Reactive oxygen species levels are high risk worker of noise induced hearing loss in hospitals

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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). Environment noise are commonly found in industrial sites but its possibility of hospitals also include noisy locations. Objectives: To analyze the correlation of reactive oxygen species and hearing impairment to employees at risk in the Hospital. Method: Participants was obtained by identifying the employees in hospital from 3,813. They were examined for baseline characteristics, hearing loss and reactive oxygen species. Hearing loss was defined as audiometry and tympanometry level. Result: 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 to 112.90 dB. The prevalence of NIHL was 47.92% (audiometry) and 70.83% (otacoustic 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). Conclusion: 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.

1. 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% sawing experience NIHL [1]. In the Tanzanian mining area reported by 12% 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 it will be very risky to experience noise-induced hearing loss and other hearing loss [3].

Enforcement of a diagnosis of hearing loss requires a tool for strengthening the diagnosis including using audiometry and optoacoustic 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 [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 instantiation, environmental sanitation installation, and nutrient installation. Based on the description above it need to be performed the correlation analysis of ROS content with audiometric and OAE measurements and at employees at risk in hospital.

2. Materials and methods

The subjects of this study were employees of Dr. Soetomo General Hospital, Surabaya, Indonesia. The subjects must meet inclusion criteria and exclusion criteria. The number of employees was 3,813 people and after being identified based on inclusion and exclusion criteria it got 56 subjects. Inclusion criteria include employees at risk of exposure to noise pollution with noise <85 dB, in hospitals (medical facility maintenance instantiation, 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 (figure 1).

The study used observational analytic study. Then, prior to conducting the data retrieval the researchers first 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 locations to be a source of noise in Dr. Soetomo General Hospital Surabaya Indonesia. Some area was value noise pollution levels. Its worker was do collection data (baseline characteristics, level of plasma ROS, audiometry, and OAE).

Baseline characteristics involved sex, age, education, environment noise, and hearing exposure. To determine the plasma ROS levels, we sample 5 ml fasting venous blood, then isolated its by 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, a pure tone audiometry (GSI Arrow, Eden Prairie MN, USA).
Assessment AOE used distortion product otoacoustic emissions (AuDX Biologic, Pleasanton CA, USA). If employers were an absent in assessment schedule, so they were drop out in research.

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 were using chi-square test (p < 0.05). Plasma ROS levels associated with audiometry used chi-square test (p < 0.05).

3. Result

3.1. 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 were age, noise exposure, and duration of work. Most of the respondents were female (58.33%), education of senior high school (81.25%), and work in nutrition room (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 22 to 58 years old. The some area in dr. Soetomo General Hospital have average of noise exposure (>85 dB) of 98.58 ± 4.54 from range 85.39 to 112.90 dB. They had duration of work in its of 19.54 ± 9.56 years and range 1 to 36 years (table 2).

| Variables          | %    |
|--------------------|------|
| 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              |      |
| Sanitation         | 14.58|
| Pollution          |      |
| Maintenance medic  | 16.67|
| Areas              |      |
| Nutrition          | 68.75|

Table 1. Frequency of demographic participant.

| Variables | Mean ± SD | p    |
|-----------|-----------|------|
| UAge      | 42.43 ± 10.72 | 0.023|
| KNoise    | 98.58 ± 4.54  | 0.167|
| Duration of work | 19.54 ± 9.56 | 0.042|

Table 2. Average of respondents characteristic.

3.2. ROS associated with hearing loss

The participant was mostly in OAE category of refer (70.83%) and mostly audiometry was category normal (52.08%). Detail about of data can be seen in table 3. So, based on of the study was most participant have noise inducted hearing loss.

| Variables | Mean ± SD | p    |
|-----------|-----------|------|
| UAge      | 42.43 ± 10.72 | 0.023|
| KNoise    | 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 Low | ROS High | \( p \) |
|---------------|----------------|---------|----------|--------|
| 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    |        |

4. Discussion

NIHL is a hearing loss that experienced by many workers. Noisy exposure over 85 dB for 8 hours is believed to interfere auditory organ function [9]. Some recent studies suggest that NIHL hearing loss is due to an increase in the production of toxic ROCs of cochlea [3] [10]. 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 [11].

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 [10].

Based on this research there was no correlation between ROS and hearing loss. It was proved by no correlation between ROS content with audiometry and OAE. The audometric examination was a subjective examination of the patient about the individual's hearing threshold [12]. Meanwhile, OAE was an examination of the inner ear objectively, especially the function of outer cochlear cell [5]. Both examinations were often used as indicators for early detection of hearing loss [4] [13].

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 [14]. ROS levels in the cochlea cause decreased hearing function by damaging the cochlear hair cells [15]. However, some research suggests the levels of ROS in cochlea have increased for the first time that exposed to the noise and decreased subsequent exposure [3] [10]. The progression of ROS to a decrease in hearing function takes a long time. The condition was supported by several studies that suggest an age increase to stimulate the number of ROS in the cochlea that causes hearing loss [11].

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 scale [16]. 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 [15] [17].

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 no significant of plasma ROS level on hearing loss. It is needed to find specific reseach influence ROS related by NIHL.

5. 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 by many factors, one of age and noise exposure. Based on evidence 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. ROS levels
in blood not specific toward hearing loss but research in vivo and evidence was significant correlation of ROS and hearing impairment.

6. References

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Acknowledgments

This project was supported by a research grant form Dr. Soetomo General Hospital Surabaya, Indonesia.