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Case Report

Unilateral diaphragmatic paralysis in a diabetic patient: A rare cause of dyspnea

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ABSTRACT

Diaphragmatic paralysis is a rare cause of dyspnea, often resulting from trauma or neuropathic conditions. Although uncommon, diabetic neuropathy can involve the phrenic nerve. Herein, we presented a case of unilateral diaphragmatic paralysis in a 62-year-old male with uncontrolled type 2 diabetes mellitus. Diagnosis was confirmed by phrenic nerve conduction studies and dynamic ultrasonography. Elevated hemoglobin A1c was noted, with no other identifiable cause. Glycemic control was prioritized in management. This case highlights the importance of considering phrenic nerve involvement in diabetic patients with unexplained dyspnea and underscores ultrasonography's diagnostic value in such cases.

Keywords: Diabetes mellitus, diaphragmatic paralysis, dyspnea, electromyography, phrenic nerve, ultrasonography.

Diaphragmatic paralysis occurs secondary to the loss of phrenic nerve function and is characterized by reduced muscle tone without any disruption in the integrity of the diaphragm itself or its attachments to the chest wall. It can be either congenital or acquired. In general, acquired diaphragmatic paralysis is rare, with an incidence of less than 0.05%.[1] While trauma, neuropathic, inflammatory, and iatrogenic causes are recognized, approximately 20% of cases remain idiopathic. Although rare, diabetic neuropathy has occasionally been identified as a cause of phrenic nerve palsy.[2]

In unilateral diaphragmatic paralysis, the most common etiologies are trauma and iatrogenic injury, with left-sided involvement being more frequent. Most patients remain asymptomatic, and the condition is often incidentally detected as an elevated hemidiaphragm on chest radiography. Less commonly, patients may experience exertional dyspnea, orthopnea, sleep disturbances, or even respiratory failure.

In this report, we presented a rare case of unilateral diaphragmatic paralysis due to uncontrolled type 2 diabetes mellitus, highlighting an uncommon cause of dyspnea in light of the existing literature.

CASE REPORT

A 62-year-old male patient was referred to the neurophysiology laboratory for phrenic nerve examination. The patient reported a one-year history of dyspnea, initially presenting with acute pain radiating to the left shoulder and periscapular region, which spontaneously resolved. Although thoracic magnetic resonance imaging was not performed, physical examination revealed multiple trigger points around the scapular region, suggesting a myofascial origin for the periscapular pain. The patient denied cough, hemoptysis, fever, wheezing, chest pain, weight loss, or sputum production and stated that the symptoms worsened with exertion and while lying supine. A written informed consent was obtained from the patient.

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The patient had a 20-pack-year smoking history but had quit 25 years earlier. He previously worked in a lumber mill and had recently been employed as a cleaner. The medical history included type 2 diabetes mellitus and hypertension for seven years. There was no travel to endemic areas or history of tick bites suggestive of Lyme disease. Family history was unremarkable.

The patient had been under follow-up in the pulmonology clinic for one year. Review of prior investigations revealed no significant pathology other than fibroatelectatic changes in the left lower lobe on thoracic computed tomography. Fiberoptic bronchoscopy with bronchoalveolar lavage cytology and transthoracic echocardiography were normal. Transthoracic echocardiography and electrocardiogram revealed no cardiac pathology, with a normal ejection fraction, effectively ruling out a cardiogenic cause for the patient's symptoms. Chest radiographs obtained approximately 15 months earlier demonstrated elevation of the left hemidiaphragm (Figure 1). Contrast-enhanced cervical magnetic resonance imaging revealed no masses or cervical disc herniation that could explain the paralysis.

On physical examination, joint range of motion was normal, and no neurological deficits were observed. Complete blood count, biochemistry, and acute phase reactants were within normal limits. Fasting plasma glucose was elevated, and hemoglobin A1c (HbA1c) was 8.4%.

Nerve conduction studies (sural, tibial, peroneal, and median nerves) performed for diabetic polyneuropathy were normal. Phrenic nerve conduction study showed delayed distal latency in the left phrenic nerve (right: 7.4 msec; left: 11.2 msec) and a reduced compound muscle action potential (CMAP) amplitude (right: 700 μV; left: 170 μV).

Pulmonary function testing showed a forced vital capacity (FVC) of 3.04 L (78% predicted), forced expiratory volume in 1 sec (FEV1) of 2.56 L (84% predicted), and an FEV1/FVC ratio of 84.2%.

To evaluate diaphragmatic contractility, dynamic ultrasonography was performed. Using a 4-10 MHz linear probe placed perpendicular to the eighth or ninth intercostal space along the anterior axillary line. The diaphragm thickness was measured during maximal expiration and maximal inspiration. The thickening fraction was calculated using the following formula: (maximal inspiration-maximal expiration)/maximal expiration.[3] The left diaphragm was thinner (0.14 cm) than the right (0.19 cm) during maximal expiration. While the right diaphragm showed a thickening fraction of 158%, paradoxical thinning of the left diaphragm was noted during maximal inspiration (Figure 2).[4]

Based on the findings of nerve conduction studies, imaging, laboratory results, and clinical history, a diagnosis of diabetic unilateral phrenic nerve paralysis was established. The patient was subsequently referred to the endocrinology department for optimal management of plasma

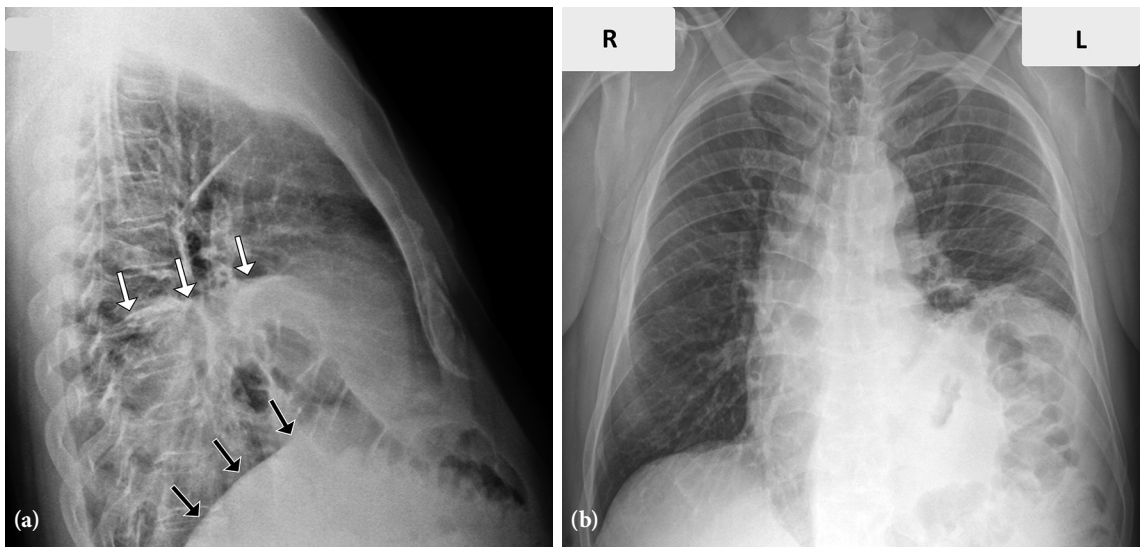

Figure 1. **(a)** Lateral and **(b)** posteroanterior chest radiographs demonstrating elevation of the left hemidiaphragm.

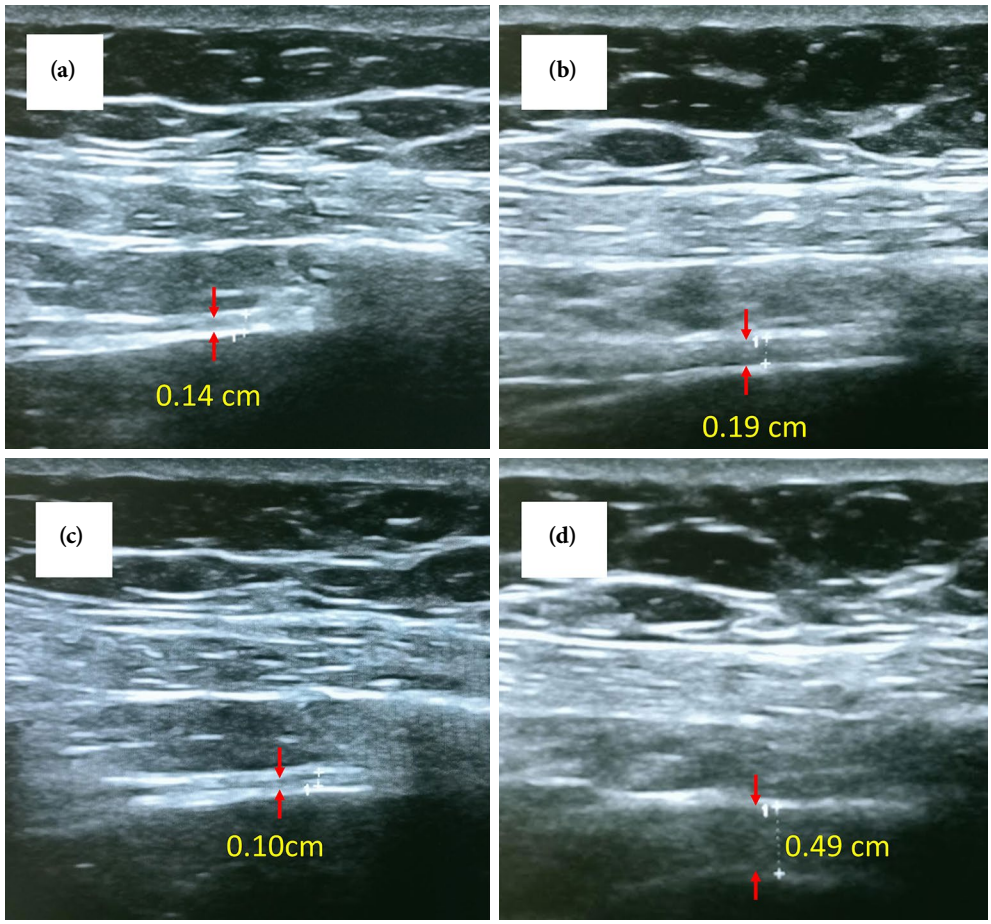

Figure 2. Diaphragmatic ultrasound images obtained from the eighth or ninth intercostal space along the anterior axillary line on the **(a, c)** left and **(b, d)** right sides. On the affected (left) side, **(c)** a paradoxical decrease in diaphragm thickness is observed during forced inspiration compared to expiration **(a)**. On the unaffected (right) side, diaphragm thickness increases **(d)** appropriately during inspiration compared to expiration **(b)**, with a thickening fraction calculated of 158%.

glucose levels. The patient has been followed by pulmonology every six months, with stable clinical status.

DISCUSSION

The diaphragm is independently innervated on each side by the phrenic nerve, which arises from the cervical nerve roots C3-C5. Although diaphragmatic paralysis may be unilateral or bilateral, unilateral involvement is more common.[5] Loss of function can result from issues affecting the diaphragm muscle itself or the phrenic nerve. Common causes include trauma, compression or infiltration (e.g., mediastinal tumors), Guillain-Barré syndrome, infections (herpes zoster, Lyme disease), Parsonage-Turner syndrome, cardiothoracic surgery, diabetes mellitus, and shrinking lung syndrome associated with systemic lupus erythematosus.

In our case, the absence of trauma, tick exposure, thoracic surgery, or rheumatologic findings, along with high glucose levels and normal imaging, led to the consideration of diabetic unilateral phrenic nerve paralysis. Electrophysiological studies play a key role in diagnosis. Phrenic nerve conduction studies assess latency and CMAP amplitudes by stimulating the nerve at the neck and recording diaphragm muscle responses. Demyelinating polyneuropathies can prolong latency, while traumatic injuries often reduce CMAP amplitudes. In healthy adults, phrenic nerve latency is typically 6 to 8 msec, with CMAP amplitudes ranging from 500 to 800 μV.[6,7] Our patient's left phrenic nerve latency was 11.2 msec,

with a CMAP of 170 μV, indicating significant impairment.

Among diagnostic tools, ultrasonography offers a noninvasive, portable, radiation-free, and rapid method to quantitatively and qualitatively evaluate diaphragmatic movement. In neuromuscular disorders affecting the diaphragm, ultrasonography has demonstrated a sensitivity of 93% and specificity approaching 100%.[3,8]

The prognosis and management of phrenic nerve injury or diaphragmatic paralysis depend on the underlying etiology and pulmonary function status. Treatment primarily targets the underlying cause and modifiable factors such as diabetes, obesity, respiratory disease, or cardiac comorbidities. Asymptomatic patients with unilateral paralysis often require no intervention. When a potentially reversible cause is identified (e.g., infection, metabolic or endocrine disorders such as diabetes or hypothyroidism), appropriate targeted therapy is indicated. Idiopathic cases may recover spontaneously; one study reported that 40 to 60% of cases with potentially reversible causes (e.g., surgical, paraneoplastic, and diabetic neuropathy) achieved spontaneous improvement in diaphragm and respiratory muscle strength over time. In cases without clinical or radiological improvement within a reasonable timeframe, surgical options such as diaphragmatic plication or phrenic nerve repair may be considered.[1,6]

Phrenic nerve paralysis is a rare complication of diabetes. Elevated HbA1c levels have been associated with the severity of neuropathy.[9] Nevertheless, this case demonstrates that phrenic nerve paralysis may occur even in the absence of diabetic polyneuropathy, suggesting that elevated HbA1c levels alone may not fully account for the development of such neuropathic complications. Most cases of unilateral diabetic phrenic nerve paralysis do not require treatment beyond appropriate glycemic control. In a study conducted by Yesil et al.,[10] bilateral phrenic nerve conduction studies performed on 37 diabetic patients, 40 prediabetic patients, and 18 healthy controls demonstrated significantly reduced amplitudes and prolonged latencies in both diabetic and prediabetic individuals. Moreover, a case report described a patient in whom diaphragmatic paralysis led to the subsequent diagnosis of previously unrecognized diabetes mellitus.[11]

In conclusion, in patients presenting with dyspnea, a thorough history and physical examination are essential. Despite its rarity, phrenic nerve paralysis in the absence of diabetic polyneuropathy should be included in the differential diagnosis. Ultrasonography is a valuable, noninvasive modality for confirming unilateral phrenic nerve palsy.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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