

Peripheral neuropathy in chronic obstructive pulmonary disease

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Received 10 August 2016

Accepted 05 September 2016

Journal of Current Medical Research and Practice

January-April 2017, 2:17–24

Introduction

Peripheral neuropathy in chronic obstructive pulmonary disease (COPD) has received scanty attention. The purpose of this study was to evaluate objectively the functional changes in the peripheral nervous system in COPD by different electrophysiological parameters and to determine the frequencies of these changes in patients with COPD.

Aim

Assessment of peripheral nerve conduction by evaluation of the motor and sensory nerve conduction (SNC) in COPD patients.

Patients and methods

In this case–control study, we recruited 25 COPD patients and matched 25 healthy controls. Motor and SNC studies for ulnar and median nerves were evaluated by means of electrophysiological nerve study. Motor nerve conduction velocity and sensory nerve conduction velocity (SNCV), distal latencies (DLs), and amplitude of compound motor action potential were recorded. Arterial blood gases including partial pressure of oxygen and carbon dioxide (PaO₂ and PaCO₂), oxygen saturation (SaO₂), and arterial pH were measured. Pulmonary function test was done and forced expiratory volume in 1 s (FEV₁), forced vital capacity (FVC), and FEV₁/FVC ratio were measured.

Results

There was a significant difference between COPD patients and the control group in all spirometric and gasometric parameters recorded, except for the arterial pH. On studying motor nerve conduction through median and ulnar nerves, there was an increase in DL, decrease in motor nerve conduction velocity, and longer F-wave latency in the COPD group than in the control group in both nerves. SNC study of the median nerve revealed a decrease in SNCV and an increase in DL in the COPD group than in the control group. Median nerve motor neuropathy was proved in 28% of patients, ulnar nerve motor neuropathy was proved in 36% of patients, whereas sensory nerve study of median nerve proved that 68% of patients have sensory axonal neuropathy and 12% have demyelinating sensory neuropathy. Median nerve Distal Latency (DL) shows negative correlation with FEV₁ and FEV₁/FVC ratio. SNCV of the median nerve was positively correlated to oxygen tension level.

Conclusion

The incidence of neuropathy is high. The rate of axonal neuropathy was significantly higher than other types. Our study showed a significant positive correlation between the degree of hypoxemia and severity of neuropathy, whereas it showed negative correlation between spirometry parameters (FEV₁ and FEV₁/FVC ratio) and median nerve DL.

Keywords:

COPD, demyelinating neuropathy in COPD, f-wave abnormalities in COPD, neurological manifestation of COPD, neuropathy in COPD

J Curr Med Res Pract 2:17–24
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2357-0121

Introduction

Chronic obstructive pulmonary disease (COPD) is defined as a common preventable and treatable disease, characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Global Initiative for Chronic Obstructive Lung Disease (GOLD) [1]. In COPD, the changes occur in peripheral nerves that are chronically subjected to hypoxemia. Hypoxemia in COPD patients is thought to have negative effects on the peripheral nervous system, as well as

on many other organs. It probably plays the foremost part, either by direct action on nerves fibers or by enhancing the effects of other neurotoxic factors [2]. Peripheral neuropathy is a failure of the nerves that carry information to and from the brain and spinal cord. There are numerous reasons for peripheral neuropathy; chronic respiratory insufficiency has

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been implicated as one of the factors in previous studies [3]. Hypoxemia, a reduction in partial oxygen tension (PaO_2), can be observed in practically every known pulmonary disease entity. Consequently, hypoxemia is an indicator of abnormal pulmonary gas exchange, and the arterial PaO_2 can also serve as a test of pulmonary function. Hypoxia, a decrease in tissue oxygen tension, is a consequence of hypoxemia [4]. The aim of our study was to investigate the peripheral nervous system in COPD patients with prospective clinical and electrophysiological study.

Patients and methods

Patients

This study was conducted at Assuit University Hospital at Chest Department and the Department of Neurology and Psychiatry in the period between May 2013 and October 2015. We enrolled 25 stable COPD patients and 25 adult age-matched and sex-matched healthy controls.

Sampling and sample size

Sampling

Sampling was done by opportunity and convenient sampling.

Sample size

There are many local studies for the estimation of prevalence of COPD among the risk group (age above 45 years with a history of smoking or ex-smoking or exposure to outdoor pollution). On the basis of results of Said *et al.* [5] and El Hasnaoui *et al.* [6], prevalence of COPD in Egypt was considered to be ~6% and our patients' and control sample size was calculated to be 25 using Open Epi V.3.01 (Open source programme, Atlanta, USA) computer program.

Chronic obstructive pulmonary disease diagnosis

A diagnosis of COPD is considered in any patient who has cough, sputum production, or dyspnea, and/or a history of exposure to known risk factors. The diagnosis is confirmed by an objective measure of airflow limitation (spirometry). Chronic cough, usually the first symptom of COPD to develop, may be intermittent in the beginning, but later it is present every day, often throughout the day. Small quantities of tenacious sputum are usually raised by COPD patients after coughing bouts. Dyspnea is the basis why most patients usually seek medical attention. As lung functions worsen, breathlessness becomes more disturbing. Wheezing and chest tightness are relatively nonspecific symptoms. Physical signs of airflow limitation are rarely present until significant

impairment of lung function has occurred. Spirometry is indicated to diagnose COPD. Spirometry should measure forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV_1), and the ratio of these two measurements (FEV_1/FVC) is then calculated. Patients with COPD classically show a decrease in both FEV_1 and FVC. The presence of a postbronchodilator FEV_1 less than 80% of the predicted value in combination with an FEV_1/FVC less than 70% confirms the presence of airflow limitation that is not fully reversible [1].

Peripheral neuropathy diagnosis

Axonal neuropathy is diagnosed by the amplitude of the recorded response greater than 80% of the expected value; conduction velocity should remain above 80% of the lower limits (80% rule). A greater loss of fast conducting fibers would result in further conduction slowing but not beyond 70% of the lower limits of the normal value [7].

Demyelinating neuropathy is diagnosed by the amplitude of the recorded response; conduction velocity should remain below 50% of the lower limits [7].

F-wave latency prolongation than normal which indicate radiculopathy [8].

Cutoff values

The cutoff values were calculated as follows:

- (1) For motor median nerve study, the least normal compound motor action potential (CMAP) is 6 mV, the least motor nerve conduction velocity (MNCV) is 49 m/s, and maximum distal latency (DL) is 3.5 ms
- (2) For motor ulnar nerve study, the least normal CMAP is 4 mV, the least MNCV is 49 m/s, and maximum DL is 4.4 ms
- (3) For sensory median nerve study, the least normal CMAP is 20 μV , the least MNCV is 53 m/s, and maximum DL is 3.7 ms
- (4) For F-wave, the cutoff point for F-wave latency of median nerve is 25 ms and for ulnar nerve it was 25.5 ms.

Inclusion and exclusion criteria

Inclusion criteria

All stable COPD patients with age ranging between 45 and 75 years who are admitted at Chest Department at Assuit University Hospital and COPD patients who are attending the outpatients' clinic were eligible to participate in this study. Healthy volunteers of same age, residence, smoking habits, and educational level were also included in the study.

Exclusion criteria

COPD patients with any of the following comorbidities were excluded from the study:

- (1) Left-sided heart failure, renal insufficiency, or liver impairment
- (2) COPD patient with exacerbation
- (3) Electrolyte disturbance that may impair the neurological studies
- (4) Diabetic patients
- (5) Chronic use of systemic steroids or any other drug affecting results
- (6) Severe decompensated respiratory failure impeding patient tolerance to complete the study
- (7) Previous cerebral stroke or any neuropsychiatric condition.

Work-up scheme

All patients were subjected to careful history taking, age, height, body weight, and BMI, as well as full chest and neurological examination. All routine investigation to apply exclusion criteria were performed. All patients eligible to participate were subjected to the following:

- (1) Spirometric evaluation: conventional spirometry was done using Zan 300 (Company nSpire Health™, Sensor Medics MGA USB, Germany), to COPD and control groups. The reference values used were those of the American Thoracic Society standards. The following parameters were observed and recorded for the research: FEV₁% of predicted and volume in liters, FEV₁/FVC in liter ratio, and FVC% of predicted and volume in liters
- (2) Gasometric evaluation: arterial blood gases sample was analyzed using Radiometer blood gas analyzer (Radiometer Medical ApS company, Demark). Arterial blood pH, partial pressure of arterial oxygen (PaO₂), partial pressure of arterial carbon dioxide (PaCO₂), arterial oxygen saturation (SaO₂), arterial bicarbonate level (HCO₃⁻), and base excess or deficit (BE/BD) were recorded
- (3) Peripheral nerve conduction study:

MNCVs, DL, and amplitude of CMAPs were measured with standard surface-stimulating and recording techniques for ulnar and median nerves. Diffuse axonal neuropathy was diagnosed by the reduction of CMAP amplitude with normal shape and duration and with normal or minimal disturbance of nerve conduction velocity. A Nihon Kohden Machine model 9400 (Nihon Kohden, Tokyo, Japan) was used to record the electromyography parameters using a band-pass of 20–1000 Hz and a recording time window of 200 ms. MNCVs were assessed using standard procedures with concentric needle electrodes. A pulse of 0.2 ms duration, at the rate of 1/s at supramaximal intensity, was used for conduction studies of median and ulnar nerves.

Sensory conduction is done by stimulating electrodes, which are ring electrodes placed around the proximal and middle phalanxes of the second or third digits and the recording electrodes are placed on the ventral aspect of the wrist, over the median nerve, usually at about 1–2 cm proximal to the proximal wrist crease. A fixed distance is preserved between the active and reference recording electrodes to avoid electrode-related changes in sensory nerve action potential amplitude and duration.

F waves were recorded from the gastrocnemius muscle after supramaximal electrical stimulation of the median and ulnar nerves at wrist. Totally, 10 stimuli were given, and the average latency value was determined for each arm.

Ethical approval

The study was approved by the Institutional Ethics Committee of Assuit University, and written informed consent was given by all patients.

Statistical analysis

Data were recorded using IBM statistical package for the social science software computer program, version 20 (SPSS; SPSS Inc., Chicago, Illinois, USA), Medcalc v. 11.6. (MedCalc Software company, Belgium), and Open Epi V.3.01 (Open source programme, Atlanta, USA). Data were described using mean ± SD and frequencies according to whether they are quantitative or qualitative, respectively. Nonparametric tests were used in the current study. Mann–Whitney test was used for comparison of results between COPD and control groups, and Spearman's correlation coefficient was used for correlation between peripheral neuropathy and spirometric and gasometric parameters of COPD patients; receiver operating characteristic (ROC) curves were plotted to investigate the probability of some gasometric and spirometric parameters being detectors of some peripheral nerve study abnormalities in COPD patients and to detect the cutoff value for these parameters. *P*-value below 0.05 was accepted as significant.

Results

We enrolled 25 stable COPD patients and compared them with 25 age-matched and sex-matched healthy controls. Age and sex were 57.28 ± 5.55 years and 14 male, respectively, in the COPD group and 56.36 ± 5.17 years and 13 male, respectively, in the control group (Table 1).

Spirometric evaluation showed that there was a significant difference between the COPD group and

the control group in all gasometric and spirometric parameters, except for blood acidity. Table 2 shows detailed spirometric and gasometric parameters.

The motor conduction velocity, latency, and amplitude of median and ulnar nerves; sensory conduction velocity, amplitude, and latency of the median nerve; and F-wave study in ulnar and median nerves in the COPD group and control group were compared using Mann-Whitney test. Detailed results are presented in Table 3 and Figs 1 and 2.

Median motor nerve conduction study shows a significant decrease in MNCV of the median nerve in the COPD group than in the control group and significant prolongation of DL of the median nerve in the COPD group than in the control group, whereas there was no significant difference in amplitude of CMAP of the median nerve in the COPD group than in the control group. There was a significant negative correlation between DL of median nerve and FEV₁ (in liter), FEV₁%, and FEV₁/FVC%, with $r = -0.516$ and $P = 0.008$, $r = -0.437$ and $P = 0.029$, and $r = -0.409$ and $P = 0.042$, respectively, as shown in Figs. 3 and 4. ROC curve was plotted to evaluate the use of a decrease in FEV₁ level as a screening tool for prediction of an increase of DL of the median nerve in the COPD group. This denoted good use of a decrease in FEV₁ level as a screening tool for the prediction of an increase of DL of the median nerve in the COPD group, as shown in Fig. 5.

Ulnar motor nerve conduction study showed a significant difference in motor conduction velocity of ulnar nerve in the COPD group than in the control group and a significant prolongation of DL of the ulnar nerve in the COPD group than in the control group, whereas there was no significant difference in amplitude of CMAP of the ulnar nerve in the COPD group (Tables 4 and 5).

Median Sensory nerve conduction (SNC) study showed a significant decrease in SNC of the median nerve in the COPD group than in the control group, whereas there was a significant prolongation in SNC latency of the median nerve in the COPD group than in the control group and there was no significant difference in SNC amplitude of median nerve in the COPD group than in the control group. There is a significant positive correlation between sensory nerve conduction velocity (SNCV) and PO₂, with $r = 0.487$ and $P = 0.038$, as shown in Fig. 6.

ROC curve was plotted to evaluate the use of a decrease in PaO₂ level as a screening tool for the prediction of a decrease of SNCV in the COPD group. This denoted

Table 1 Detailed demographic data of chronic obstructive pulmonary disease and control groups

	COPD group (n=25)	Control group (n=25)	P
Sex			
Male	14	13	0.777
Female	11	12	
Age (mean±SD) (years)	57.28±5.55	56.36±5.17	0.448
Smoking			
Smoker	5	4	0.691
Ex-smoker	11	9	
Nonsmoker	9	12	
Residence			
Urban	8	10	0.556
Rural	17	15	
Dominant hand			
Right handed	22	21	0.684
Left handed	3	4	
Education			
Literate	9	11	0.627
Illiterate	16	14	
Duration of illness			
Mean±SD		14.6±4.6	

COPD, chronic obstructive pulmonary disease.

Table 2 Detailed spirometric and gasometric parameters

	COPD group (n=25) (mean±SD)	Control group (n=25) (mean±SD)	P
FEV ₁ (l)	1.28±0.55	3.18±0.92	<0.0001*
FEV ₁ %	45.72±16.65	93.88±8.91	<0.0001*
FVC (l)	2.77±0.71	3.65±0.96	0.001*
FVC%	82.64±15.67	95.40±11.13	0.003*
FEV ₁ /FVC%	46.16±13.27	85.68±4.76	<0.0001*
BMI (kg/m ²)	22.74±5.52	28.50±6.36	0.001*
pH	7.40±0.04	7.41±0.02	0.152
PaCO ₂ (mmHg)	67.32±12.98	39.64±3.67	<0.0001*
PaO ₂ (mmHg)	61.88±5.18	84.64±5.97	<0.0001*
SaO ₂ %	91.12±2.05	96.80±1.19	<0.0001*
HCO ₃ (mEq/l)	38.23±4.71	18.00±2.27	<0.0001*
BE/BD	13.48±5.09	2.16±1.40	<0.0001*

*Presence of statistically significant difference between the two groups. BE/BD, base excess/deficit expressed in mmol/l; FEV₁%, forced expiratory volume % predicted; FEV₁/FVC, forced expiratory volume/forced vital capacity ratio; FEV₁, forced expiratory volume in first second; FVC%, forced vital capacity percent of predicted; FVC, forced vital capacity in liter; HCO₃, serum bicarbonate level; PCO₂, partial arterial pressure of carbon dioxide in mmHg; pH, blood arterial acidity; PO₂, arterial partial pressure of oxygen in mmHg; SaO₂, arterial oxygen saturation percent.

good use of the decrease in PaO₂ as a screening test for predicting decrease of SNCV in the COPD group, as shown in Fig. 7.

F-wave study showed that there is a significant difference in F-wave study of median nerve in the COPD group (mean: 32.36 ± 6.25) than in the control group (mean: 26.8 ± 3.23), with P less than 0.0001, and that there is a significant difference in F-wave study of the ulnar nerve in the COPD group (mean: 30.8 ± 5.8) than in the control group (mean: 27 ± 1.9), with a P less than 0.001.

Table 3 Detailed nerve conduction study in median and ulnar nerves

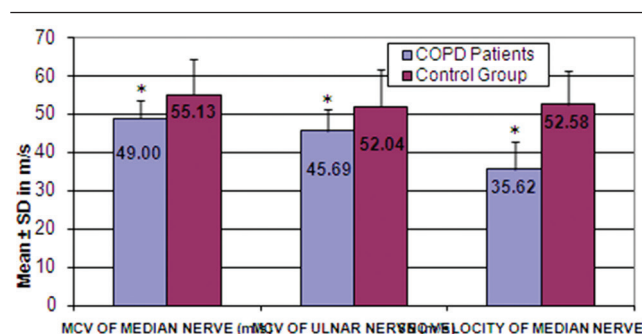
Groups	COPD groups (mean±SD)	Control group (mean±SD)	P
Distal latency median nerve (ms)	3.66±0.39	2.98±0.78	<0.0001*
Distal latency ulnar nerve (ms)	3.73±1.37	2.77±0.47	0.001*
SNC latency median nerve (ms)	4.01±0.47	3.28±0.45	<0.0001*
Amplitude of CMAP of median nerve (μv)	7.84±3	7.72±1.77	0.90
Amplitude of CMAP of ulnar nerve (μv)	7.08±2.1	6.82±2.04	0.58
SNC amplitude median nerve (μv)	20.3±8.2	17.58±5.94	0.347
MNCV median (m/s)	49±4.6	55.13±9.41	0.006*
MNCV ulnar (m/s)	45.6±5.4	52.04±9.81	0.014*
SNC median (m/s)	35.6±7	52.58±8.57	<0.0001*
F-wave latency median nerve (m/s)	32.36±6.52	26.8±3.23	<0.0001*
F-wave latency ulnar nerve (m/s)	30.8±5.8	27±1.9	0.001*

*Presence of statistically significant difference between the two groups. CMAP, compound motor action potential; COPD, chronic obstructive pulmonary disease; MNCV, motor nerve conduction velocity; SNC, sensory nerve conduction.

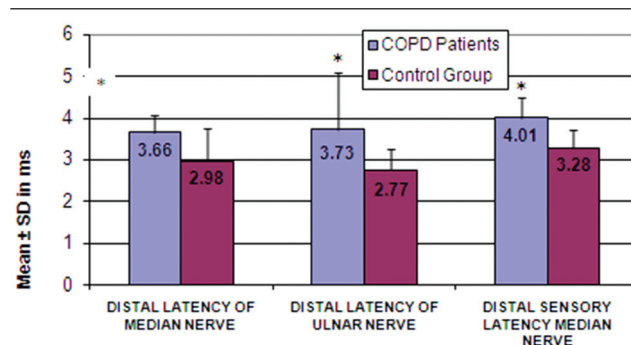
Peripheral neuropathy pattern: the motor median nerve study showed that 28% of COPD patients have axonal neuritis, the motor ulnar nerve study showed that 36% of patients have axonal neuritis, and the sensory median nerve study showed that 68% of COPD patients have axonal neuritis, and 12% have demyelinating neuritis. Overall incidence of neuritis in the COPD group was 88%.

Discussion

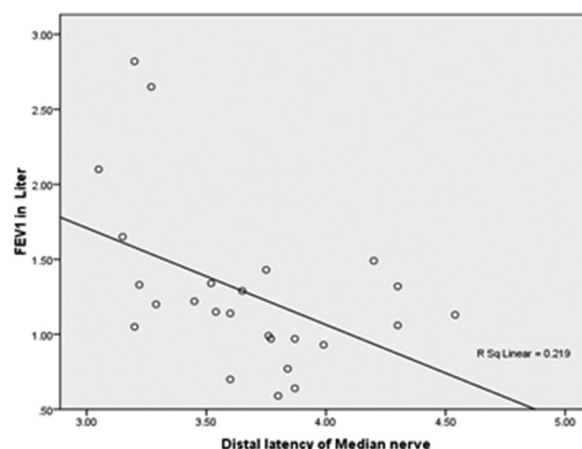
The association of polyneuropathy (PNP) with COPD is described in literature with a prevalence rate of 36–80% [9]. It accompanies COPD frequently and complicates it, worsening the quality of life and possibly the prognosis. Several studies have been conducted to determine the factors that are responsible for neuropathy in COPD patients [10]. In our study, we studied the motor nerve conduction through median and ulnar nerves and SNC through median nerve. The results of our study showed incidence of preclinical polyneuritis mostly in the form of mixed axonal demyelinating neuritis (evidenced by an increase in DL of median and ulnar nerves and decrease in nerve conduction velocity and decrease in CMAP of ulnar and median nerves) and early radiculopathy (evidenced by impairment of F-wave study through ulnar and median nerves). Our results of nerve conduction in COPD patients were strongly positively correlated to the level of oxygen tension in blood and SNC of the median nerve. In addition, there was a negative correlation between FEV₁% and FEV₁/FVC% and DL of the median nerve.

Figure 1

Nerve conduction velocity in median and ulnar nerves. The figure shows a significant decrease in motor nerve conduction velocity of the median nerve in the chronic obstructive pulmonary disease (COPD) group than in the control group with a $P = 0.006$; a significant decrease in motor conduction velocity of ulnar nerve in the COPD group than in the control group, with $P = 0.014$; a significant decrease in SNC of median nerve in the COPD group than in the control group, with P -value less than 0.0001.

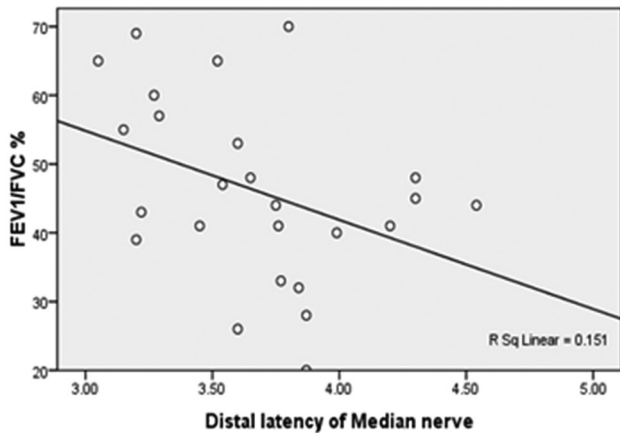
Figure 2

Distal latency of median and ulnar nerves. Figure shows the increase in distal latency of median and ulnar nerves. There was significant prolongation of distal latency of median nerve in the chronic obstructive pulmonary disease (COPD) group than in the control group, with a P -value less than 0.0001; a significant prolongation of distal latency of ulnar nerve in the COPD group than in the control group, with $P = 0.001$; and a significant prolongation in sensory nerve conduction latency of median nerve in the COPD group than in the control group, with P -value less than 0.0001.

Figure 3

The negative correlation between forced expiratory volume in 1 s in liter and distal latency of median nerve.

Figure 4

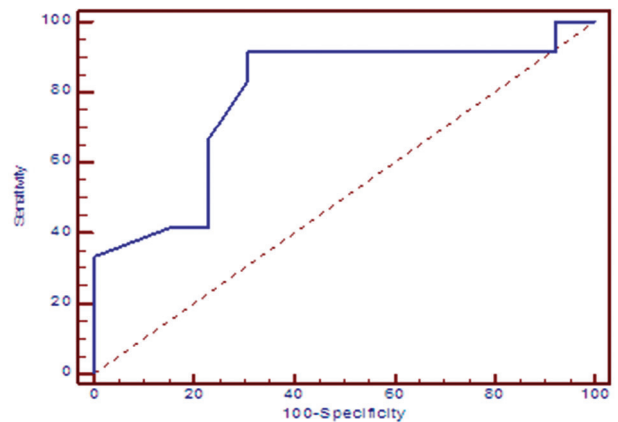


The negative correlation between forced expiratory volume in 1 s and forced vital capacity and distal latency of median nerve.

To understand the mechanism of peripheral neuritis in COPD in which hypoxemia was the cornerstone, studies that included patients having clinical evidence of peripheral neuropathy reported a higher prevalence of peripheral neuropathy upon neurophysiological investigation. Similarly, studies involving patients with severe hypoxemia and/or hypercapnia observed a higher prevalence of peripheral neuropathy upon neurophysiological analysis [3,11,12]. Stoebner *et al.* [13] also observed that the microangiopathy in peripheral nerves in patients with COPD appears to be diffuse and essentially related to hypoxia. Hypoxic neuropathies are associated with nerve capillary endothelial cell hyperplasia and hypertrophy, predisposing to luminal occlusion. This may impede the transport of nutrients and oxygen. These mechanisms seem to be applicable to peripheral nerve dysfunction and lesions, resulting from impaired axonal transport and causing axonal degeneration [14]. In animal models, chronic hypoxemia causes a deceleration in nerve conduction velocity. Studies of the oxygen consumption in the microenvironment of the peripheral nerve under conditions of nerve edema and experimental diabetic neuropathy show that the peripheral nerve function is oxygen dependent [15]. Axonal transport is an energy-requiring process and its impairment by hypoxemia can enhance axonal degeneration [16].

Most of the studies conducted support our results. Narayan and Ferranti [17] studied 16 patients with chronic respiratory insufficiency and severe hypoxemia. When matched with a control group, a statistically significant slowing of nerve conduction was noted in the motor median, ulnar, peroneal, and tibial nerves, and also in the sensory median nerve [17]. In a different study by Faden *et al.* [18], 20 out of 23 COPD patients

Figure 5



Receiver operating characteristic curve was plotted to evaluate the use of a decrease in forced expiratory volume in 1 s (FEV_1) level as a screening tool for the prediction of an increase of distal latency of median nerve in the chronic obstructive pulmonary disease group. This denoted good use of the decrease in FEV_1 level as a screening tool for prediction of increase of distal latency of median nerve in the chronic obstructive pulmonary disease group with a sensitivity of 91%, specificity of 69%, positive predictive value of 73%, negative predictive value of 90%, cutoff value lower to 89% of predicted of FEV_1 , and area under the curve of 0.788.

Table 4 Receiver operating characteristic curve to evaluate the use of decrease in percentage of forced expiratory volume in first second as a screening tool for prediction of increase of distal latency of median nerve in the chronic obstructive pulmonary disease group

Cutoff	Sensitivity	Specificity	PPV	NPV	AUC
$\leq 46\%$ of predicted of FEV_1	91.67	69.23	73.3	90.0	0.788

AUC, area under the curve; FEV_1 , forced expiratory volume in 1 s; NPV, negative predictive value; PPV, positive predictive value.

Table 5 Receiver operating characteristic curve to evaluate the use PaO_2 level as a screening tool for prediction of increase of sensory nerve conduction velocity in the chronic obstructive pulmonary disease group

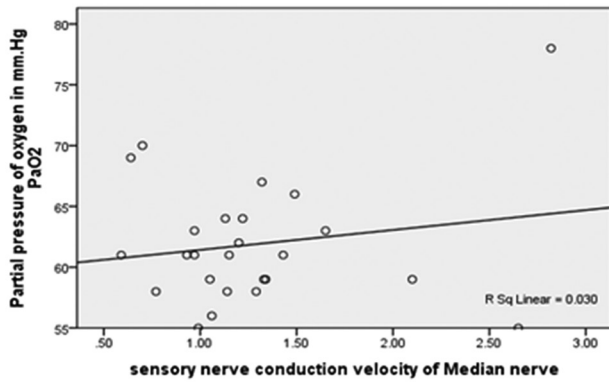
Cutoff	Sensitivity	Specificity	PPV	NPV	AUC
$PaO_2 \leq 59$ (mmHg)	80%	70%	40%	93.3%	0.785

AUC, area under the curve; NPV, negative predictive value; PPV, positive predictive value.

showed electrophysiologic evidence of peripheral nerve dysfunction. Abnormalities of SNC were most common affecting the sural nerve (20 patients), ulnar nerve (11 patients), radial nerve (eight patients), and median nerve (seven patients). Six patients had impairment of both sensory and motor nerve function; the common peroneal was the most frequently affected motor nerve. Clinical signs of neuropathy were found in four patients. COPD-related neuropathy was established to be correlated with cigarette consumption [18].

In agreement with our results, Valli *et al.* [19] investigated 19 patients with chronic respiratory insufficiency without conditions known to cause

Figure 6



Positive correlation between sensory nerve conduction velocity of median nerve and PaO₂ level.

PNPs. The motor and sensory conduction studies showed only reduced mean amplitude of the ulnar nerve sensory nerve action potential and of the CMAP of the abductor pollicis brevis muscle. The electromyography was abnormal in 94.7% of the cases. The data from this study support the hypothesis of an involvement of motor neurons in COPD.

Our data were supported further on by many studies [9,20–22].

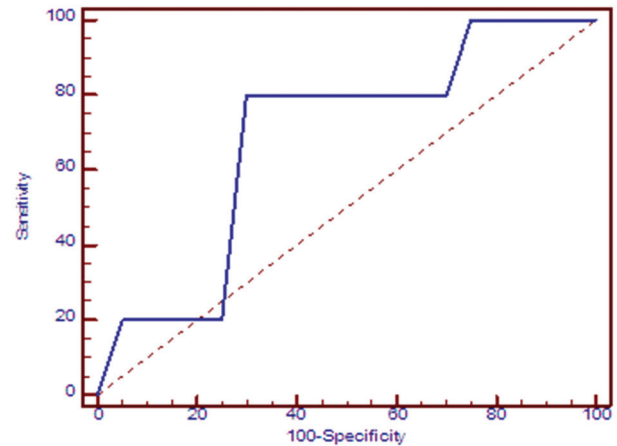
Agrawal *et al.* [23], in a group data analysis of COPD patients, revealed no motor nerve impairment, which contradicts our results. However, individual data analysis of the five patients with electrophysiological evidence of peripheral neuropathy suggests a predominantly sensory and axonal PNP. A possible explanation may be that the slight motor abnormalities in the five patients are masked by the normal values of the remaining 25 patients.

Some authors did not find any correlation between the electrophysiological and spirometric findings or blood gas analysis results [24]. However, others have implicated chronic severe hypoxemia as the causative factor for PNP [25,26] and that is consistent with our results.

Conclusion

Peripheral neuritis is a very common comorbidity in COPD patients. All types of neuropathy can take place. Sensory affection is more evident than motor affection in most studies. Mixed axonal demyelination type is the most common type of observation noticed. Most affections are subclinical. Chronic hypoxemia is the cornerstone of pathogenesis of neuropathy in COPD.

Figure 7



Receiver operating characteristic curve was plotted to evaluate the use of a decrease in PaO₂ level as a screening tool for prediction of a decrease of sensory nerve conduction velocity in the chronic obstructive pulmonary disease group. This denoted good use of a decrease in PaO₂ as a screening test for predicting decrease of sensory nerve conduction velocity in the chronic obstructive pulmonary disease group with a sensitivity of 80%, specificity of 70%, positive predictive value of 40%, negative predictive value of 93%, cutoff value lower to 59 mmHg, and area under the curve of 0.785.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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