

Assessment of the risk factors for hearing loss in adult Nigerian population

Olusola Ayodele Sogebi

ENT Unit, Department of Surgery, College of Health Sciences, Olabisi Onabanjo University, Sagamu, Nigeria

ABSTRACT

Background: A reduction in hearing sensitivity is common in adults and was previously considered to be normal as age increases. However, other health variables may play a role in the sensory changes. This prospective, comparative, hospital-based study assessed the risk factors (RFs) associated with sensorineural hearing losses (SNHL) in adult patients in a specialized tertiary hospital clinic in South-western Nigeria. **Materials and Methods:** Patients with clinical diagnosis of hearing impairment (bilateral SNHL) were the test subjects and age and sex-matched comparable group without SNHL were the Controls. Using a structured questionnaire, variables assessed included current and past medical history, family and social history, use of medications including ototoxic drugs, and prolonged medications. **Results:** One hundred and twenty-seven patients participated in the study comprising of 76 test subjects with SNHL (including 14 with suspected ARHL) and 51 controls. 59.8% of the participants were males. Univariate analysis revealed statistically-significant differences in family history, alcohol consumption, smoking, exposure to noise, previous ear discharge, previous head injury, hypertension, diabetes, osteoarthritis, ototoxic drugs usage, prolonged medication and obesity between the two categories of subjects. Logistic regression analysis revealed family history, smoking, noise exposure, head injury, hypertension had significantly increased odds of developing SNHL. **Conclusion:** It was concluded that the RFs for SNHL in adult Nigerians were multifactorial while some of the RFs may be amenable to primary prevention. Legislation and public health education could facilitate reduction of SNHL in our community.

Key words: Adults, elderly, hearing loss, Nigerians, risk factors

Address for correspondence:

Dr. Olusola Ayodele Sogebi,
Department of Surgery,
OACHS, Olabisi Onabanjo
University, Sagamu, Nigeria.
E-mail: ayosogebi2000@yahoo.com

INTRODUCTION

Hearing loss is one of the most prevalent chronic conditions in adults worldwide¹ and it is classified as conductive, sensorineural or mixed in type. Conductive hearing loss has readily identifiable causes and is easily amenable to treatment but sensorineural hearing loss has more grievous consequences on the individual. Good hearing function is particularly required for adults in the working population who are exposed to noise and other challenging listening situations at work. Research has shown that hearing loss is associated with a greater need for recovery after work.² Such a need for recovery may increase the request for sick leave, suggesting that hearing loss may have adverse

economical consequences. Furthermore the likelihood of the hearing impairment to continue into older age after retirement is a concern.

Progressive bilateral sensorineural hearing loss which starts from the middle age and continues into the older years is characteristic of age-related hearing loss (ARHL). ARHL is the most common sensory impairment associated with ageing.³ Global incidence and prevalence of ARHL are projected to increase with the increase in average life expectancy. Age-related hearing loss is not reversible and it imparts on the health of elderly persons with adverse consequences which include physical dependence, domestic accidents, emotional and psychological disturbances; in addition to limitation in social interactions.⁴

Hearing aids, cochlear implants, assistive listening devices and other aural rehabilitation methods are the means of treatment. Advances in technology have refined hearing aids to be more comfortable with better amplification which should enable patients to receive assistance. Unfortunately many sufferers of hearing impairment particularly in sub-Saharan Africa cannot afford the cost of these treatments.

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A reduction in hearing sensitivity was previously considered to be a normal age-related occurrence. However, recent studies have suggested other health variables, apart from age, which play significant role in the sensory changes.⁵ Variables like sex, previous illnesses, vascular alterations or exposure to noise⁶ have been mentioned in recent literature to affect hearing and could favor the progression of hearing loss including ARHL.⁷ Consequently some authors have suggested that ARHL could be preventable by avoidance or control of these risk factors.

Most of the reports on hearing impairment in adults were from studies done in Europe and America while few studies have been done on hearing impairment among black population. However, not much work has been done on the risk factors associated with purely sensorineural hearing loss among adults and elderly patients in Nigeria. This study, therefore, aims to assess and identify the risk factors associated with SNHL in patients in a specialized clinic in South-western Nigeria. This knowledge will assist in the possibility of reducing the incidence and probably prevent the development of SNHL and by extension, ARHL.

MATERIALS AND METHODS

This is a prospective, comparative, hospital-based study that was conducted at the ENT Clinic of Olabisi Onabanjo University Teaching Hospital (OOUTH), Sagamu, Nigeria between July 2007 and June 2011. Consecutive adult patients attending the ENT clinic of OOUTH with diagnosis of hearing impairment were approached for recruitment into the study as subjects.

Consent was sought and obtained from the patients with clinical diagnosis of hearing impairment confirmed with pure tone audiograms (PTA) of bilateral sensorineural hearing losses with pure tone average of at least 25 dB. The general nature of the study, the benefits and potential risks were explained to the subjects. Voluntary participation and the possibility of withdrawal from the study at any time without affecting medical care and treatment, and maintenance of confidentiality of information were all emphasized to the subjects before consent was obtained. Other patients of comparable ages above 41 years but with no symptoms of hearing impairment and with normal pure tone audiograms were counseled as controls and consents obtained from them.

Non-consenting patients, patients with Meniere's disease, vestibular neuronitis, tympanic membrane perforations and those that previously had middle ear surgeries were excluded. Others excluded were patients without PTAs or with audiograms that revealed other forms of hearing impairment like conductive or mixed hearing loss.

Information was obtained from the patients using structured questionnaire. The information sought

included socio-demographics of the patients, otological symptoms experienced and duration, social habits, and medical histories. Medical history included history of previous ear discharge in childhood or adolescence, previous head injury with associated loss of consciousness, previously diagnosed hypertension (blood pressure of $\geq 140/90$ mmHg at least two consecutive clinic attendances or on anti-hypertensives), diabetes (fasting blood sugar ≥ 126 mg, on oral hypoglycemic agents or on insulin), osteoarthritis, epilepsy and sickle cell disease. Family history of ear disease was marked positive if a parent, sibling or a first-degree relative had a hearing impairment. Social history included consumption of alcohol and its frequency, history of past or current smoking within the previous six months, and exposure to noise especially at work like factory that use heavy, noisy and vibrating machines, a blacksmith shop, radio room/disco room, or welding shop for at least 2 hours per day for at least 5 days a week was also obtained. The history of medications including ingestion of ototoxic drugs like acetylsalicylate, aminoglycosides, diuretics, and 4-aminoquinolines antimalarial drugs, and prolonged use (at least for 6 months) of any medications especially for chronic diseases like hypertension and diabetes were obtained.

PTA was done in a sound proof room by using a diagnostic audiometer GSI67. The air and bone conduction hearing thresholds were measured at different frequencies from 0.5 to 8.0 kHz with masking done for the bone threshold measurements. The Pure tone average (PTAv) was calculated as the arithmetic mean for the air conduction thresholds at the six frequencies between 0.5 and 8.0 kHz. A subject was considered to have ARHL when in addition to the bilateral SNHL, the pure tone audiogram had an accentuation of the slope at the high tone frequencies starting from 2.0 kHz.

The weight and heights of the subjects were measured using the Surgifriend Medicals scale (Surgifriend Medicals, England) and the Body Mass index (BMI) was calculated by using the formula: $\text{Weight in kg}/(\text{Height in metres}^2)$.

The study protocol was approved by the OOUTH- Health Research and Ethics Committee (HREC) and the study was conducted in accordance with the principles of the Helsinki declaration.

The information obtained was entered into the spreadsheet. The participants were divided into two groups as Test and Control based on the diagnosis of SNHL (Present or Absent). Frequency tables were utilized in the description of variables while cross-tabulation was done to demonstrate the relationship between variables. Associations between continuous variables were examined by the Student's *t*-test while discrete variables were treated using Chi-square test. SNHL, as an independent predictor of

the variables, was evaluated by a series of bivariate logistic regression models in which each parameter was treated as the outcome variable and SNHL was regarded as the independent predictor. The data obtained was analyzed using the SPSS version 17.

RESULTS

One hundred and twenty-seven patients participated in the study comprising seventy-six subjects with SNHL (including 14 with suspected ARHL) and 51 controls. The age ranged between 45 and 94 years, with a mean age of 69.6 years (SD = 8.9). 59.8% of the participants were males with a Male:Female ratio of 1.5:1. 70.9% of the participants were married at the time of the study. Almost three-quarters of the participants had minimum of secondary school education, although 3 (2.4%) had no formal education. 81.1% of participants were semi-skilled workers or professionals while 18.9% were not skilled workers, as seen in Table 1.

Table 2 explored the relationships of the variables between the test subjects and the controls. There were no significant differences in age ($P = 0.321$), sex ($P = 0.848$), marital status ($P = 0.459$), level of education ($P = 0.426$), occupation group ($P = 0.100$), heavy alcohol consumption ($P = 0.775$), history of sickle cell disease ($P = 0.229$), epilepsy ($P = 0.402$) and BMI ($P = 0.141$) between the test subjects and controls. Other parameters including family history ($P = 0.031$), alcohol consumption patterns (no; $P < 0.001$, stopped; $P = 0.019$, occasional; $P < 0.001$), smoking ($P = 0.042$), exposure to noise ($P = 0.031$), previous ear discharge ($P = 0.022$), previous head injury ($P = 0.036$),

hypertension ($P = 0.022$), diabetes ($P = 0.032$), osteoarthritis ($P = 0.032$), ototoxic drugs usage ($P = 0.001$), prolonged medication ($P = 0.011$) and obesity (BMI ≥ 30 ; $P = 0.040$) revealed statistically-significant differences between the two categories of subjects.

Further subjection of variables to multivariate (logistic regression) analysis with SNHL as predictor and each of the significant parameters as the outcome variable is shown in Table 3. Previous ear discharge (OR = 3.7, $P = 0.087$), diabetes (OR = 3.8, $P = 0.102$), osteoarthritis (OR = 1.4, $P = 0.692$), prolonged medication (OR = 0.8, $P = 0.779$) and obesity (OR = 2.1, $P = 0.485$) dropped out and were not considered as risk factors. However, family history (OR = 26.3, $P = 0.038$), smoking (OR = 19.2, $P = 0.009$), noise exposure (OR = 17.3, $P = 0.001$), head injury (OR = 56.8, $P = 0.009$), hypertension (OR = 7.5, $P = 0.010$) and ototoxic drugs (OR = 6.3, $P = 0.013$) had significantly increased odds of developing SNHL. Conversely, stoppage of alcohol (OR = 0.03,

Table 1: Sociodemographic characteristics of the patients

Variable	n (%)
*Age	45-94 (69.6±8.9)
Sex	
Male	76 (59.8)
Female	51 (40.2)
Marital status	
Married	90 (70.9)
Others (divorced, separated, widow (er))	37 (29.1)
Education	
No formal education	3 (2.4)
Primary school	29 (22.8)
Secondary school	67 (52.8)
Tertiary	28 (22.0)
Occupational group	
Unskilled	24 (18.9)
Semi-skilled	52 (40.9)
Professional	51 (40.2)
Category of patient	
Control	51 (40.2)
Test	76 (59.8)

*Age range, mean±SD

Table 2: Comparison of characteristics in relation to patient category

Variable	Control n=51 (%)	Test n=76 (%)	P
*Age (average)	68.6	70.2	0.321
Sex (male)	58.8	60.5	0.848
Marital status (married)	74.5	68.4	0.459
Level of education (minimum secondary school)	82.4	69.7	0.426
Occupational group (professional)	45.1	36.8	0.100
Family history	7.8	22.4	0.031
Social history			
Consumption of alcohol			
No	35.3	76.3	<0.001
Stopped	35.3	17.1	0.019
Occasional/light	27.5	5.3	<0.001
Heavy	2.0	1.3	0.775
Smoking (previous or current)	7.5	18.4	0.042
Exposure to noise	23.5	42.1	0.031
Medical history			
Previous ear discharge	11.8	28.9	0.022
Head injury	3.9	15.8	0.036
Elevated blood pressure	23.5	43.4	0.022
Diabetes	11.8	27.6	0.032
Osteoarthritis	11.8	27.6	0.032
Sickle cell disease	2.0	6.6	0.229
Epilepsy	1.0	nil	0.402
Medications			
Ototoxic drugs	13.7	40.8	0.001
Prolonged medication	21.6	3.4	0.011
*BMI	25.8	26.8	0.141
BMI ≥ 30	7.8	21.1	0.040

*Values are means and statistics is student t-test; BMI – Body mass index

Table 3: Risk of developing SNHL

Variable	*OR	95% C.I.	P
*Family history	26.259	1.205-572.458	0.038
Alcohol consumption			
*Stopped	0.029	0.005-0.167	<0.001
*Occasional/light	0.003	0.000-0.042	<0.001
Heavy	0.019	0.000-4453.789	0.529
*Smoking	19.162	2.091-175.569	0.009
*Noise	17.298	3.120-95.902	0.001
Previous ear discharge	3.700	0.826-16.528	0.087
*Head injury	56.806	2.727-1183.512	0.009
*Elevated blood pressure	7.518	1.614-35.024	0.010
Diabetes	3.853	0.765-19.412	0.102
Osteoarthritis	1.394	0.269-7.217	0.692
*Ototoxic drugs	6.254	1.468-26.643	0.013
Prolonged medication	0.821	0.207-3.257	0.779
Obesity	2.053	0.273-15.435	0.485 ³

*ORs are Logistic Regression ORs; SNHL – Sensorineural hearing losses

$P < 0.001$), and occasional or light consumption of alcohol (OR = 0.003, $P < 0.001$) were significantly protective for developing SNHL.

DISCUSSION

This study indicated that the risk factors for SNHL in middle aged and elderly Nigerians are similar to those reported elsewhere. The risk factors associated with SNHL are multi-factorial and included family/genetic disposition, social and environmental factors, chronic medical conditions and use of medications. It is noteworthy that modifiable factors such as noise exposure, use of ototoxic drugs and smoking were major contributors to SNHL in this study.

It has been established that some degree of sensory hearing loss is inevitable as age advances⁶ and SNHL tends to be accelerated by a combination of genetic and environmental factors. In this study, subjects that had family histories of hearing loss had statistically significant increased odds of developing SNHL. It is hypothesized that there may be genetically-mediated links within family members who develop SNHL. Hereditary studies on hearing impairment using linkage analysis showed that hereditary hearing loss is genetically heterogeneous.⁸ The influence of heredity seems to be more pronounced at higher degrees of hearing losses. For instance, McMahan *et al.*⁹ reported that family history was most strongly associated with moderate to severe hearing loss. Similarly, heritability estimates by comparing different parameters (Total Hearing Loss (size), Uniform Hearing Loss (percentage of frequency-dependent hearing loss) and Bulge Depth) suggest a higher heredity for severe types of presbycusis compared to moderate or mild types.¹⁰

Noise exposure is a well recognized and probably most studied environmental factor causing hearing loss¹¹

manifesting with loss of hair cells with abrupt high-tone SNHL. Continual exposure to high noise levels will potentiate the development and perpetuate SNHL leading to an irreversible impairment.¹² Thus after a lifetime of noise exposure, it may be difficult to distinguish between noise induced hearing loss and ARHL both audiometrically and histologically.¹³ Studies on animals raised in augmented acoustic environment revealed protective anatomic and physical functions at both peripheral and central levels when compared with the controls that developed in a normal quiet environment.^{14,15} It is reasonable not to discountenance the effect of noise regarding hearing loss of any type.

In contrast, other studies did not find an association between history of noisy jobs and change in rate of hearing thresholds.^{16,17} Subjects that are prone to environmental noise include those involved in the use of heavy machinery, in recreation and sports, in military operations, firefighters and people working at the airports.¹⁸⁻²⁰ There is an almost uniform exposure to environmental noise from power generating equipment in our environment which may potentially increase the incidence of middle aged SNHL in the near future unless noise regulation is enforced. Avoidance of hazardous noise logically reduces development and the progression of sensory hearing loss. Public health education concerning noise pollution hazards and use of personal ear protection devices are other measures which can be deployed in controlling this hazard. Personal protection with simple measures of insert ear plugs provide about 15-25 dB attenuation in noise level and, can thus permit people to work in otherwise hazardous areas.⁶

In this study, current smokers had significantly increased odds of developing SNHL compared with non smokers. This is similar to reports from Asia,²¹ North America²² and Australia.²³ Cigarette smoking may affect hearing through its effects on antioxidative mechanisms or on the vasculature supplying the auditory system.²⁴ Animal studies identified nicotinic-like receptors in the hair cells, which suggested that smoking may have direct ototoxic effects on hair cell function through its potential effect on the neurotransmission of auditory stimuli.^{25,26} It is emphasized that smoking may play a role in hearing loss and modification of smoking habits may prevent, delay or ameliorate declines in hearing sensitivity.²²

It was noted in this study that hypertensives had significantly increased odds of developing SNHL compared with non-hypertensives. The effect of hypertension transcends almost all of the cardiovascular (CV) system. Hutchison *et al.*,⁵ examined the association between cardiovascular health and hearing function and found that low cardiovascular fitness in the old age group had significantly worse pure-tone hearing at high frequencies (2000 and 4000 Hz). They suggested a potentially positive impact of CV health on hearing

sensitivity over time. Fang²⁷ conducted image analysis of arterial vessels of the internal auditory meatus among the pre-senile and the aged with hypertension and atherosclerosis and found that the progress of the vessel changes in patients with presbycusis might be accelerated by atherosclerosis and hypertension. These changes result from endothelial dysfunction which predisposes to the development of a pro-thrombotic state due to the increase in the vascular wall.²⁸ When the endothelium cannot explicitly perform its duties, adhesion molecules, endothelial progenitor cells and pro-inflammatory vascular conditions develop leading to distortion of the ear microcirculation. These consequently alter the blood supply to the ear and impair cochlear membrane functions.²⁸

SNHL can be a sequel of previous head injuries especially at middle age. Hearing loss from head (which includes brain) injuries may be due to a disruption of the membranous portion, disturbance in the microcirculation, or hemorrhage into the fluids of the cochlea.⁸ Munjal *et al.*,²⁹ observed a higher prevalence of hearing impairment in the group of patients with closed head injury compared with control group, and also an association between the extent of auditory dysfunction and severity of traumatic brain injury.³⁰ It is expedient to prevent head injuries as much as practicable.

Ototoxic drugs are a heterogeneous group and cause hearing loss through different pathways. However, all the drugs have the tendency to initiate hearing loss which could become irreversible. The use of NSAIDs may transiently cause tinnitus and mild to moderate hearing loss as the drug impairs the active process of the outer hair cells and affects both peripheral and central auditory neurons. Aminoglycosides antibiotics can damage hair cells causing a non-reversible hearing loss predominantly in the high frequencies. Similarly cisplatin, a chemotherapeutic agent, appears to damage the outer hair cells first, and if exposure continues the inner cells. Loop diuretics such as furosemide or ethacrynic acid cause reversible hearing loss due to disruption of the ion balance in the stria vascularis. Quinine exposure to guinea pig cochlea led to a dose-dependent and reversible hyperpolarization followed by a depolarization of the hair cells' membrane potential.³¹

Some medications like Heat shock protein (Hsp) 70,³² peptide inhibitor of c-Jun N-terminal kinase,³³ Ebselen³⁴ and Heme oxygenase-1 (HO-1)³⁵ have been shown to attenuate the effects of ototoxic drugs. While these findings raise some hope, the practicability in all situations is doubtful and hence the use of drugs should be regulated. Health education of the general populace on early signs of toxicity of drugs, monitoring of the blood levels of drugs especially those that have dose-dependent side effects like gentimycin are other means of controlling hearing loss from ototoxic drugs. The need for all physicians to

be circumspect in prescription of medications cannot be overemphasized.

In this study, consumption of alcohol appears not to be significant, although it was mentioned as a risk factor for presbycusis.³⁶ Our study found that while heavy consumption of alcohol did not increase the risk of SNHL, occasional or light consumption was actually protective. This observation should be considered with reservation due to small number of patients employed in this study. In Japan, occasional or light drinkers showed significantly decreased risks of hearing loss while heavy drinkers did not have increased odds compared with non-drinkers.²¹ The risks of other diseases like carcinoma of the liver and other malignancies associated with alcohol consumption, with potentiated effect when combined with smoking precludes alcohol ingestion as a possible factor in reducing prevalence of SNHL.

Some limitations should be noted in this study. This is a hospital-based study and findings thereof may not represent occurrence in the community. Possibility of co-existing diseases in both the subjects and controls might affect results and healthy comparable individuals may not have similar results. Some parameters like diabetes and dyslipidaemia which confirm cardiovascular diseases could not be ascertained by only asking questions. The fact that only peripheral and not central auditory functions were measured is also a limitation as SNHL was suggested to represent a combination of deteriorated function of the auditory periphery and the central auditory system.³⁷

Despite the foregoing, this study has allowed us to quantify the risk factors associated with SNHL in a suburban middle-aged and elderly population. Results also suggested that some of the risk factors may be amenable to primary prevention. Legislation and public health education could facilitate reduction in SNHL in our community.

The need for further research especially longitudinal and cohort population-based studies on adults will be necessary to fully quantify the risk in the population. There is also a need for studies which will assess the progression of SNHL and possibly clarify the differences between ARHL and other types of SNHL as well as consider the possibility of arrest and reversal of the disease.

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