Egyptian Journal of Ear, Nose, Throat and Allied Sciences xxx (2016) xxx–xxx

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Contents lists available at ScienceDirect

Egyptian Journal of Ear, Nose, Throat and Allied Sciences

journal homepage: www.ejentas.com

Original article

Cognitive profile in patients with bronchial asthma and chronic obstructive pulmonary disease (COPD)

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ARTICLE INFO

Article history:
Received 24 February 2016
Accepted 2 June 2016
Available online xxxx

Keywords:
Bronchial asthma
COPD
Cognitive profile
P300
MoCA

ABSTRACT

Objective: To evaluate the cognitive status in patients with bronchial asthma and chronic obstructive pulmonary disease (COPD).
Methods: 40 patients with bronchial asthma, 40 patients with COPD and 20 healthy subjects (control) were included in the study. Comparison was done between the three groups in both Montreal Cognitive Assessment (MoCA) test and P300 latency. Also, correlation between these scores and patient characteristic parameters were evaluated.
Results: There was a significant prolongation in P300 latency ($P < 0.04$) and reduced MoCA scores in COPD group compared to asthma group ($P < 0.002$). 34/40 COPD patients had prolongation of P300 latency and reduced MoCA scores. However, 20/40 asthma patients had prolongation of P300 latency and 24/40 asthma patients had reduced MoCA scores. P300 latency correlated significantly with age ($r = 0.423$, $P < 0.007$), duration of disease($r = 0.622$, $P < 0.0001$) PaO2 ($r = -0.490$, $P < 0.001$), SaO2 ($r = -0.496$, $P < 0.003$) and degree of the disease (FEV1/FVC) ($r = -0.353$, $P < 0.026$) in COPD group. MoCA score was significantly correlated with WBC ($r = 0.45$) in COPD group and with BMI ($r = 0.236$, $P < 0.05$) in asthma group. There was no correlation between P300 latency and patients' characteristics in asthma group ($P > 0.05$).
Conclusions: COPD significantly decreased the cognitive status compared to bronchial asthma. Longer latency of P300 appears to be an expected sequel of COPD. MoCA abnormalities were comparable to P300 abnormalities in COPD and asthma patients.

1. Introduction

Chronic diseases of respiratory system become a serious public health problem all over the world. The most prevalent are obstructive diseases (asthma and COPD). The common denominator of both diseases is chronic inflammation leading to airway obstruction. The main differences are the distinct features of inflammation and reversibility of obstruction as it is reversible in asthma, while it progressively worsens and is irreversible in COPD. In asthma, the inflammatory infiltrate is predominantly composed of eosinophils, mast cells and CD4 T (Th2) lymphocytes, while in COPD it is mainly formed by neutrophils, macrophages and CD8 T (Th1) lymphocytes.[1]. Both diseases have been found to be co morbid with cognitive disorders.[2,3] A mechanism proposed for the cognitive impairment in COPD patients is the neuronal damage mediated by hypoxia as a result of the pulmonary disease or the co morbidities that adversely affect the brain, such as vascular disease and smoking.[4] Mild cognitive impairment(MCI) is defined as a clinical condition characterized by the decline of cognitive function greater than expected for a certain age and educational level of the individual but not severe enough to interfere with their daily activities.[5] The Montreal Cognitive Assessment test is an efficient instrument to use for screening, diagnosis and tracking of MCI. It assesses different cognitive domains, has good psychometric properties and has become a widely used screening instrument for MCI.[6] Also, P300 is an electrophysiological test which is a valuable tool for assessing cognitive function.[7] The changes in P300 amplitude reflect the degree and quality of information processing. P300 latency is related with cognition ability, attention, and the capacity

Peer review under responsibility of Egyptian Society of Ear, Nose, Throat and Allied Sciences.
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http://dx.doi.org/10.1016/j.ejenta.2016.06.004
2090-0740/

Please cite this article in press as: Mourad S., et al. Cognitive profile in patients with bronchial asthma and chronic obstructive pulmonary disease (COPD). Egypt J Ear Nose Throat Allied Sci (2016), http://dx.doi.org/10.1016/j.ejenta.2016.06.004

of intellect memory.[8] To our knowledge there are few studies which compare the cognitive function in asthma and COPD patients. We adopted both P300 and MoCA test to assess cognitive functions in these patients simultaneously to study their relative relevance in clinical setup in addition to measure the correlation between these scores and patient characteristic parameters.

2. Materials and methods

2.1. Study design

It is cross-section, observational study. Our patients were selected from the patients admitted in the Department of Chest Medicine, Sohag University Hospitals, while the control group was selected from healthy volunteers. All cognitive assessment tests were performed in audiology unit, Sohag university Hospitals. The Ethics Committee of Sohag faculty of Medicine reviewed and approved the protocol, and all patients signed informed consent after the nature of the study had been fully explained prior to their participation.

2.2. Subjects

Our participants were divided into 3 groups, based on spirometry with reversibility test and physical examination, patients with asthma, COPD and control group. In asthma group; they were 40 patients, all of them selected according to the GINA criteria for asthma – typical symptoms, typical interview, variable and reversible airway obstruction on spirometry tests – recently or documented in the past and were in a stable period of the disease (based on medical history and physical examination). Asthma patients only inhaled Steroid which have no systemic effect. 40 COPD patients were selected according to the Gold criteria for COPD. All patients in this group had typical interview (exertional dyspnea, progressive, chronic, productive cough), FEV1/FVC < 40 and >80 years, illiteracy, alcoholism, obesity, long term oxygen therapy, severe cardiovascular co-morbidities, cancer, uncontrolled diabetes, major cognitive impairment, history of head injury or brain tumor, dementia, or epilepsy. COPD patients are not using steroid. All subjects underwent the following: 1-An extensive physical examination. 2-Hearing assessment through; otological examination, two cannel pure tone audiometer, Madsen model, 922 and Immittancemetry, Amplaid model 775. The oxygen pressure (PaO2), oxygen saturation (SaO2) and carbon dioxide pressure (PaCO2) were analyzed in radial artery blood samples. The respiratory function tests were performed using a spirometry device (Superspiro, Micromedical Limited, Rochester, England). Forced expiratory volume (FEV1), forced vital capacity (FVC), FEV1/FVC parameters were measured.

2.3. Assessment of Cognitive Ability

Montreal Cognitive Assessment: it is a 30 point test that takes 10 to 15 min to be completed. It assesses several cognitive domains. A score below 26 points indicates mild cognitive impairment. It assesses visuospatial abilities using a clock drawing task (3 points), and a three-dimensional cube copy (1 point). Multiple aspects of executive functions are assessed using a trail making task (1 point), a phonemic fluency task (1 point), and a two item verbal abstraction task (2 points). MoCA test was validated to be used as screener as the author used the Montreal Cognitive Assessment, but not the Mini-Mental State Examination, ahichhas adequate psychometric properties as a screening instrument for the detection of mild cognitive impairment. Language is assessed using a three item confrontation naming task with familiar animals (3 points) and repetition of two syntactically complex sentences (2 points). Short term memory is evaluated with a task that involves two learning trials of five nouns and delayed recall after approximately 5 min (5 points). Attention, concentration and working memory are evaluated using a sustained attentions task (target detection using tapping, (1 point), digits forward and backward (1 point each) and a serial subtraction task (3 points). At the end of the test, orientation and place is assessed (6 points). In all patients, the P300 ERP recordings were taken before breakfast (at 8:30 AM) after an optimal overnight sleep. The stimulus was tonal stimuli as the speech stimuli is not present in our instrument. The 'odd-ball paradigm' was applied to all subjects. In this paradigm, standard (1000 Hz) and target (4000 Hz) auditory stimuli with duration of 1000 ms were presented to the subject binaurally over headphones. The deviant tone was designated as target and was counted by the patient. The target tone occurred regularly with a 0.20 probability. The rise and fall time of each tone was 5 ms. Recording conditions: Electroencephalogram (EEG) activity was recorded at the frontal (Fz), central (Cz) and parietal (P3 and P4) electrode sites of the 10/20 international system using Ag/AgCl electrodes, fixed with electrode paste and tape, with an impedance of 5 kΩm or less. The reference electrode was attached to the right mastoid and the ground electrode was attached to the left mastoid. The signals from the electrodes were amplified and band-passed between 0.3 and 100 Hz. EEG was digitized at 1000 Hz with a 1024 ms pre-stimulus baseline. The P300 targeted/frequent stimulus latency expressed numerically with reference value 300 ± 10 ms. The P300 amplitude was not calculated as it is highly variable in our instrument.

2.4. Statistical analysis

Data was collected and analyzed using SPSS v.17.0 (SPSS Inc, Chicago, IL, USA) and is presented as mean ± standard deviations. To assess the significance of the differences between groups, the ANOVA along with Bonferroni post-hoc.was used. The strength of correlations between variables was assessed using Spearman's correlation coefficient and its statistical significance with t-distribution test. A P value of <0.05 was considered the threshold for statistical significance.

3. Results

3.1. Demographic characteristics in the control

Asthma and COPD Groups Demographic features of asthma, COPD and control groups are shown in Table 1. This study included 100 participants, 46 women and 54 men. They were 80 patients with obstructive lung diseases (40 adult onset asthma and 40 COPD patients), in addition to 20 healthy subjects as a control group. The average ages in asthma, COPD and control groups were 44.5 ± 13.5, 60.85 ± 19.8 and 53.5 ± 14.8 years respectively.

3.2. MCI Frequency

MoCA mean score in asthma, COPD and control groups were 20.4 ± 4.65, 16.4 ± 6.3 and 28.4 ± 5.85 (Fig. 1) and prevalence of MCI in these groups were 60%, 85% and 15% respectively (Table 2). P300 latency in asthma, COPD and control groups were 312 ± 28.99, 324.25 ± 21.91 and 305 ± 10.27 (Fig. 2) and prevalence of MCI were 50%, 85% and 10% respectively (Table 2). The MoCA score was significantly lower in asthma and COPD groups (P < 0.0001) compared with the score in the control group. Furthermore, the MoCA score declined significantly in the COPD compared with that in the bronchial asthma (P < 0.001). Mean scores on the

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Table 1
Comparison of patients' parameters between studied groups.

Parameter	Asthma (A) N = 40	COPD (B) N = 40	Controls (C) N = 20	P value A vs. C	P value A vs. B
Age	44.5 (13.5)	60 (19.85)	53.5 (14.8)	0.0001	0.0001
Sex					
Females	28 (70.00%)	10 (25%)	8 (40.00%)	<0.0001	<0.0001
Males	12 (30%)	30 (75%)	12 (60.00%)		
BMI	22.77 ± 4.18	23.22 ± 3.97	22.54 ± 3.18	0.79	0.62
Duration of the disease	16.2 ± 9.54	13.2 ± 5.62	–		0.32
WBCs	10.67 ± 3.92	9.34 ± 3.53	10.52 ± 4.53	0.28	0.11
CRP	6.1 (2)	6 (4)	6 (2)	0.89	0.80
PH	7.42 ± 0.03	7.38 ± 0.07	7.40 ± 0.05	0.003	0.0007
$PaCO_2$	32.78 ± 4.22	45.15 ± 10.96	38.3 ± 4.19	<0.0001	<0.0001
PaO_2	83.05 ± 15.29	64.29 ± 15.16	88.14 ± 8.01	<0.0001	<0.0001
SaO_2	93.75 ± 3.10	83.35 ± 14.34	94.15 ± 3.16	<0.0001	<0.0001
FEV_1	44.85 ± 17.89	47.35 ± 13.73	85.3 ± 2.87	<0.0001	0.49
FVC	63 ± 14.84	55.4 ± 16.03	83.4 ± 9.03	<0.0001	0.03
FEV_1/Fvc	65.15 ± 13.02	61.25 ± 17.51	86.05 ± 4.36	<0.0001	0.26

Results are presented as mean ± standard deviation. P values were calculated with unpaired *t*-student and ANOVA tests. Column 5 is the correlation between A, B, C.
CRP – C-reactive protein, BMI – body mass index, WBC – White blood cell, FVC – Force vital capacity, FEV1 – Forced expiratory volume.

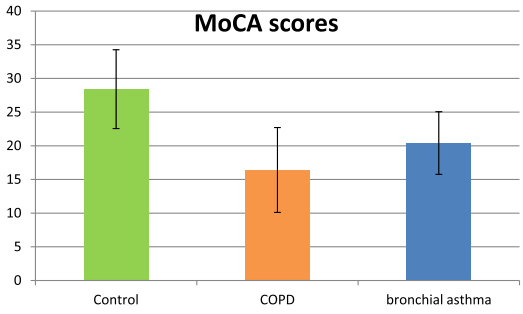

Fig. 1. MoCAmean scores in control, COPD and bronchial asthma. MCI Frequency MoCAmean score in control, COPD and asthmagroups were 20.4 ± 4.65, 16.4 ± 6.3 and 28.4 ± 5.85.

MoCA scale corresponding to moderate to severe cognitive impairment in COPD patients, while scores in bronchial asthma corresponding to the result of mild cognitive impairment. It was found that the latency of P300 was prolonged in COPD group compared to asthma group (P < 0.04).

3.3. Correlation between cognitive function and clinical parameters

There was a significant correlation between MoCA score and WBC (r = 0.450, P < 0.003), in patient with COPD group and with BMI(r = 0.427, P < 0.006) in asthma group (Table 3). P300 latency was correlated significantly with age (r = 0.423, P < 0.007), sex (r = −0.304, P < 0.059), PaO2 (r = −0.490, P < 0.001), SaO2 (r = −0.496, P < 0.003), FEV1/FVC (r = −0.353, P < 0.026) values. Interestingly, there was a significant correlation between P300 latency, and duration of the disease(r = 0.622, P < 0.0001). However, there was no statistical significant correlation between P300 abnormalities and asthma patients' characteristics (P > 0.05 for all) (see Table 4).

4. Discussion

The cognitive impairment is a significant concern for elderly because it decreases the quality of life and, in advance stages, it might cause functional disabilities. These findings are of special clinical relevance because cognitive dysfunctions among asthma and COPD patients remain often undetected and untreated in daily clinical practice. This may be because the screening tools for cognitive dysfunctions are not routinely employed and that on the other hand, patients often tend to deny that they are suffering from these conditions due to the stigma attached to them. To our knowledge this is the first study which uses MoCA test and P300 simultaneously to assess the cognition in patients with asthma and COPD. Comparing MOCA test to MiniMental State Examination test (MMSE) in detecting the earliest stages of impairment. Dong et al. (2012) and Villeneuve et al. (2012) compared these tests and concluded that MoCA is superior to MMSE in detection of patients with cognitive impairment.[9,10] In the present study P300 and MoCA were used simultaneously to assess cognitive functions; the utility of each one of them for assessment of cognitive functions has been established in prior studies. Al Tahan et al. (2010) found that MMSE alone is an insensitive tool and they recommended P300 latency to assess cognitive dysfunctions.[11] For these reasons, we used P300 in addition to MoCA for cognitive assessment. In our study MoCA mean score in asthma, COPD and control groups were 20.4 ± 4.65, 16.4 ± 6.3 and 28.4 ± 5.85 and prevalence of MCI in these groups were 60%, 85% and 15% respectively. P300 latency in asthma, COPD and control groups were 312 ± 28.99, 324.25 ± 21.91 and 305 ± 10.27 and prevalence of MCI were 50%, 85% and 10% respectively. The mean MoCA score in COPD patient's

Table 2
Percentage of MCI and P300 in COPD and asthma subjects.

Items	Asthma	COPD	Control	P-value
Montreal cognitive assessment	20.4 ± 4.65	16.4 ± 6.30	28.4 ± 5.85	0.002
Normal	16 (40.00%)	6 (15.00 %)	17 (85%)	<0.01
Abnormal	24 (60.00%)	34 (85.00%)	3 (15%)	
P300	312 ± 28.99	324.25 ± 21.91	305.13 ± 10.27	0.04
Normal	20 (50.00%)	6 (15.00%)	18 (90%)	0.001
Abnormal	20 (50.00%)	34 (85.00%)	2 (10%)	

Results of MoCA in control group was 15% as three case with cognitive impaired was removed and the test was repeated with 3 other healthy volunteers with normal cognition.

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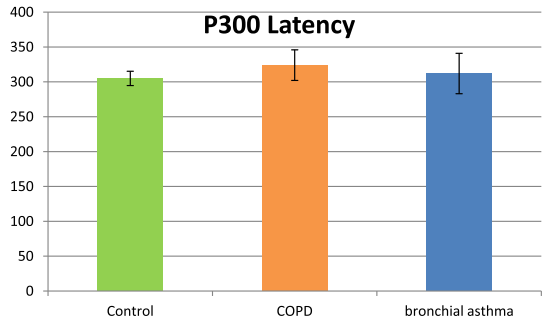

Fig. 2. P300 Latency in control, COPD and bronchial asthma. P300 latency in control, COPD and asthma groups were 312 ± 28.99, 324.25 ± 21.91 and 305 ± 10.27.

Table 3
Correlation between MoCA score and clinical parameters.

Test parameter	Asthma		COPD	
	r	*P*	r	*P*
Age	0.083	0.609	−0.274	0.087
Sex	0.193	0.347	−0.201	0.214
Duration of disease	0.04	0.79	0.15	0.35
BMI	−0.427	0.006	0.14	0.40
CRP	0.01	0.95	0.09	0.58
WBC	0.03	0.86	0.45	0.003
FEV_1	−0.27	0.09	0.16	0.31
FVC	−0.141	0.387	0.264	0.099
FEV_1/FVC	0.250	0.120	−0.281	0.079
SaO_2	0.065	0.692	0.074	0.651
PaO_2	0.14	0.39	−0.07	0.66
$PaCO_2$	−0.15	0.34	0.08	0.64

Table 4
Correlation between P300latency and clinical parameters.

Test parameter	Asthma		COPD	
	r	*P*	*r*	*P*
Age	0.091	0.576	0.423	0.007
Sex	−0.057	0.726	−0.304	0.059
Duration of disease	0.083	0.691	0.622	0.0001
BMI	0.032	0.887	0.290	0.070
CRP	0.180	0.265	−0.005	0.974
WBC	−0.038	0.818	−0.045	0.741
FEV_1	−0.192	0.236	−0.238	0.139
FVC	−0.020	0.901	−0.093	0.568
FEV_1/FVC	0.076	0.0639	−0.353	0.026
SaO_2	−0.048	0.769	−0.496	0.003
PaO_2	−0.037	0.822	−0.490	0.001
$PaCO_2$	0.283	0.077	0.178	0.272

was 16.4 ± 6.30, consistent with moderate to severe cognitive impairment,[12] while scores in bronchial asthma patients scale was 20.4 ± 4.65, corresponding to the result of mild cognitive impairment. These results are in agreement with the results of Preda et al. (2013) for MoCA test.[13]

In our study, P300 abnormalities were comparable to MoCA abnormalities in COPD but less in asthma. The MoCA has been found to be superior to the P300 in detecting MCI in asthma populations. Hence, compared with the P300, the current study showed that the MoCA is an appropriate and validated brief screening test for detecting MCI in patients with asthma and COPD. This has the potential to be clinically important, to detect hidden cognitive problems in asthma and COPD patients. Nevertheless, our findings are largely in agreement with the published data of other studies. Cognitive dysfunction is highly prevalent in both COPD and asthma patients.[2,3] The first study providing information on cognitive dysfunctions in asthma patients was conducted in 1981 by Schraa et al. they revealed that a majority of the asthmatics manifested definite signs of memory impairment when they were asked to recall.[14] In the largest sample cross sectional study so far asthma was associated with 78% increased risk of cognitive impairment when controlling for demographic characteristics, self-rated health status, inhaled corticosteroid use and FEV1/FVC.[15] The underlying mechanisms of cognitive impairments in COPD remain controversial issue. Hypoxemia, hypercapnia, or vascular co morbidities have been proposed as possible causes of brain alterations in patients with COPD.[16] Guo et al. (2013) reported that chronic asthma impairs cognitive function and synaptic plasticity, down-regulation of c-fos, Arc and neurogenesis induced by intermittent hypoxia during chronic asthma might contribute to learning and memory dysfunctions.[17] In our study, there is a significant correlation between MoCA test and WBC count in COPD patients. Patients who had a high level of WBC had a lower MoCA score. It is well known that the most important risk factor that leads to COPD is smoking. All our COPD patients in this study were smokers, so we assume that the inflammatory cytokines released by neutrophils over years because of smoking might have led to synaptic alteration and so, the destruction on existing neurons, led to a decrease of cognitive function.[18] MoCA is only correlated with BMI($r = -0.427$, $P < 0.006$) in asthma group. The scores of MoCA progressively decreased across the spectrum from mild overweight to obese groups in addition[19] P300 latency was significantly prolonged in COPD groups compared to asthma group. This result may be related to one or any number of the factors such as oxygen saturation, PaO2, PaCO2 and FEV1. In fact, in this study, P300 latency correlated significantly with PaO2, SaO2, FEV1/FVC values and age. Interestingly, there was a significant correlation between P300 latency, and duration of the disease($r = 0.622$, $P < 0.0001$). Our results were compatible with other studies. A positive correlation was found between P300 latency and age ($r = 0.423$, $P < 0.007$). It is well known that cognitive skills decline with age.[20]. It has been proposed that the mechanism underlying the positive relationship between age and cognitive functioning is cerebral oxygenation.[21]. P300 latency negatively correlated with FEV1/FVC ($r = -0.353$, $P < 0.026$). Reeves et al. (1999)[22] reported that auditory P300 latency was negatively correlated with pulmonary function in COPD patients. We found a negative correlation between P300 latency and both PaO2 and SaO2 [PaO2 ($r = -0.490$, $P < 0.001$), SaO2 ($r = -0.496$, $P < 0.003$)]. Grant et al. 1982[23] found an inverse correlation between neurologic decline and rest O2 saturation. There are some studies that found a significant correlation between low PaO2 and measures of cognitive performance.[21,24] We did not found any statistical significant correlation between P300 abnormalities and asthma patients' characteristics ($P > 0.05$ for all). The identification of existence of subclinical cognitive dysfunctions in asthma and COPD patients has significant impact over the disease as patients with untreated cognitive difficulties may deteriorate with faster rates and have worse health outcomes than cognitively intact patients.[24] Moreover, the cognitive dysfunctions are also recognized to be associated with increased mortality and disability. The influence of COPD on cognitive performance is partially reversible using oxygen therapy and physical activity, which is often not, appreciated enough.

5. Conclusion

Both bronchial asthma and COPD patients are at a great risk of cognitive disorders so, self administering MoCA test, Consultation and medical therapies are necessary for screening of these patients.

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