The Association of Reactive Oxygen Spesies Levels on Noise Induced Hearing Loss of High Risk Workers in Dr. Soetomo General Hospital Surabaya, Indonesia.

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ORIGINAL ARTICLE

The Association of Reactive Oxygen Species Levels on Noise Induced Hearing Loss of High Risk Workers in Dr. Soetomo General Hospital Surabaya, Indonesia

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Abstract Excessive noise exposure could increase the production of reactive oxygen species in the cochlea, thus causing the risk of noise-induced hearing loss (NIHL). Noise is commonly found in the industrial sites. However, public places like hospital also can have noisy location which risk the workers of NIHL. To analyzed the correlation of reactive oxygen species and hearing impairment to employees at risk in the hospital. Participants were obtained by identifying the employees in hospital from 3813. They were examined for baseline characteristics, hearing loss and reactive oxygen species. Hearing loss was defined as audiometry and tympanometry level. The statistical test that used in this study is Chi square test ($p < 0.05$). The proportion of participant was 42.43 ± 10.72 years old in women (58.33%) and noise levels at Dr. Soetomo General Hospital was 98.15 ± 8.16 dB in range 85.39–112.90 dB. The prevalence of NIHL was 47.92% (audiometry) and 70.83% (otoacoustic emission). Reactive oxygen species estimated 5.55 ± 4.39 ng/ml. Statistical analysis of reactive oxygen species to audiometry ($p = 0.993$) and reactive oxygen species to otoacoustic emission ($p = 0.647$). Increased production of reactive oxygen species that cause hearing loss, but there was no correlation between reactive oxygen species and hearing loss in risk worker at the hospital.

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Introduction

Noise possibly cause health problems in the form of noise-induced hearing loss (NIHL). In Nepal, it was reported that 31% of carpenters and 44% sawmill workers experience NIHL [1]. In the Tanzanian mining area reported that 12% workers have poor hearing and 35% have a mild hearing loss. Every year the NIHL population increases due to the hearing exposure [2].

Noisy exposure could increase the production of reactive oxygen species (ROS) in the cochlea. ROS is a mediator of cochlear cell damage, in addition biochemically ROS causes peroxidation of cochlear lipids and produces toxic substances. ROS itself was reported to persist cochlear for 7–10 days by post-exposure to the noise. If individuals are exposed to noise every day, they will have a higher risk to experience noise-induced hearing loss and other hearing loss [3].

Medical examination of hearing loss requires a tool for strengthening the diagnosis including using audiometry and otoacoustic emission (OAE) [4]. Audiometry is used to measure the subjective hearing impairment of the patient while OAE is used to evaluate the patient's hearing loss objectively [4, 5]. Thus, both examinations are important as the basis for effective therapy.

Indonesia is a country that has a large area and has various tribes [6]. Characteristics of workers in Indonesia is very unique, often found its workers tend not to comply for in the use of Personal Protective Equipment. Government regulations related to occupational health especially noise

thresholds in the workplace (PMK No. 70 tahun 2016 and per.13/men/x/2011) [7, 8]. Hospitals are one of the places to work and where to seek medical help, but some places in the hospital are a source of noise pollution. Dr. Soetomo General Hospital, Surabaya Indonesia is one of the places of work that has several noisy locations such as medical facility maintenance installation environmental sanitation installation, and nutrient installation. Based on the description above it need to be investigated and analyzed the association of reactive oxygen species levels on noise induced hearing loss of high risk workers in Dr. Soetomo General Hospital Surabaya, Indonesia.

Methods

The subjects of this study were employees of Dr. Soetomo General Hospital (RSUD Dr. Soetomo), Surabaya, Indonesia. The subjects must meet inclusion criteria and exclusion criteria. The number of employees were 3813 people and after being identified based on inclusion and exclusion criteria, 56 subjects were obtained. Inclusion criteria include employees at risk of exposure to noise pollution with noise < 85 dB, in hospitals (medical facility maintenance installation, environmental sanitation installation, and nutrient installation) and willing to participate the research by filling out informed consent sheets. Exclusion criteria include having ear abnormalities, results of type B tympanometric screening, and moderate or ever taking muscle-drugs drugs (Fig. 1).

The study used observational analytic study. Prior conducting the data retrieval the researchers conducted a test of ethics with the ethics committee of Dr. Soetomo General Hospital, Surabaya Indonesia (219/Panke.KKE/III/2017). Data collection was done in some potential noise locations in Dr. Soetomo General Hospital Surabaya Indonesia. The noise pollution levels was measured and its workers were examined (baseline characteristics, ROS level, audiometry, and OAE).

Baseline characteristics involved sex, age, education, environment noise, and hearing exposure. To determine the plasma ROS levels, we sampled 5 ml fasting venous blood, then isolated it in room of temperature 10–15 °C. The blood was analyzed using Enzyme Linked Immune Sorbent Assay (ELISA) reader (Bio-rad laboratories, Inc, Hercules, USA) and used *Ferric Reducing Ability of Plasma* (*FRAP*) reagent kit. Furthermore, audiometry examination was done using audiometry set (GSI Arrow, Eden Prairie MN, USA). AOE assessment used distortion product otoacoustic emissions (AuDX Biologic, Pleasanton CA, USA). If workers were absent during assessment schedule, they will be drop out from the samples.

The research data were presented as mean ± standard error and frequency. All data of inspection result was processed using SPSS version 23.0 (SPSS, Inc., Chicago, IL). Statistical evaluation of plasma ROS levels and OAE by using Chi square test ($p < 0.05$). Chi square test ($p < 0.05$) analysis was used to analyze plasma ROS levels.

Result

Demographic Data

Demographic data was divided into two base on frequency and average data. Frequency data were sex, education, and work area in Dr. Soetomo General Hospital. Average data

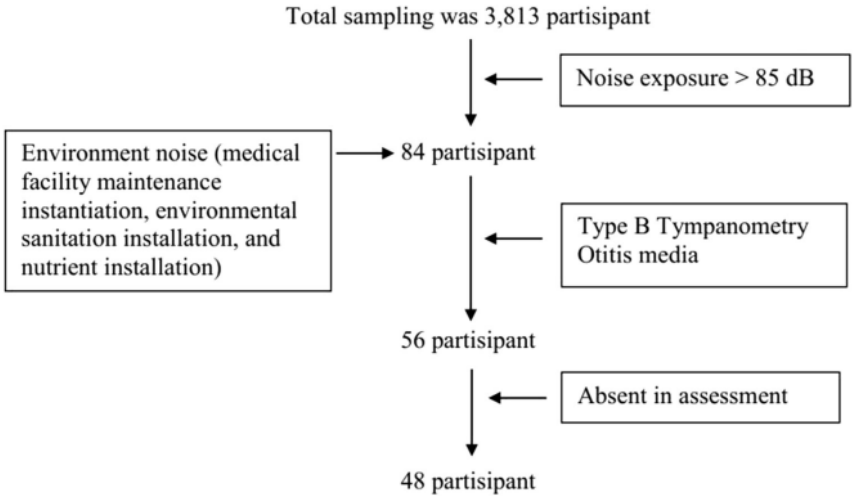

Fig. 1 Sampling of participants

Table 1 Frequency of demographic participant

Variables	Characteristics	%
Sex	Male	41.67
	Female	58.33
Education	Elementary school	2.08
	Junior high school	0.00
	Senior high school	81.25
	Bachelor degree	16.67
Noise pollution areas	Sanitation	14.58
	Maintenance medic	16.67
	Nutrition	68.75

Table 2 Average of respondents characteristic

Variables	Mean ± SD	*p*
Age	42.43 ± 10.72	0.023
Noise	98.58 ± 4.54	0.167
Duration of work	19.54 ± 9.56	0.042

Table 3 ROS associated with hearing loss

Characteristic	Category	ROS (%)		*p*
		Low	High	
OAE	Refer	54.17	16.67	0.993
	Pass	14.58	14.58	
Audiometry	Hearing loss	25.00	22.92	0.647
	Normal	35.42	16.66	

$p < 0.05$

were age, noise exposure, duration of work. Most of the respondents were female (58.33%), mostly senior high school graduate (81.25%), and work in kitchen (68.75%). Frequency data in this study can be seen detail in Table 1. The age of participant were 42.43 ± 10.72 years old and range from 22 to 58 years old. Some area in Dr. Soetomo General Hospital have average of noise exposure (> 85 dB) of 98.58 ± 4.54 and range from 85.39 to 112.90 dB. They have been working in the Dr. Soetomo General Hospital around 19.54 ± 9.56 years and range 1–36 years (Table 2).

Value of ROS was 5.55 ± 4.38 ng/dl with range from 3.99 to 37.60 ng/dl. Value of OAE was mostly in Refer category (70.83%) and mostly audiometry was category normal (52.08%). Detailed information can seen be Table 3. So, base on of the study was most participant have noise inducted hearing loss.

ROS and Hearing Loss

The participant was mostly in OAE category of Refer and ROS category of low (54.17%). There is no significance correlation between OAE characteristic and ROS. Most audiometry cattery was found normal with low percentage of ROS value (35.42%). The result of the statistical test with Chi square found no correlation between plasma ROS level and audiometry value ($p = 0.993$). The result of the statistical test with Chi square found no significant correlation between serum ROS level and audiometry ($p = 0.647$). The result of analyzed was shown in Table 3.

Discussion

NIHL is a hearing loss that experienced by many workers. Noisy exposure over 85 dB for 8 h is believed to interfere auditory organ function [9, 10]. Some recent studies suggest that NIHL hearing loss is due to an increase in the production of toxic ROCs of cochlea [3, 11]. In vivo studies also show that ROS causes mutation of the apoptotic-inducing factor (AIF) gene. AIF regulation reduction in neuronal cells makes it more sensitive and easily damaged [12].

ROS is considered to be a toxic cellular metabolic product but it also serves as a molecule that regulates many physiological processes. ROS plays an important role in the induction of apoptosis under physiological and pathological conditions. Increased ROS formation and subsequent apoptotic induction have been implicated in the development of some hearing loss pathologies. Furthermore, mitochondrial dysfunction plays an important role in some types of hearing loss [11].

Based on this research there was no correlation between ROS and hearing loss. It was proved by statistical analysis that no correlation between ROS level and audiometry and OAE was found. The audiometric examination is a subjective examination of the patient about the individual's hearing threshold [13]. Meanwhile, OAE is an examination of the inner ear objectively, especially the function of outer cochlear cell [5]. Both examinations are often used as indicators for early detection of hearing loss [4, 14].

Currently, ROS was alleged to be one of the main causes of impaired hearing function. ROS has characteristics as an unpaired electron, activating toxic chemical reactions to cellular and subcellular structures [15]. ROS levels in the cochlea cause decreased hearing function by damaging the cochlear hair cells [16]. However, some research suggests the levels of ROS in cochlea increased when it exposed to the noise first time and decreased subsequently after exposure [3, 11]. The progression of ROS to a decrease in hearing function takes a long time. The condition was

supported by several studies that suggest age increase to stimulate the number of ROS in the cochlea that causes hearing loss [12].

The main sources of ROS production within the cochlea appear to be the hair cells' mitochondria, or enzymes such as xanthine oxidase and NADPH oxidase. Once generated, ROS are responsible for direct cellular damage to lipids, proteins, and DNA, triggering apoptosis or necrosis and may also diffuse among the inner ear scalae [17]. ROS was produced by mitochondria, cytochrome p450 metabolism, microsomes, nitric oxide synthase and other inflammation-related processes. The overproduction of free radicals is highly toxic and triggers a complex mechanism of damage due to the peroxidation of membrane lipids, denaturation of cellular proteins, damage of DNA and cell death in several pathological conditions such as mutagenesis, carcinogenesis, aging, neurodegenerative and inflammatory diseases [16, 18].

There is increasing evidence that ROS-mediated damage in the cochlear may be a common factor for hearing loss caused by many factor. Based on the results obtained there is no significant effect of plasma ROS level on hearing loss, further investigation on the role of ROS on hearing should be conducted.

Conclusion

The number of workers who experience hearing loss in some of the noisy environment in the hospital was quite a lot. ROS production was influenced many factors like age and noise exposure. Based on evidence there was an increased production of ROS causing of hearing loss, but no correlation of reactive oxygen species with hearing impairment in high risk employees at the Hospital.

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PAGE 1

PAGE 2

PAGE 3

PAGE 4