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CASE REPORT

Peripheral Neuropathy in Chronic Obstructive Airway Disease

Neeraj Gupta[1], Chetan Basavaraj Patil[1], Rakesh C. Gupta[1], Shahir Asfahan[1]

[1]*Department of Respiratory Medicine, J L N Medical College, Ajmer, Rajasthan, India*

Peripheral neuropathy in COPD has received scanty attention despite the fact that very often clinicians come across COPD patients having clinical features suggestive of peripheral neuropathy while this comorbidity is often overlooked & considered a separate entity. A number of studies have now confirmed the association of COPD and peripheral neuropathy with hypoxaemia being a dominant etiopathogenic factor among others. We report a case a demyelinating polyradiculopathy in a patient with COPD along with a brief review of literature.

Key words: Chronic obstructive pulmonary disease, peripheral neuropathy, demyelinating polyradiculopathy

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a disease characterized by airflow limitation, which is not fully reversible. 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.[1] Although few series of studies are available in the literature describing COPD and peripheral neuropathy (PNP), our case report will probably recall for inclusion of this manifestation in the list of systemic manifestations of COPD, in which it has not yet find a place even in the latest review published.[2]

CASE REPORT

A 60-year-old male, smoker, nondiabetic, normotensive, known case of COPD presented with a history of severe breathlessness, increased cough and expectoration since past 8 days. He had several episodes of acute exacerbations in the past. He was on inhaled ipratropium + salbutamol since past 7 years prior to admission.

During the admission, he complained of progressive weakness in all four limbs followed by tingling sensation and numbness, predominantly in the lower limbs since past 5 weeks, which made him to reach the moribund state. These complaints restricted him to perform any kind of routine, apart from breathlessness. No history of similar complaints could be traced in his family. His bowel and bladder habits were regular.

Respiratory system examination revealed barrel shaped chest, distant breath sounds, early inspiratory crepitations and expiratory rhonchi with raised jugular venous pressure.

Central nervous system examination revealed diminished deep tendon reflexes including plantar response. Power and tone in all four limbs were impaired (UL-3/5, LL-2/5) with reduced to nil sensation to fine touch but responded to painful stimuli. There were no signs of cranial nerve involvement. Other system examination was normal.

Laboratory investigations of blood were within normal limits except for slightly elevated leucocytes count (13,200). Chest radiograph was suggestive of emphysema. Spirometry revealed severe stage of Global Initiative for Chronic Obstructive Lung Disease (class III); electrocardiography was suggestive of P pulmonale. HIV test by ELISA was nonreactive, and Vitamin B_{12} level was 560 pg/mL. Computed tomography of the head was normal, magnetic resonance imaging dorsolumbar spine showed disc degenerative changes. Cerebrospinal fluid (CSF) protein was slightly elevated (55 mg/dL); rest of CSF picture was within normal limits. SpO_2 on different occasions was in the range of 80-90%. Arterial blood gas report was suggestive of chronic respiratory acidosis.

Motor nerve conduction studies in both upper and lower limbs revealed absent F-wave, prolonged distal latencies and slowed conduction suggestive of demyelinating polyradiculopathy.

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Corresponding Author: Dr. Chetan Basavaraj Patil, No. 19, Suraj Vihar, Near Laxmi Nain, Lohagal Road, Ajmer - 305 001, Rajasthan, India. Tel: 7737441009; Fax: +917737-441009.
E-mail: chetan.colors@gmail.com

DISCUSSION

WHO defines COPD as a lung disease characterized by chronic obstruction of lung airflow that interferes with normal breathing and is not fully reversible. Emphysema and chronic bronchitis being the two common phenotypes that makes up COPD. It's a major cause of chronic morbidity and mortality throughout the world and the fourth most common cause of death worldwide.[3]

Peripheral neuropathy describes damage to the peripheral nervous system and distorts and interrupts messages between the brain and the rest of the body. Some forms of neuropathy, which damage only one nerve are called mononeuropathies. More often though, multiple nerves affecting all limbs are affected-called polyneuropathy. Occasionally, two or more isolated nerves in separate areas of the body are affected-called mononeuritis multiplex.

The actual prevalence of PNP in COPD is currently unknown, although there are several studies that indicate peripheral nervous system is commonly involved, which on most occasions is subclinical. The incidence has been reported to be as high as 93.8% in one study.[4] About one-third of patients of COPD have clinical evidence of polyneuropathy, and two-thirds have electrophysiological abnormalities. Some patients with no clinical evidence of polyneuropathy still have electrophysiological deficit suggestive of PNP. The appearance consists of a polyneuropathy often subclinical or with primarily sensory signs, which has the neurophysiological and pathological features of mainly axonal neuropathy.

Poza and Martí-Massó[5] also observed that microangiopathy in peripheral nerves in COPD patients appears to be diffuse and essentially related to hypoxia. Agrawal *et al*.[6] suggested that tobacco smoking; malnutrition; and drugs used in COPD treatment, may be possibly associated with neuropathy seen in COPD patients.

Our patient was diagnosed to be having demyelinating polyradiculopathy based on nerve conduction studies of the limbs. Other common causes of acquired PNP were excluded by careful clinical history, which included family history, history of drug abuse, illnesses such as hypothyroidism, collagen vascular disorders, chronic gastrointestinal disorders, viral infections like HIV, varicella and leprosy. The presence of sensory motor neuropathy in our case has prevented him in doing routine activities for last few weeks. Hence, we propose that in the absence of any other definitive causes of acquired PNP, it was probably a result of poorly managed COPD with chronic hypoxemia which resulted in peripheral neuropathy.

After carefully reviewing the literature in this regard, following prominent features of PNP in COPD need to be remembered and applied in such kind of patients:

a. Abnormality of sensory nerve conduction is most common manifestation.[7] It is usually mild, distal and leg accentuated.[8]
b. The rate of severity of neuropathy correlates with the severity of hypoxemia.[9]
c. The severity of PNP has also been shown to be correlated with hypercapnia, the degree of disability and thus with the severity of COPD.[10]
d. Tobacco smoking, alcoholism, malnutrition and adverse effects of certain drugs may be the other etiopathogenetic forms.

CONCLUSION

Based on the above facts published in a series of studies, we propose that:

a. PNP is not an uncommon systemic manifestation of COPD and must be included in the list along with other relatively more common systemic manifestations of COPD.
b. Knowledge of its coexistence may be valuable in early diagnosis of PNP, thereby framing the preventive strategies.
c. A well-controlled chronic airway obstruction with adequate bronchodilator therapy, keeping the PaO_2 levels above the definition of hypoxemia (i.e., PaO_2 >60 mmHg) may probably be helpful in preventing or slowing down the manifestations of PNP in COPD patients, thereby allowing them to avoid another disability.

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