Spontaneous idiopathic hemi-diaphragmatic paralysis: case report

Ogheneyoma Akpoviroro[1*], Jeremy Freiwald[2]

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ABSTRACT

Background: The diaphragm is a major muscle of respiration that is innervated by the phrenic nerve. Dysfunction of this muscle could lead to respiratory failure of varying degrees, depending on whether the bilateral diaphragms or a unilateral diaphragm (i.e., hemidiaphragm) are affected. Such respiratory dysfunction could be so severe as to result in symptomatic hypercapnia requiring medical and/or surgical intervention for amelioration. Diaphragmatic paralysis occurs when underlying pathology results in a failure of the phrenic nerve to control diaphragmatic function; however, in some cases, there are no known precipitating pathologic etiologies. Diaphragmatic paralysis is an uncommon presentation in the clinical setting that often leads to delayed diagnosis.

Case Presentation: This report depicts a case of acute-onset dyspnea due to spontaneous idiopathic hemidiaphragm paralysis. A 71-year-old Caucasian man presented via telemedicine to his primary care physician with complaints of dyspnea ongoing for 2 days. The patient subsequently underwent multiple tests, including a course of antibiotics, multiple imaging studies, and several visits to the emergency department. Despite extensive lab and imaging studies, the diagnosis of hemi-diaphragmatic paralysis was delayed for several months before a final diagnosis by the pulmonary medicine clinic.

Conclusion: This case portrays the significance of looking deeper beyond the typical cardiopulmonary etiologies in patients with unexplained acute dyspnea. It specifically highlights the importance of working up the respiratory muscles, especially the diaphragm, as the possible origin of acute unexplained dyspnea so as not to significantly delay diagnosis.

Keywords: Hemidiaphragm paralysis, unilateral diaphragm paralysis, phrenic nerve palsy, unilateral phrenic nerve palsy, phrenic nerve dysfunction, acute dyspnea of undetermined origin, unexplained dyspnea, case report.

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Correspondence to: Ogheneyoma Akpoviroro
*Internal Medicine Department, Geisinger Northeast, Wilkes Barre, PA.
Email: oakpoviroro@geisinger.edu
Full list of author information is available at the end of the article.

Background

Acute-onset dyspnea commonly prompts investigation of the cardiovascular and pulmonary systems. Diaphragmatic paralysis is a diagnosis that is often missed, sometimes for months, when patients are being evaluated for acute-onset dyspnea. Diaphragmatic paralysis is attributed to phrenic nerve dysfunction secondary to compression from adjacent pathology, surgical intervention (i.e., iatrogenic origin), neuropathy, trauma [1,2], inflammation, and even as a complication of infections such as COVID-19 [3,4]. The resulting paralysis may be asymptomatic when a hemi-diaphragm is affected, or it may result in hypoventilation with resulting hypercapnia and dyspnea [4]. In symptomatic patients with hemi-diaphragm paralysis (HDP), it is thought that symptoms arise due to a combination of a loss of inspiratory forces generated by the paralyzed half of the diaphragm and decreased pressure generation by the functional diaphragm [5]. This report depicts a case of acute-onset dyspnea due to spontaneous idiopathic HDP, an uncommon pathology, especially in the primary care setting, and a diagnosis that is frequently missed as an etiology of dyspnea in adults [6]. This case demonstrates the importance of keeping the respiratory muscles in the differential when assessing patients with new-onset dyspnea. This is especially significant when dyspnea is unexplained, even in patients without known precipitators of diaphragmatic injury.

Case Presentation

A 71-year-old Caucasian man presented via telemedicine to his primary care physician (PCP) with complaints of dyspnea ongoing for 2 days. Past medical history was significant for basal cell carcinoma of the skin status post Mohs microscopic surgery, colonic polyp, hypertension, hyperlipidemia, prostate cancer, plantar fasciitis, prediabetes, transient ischemic attack, thyroid nodule status post partial thyroidectomy, tobacco use disorder (>30

pack years), and vitamin D deficiency. Symptoms were reported to have started as a sudden onset of pain in the left chest and epigastric region, with associated dyspnea. The patient was previously seen at an emergency department (ED) 2 days before his primary care visit, without a resulting diagnosis. The patient was discharged home without treatment. The patient was seen via telemedicine by his PCP 2 days after this ED visit. A computed tomography (CT) angiogram was ordered by the PCP and performed on the same day; this test revealed right lower lobe opacification concerning infiltration such as pneumonia, versus atelectasis. There was no evidence of pulmonary embolism. Labs ordered following this initial telemedicine visit, including a basic metabolic panel and a COVID-19 polymerase chain reaction test, were negative.

Therapeutic intervention

The patient was seen via telemedicine following his first telemedicine primary care visit, with no improvement in dyspnea. A 10-day course of twice daily amoxicillin-clavulanate (875/125 mg) and a 6-day tapering course of methylprednisolone were ordered by the PCP, given the patient's significant smoking history, and suspected acute exacerbation of chronic obstructive pulmonary disease (COPD) due to underlying lung infection.

Follow-up and outcomes

The patient reported some improvement in dyspnea via a telemedicine visit 1 day after starting therapy but presented to the ED 12 days later with significant dyspnea, worse on exertion, and an occasional dry cough. Physical examination noted clear lungs to auscultation bilaterally and unremarkable findings in other systems. Vitals were stable and non-contributory (Table 1). Multiple labs were ordered and were mostly within normal limits (Table 2). An electrocardiogram showed normal sinus rhythm without any acute ischemic changes. An anterior-posterior chest X-ray was significant for right lung base atelectasis and elevation of the right hemidiaphragm (Figure 1). The patient was discharged from the ED with a diagnosis of dyspnea of undetermined etiology.

The patient was seen by his PCP in person, 14 days after being discharged from the ED. This was the fourth visit with the PCP. He reported continued dyspnea worse with

Table 1. *Vitals taken on arrival at the ED.*

VITALS	VALUE
Oxygen saturation	95%
Temperature	97.8 F
Pulse	106 (repeated 80)
Respiratory rate	32 (repeated 20)
Blood pressure	160/91 (repeated 133/78)

Table 2. *Results of lab tests drawn at the ED.*

LAB TEST		RESULT
Troponin		4.2 pg/ml
Comprehensive metabolic panel	Calcium	9.3 mg/dl
	Glucose	121 mg/dl
	Urea nitrogen	12 mg/dl
	Creatinine	0.9 mg/dl
	Sodium	138 mmol/l
	Potassium	3.6 mmol/l
	Chloride	105 mmol/l
	CO_2	21 mmol/l
	Estimated glomerular filtration rate	83 ml/min-ute/1.73 m^2
	Creatine phosphokinase	39 IU
	Phosphorus	2.9 mg/dl
	Magnesium	1.8 mg/dl
	Total protein	7.1 g/dl
	Albumin	4 g/dl
	Alkaline phosphatase	60 IU/l
	Aspartate transaminase	17 IU/l
	Alanine transaminase	33 IU/l
	Total bilirubin	0.8 mg/dl
Prothrombin time		13.5 seconds
Partial thromboplastin time		29.7 seconds
International normalized ratio		1 second
D-Dimer		<0.50 mcg/ml
Complete blood count	White blood cell	10.1 K/cmm
	Red blood cell	4.68 l M/cmm
	Hemoglobin	15.0 g/dl
	Hematocrit	44.1%
	Mean corpuscular volume	94.2 H u/cmm
	Mean corpuscular hemoglobin	32.0 pg
	Mean corpuscular hemoglobin concentration	34.0 g/dl
	Red cell distribution width	15.4 H%
	Platelet	156 K/cmm
	Mean platelet volume	9.9 u/cmm
	Neutrophil %	60.0%
	Lymphocyte %	26.2 l %
	Monocyte %	6.0%
	Eosinophil %	7.1 H%
	Basophil %	0.7%
	Neutrophil #	6.0 K/cmm
	Lymphocyte #	2.6 K/cmm
	Monocyte #	0.6 K/cmm
	Eosinophil #	0.7 H K/cmm
	Basophil #	0.1 K/cmm
Brain natriuretic peptide		22 pg/ml

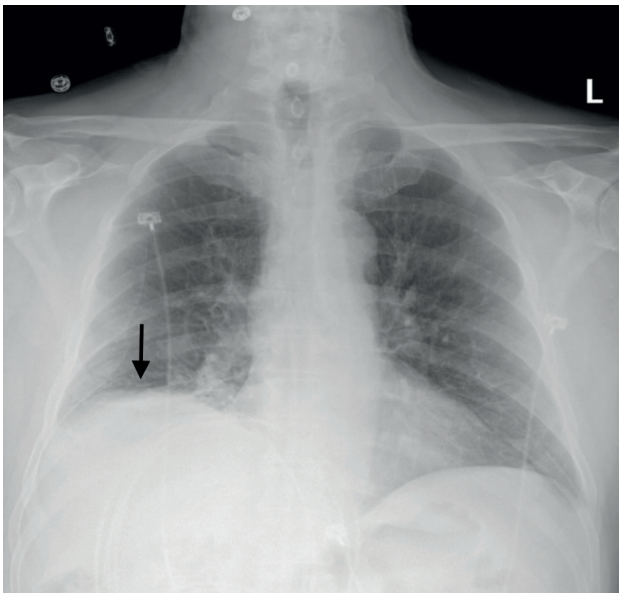

Figure 1. *Anterior posterior chest radiograph taken at the institutional-affiliated ED.*

minimal exertion, orthopnea while laying supine and on his left side, and nocturnal episodes of significant dyspneic exacerbations, causing nighttime awakenings. The patient reported persistent oxygen saturation of 93% throughout the day at home. At this point, the patient's PCP started him on the combination inhaler, ipratropium bromide, and albuterol, to be used every 6 hours, as needed for symptomatic relief of dyspnea. A 6-minute walk test to determine exercise tolerance, a chest CT scan without contrast, and a pulmonary function test (PFT) were ordered; a consult to pulmonology was placed. The CT scan without contrast showed similar findings to the prior CT angiogram of the chest, with right middle and right lower lobar atelectasis versus infiltration, air bronchograms, and elevation of the right hemidiaphragm favoring atelectasis over infiltration; lingula atelectasis was also noted to be present.

The 6-minute walk test and PFT were performed 1 week after the fourth PCP office visit. The patient walked a distance of 550 feet without significant desaturation (Table 3). The PFT results were consistent with a restrictive pathology, with a notably low inspiratory capacity of 1.90 l (Table 4).

The patient was seen by the pulmonary medicine clinic 1 month later; a fluoroscopy of the diaphragm, a ventilation-perfusion (VQ) scan of the lung to rule out any residual vascular abnormalities, a transthoracic echocardiogram, a posterior-anterior chest radiograph and a repeat D-dimer level (<0.5 mcg/ml) were ordered. In the interim, the patient had persistent dyspneic symptoms. The repeat chest X-ray revealed persistent right basilar atelectasis with an associated elevation of the right hemidiaphragm, similar to the previous chest radiograph. The VQ scan showed no ventilation defects, or segmental perfusion defects, corresponding to a low probability of pulmonary embolism. The transthoracic echocardiogram was significant for mild concentric

Table 3. *Six-minute walk test.*

TIME FROM START OF EXAMINATION (MINUTES)	OXYGEN SATURATION (%)	HEART RATE (BEATS PER MINUTE)
1	94	105
2	93	102
3	92	105
4	92	103
5	92	102
6	92	102

Baseline oxygen saturation: 95%; baseline heart rate: 97; walking pace: moderate; number of stops: 0.

left ventricular hypertrophy, impaired relaxation pattern of left ventricular filling, and mild-to-moderate aortic stenosis without hemodynamic significance. The left ventricular ejection fraction (LVEF) was 60%-65% and there was no evidence of pericardial effusion.

The patient was seen 2 weeks later at the pulmonary medicine clinic before the fluoroscopy was performed. The patient reported continued dyspnea worse with exertion and orthopnea. At this point, it was suspected that the patient had some diaphragmatic dysfunction given his poor inspiratory function on PFTs (Table *4*) and otherwise noncontributory pulmonary and cardiovascular testing. A nighttime positive pressure device was recommended but the patient refused this therapy at this time; the patient was sent home with an inspiratory muscle trainer and an order for a nuclear regadenoson stress test. The nuclear stress test showed no regional wall motion abnormalities of the left ventricle, a normal LVEF of 75%, no fixed or reversible perfusion defects, no evidence of transient ischemic dilatation of the left ventricle, and the stress electrocardiogram (EKG) was non-diagnostic. The CT component of this test showed some improvement in the right middle and right lower lobar atelectasis compared to the prior CT scan of the chest. Chest fluoroscopy showed atelectasis of the right lung base above the elevated right hemidiaphragm, with minimal right diaphragmatic excursion during inspiration and expiration. Paradoxical motion during forced inspiration was also noted, consistent with a positive sniff test, and confirming the diagnosis.

The patient was seen by the PCP about 4 months after his initial in-person visit; he complained of worsened dyspnea now worse with speech. The patient also reported sharp intermittent right upper quadrant pain. He was referred to the thoracic surgery clinic to discuss treatment options. The patient was seen by surgery and a decision for diaphragmatic plication was made about 6 months after the onset of the patient's symptoms, and the patient underwent the aforementioned procedure as planned.

Discussion

This case highlights a manifestation of idiopathic HDP occurring spontaneously as sudden-onset dyspnea, without

Table 4. *Pulmonary function test.*

SPIROMETRY					
	Predicted	**Upper limit of normal**	**Lower limit of normal**	**Pre-bronchodilator**	**Pre-bronchodilator % of predicted**
FVC (l)	3.71	4.69	2.74	2.57	69%
FEV 1 (l)	2.83	3.57	2.03	1.93	68%
FEV1/FVC (l)	76	89	63	75	
FEF 25%-75% (l/second)	2.19	3.96	0.94	1.50	69%
FEF 50% (l/second)	3.85	6.02	1.68	2.15	56%
FIF 50% (l/second)				2.96	
PEF (l/second)	7.49	9.58	5.40	4.61	62%
FEV0.5/FIV0.5				1.05	
PIF (l/second)	6.92	10.48	3.36	3.08	44%
Lung volumes					
TLC (l)	6.42	7.57	5.26	4.50	70%
VCMAX_pl (l)	3.71	4.69	2.74	2.72	73%
IC (l)	2.71	2.71	2.71	1.90	70%
FRCpleth (l)	3.50	4.49	2.51	2.60	74%
ERV (l)	0.96	0.96	0.96	0.82	86%
RV (l)	2.54	3.22	1.87	1.78	70%
RV % TLC (%)	42	51	33	40	95%
Airway resistance					
Raw (cmH_2O*second/l)	3.06	3.06	3.06	1.11	36%
sGaw 1/(cmH_2O*second)	0.08	0.08	0.08	0.27	329%

Diffusing capacity	**Predicted**	**Lower limit of normal**	**Pre-bronchodilator**	**Pre-bronchodilator % of predicted**
DLCO_SB ml (minute*mmHg)	23.04	16.94		53%
DLCOcSB ml (minute*mmHg)	23.04	16.94	12.08	52%
DL/VA ml (minute*mmHg)	4.12	3.05	3.08	75%
VIN%VCmax (%)			94%	
VA_SB (l)	5.63	4.51	3.96	70%
Hb g(Hb)/dl			15.00	

FVC: forced vital capacity; FEV1: forced expiratory volume in 1 second; FEV1/FVC: ratio of forced exploratory volume in 1 second to forced vital capacity; FEF 25%-75%: forced expiratory flow between 25% and 75% of the vital capacity; FEF 50%: forced expiratory flow at 50% of the vital capacity; FIF 50%: forced inspiratory flow at 50% of the vital capacity; PEF: peak expiratory flow; FEV0.5/FIV0.5: the ratio of the forced expiratory volume to the forced inspiratory volume in the first half a second; PIF: peak inspiratory flow; TLC: total lung capacity; VCMAX_ pl: the largest measured vital capacity; IC: inspiratory capacity; FRCpleth: forced residual capacity; ERV: expiratory reserve volume; RV: residual volume; RV % TLC: percentage ratio of the residual volume to the total lung capacity; Raw: airway resistance; sGaw: specific airway conductance; DLCO_SB: single-breath diffusing capacity of the lung for carbon monoxide; DLCOcSB: single breath diffusing capacity of lung for carbon monoxide corrected for hemoglobin; DL/VA: the diffusing capacity of the lung for carbon monoxide divided by the alveolar air; VIN%VCmax: the percentage ratio of the volume of inspired air to the largest measured vital capacity; VA_SB: alveolar volume measured in a single breath; Hb: hemoglobin.

any known precipitating factors, resulting in two separate ED visits and multiple visits with the PCP. Multiple imaging studies did not reveal any anatomical precipitants, and the patient had neither undergone any preceding surgical procedures nor did he have any other apparent attributable etiologies for this pathology.

This patient underwent extensive testing to rule out cardiovascular and pulmonary etiologies for his dyspnea.

In the acute setting, ischemia was ruled out by normal cardiac biomarkers and a normal EKG. Metabolic origins for his dyspnea were also excluded with a persistently normal metabolic panel. This patient's blood count was consistently within the normal range for all cell lines, so significant anemia was ruled out as the cause of his symptoms. One would also expect anemia to cause chronic dyspnea, rather than acute-onset dyspnea. The patient had no localizing symptoms to indicate a central neurologic cause such as a stroke, as such, there was no indication for brain imaging. The final diagnosis, however, was a likely peripheral neurologic cause, in the form of phrenic nerve dysfunction causing the right HDP without any known precipitators. An underlying

Table 5. *Timeline of events.*

HISTORY	TIME
Onset of symptoms	January 2022
First ED visit	January 2022
First PCP visit	January 2022
CTPE negative, labs non-contributory	January-February 2022
Antibiotic and steroid course	January-February 2022
Second PCP visit	January 2022
Third PCP visit	February 2022
Second ED visit	February 2022
Fourth PCP visit	February 2022
Started on bronchodilator treatment	February 2022
6-minute walk test, CT scan, PFTs	February-March 2022
Pulmonary medicine referral	April 2022
VQ scan, fluoroscopy	April-May 2022
Diagnosis	May 2022
Referred to thoracic surgery for plication	May 2022

PCP: primary care physician; CTPA: computed tomography pulmonary angiogram; PFTs: pulmonary function tests; VQ scan: ventilation-perfusion scan.

neuromuscular disorder may lead one to consider the diagnosis of HDP in a case of acute dyspnea without apparent etiology, but this patient did not have a history of neuromuscular disease.

Further cardiac testing with a transthoracic echocardiogram and nuclear stress test did not support a cardiogenic culprit. The transthoracic echocardiogram did not reveal any wall motion abnormality or significant valvular pathology that was hemodynamically significant, or that affected the LVEF in such a way that pulmonary compromise would occur. In addition, there was no restrictive pathology surrounding the heart on imaging, such as pericardial effusion, to support an extra-cardiac culprit. This patient also had no other symptoms consistent with heart failure, which could also have been a cardiogenic origin of his dyspnea. Physical examination was equally non-contributory.

A CT angiogram was initially performed and was followed by a VQ scan to confirm the absence of segmental and sub-segmental perfusion defects and ventilation abnormalities. This patient had an extensive smoking history (40 pack years), quitting only a few months before his symptom onset, which could have led one to suspect a pulmonary origin of his symptoms. Pulmonary infectious etiology was initially suspected given findings of right lung base infiltration versus atelectasis. Following a lack of response to appropriate antibiotic therapy, negative respiratory viral tests, and persistent right lung basilar findings, infectious etiologies were ruled out. This patient also had no convincing symptoms to support a persistent or untreated infection, including a pulmonary infection due to atypical organisms such as fungi. The patient consistently had stable vitals, normal cell counts, and no elevation of inflammatory markers. A COPD exacerbation was also ruled out, given a lack of precipitators for the exacerbation, and the patient had received appropriate antibiotic and oral steroid therapy without an appropriate response. In addition, the PFT did not support underlying obstructive pathology and the chest CT was not consistent with underlying COPD. Also, the prolonged, persistent, and gradually worsening dyspnea was not compatible with a COPD exacerbation, as was the lack of any response to, or symptomatic relief from bronchodilator treatment. Although the PFTs were consistent with restrictive pathology, this patient's chest imaging did not support any intrinsic pulmonary pathology as the origin of this restriction or dyspnea.

The patient was overweight (BMI 29.95) but not obese that dyspnea could be attributed to his weight. In addition, the sudden onset of symptoms makes obesity a very unlikely culprit for his dyspnea, as one would expect that obese patients would have chronic baseline dyspnea.

In HDP, PFT, which is more likely to be ordered before fluoroscopy during workup, would reveal a restrictive pattern, with a ratio of forced exploratory volume in 1 second to forced vital capacity (FEV1/FVC) ration of >0.7, decreased inspiratory forces and decreased lung capacity owing to associated atelectasis. If both imaging and baseline PFTs are equivocal, PFTs measured with a change in position may help determine diaphragmatic paralysis as the origin of unexplained dyspnea. We would expect a decrease of >20% in the vital capacity when a patient changes from a standing to a supine position, owing to a combination of a paralyzed diaphragm decreasing the thoracic capacity, and an upward displacement of intrabdominal organs into the chest cavity [7,8]. Performing such a test might be limited by a patient's orthopnea severity. Ultrasound imaging is also an option to further evaluate the diaphragm and may be a useful tool in primary care clinics with the availability of point-of-care ultrasound. Transdiaphragmatic pressure measurements and electrical or magnetic stimulation of the phrenic nerve are tests that could be performed if HDP is suspected but all other tests have been noncontributory; however, these tests are not commonplace and require trained experts to be appropriately performed [9].

Management options for HDP range from more conservative options including pulmonary rehabilitation with diaphragm strength training, to surgical management in the form of pacing or diaphragm plication. Diaphragm plication has been shown to be associated with recovery of diaphragmatic function with time [10-12]. Some physicians might also decide to monitor the patient without any definitive treatment, as some cases may spontaneously resolve [13-15]. Symptomatic management with

non-invasive ventilation to counteract hypercapnia may also be considered. In this case, the patient opted for diaphragmatic plication after 6 months of persistent and significant dyspnea impacting his daily activities. Diaphragm pacing was not an option for the patient as this procedure is typically reserved for bilateral diaphragmatic paralysis. Diaphragmatic pacing in HDP would lead to antagonism between the paced and unpaced diaphragms with resulting dyspnea.

Several cases of HDP have been reported, one attributed to amyotrophic neuralgia [16], another associated with respiratory disease, specifically asthma [17], a case following electrical cardioversion [18], several cases with associated underlying pathologic etiologies [6], and others without any neuromuscular or other etiology discovered [6,9]. Bilateral diaphragmatic paralysis appears to be a more common diagnosis; however, this is likely because 75% of HDP cases are asymptomatic and bilateral palsy is more likely to cause symptoms prompting presentation to a clinician than unilateral paralysis [6]. Some patients with HDP may be asymptomatic, while others may present with varying degrees of respiratory distress. As HDP may be asymptomatic, it may cause the diagnosis in a patient presenting with acute dyspnea to become obscure, especially when it is an idiopathic HDP case. It is uncertain what precipitating factors are associated with idiopathic HDP, or which factors influence the severity of symptoms in patients who present with respiratory distress. Further studies to determine risk factors associated with idiopathic HDP and its severity spectrum may be warranted.

Conclusions

Diaphragmatic paralysis is an often-missed diagnosis in patients with acute onset of dyspnea, especially due to ambiguous imaging findings and generally normal or baseline lab values. Our patient had a diagnosis that was delayed for months (Table 5) due to a lack of preceding etiologic factors for diaphragmatic paralysis. It is important that we keep this diagnosis on our list of differentials, especially after ruling out cardiopulmonary pathologies. It is also important to recognize that some cases of diaphragmatic paralysis may be completely idiopathic and to ensure that this diagnosis does not fail to be considered when a patient has unexplained dyspnea.

What is new?

Diaphragmatic paralysis is a rare cause of acute dyspnea; however, it is commonly associated with underlying pathology that may help lead to a diagnosis. Idiopathic unilateral diaphragmatic paralysis is a rarer presentation. The majority of patients with unilateral diaphragm paralysis are asymptomatic, and an idiopathic etiology makes diagnosis obscure in many patients who are symptomatic, which leads to delayed diagnosis.

List of Abbreviations

HDP Hemidiaphragm paralysis
PCP Primary care physician

Conflict of interest

The authors declare that there are no conflicts of interest to disclose.

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None.

Consent for publication

Written informed consent was ***not*** obtained from the patient for publication of this case report, as this requirement was waived by the institutional review board of the Veteran's Affair Medical Center at Wilkes Barre, Pennsylvania, with the requirement of complete anonymity of the patient and complete de-identification of any patient data published in association with this case report.

Ethical approval

The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures described here were performed in accordance with the ethical standards of the institutional research committee and with the Helsinki Declaration (as revised in 2013). This case report was approved by the institutional review board of the Veteran's Affair Medical Center at Wilkes Barre, PA.

Author details

Ogheneyoma Akpoviroro[1], Jeremy Freiwald[2]

1. Internal Medicine Department, Geisinger Northeast, Wilkes Barre, PA
2. Veteran's Affairs Medical Center, Wilkes Barre, PA

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Summary of the case

1	**Patient (gender, age)**	Male, 71
2	**Final diagnosis**	HDP/unilateral diaphragmatic paralysis
3	**Symptoms**	Acute-onset and progressive dyspnea
4	**Medications**	Short course of methylprednisolone, amoxicillin-clavulanate
5	**Clinical procedure**	Diaphragmatic plication
6	**Specialty**	Internal medicine