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IDIOPATHIC DIAPHRAGMATIC PALSY ASSOCIATED WITH MOTOR AXONAL NEUROPATHY: A RARE CASE REPORT

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ABSTRACT

The loss of diaphragmatic muscle function, known as diaphragmatic palsy, is brought on by either phrenic nerve damage or muscle fiber weakening. Both unilateral and bilateral, it is frequently linked to generalized neuromuscular problems, although it can also be idiopathic, caused by trauma, compression, infection, or inflammation. The source, length, and degree of involvement all affect the symptoms, but frequent ones include anxiety, morning headaches, sleep difficulties, daytime somnolence, dyspnea, and orthopnea. Reduced thoracic muscular tone during rapid-eye-movement (REM) sleep frequently exacerbates these symptoms, resulting in hypoxemia and hypercapnia. We report a case of a 69-year-old male who presented with the symptoms of idiopathic diaphragmatic palsy and with a known history of motor axonal neuropathy.

KEYWORDS: Diaphragmatic Palsy, Neuromuscular Disorders, Idiopathic, Dyspnea.

INTRODUCTION

The main respiratory muscle in humans is the diaphragm, a dome-shaped musculo-fibrous structure that divides the thoracic and abdominal chambers. Diaphragmatic palsy (DP) is the term used to describe the loss of diaphragmatic muscular function brought on by either phrenic nerve damage or muscle fiber weakening. DP may be either transient or permanent,

depending on the degree of involvement and whether it is bilateral or unilateral.[1] It can be caused by trauma, compression, infection, inflammation, or idiopathic reasons, although it is frequently linked to widespread neuromuscular problems.[2] Approximately 20% of cases are classified as "idiopathic" since, even after thorough studies, no obvious cause can be found.[1] Other reasons include exposure to botulinum toxin, myasthenia gravis, spinal cord injuries, Guillain-Barre syndrome, and metabolic disorders such as hypophosphatemia, hypomagnesemia, hypokalemia, and hypocalcemia.[3] According to estimates, there are two cases of idiopathic diaphragmatic palsy for every 100,000 people per year.[6]

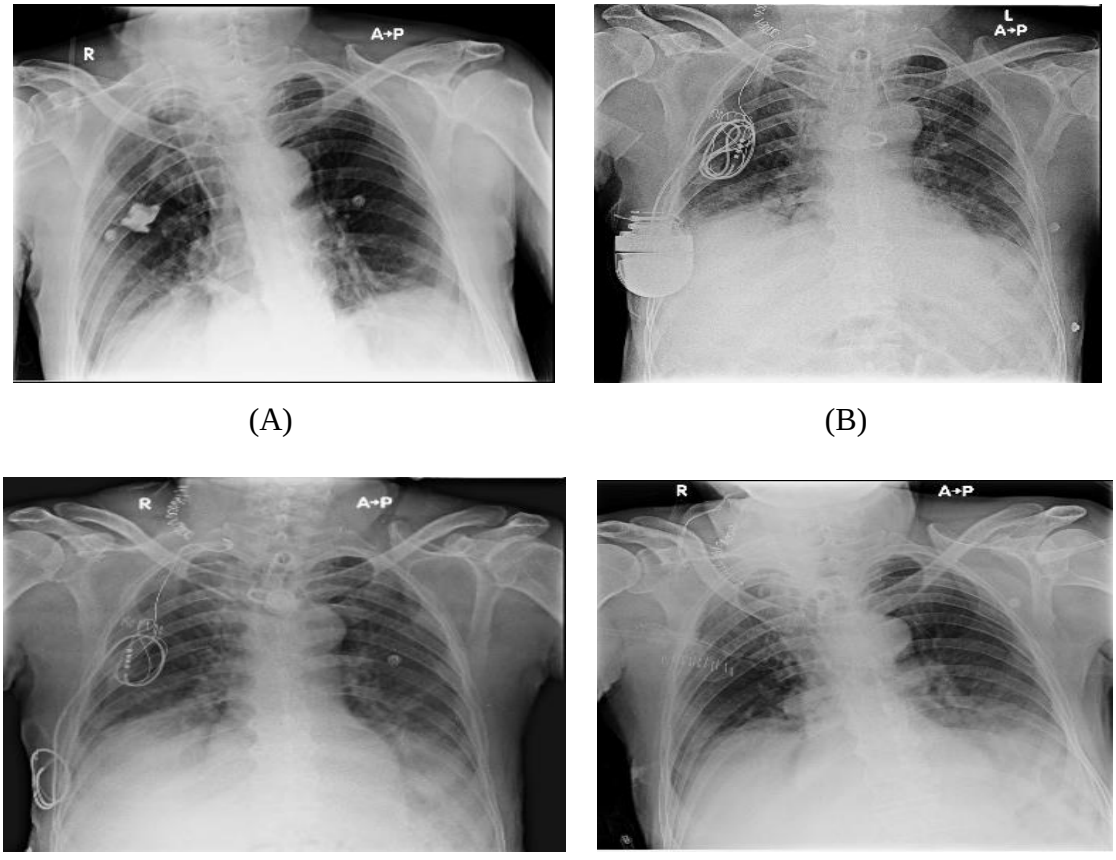
Diaphragmatic weakness symptoms vary depending on the origin, length of time, and involvement of one or both hemidiaphragms. Most people with unilateral diaphragmatic weakness are asymptomatic and are discovered by chance when a chest X-ray reveals an inflated hemidiaphragm. On the other hand, orthopnea, exertional dyspnea, or disturbed sleep may occur in those with impaired cardiopulmonary reserve.[1] Anxiety, morning headaches, sleep disruption, daytime somnolence, orthopnea, and dyspnea (81%) are common symptoms. Thoracic muscular tone is reduced during REM sleep, which exacerbates hypercapnia and results in hypoxemia.[6,7,8] In situations of unexplained dyspnea, evaluation usually starts with pulmonary function tests and chest radiography. Despite being widely utilized, dynamic imaging techniques like diaphragm fluoroscopy have poor specificity for both unilateral and bilateral diaphragmatic paralysis (UDP and BDP).[2] Diaphragmatic palsy is seen to be highly suggestive when there is a reduction in vital capacity (VC) of more than 25% from an upright to a supine posture.[4,5]

Treatment of diaphragmatic palsy depends on its cause and the extent of respiratory impairment. Concurrent conditions like obesity and chronic obstructive pulmonary disease should be managed prior to specific treatment. Most unilateral cases require no intervention, as they are often transient and resolve over time. Some patients may benefit from nocturnal ventilator support. In cases of bilateral diaphragmatic weakness, non-invasive positive pressure ventilation is the preferred non-surgical treatment option, offering effective respiratory support and symptom relief.

CASE REPORT

A 69-year-old guy with a known history of severe depressive illness and hypertension was brought to the emergency room on December 14, 2024, complaining of decreased activity, dysphagia, and trouble speaking over the previous month. A nerve conduction investigation

on September 20, 2024, found length-dependent motor axonal neuropathy with early demyelination. He was then admitted to NIMHANS' psychiatric department and slated for electroconvulsive treatment (ECT). On October 5, 2024, he had an MRI under general anesthesia, during which he had respiratory difficulty, hypothermia, and desaturation. He was intubated and later had a tracheostomy for airway protection and extended ventilation. Upon evaluation, he exhibited raised blood pressure and fever, 100% oxygen saturation on a ventilator with CPAP, and a GCS of E4V5M6. The remaining part of the clinical evaluation was normal. Investigations, including a chest X-ray (Fig. 1), serum electrolytes (Table 1), and arterial blood gas analysis (Table 3), revealed idiopathic diaphragmatic palsy coupled with motor axonal neuropathy.



(A) (B)

(C) (D)

Fig. 1: X-Ray chest AP view shows.

A: Patchy homogeneous opacity noted in bilateral lower lung zones S/O consolidation on 12/14/2024.

B: Diffuse haziness noted in bilateral lower zones on 12/18/2024.

C: Patchy homogeneous opacity noted in bilateral lower lung zones S/O consolidation on 12/18/2024.

D: Mild haziness noted at left lower and right upper lung zones s/o infective etiology on 26/12/2024.

Table 1: Serological workup of the patient from Day 1 to Day 14.

LAB PARAMETERS (NORMAL RANGE)	D 1	D 2	D 3	D 4	D 5	D 6	D 7	D 8	D 9	D 10	D11	D12	D 14	
HB(13-17gm/dl)	11.1↓												11.0↓	
RBC COUNT(4.5-5.5 mill/cmm)	3.66↓												3.68↓	
PCV(40-50%)	31.9↓												32.5↓	
MCHC(31.5-34.5 g/dl)	34.7												33.8	
Lymphocytes(20-40%)	7.7↓												10.3↓	
Neutrophils(40-80%)	87↑												80.1	
WBC COUNT(4.0-10.0 K/uL)	7												9.2	
PC(150-400 K/uL)	215												439↑	
CRP(<5 mg/L)	49.4↑												12.2↑	
Sodium(135-148 mmol/L)	135.4	137.4	135.3	139.9	137.2	135.5	134.8	140.4	135.9	137.2	134.6	141.4	142.5	
Chloride(98-107mmol/L)	97.6	100.6	98.6	104.2	101.1	101.1	96.8↓	100.7	96.0↓	97.9↓	99.1	102.5	106.1	
Bicarbonate(22-29mmol/L)	30.6↑	26.7	26.2	24.0	25.7	25.4	29.1↑	30.4↑	29.3↑	30.3↑	23.7	27.2	23.0	
Potassium(3.5-5.5 mmol/L)	4.23	3.97	3.72	4.21	3.77	4.19	4.45	5.41	4.76	4.23	4.30	3.63	3.33	

D: Day; WBC: White Blood Cell; PCV: Packed cell volume; PC: Platelet count; CRP:C-reactive protein; MCHC: Mean cell haemoglobin concentration; Hb: haemoglobin; ↓: Decreased; ↑**:** Increased;

Table 2: Other laboratory reports.

TEST	RESULT
Blood culture, tracheal aspirate and Urine culture	No growth
HIV	Negative
HCV	Negative
HBsAg	Negative
HIV - Human Immunodeficiency Virus; HCV - Hepatitis C Virus; HBsAg - Hepatitis B Surface Antigen;	

Table 3: Routine workup of Arterial blood gas (ABG) analysis.

ABG Components	14/12	15/12	16/12	18/12	19/12	23/12	24/12	25/12	26/12	
pH [7.350-7.450]	7.465↑	7.422	7.438	7.430	7.463↑	7.36	7.39	7.622↑	7.55↑	
pCO2 [35-45mmHg]	40.3	41.7	39.8	42.2	35.5	55.9	46.8↑	25.2↓	26.6↓	
pO2 [75-100mmHg]	102.3↑	116.9↑	144.9↑	103.3↑	112.4↑	137.2↑	105.6↑	119.3↑	113.9↑	
HCO3 [22-26 mmol/L]	28.3	26.2↑	26.3↑	27.0↑	25.7	28.0↑	26.9↑	28.7↑	26.7↑	
BE [-2.0 to +2.0 mmol/L]	4.3↑	1.9	2.0	2.8↑	1.1	4.0↑	2.7↑	4.7↑	2.1↑	
Hct [35.0-50.0%]	32↓	32↓	27↓	27↓	13.5↓		28↓	29↓	39.4	
tHb [13.0-17.0 g/dL]	11.0↓	10.8↓	9.1↓	9.2↓			9.1↓	9.8↓		
COHb [0.5-2.5%]	0.3↓	0.2↓	0.1↓	0.2↓			1.0	0.3↓		
O2Hb [95.0-99.0%]	97.2	98.0	98.9	97.3			96.8	98.4		

MetHb [0.4-1.5%]	0.3↓	0.3↓	0.3↓	0.3↓			0.3↓	0.3↓		
SO2 [75.0-99.0%]	97.8	98.5	99.3	97.8	98.4		98.1	99.0	98.7	
AnGap [8-16 mEq/L]	10.3	12.6	6.5↓	10.1	9.9	12.9	6.7↓	9.7	5.8↓	
Glu [74-106 mg/dL]	67↓	63↓	33↓	76		58↓	114↑	237↑		
Lac [0.4-2.2 mmol/L]	1.22	1.42	1.07	1.14	0.7	1.29	1.41	1.75↑	1.1	

pH: Potential of Hydrogen; pCO2: Partial Pressure of Carbon Dioxide; pO2: Partial Pressure of Oxygen; HCO3: Bicarbonate; BE: Base Excess; Hct: Hematocrit; tHb: Total Hemoglobin; COHb: Carbon Monoxide Hemoglobin; O2Hb: Oxygenated Hemoglobin; MetHb: Methemoglobin; SO2: Oxygen Saturation; AnGap: Anion Gap; Glu: Glucose; Lac: Lactate; ↓: Decreased; ↑**:** Increased;

The patient was hospitalized with the aforementioned history. Relevant scans and investigations have been done. He experienced sporadic temperature spikes; cultures were submitted but revealed no growth. On December 17, 2024, the patient received a right phrenic nerve stimulation trial while under general anesthesia. He was lying supine, with his head rotated left to reveal the anterior border of the right sternocleidomastoid muscle. The right phrenic nerve was located using ultrasound guidance, and an incision was made appropriately. Dissection proceeded until the anterior scalene muscle was discovered and the phrenic nerve was precisely isolated. Using an external stimulator, nerve stimulation caused observable diaphragmatic contraction, which was validated by neuromonitoring and spontaneous breathing effort during ventilator stoppage. Electrodes were sutured around the nerve, and the leads were subcutaneously tunneled and connected to a percept battery. The wound was closed in layers, and sterile dressing was applied.

After the surgery, the patient was sent to the Neuro ICU. The patient was tolerating life without a ventilator when the battery simulation was conducted on December 18, 2024. The patient self-removed the battery on 18-12-24 due to restlessness. After a re-examination, the patient's phrenic nerve stimulation trial and electrode removal under GA failed on December 23, 2024. However, diaphragm contraction was not seen upon stimulation. The process was dropped. Leads were taken out. The patient was returned to the Neuro ICU after the operation. There was routine physical treatment. Assessments for speech and swallow treatment were conducted, and recommendations were complied with. The patient is well tolerated, according to BIPAP trials.

On December 26, 2024, after hemodynamic stabilization, the patient was moved from the Neuro ICU to the ward for continued therapy. IV antibiotics (Inj ceftriaxone 2 mg BD and Inj piperacillin+tazobactum 4.5 mg QID), mucolytic agents (Inj Acetylcysteine 400 mg TID, Tab Acetylcysteine 600 mg), antihypertensive medications (Inj Amlong 10 mg BD, Tab

Spironolactone 25 mg OD), Tab pyridostigmine 30 mg TID, and other supportive measures such as antipyretics and multivitamins were used in the patient's conservative care. As the patient's symptoms improved and her hemodynamics stabilized, she was discharged with regular follow-up and proper treatment, including IV antibiotics, oral hypertension medications, mucolytics, and multivitamins.

DISCUSSION

Diaphragmatic palsy refers to the loss of diaphragmatic muscular function due to either weakness of the muscle fibers or phrenic nerve injury. The condition may be temporary or permanent, depending on the underlying cause.[1] Diagnosis typically involves chest radiography and pulmonary function tests in patients with unexplained dyspnea. Although dynamic imaging like diaphragm fluoroscopy is used, its specificity for both unilateral and bilateral diaphragm paralysis remains low.[2] In this case, a 69-year-old male presented with reduced activity, dysphagia, and difficulty speaking. Chest X-ray revealed patchy homogeneous opacity in the bilateral lower lung zones, indicative of consolidation and consistent with idiopathic diaphragmatic palsy. Diaphragmatic paralysis is often associated with generalized neuromuscular disorders[2], and this patient had a known history of motor axonal neuropathy. This highlights a possible correlation between idiopathic diaphragmatic palsy and underlying neuromuscular conditions in elderly patients.

CONCLUSION

Diaphragmatic palsy is a rare but significant condition that may result from diaphragmatic muscle weakness or phrenic nerve injury. It can be either temporary or permanent depending on the underlying etiology and may present independently or in association with neuromuscular disorders. Timely diagnosis through appropriate imaging and pulmonary function assessment is essential, especially in patients with unexplained dyspnea. In this case, the diagnosis of idiopathic diaphragmatic palsy in a patient with pre-existing motor axonal neuropathy highlights the importance of considering diaphragmatic dysfunction in patients with neuromuscular symptoms. Early recognition and appropriate management can improve respiratory function and overall patient outcomes.

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