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Auditory function assessment in patients with chronic obstructive pulmonary disease

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Abstract

Background: Chronic obstructive pulmonary disease is a multisystem disease with multiple comorbidities. Hearing is dependent on the cochlear functions that may be affected by oxygenation. Affection of hearing is problematic and represents a major concern that should be seriously investigated as an important comorbidity in chronic obstructive pulmonary disease patients.
Objective: To assess auditory status among chronic obstructive pulmonary disease patients.
Methodology: The current study was carried out at Al-Azhar University Hospitals, Cairo, from 1 August 2021 to 2022, including 120 participants. In addition to the control group (60 healthy participants), there were two study groups: chronic obstructive pulmonary disease patients with respiratory failure group (30 patients) and non-respiratory failure group (30 patients). Hearing functions were studied using pure tone audiometry, and auditory brain stem response.
Results: There was statistically significant hearing impairment in chronic obstructive pulmonary disease patients in comparison to control group. The hearing impairment was more significant in chronic obstructive pulmonary disease with respiratory failure group in comparison to chronic obstructive pulmonary disease without respiratory failure group. The auditory impairment shows a negative interrelationship with oxygen tension (PaO2) and a positive interrelationship with the smoking index.
Conclusion: Hearing affection was meaningfully higher among chronic obstructive pulmonary disease patients and more prominent in patients with respiratory failure. Hypoxia results in deterioration of pure tone audiometry and increased absolute and interpeak latencies in auditory brain stem response. At every frequency, the mean pure tone audiometry thresholds were higher for chronic obstructive pulmonary disease groups than control group albeit remaining in the mild to moderate area of hearing loss. Retro-cochlear affection was suggested among patients with chronic obstructive pulmonary disease as evidenced with the prolongation of auditory brain stem response waves latencies.

Keywords

COPD, hearing loss, auditory brainstem response, hypoxia

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Introduction

Chronic obstructive pulmonary disease (COPD) as a multisystem disease has a myriad of significant comorbidities that could include the central nervous system.[1]

Attention, and psychomotor speed, as well as other cognitive functions like memory, learning, visuospatial skills, and auditory perception were found to be insulted in COPD patients.[2] Hypoxemia may result in substantial deleterious consequences via disturbances insulting the central as well as the peripheral nervous system that subsequently include the auditory system which is very sensitive to the sequel of the hypoxemic insult. Adequate blood and tissue levels of oxygenation are mandatory for efficient functioning of the peripheral and central auditory system.[3]

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The transduction process of the inner ear and the transmission of impulses through the auditory conduit is known to rely on the blood and tissue oxygenation, therefore any substantial deficiency in the oxygenation level can compromise the developments of generation and transmission of auditory nerve impulses at the level of the auditory system with subsequent impairment of the hearing process.[4]

The hypoxic consequences on the cochlea have been ascribed to the metabolic disturbances of different electrochemical potentials in the ear which are formed by the metabolic activity of Na/K ion potentials, and it is recognized that such process is offended by the deficient oxygenation.[5]

Hypoxia and smoking both disturb the antioxidants production pathways. The production of the antioxidant substances continuously guards against the oxidative stress and the reactive oxygen species. When the oxidative stress is sufficiently severe, direct damage to carbohydrates, proteins, lipids, and nucleic acids occur with subsequent cellular death, at this point the cochlear destruction becomes unavoidable and usually persistent even after restoration of the antioxidant defense mechanisms.[6]

Smoking is well known to be associated with many derangements to the human health such as lung cancer and cardiovascular diseases; however, its contribution to the impairment of hearing is still a controversy. Many studies have revealed a positive contribution while others did not reveal this. Different theories have been presumed to explain the effect of nicotine on the auditory apparatus, including direct ototoxicity, vasculopathy of the small blood vessels in the inner ear, and higher blood viscosity which amplify the cochlear ischemia.[7]

Subjects and methods

The current study is a hospital-based cross-sectional analytical study, conducted at outpatient clinics of chest diseases and the audio-vestibular unit at Al-Azhar University Hospitals, Cairo, Egypt from 1 August 2021 to 2022.

Studied groups

This study was carried out on 120 participants; they were classified into three groups:

(1) COPD patients with non-respiratory failure group: included 30 patients.
(2) COPD patients with respiratory failure group: included 30 patients.
(3) Control group: included 60 subjects who were non-smokers and apparently well, with age and sex matched. All of them had no clinical or spirometric data suggestive of any chest diseases.

The study included 60 COPD patients on their regular therapy with clinical stability at the time of assessment. They were assessed for study eligibility (inclusion and exclusion criteria) by the history taken and full clinical assessment. The COPD patients were classified into two equal groups, respiratory failure and non-respiratory failure according to the PaO2 level (PaO2 less than 60 mmHg and PaO2 equal or more than 60 mmHg). The 60 patients were subjected to clinical examination, spirometry (FEV1/FVC, FEV1, FEF 25%–75%, VC and FVC) and arterial blood gases (ABG) as well as audiological assessment involving the pure tone audiometry (PTA), speech audiometry, tympanometry, and auditory brain stem response (ABR).

Methods

Full clinical evaluation was performed for all participants with particular attention for age, sex, medical history, the sum of cigarette smoked per day, and the duration of smoking.

- *The smoking index* (pack/year) was estimated as the number of packs smoked per day multiplied by the sum of years of smoking.
- *The body mass index* (BMI) was evaluated according to the following equation: weight (kg)/height $(m)^2$.
- *Spirometry* was carried out by the SPIROSIFT SP5000, (Japan). The following parameters were obtained; vital capacity (VC%), forced vital capacity (FVC%), forced expiratory volume in the first second (FEV1%), FEV1\FVC ratio, and forced expiratory flow rate 25–75 (FEF 25%–75%). Spirometric values were calculated using the best out of three technically acceptable presentations in harmony with the European respiratory society recommendations.
- COPD patients were defined as patients with FEV1/FVC ratio less than 70% after bronchodilator therapy according to GOLD definition for COPD.[8]

Arterial blood gases. ABG investigation was done following a 15-min rest in room air using a Rapid Lab 248 blood gases analyzer (Siemens Medical Solutions, Malvern, PA, US); O2 saturation, Partial pressure of oxygen (PaO2) mmHg, and Partial pressure of carbon dioxide (PaCO2) mmHg, power of hydrogen (pH), and bicarbonate (HCO3) mmol/L were documented.

Audiological evaluation

- The external auditory canal and the tympanic membrane were investigated by otoscopy.
- Tympanometry at 226 Hz and ipsilateral acoustic reflex thresholds at 1, 2, and 4 kHz were recorded using an immittance meter (Maico, Diagnostic GmbH, MI 44).
- Pure tone auditory examination was conducted for each ear by the clinical audiometer (Piano Plus INVENTIS), using calibrated TDH39 headphones, at

the subsequent frequencies of: 250, 500, 1000, 2000, 4000, and 8000 Hz. All Audiometric evaluations were completed by a well-trained investigator in a specific sound-treated room in the audiology unit at Al-Zahraa University Hospital.

- Interacoustics Eclipse (EP25, Inc., Middlefart, Denmark) was used to obtain ABR through click stimuli with the following measures: 100 µs, 80 dBnHL, rarefaction, and broadband click, given at a rate of 21.1/s, 1200 total sweeps, and a 20 ms time window. The absolute latencies of waves I, III, and V on both sides and the IPLs of waves I–III, III–V, and I–V were recorded.

Sample size

The size of sample and sampling method:

Sample size was evaluated through the following equation:[9]

$$\text{Sample size}\ (\text{N}) = (Z\alpha/2+1-\beta)2\ *2*\text{SD}2/\ \Delta 2$$

The statistical level of significance was taken at α level 0.05, and the study power was 80%. The standard deviation (SD) of hearing thresholds from the previous study was 8.9 dBHL[10] with an estimated difference in the mean hearing thresholds between COPD patients and the control group being 5.0 dBHL. Sample size of 50 in each group was calculated and increased by 20% for possible drop out. Finally the sample size was 60 individuals in each group with a total number of 120.

Exclusion criteria. Patients with diabetes mellitus, hypertension, cardiovascular diseases, neurological diseases, patients known to have chronic ear diseases, patients with history of ear surgery or ototoxicity, patients with a familial history of hearing loss, and patients with middle ear diseases, all of them were excluded from the study.

Ethical considerations. This study was conducted in agreement with the Declaration of Helsinki. This study was conducted after approval by the institutional review board, faculty of medicine for girls Al-Azhar University, Cairo, Egypt (approval number 1074). Purposes and tools of this study were elucidated to all participants. Consent was taken from all individuals keeping their rights to leave the study at any point while preserving their rights of medical care. All records were anonymous and coded to declare the privacy of participants.

Statistics analysis. Data was collected and fed to the computer. Data analysis was done with the aid of Statistical package for social science software (SPSS version 16.0 Inc, Chicago, IL, USA). A descriptive evaluation of the study variables was conducted using mean and SD for quantitative records and frequencies of occurrence for qualitative facts.

Quantitative data were evaluated through the Analysis of Variance test when data were typically distributed while Chi square-test was used for comparative evaluation of qualitative data. Pearson's correlation coefficient was done to determine the degree of correlation between two quantitative records.

Simple linear regression analyses were carried out to examine associations between hearing outcomes (PTA and ABR), and their possible affecting covariates. Multiple linear regressions were then performed to test the association between each of the hearing outcome measures and the risk factors for auditory dysfunction tested in the simple linear regression models. A backward elimination technique was used with each model to select those risk factors remaining significant in the adjusted analysis, using a selection criterion of $\alpha < 0.05$.

p-value of < 0.05 was identified to be statistically significant with 95% confidence level. Results were displayed in tables.

Results

The study included 60 COPD patients and 60 healthy control persons. COPD patients were divided into respiratory failure and non-respiratory failure patients. There was no statistically significant difference between non-respiratory failure group, respiratory failure group, and control group concerning the age, sex, and BMI ($p > 0.05$). FEV1/FVC ratio, FEV1%, FVC%, and FEF 25%–75%, O2 saturation, and PaO2 were significantly reduced in the group that included respiratory failure patients when compared to the non-respiratory failure patients group and control group ($p = 0.001$), Table 1.

The average hearing thresholds were significantly increased in respiratory failure group (31.11 ± 8.28) (25.55 ± 7.12) compared to non-respiratory failure group (25.55 ± 7.12) and control group that means more auditory affection in COPD patients especially in respiratory failure group. Among respiratory failure COPD group, 22 (73.3%) patients had hearing impairment, 60% diagnosed with mild hearing impairment, and 13.3% had moderate hearing impairment in comparison to non-respiratory failure group where 14 (46.7%) had hearing impairment, Table 2.

PTA results revealed a statistically significant difference in all measures between the COPD patients and the control subjects. On comparing the PTA test results, the hearing thresholds were elevated at all frequencies (250, 500, 1000, 2000, 4000, and 8000 Hz) in left ears of COPD patients. The same results were obtained from the right ear, sparing only 250 Hz, which indicates more hearing affection in COPD groups. As regard the word discrimination score (WD%), the statistical difference was insignificant between COPD patients and the group of control subjects (Table 3).

Table 1. Characteristics of the studied groups.

Variables	Non-respiratory failure group (30)	Respiratory failure group (30)	Controls (60)	*p*-Value
Age/years (mean ± SD)	68.40 ± 3.66	64.20 ± 5.91	67.75 ± 4.62	0.059
Sex				
Male	28	22	48	0.345
Female	2	8	12	
BMI (mean± SD)	28.66 ± 3.81	27.93 ± 3.57	26.50 ± 5.09	0.475
Smoking (pack years) (mean ± SD)	27.85 ± 6.32	28.53 ± 3.64	27.53 ± 3.64	0.725
Spirometry				
VC (mean ± SD) (percentage of predicted value)	77.93 ± 3.65a69	69.46 ± 10.22	82.75 ± 2.43	0.000*
FVC (mean ± SD) (percentage of predicted value)	75.80 ± 4.45	65.73 ± 8.98	81.00 ± 2.44	0.000*
FEV1 (mean ± SD) (percentage of predicted value)	46.40 ± 6.16	32.60 ± 10.26	82.00 ± 1.51	0.000*
FEV1/FVC (mean ± SD) (percentage)	57.86 ± 9.68	43.33 ± 8.70	80.62 ± 1.99	0.000*
FEF 25–75 (mean ± SD) (percentage of predicted value)	22.13 ± 6.83	17.00 ± 6.78	40.62 ± 4.43	0.000*
Blood gases				
PH (mean ± SD)	7.38 ± 0.03	7.37 ± 0.02	7.37 ± 0.01	0.279
PaCO2 (mean ± SD) (mm Hg)	48.00 ± 9.07	47.20 ± 7.04	45.62 ± 5.31	0.779
PaO2 (mean ± SD) (mm Hg)	72.86 ± 6.27	50.93 ± 3.30	88.50 ± 3.46	0.000*
HCO3 (mean ± SD)	25.27 ± 3.93	23.93 ± 1.75	22.62 ± 0.74	0.094
O2 saturation (mean ± SD)	94.66 ± 0.97	80.86 ± 2.66	96.87 ± 0.83	0.000*

*Significant difference (p-value < 0.05).

Table 2. Hearing data among the studied groups.

Hearing parameters	Respiratory failure group (30)	Non-respiratory failure group (30)	Controls (30)	*p*-Value
Average hearing thresholds (mean ± SD)	31.11 ± 8.28	25.55 ± 7.12	19.88 ± 0.77	0.000*
Hearing loss				
Yes	22 (73.3%)	14 (46.7%)	0 (0.0%)	0.000*
No	8 (26.7%)	16 (53.3%)	30 (100%)	
Degree of hearing				
Normal hearing (⩽25 dB)	8 (26.7%)	16 (53.3%)	30 (100%)	0.000*
Mild hearing loss (26–40 dB)	18 (60.0%)	14 (46.7%)	0 (0.0%)	
Moderate hearing loss (41–55 dB)	4 (13.3%)	0 (0.0%)	0 (0.0%)	

ABR test results show increased absolute latencies of waves I, III, and V in COPD subjects, with marked prolongation of the absolute latencies of the respiratory failure group (more severe hearing affection in the respiratory failure group; Table 4).

The interpeak latencies among the studied groups showed nonsignificant difference on comparing with the normal control group (Table 5).

The hearing thresholds show a positive correlation with smoking index ($p < 0.05$) (hearing affection positively correlates with smoking index) and negative correlation with PaO2 ($p < 0.05$) (hearing affection negatively correlates with oxygen tension; Table 6) (Figures 1 and 2).

Using simple (bivariate) linear regression analyses, the variables significantly associated with the mean hearing thresholds were BMI and PO2, while by using multiple linear regression analyses for these significant factors, PO2 only shows association with PTA outcome. Regarding bivariate linear regression analyses, PO2 was the only variable significantly associated with the mean ABR (Table 7).

Discussion

COPD is a multisystem disease that often coexists with comorbidities.[11] Some of the comorbidities may arise independently of the disease, while others may be causally related, either with same predisposing factors or by increasing the risk or the severity of the other disease.[8]

Both systemic inflammation and chronic hypoxia can trigger each other. Both can cause a marked decrease in the blood supply and, consequently, the oxygen supply to the peripheral nerves. The functions of the inner ear are widely

Table 3. Pure tone audiometry among the studied groups.

Air conduction	Right ear			Left ear		
	Non-respiratory failure group	Respiratory failure group	Controls	Non-respiratory failure group	Respiratory failure group	Controls
	Mean ± SD (dB)			Mean ± SD (dB)		
250 Hz	22.00 ± 5.60	23.33 ± 6.17	18.33 ± 5.56	23.33 ± 6.72	24.33 ± 5.62	17.00 ± 4.14
p-value	0.052			0.002*		
500 Hz	23.33 ± 6.98	25.00 ± 5.00	16.33 ± 4.41	24.66 ± 6.11	25.00 ± 5.97	17.00 ± 4.55
p-value	0.001*			0.000*		
1000 Hz	25.33 ± 7.66	26.33 ± 6.93	19.33 ± 4.95	25.33 ± 6.67	26.33 ± 6.11	20.66 ± 3.19
p-value	0.015*			0.017*		
2000 Hz	24.66 ± 8.75	29.00 ± 14.90	20.66 ± 3.19	23.66 ± 7.89	29.33 ± 10.83	21.33 ± 2.96
p-value	0.184			0.025*		
4000 Hz	25.33 ± 10.25	40.66 ± 15.33	22.00 ± 3.16	25.66 ± 11.93	40.00 ± 14.14	21.33 ± 2.96
p-value	0.001*			0.000*		
8000 Hz	31.66±11.75	41.00 ± 17.94	22.66 ± 2.58	29.00 ± 12.27	45.66 ± 16.78	22.00 ± 3.16
p-value	0.012*			0.000*		
WD%	92.26 ± 5.11	92.80 ± 4.05	100.00 ± 0.00	91.20 ± 5.69	94.13 ± 4.24	100.00 ± 0.00
p-value	0.000*			0.000*		

*Significant difference between the three groups (*p*-value < 0.05).

Table 4. ABR absolute latency among the studied groups.

Air conduction	Right ear			Left ear		
	Respiratory failure group	Non-respiratory failure group	Controls	Hypoxic group	Non-hypoxic group	Controls
	Mean ± SD			Mean ± SD		
Wave I	1.78 ± 0.09	1.66 ± 0.10	1.57 ± 0.06	1.81 ± 0.13	1.76 ± 0.12	1.58 ± 0.04
p-Value	0.000*			0.000*		
Wave III	3.79 ± 0.11	3.67 ± 0.14	3.57 ± 0.06	3.82 ± 0.12	3.71 ± 0.13	3.58 ± 0.04
p-Value	0.000*			0.000*		
Wave V	5.83 ± 0.13	5.70 ± 0.14	5.58 ± 0.06	5.84 ± 0.13	5.68 ± 0.14	5.45 ± 0.49
p-Value	0.000*			0.006*		

*Significant difference between the three groups (*p*-value < 0.05).

dependent on the cochlear oxygen and blood supply; therefore, any affection in oxygen and blood supply to the cochlea leads to a marked reduction in its sensitivity.[12]

Therefore, the current study was conducted to assess auditory functions in COPD patients, investigate the possible effects of hypoxia, and assess the possible correlation with smoking index.

In the current study we selected both COPD patients and controls matched regarding age, sex, BMI, and smoking index (Table 1) to avoid the co-effects of these parameters on hearing.

As expected, the mean percentage of FEV1 was significantly reduced in patients with COPD (46.40 ± 6.16, 32.60 ± 10.26) as well as FEV1/FVC (mean ± SD: 57.86 ± 9.68, 43.33 ± 8.70) in non-respiratory failure and respiratory failure groups, respectively, in comparison to the control group (82.00 ± 1.51 for FEV1, and 80.62 ± 1.99 for FEV1\FVC; Table 1).

According to the above results, our study proved a significant difference in auditory measures with mild to moderate hearing loss in COPD group in comparison with control group. The hearing loss is significantly higher in respiratory failure COPD group in comparison to non-respiratory failure group (Table 2).

These results are consistent with many studies in the literature reporting that COPD-induced hypoxemia may affect the auditory functions.[10,13]

PTA test is a test that used to assess hearing sensitivity and measure the degree and type of auditory impairment.[14] There are many conflicting reports regarding the alterations

Table 5. Interpeak latency among the studied groups.

Air conduction	Right ear			Left ear		
	Respiratory failure group	Non-respiratory failure group	Controls	Respiratory failure group	Non-respiratory failure group	Controls
	Mean ± SD			Mean ± SD		
Waves I–III	2.00 ± 0.03	2.01 ± 0.12	2.00 ± 0.01	1.99 ± 0.04	1.95 ± 0.11	1.98 ± 0.03
p-Value	0.953			0.311		
Waves III–V	2.02 ± 0.06	2.02 ± 0.09	2.00 ± 0.01	2.01 ± 0.06	1.97 ± 0.08	2.00 ± 0.00
p-Value	0.699			0.239		
Waves I–V	4.03 ± 0.06	4.04 ± 0.11	3.74 ± 0.68	4.00 ± 0.06	3.92 ± 0.10	3.85 ± 0.29
p-Value	0.090			0.394		

Table 6. Correlation of average hearing threshold and average ABR with O2 level and smoking index.

Correlation of mean hearing threshold with	Correlation coefficient	*p*-Value
1 PaO2 (mm Hg)	−0.49	0.002*
2 smoking index (Pack years)	0.42	0.021*
Correlation of mean ABR with		
1 PaO2 (mm Hg)	−0.64	0.000*
2 smoking index (pack years)	0.07	0.703

*Correlation is significant at the level of 0.05 (two tailed).

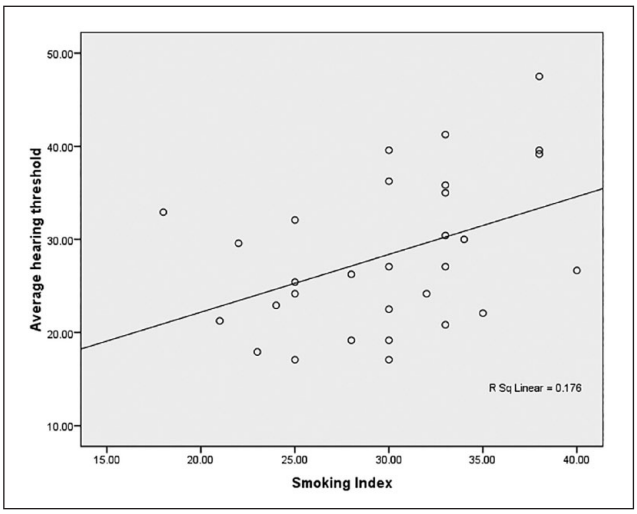

Figure 1. Correlation between hearing threshold and smoking index.

of hearing in COPD. Kamenski et al.,[15] reported that there is no correlation between COPD and elevated hearing thresholds as measured by PTA ($n=194$), while Schnell et al.,[16] reported a significantly higher incidence of hearing affection in patients with COPD according to self-reported hearing problems.

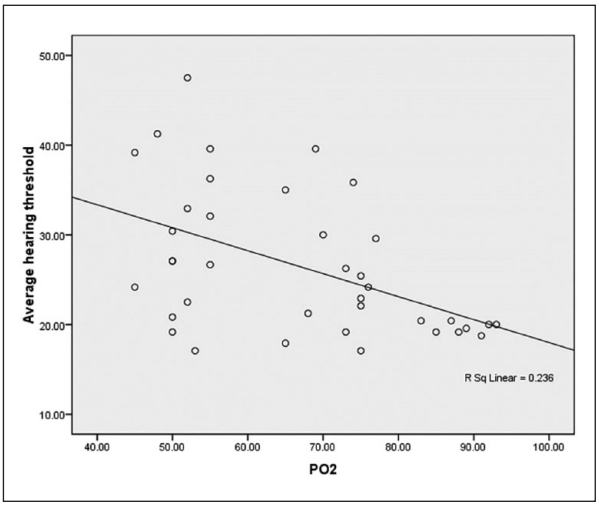

Figure 2. Correlation between hearing threshold and PaO2 level.

The current study revealed a significant increase in pure tone audiometric thresholds for COPD patients than in control persons. Also, the hearing thresholds were significantly higher in COPD patients when compared with control persons within the same frequency, with increasing magnitude with the rising of frequencies (Table 3), also pure tone audiometric thresholds increased more in respiratory failure group in comparison to non-respiratory failure COPD group.

A recent meta-analysis research for four large studies reported that the overall pooled mean audiometry thresholds were significantly higher in COPD patients than control persons and another large study reported significant affection of cognitive functions in general in those patients in comparison to control.[17–21]

Abdel Dayem et al.[19] reported that the auditory functions affection among COPD patients was apparent in the high frequency range. Also, vestibular affection was significant in tests that assessed postural state.

Table 7. Bivariate and multivariate linear regression analysis of PTA and ABR outcomes.

Bivariate linear regression model of PTA outcome			Bivariate linear regression model of ABR outcome	
Characteristics	Beta coefficient	*p*-Value	Beta coefficient	*p*-Value
Age	0.07	0.668	0.10	0.532
Sex (male)	−0.02	0.892	−0.04	0.785
BMI	**0.32**	**0.04***	0.038	0.823
Smoking index	0.28	0.128	0.07	0.703
PO2	−0.46	**0.004***	−0.64	**0.000***
Multiple linear regression of significant factors affecting PTA outcome				
BMI	**0.26**	**0.073**		
PO2	−0.42	**0.006***		

*Significant regression (p-value < 0.05).

Many previous large studies have reported that the transduction function of the inner ear is largely affected by the cochlear oxygen supply and that hypoxia will be associated with more loss of cochlear sensitivity. Other studies suggested worse central auditory functions in more hypoxemic patients in comparison to normal non-hypoxic individuals.[19]

ABRs are auditory-evoked potentials, representing the synchronous neural activation in the auditory pathways that started from the hearing nerves up to the level of brainstem centers. The latencies of ABR waves represent the speed of electrical sound signals while being transmitted through different parts of the hearing pathway. Hence, the prolongation of that latencies indicates that there is slower conduction of hearing signals.[22]

The current study (Table 4) reported that COPD patients have a specific insult to the auditory brainstem pathways as ABR test results declared increased absolute latencies of wave I, III, and V in COPD patients with marked prolongation of the absolute latencies of the respiratory failure group. However, the inter peak latencies showed nonsignificant difference on comparing with the normal control group.

Consistent with our results, Atis et al.,[23] reported significant ABR abnormalities (76.1%) among patients with severe COPD. Furthermore, Gupta et al.,[4] reported that 65% (26/40) of COPD patients had ABRs abnormalities, with prolonged absolute peak latencies (waves I, III, and V) and prolonged interpeak intervals (I–III and III–V) in both sides. Recently Bayat et al.,[21] found that ABR waves were significantly delayed in COPD patients. In disagreement with our study, Nakano et al.[24] and Barbieri et al.,[25] reported nonsignificant differences in ABR tests between COPD patients and control subjects.

Smoking has both direct and indirect effects on the hearing system. Smoking increases the reactive and free oxygen radicles that damage the hair cells. Smoking elevates the carboxyhemoglobin level and decreases the perfusion of the hearing organs. Rabhu et al.[26] and Pezzoli et al.,[27] reported hearing loss that was precipitated by smoking at high frequencies. Chang et al.,[7] studied the relation between smoking and auditory affection with PTA. They reported that smoking (active and passive) precipitated hearing loss at high frequencies, especially more prominent after 40 years of age. Uchida et al.,[28] reported that smoking causes auditory problems at high frequencies (4 kHz), which occur with increased dose-dependence. Also, Noorhassim and Rampal,[29] reported the same effects of smoking on hearing, especially with high frequencies, in a dose-dependent relationship.

In the bivariate model, the PO2 and BMI in COPD patients were significantly associated with the mean hearing thresholds while in the multivariate model only PO2 was significantly associated with the mean hearing threshold. As regards ABR, the PO2 was the variable that showed a significant association with the mean absolute latencies of wave V (Table 7). It was suggested that the transduction functions of the inner ear and the nerve impulses along the hearing pathways are affected by cochlear oxygen supply. Therefore, any considerable reduction in oxygen supply can lead to significant changes in both PTA and ABR.[30] Our results support this hypothesis. This agrees with the studies Cichosz et al.,[31] and Cicek et al.[32]

Limitation

A few limitations to the present study already exist. First, the size of sample was not large and it was a single-center study. The analysis of this study largely relied on the data of observational studies; therefore, it is difficult to resist residual confounders connecting COPD and hearing affection. Large multicenter studies are recommended in the future to study this important relationship between hearing affection and COPD disease.

Conclusion

In comparative estimates with controls, the hearing impairment was meaningfully higher among COPD patients with more significant affection in patient with respiratory failure. Hypoxemia affects the auditory functions resulting in

deterioration of PTA and increased absolute and interpeak latencies in ABR. At every frequency the mean PTA thresholds were higher for COPD groups when compared to control group albeit remaining in the mild to moderate area of hearing loss. Retro-cochlear affection was suggested among patients with COPD as evidenced with the prolongation of ABR wave latencies. Hypoxemia aggravates hearing loss in COPD patients and preventive measures should be considered to minimize the damaging consequences of hypoxemia on the auditory apparatus.

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