-two patients from the sudden SNHL group and 6 patients in the control group had a CT. Findings in patients from the idiopathic sudden SNHL include: 4 patients (18.2%) with cerebral or brainstem parenchymal disease, 3 (13.6%) with arterial malformations, and 2 (9.1%) with temporal bone aberrations.

Conclusions

- Our MRI and CT findings support microvascular disease, cerebral parenchymal disease, and cranial nerve changes as possible etiologies in the development of idiopathic SSNH, however, a larger sample is needed to detect these differences.
 - We will review many more images to increase the power of our study
- Otolaryngologists can provide supporting diagnostic information during the initial care and work-up of patients presenting with signs and symptoms consistent with idiopathic SSNHL by utilizing MRI as well as CT imaging modalities.

Contact

Huseyin Isildak, MD
Division of Otolaryngology - Head and Neck Surgery
Phone: 717-531-8946
Fax: 717-531-6160
Email: hisildak@hmc.psu.edu

References

- Schreiber B, Charlotte A, Haskard, DO, Luxon LM. Sudden sensorineural hearing loss. *Lancet*. 2010;375:1203-11.
- Swartz JD. Sensorineural hearing deficit: a systematic approach based on imaging findings. *Radiographics*. 1996;16(3):561-574.
- Wilson YL, Gandolfi MM, Ahn IE, et al. Cost analysis of asymmetric sensorineural hearing loss investigations. *Laryngoscope*. 2010;120(9):1832-6.
- Prasad J, Cousins VC. Asymmetrical hearing loss. *Aust Fam Physician*. 2008;37(5):312-6, 319-20.
- Stachler RJ, Chandrasekar SS, Archer SM, et al. Clinical practice guideline: sudden hearing loss. *Otolaryngol Head Neck Surg*. 2012; 146(suppl 3):S1-35
- Rauch SD. Clinical practice. Idiopathic sudden sensorineural hearing loss. *N Engl J Med*. 2008;359:833-40
- Swartz JD. Sensorineural hearing deficit: a systematic approach based on imaging findings. *Radiographics*. 1996;16(3):561-574.
- Cadoni G, Cianfoni A, Agostino S, et al. Magnetic resonance imaging findings in sudden sensorineural hearing loss. *J Otolaryngol*. 2006;35(5):310-316.
- Aarnisalo AA, Suoranta H, Ylikoski J. Magnetic resonance imaging findings in the auditory pathway of patients with sudden deafness. *Otol Neurotol*. 2004;25(3):245-249.