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CASE SERIES

Diaphragmatic Eventration Caused by SARS-CoV-2 and Influenza A Virus Infection: Two Case Reports

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Abstract: Diaphragmatic eventration (DE) denotes abnormal elevation of the diaphragm due to paralysis, hypoplasia, or atrophy and is classed as either congenital or acquired. In this report, one case involved a patient with congenital localized DE who developed persistent symptoms such as cough, sputum production, chest tightness, and shortness of breath after infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Imaging studies ultimately revealed progression to complete DE. The patient underwent surgical intervention and recovered well postoperatively. The other case involved a patient who developed chest tightness, shortness of breath, cough, and sputum production after infection with Influenza A virus. Chest CT imaging confirmed localized DE. Although antiviral treatment alleviated the symptoms, the diaphragmatic elevation remained. This study demonstrates that both SARS-CoV-2 and influenza virus infections can potentially damage the phrenic nerve, leading to subsequent diaphragmatic dysfunction. Early recognition and intervention can effectively prevent progression of DE and ensure prompt, effective treatment.

Keywords: diaphragmatic eventration, SARS-CoV-2, influenza A virus, diaphragmatic dysfunction, case report

Introduction

Diaphragmatic eventration (DE) is a rare disease, with an estimated incidence of 1 in 10,000.[1] It is a condition in which the diaphragm becomes abnormally thinner and looser than normal and bulges owing to congenital developmental anomalies or acquired injuries, potentially compromising the respiratory and digestive systems. The core distinction between a normal diaphragm and DE is the abnormal elevation of the diaphragm in the absence of true structural defects. Congenital factors often result from incomplete or absent myogenesis of the pleuroperitoneal membrane during embryonic development, often associated with developmental abnormalities, and some patients may also have chromosomal abnormalities.[2] It is often related to genetic or prenatal environmental factors (such as infection, hypoxia). Genetic predisposition and prenatal environmental factors, such as infection or hypoxia, are commonly implicated. Acquired factors are mostly due to injury to the phrenic nerve, including surgical trauma (such as heart surgery), cervical trauma, tumor invasion, direct cardiac surgery, mediastinal incision, and high cervical spinal cord trauma can cause phrenic nerve paralysis, leading to diaphragmatic relaxation. Infections such as herpes zoster can invade the phrenic nerve. Diaphragm inflammation also occurs. Chronic lung disease and raised intra-abdominal pressure from obesity or pregnancy are additional factors.[3] In clinical practice, acquired causes are more common and affect a broader population than congenital ones. Whether congenital or acquired, localized DE frequently lacks typical clinical symptoms, which can easily lead to delayed diagnosis and treatment.[4]

In this report we described two cases: pre-existing localized DE evolved into complete paralysis after SARS-CoV-2 infection, whereas a previously healthy individual developed focal eventration following influenza A. In both, relentless post-viral cough and systemic inflammation are thought to have altered thoraco-abdominal pressures and injured the

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phrenic nerve, underlining that both viruses can precipitate diaphragmatic dysfunction. This study demonstrates that SARS-CoV-2 and influenza infections can damage the phrenic nerve; timely diagnosis and treatment prevent progression of diaphragmatic dysfunction and improve outcome.

Case Presentation

This report has been approved by the ethics committee of the affiliated people's hospital of Ningbo University (No: 2025-N-012). Two patients both provided written informed consent for the publication of medical data.

Case 1

In February 2023, a 74-year-old female was admitted to our hospital due to intermittent cough for 2 months, chest tightness and abdominal distension for over 1 month. Two months before admission, the patient was infected with SARS-CoV-2 and experienced persistent coughing, especially at night, which she found intolerable. She self-medicated with cough suppressants but did not experience significant improvement. One month before admission, she developed continuous chest tightness and abdominal distension, with symptoms worsening in the supine position and alleviating in the sitting position. The patient had no history of other non-communicable diseases.

Through medical history inquiry, it was found that the patient had undergone a physical examination at the local hospital one year ago, and the chest X-ray showed that her left diaphragm was elevated to the level of the seventh posterior rib (Figure 1A). As the patient had no complaints at that time, no special treatment was given, and she was

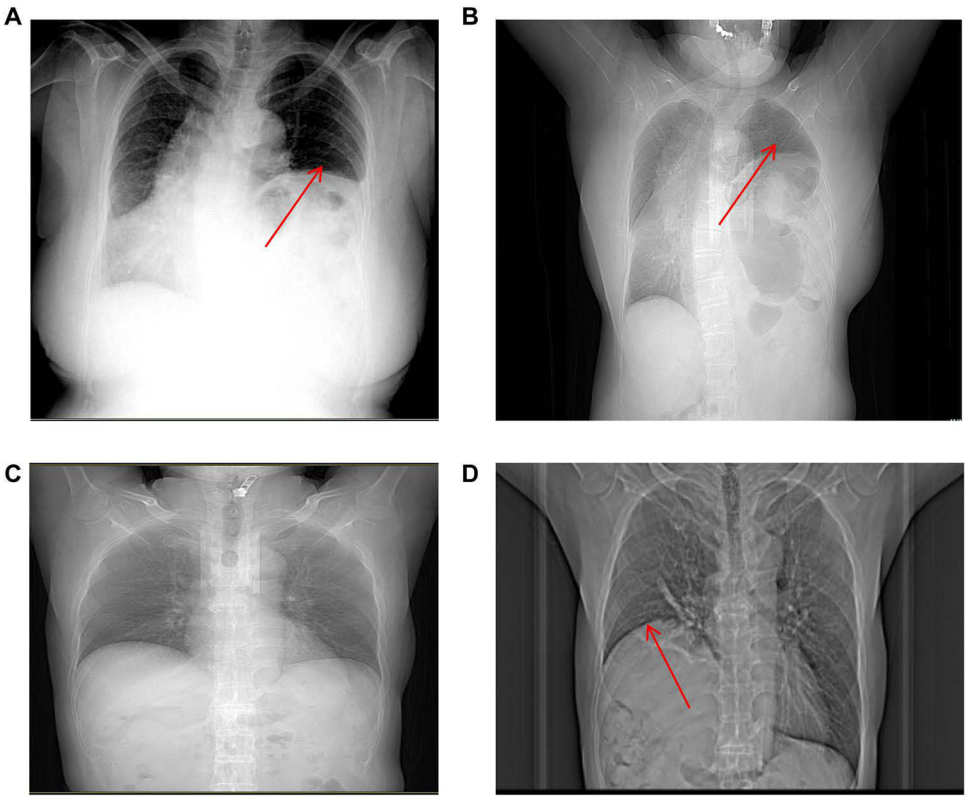

Figure 1 The chest x-ray results for the two patients. (**A**) For patient 1, a chest X-ray in June 2022 showed that the left diaphragmatic surface was significantly elevated (as the red arrow pointed), extending to the level of the seventh posterior rib; The mediastinum shifts to the right, and the right diaphragmatic surface undergoes a complete thoracolumbar scoliosis change; (**B**) For patient 1, a chest X-ray in February 2023 revealed that the left diaphragm was significantly elevated, extending upwards to the fourth posterior rib (as indicated by the red arrow), the contents of the left abdominal cavity moved upward, the mediastinum shifted to the right, and the right diaphragm surface underwent a change in thoracolumbar scoliosis; (**C**) For patient 2, in October 2024, no abnormalities were found in the chest X-ray examination; (**D**) For patient 2, a chest X-ray in February 2025 showed that the trachea was centered, the thoracic cage was symmetrical, and the right diaphragm partially protruded and extended to the fourth posterior rib (Red arrow).

diagnosed with localized DE. The patient had no history of trauma to the phrenic nerve or diaphragm, no surgical history, and no history of mechanical ventilation.

The patient's vital signs, clinical symptoms, and laboratory results at admission are presented in Table 1. The chest X-ray showed that the left diaphragm was significantly elevated, extending upward to the fourth posterior rib (Figure 1B). In addition, the chest CT scan showed that the trachea was deviated to the right, the left lung was compressed, and the mediastinum was shifted to the right, with abdominal contents displaced upward, and the diaphragm was continuous without disruption (Figure 2A). The patient was ultimately diagnosed with complete left DE. After medical treatment, the patient's symptoms did not improve, and she was transferred to the thoracic surgery department for diaphragmatic plication.[5] The patient's condition was stable after surgery, and she was discharged one week later. During the six-month follow-up, the patient did not report any significant discomfort.

Case 2

In January 2025, a 61-year-old male was admitted to our hospital due to persistent fever with chest tightness and shortness of breath for one week. One month ago, the patient was treated at a local hospital for fever with productive cough, and nucleic acid test subsequently confirmed influenza A infection.

On admission, the patient's general condition, clinical symptoms, and laboratory results were presented in Table 1. The chest CT images revealed patchy opacities in the lower lobe of the right lung and partial elevation of the right diaphragm extending to the level of the fourth posterior rib (Figure 1D). No diaphragmatic rupture or destruction was observed (Figure 2B). The right lung was compressed, and no significant mediastinal displacement was noted. By inquiring about the patient's past medical history and reviewing previous imaging data, no diaphragmatic abnormalities were found in the past (Figure 1C). The patient had no history of congenital disorders, surgery, or trauma. The respiratory

Table 1 The Baseline Characteristics and Clinical Test Results for the Two Patients

Variable	Patient 1	Patient 2	Normal Range
General condition			
Age (Years)	74	61	
Sex	Female	Male	
Pulmonary disease	Bronchial asthma	Bronchiectasis	
Clinical symptoms			
Chest tightness	Yes	Yes	
Abdominal distension	Yes	No	
Cough and sputum	Yes	Yes	
Fever	No	Yes	
Blood pressure (mmHg)	121/78	141/89	<140 / <90
Laboratory results			
RBC (*10^12/L)	4.98	5.18	4.3–5.8
WBC (*10^9/L)	7.3	9.6	4.0–10
Neutrophil percentage (%)	70.2	77.6	40-75
Lymphocyte percentage (%)	24	21.5	20-40
CRP (mg/L)	3.44	13.3	<8
Procalcitonin (ng/mL)	0.01	0.03	<0.05
Culture tests	/	/	
PCR test	SARS-CoV-2	Influenza A virus	
Blood gas analysis			
PaO2 (mmHg)	90	95	80-100
PaCO2 (mmHg)	41	36	35-45
Pulmonary function			
FEV1 (L)	1.93	2.47	
MVV (L/min)	38.37	65.15	
DLCO (mmol/min/kPa)	4.98	6.42	

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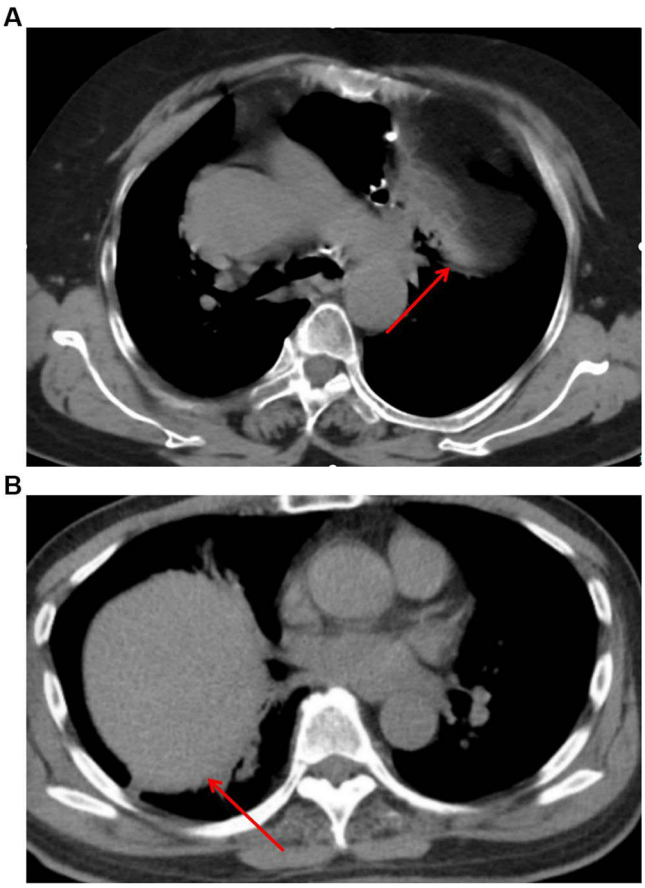

Figure 2 The chest CT results for the patients. (**A**) For patient 1, a chest CT scan in February 2023 showed that the thoracic cage was symmetrical, the trachea was deviated to the right, the left lung was compressed, the mediastinum was displaced to the right, and the diaphragm was continuous and intact (Red arrow); (**B**) For patient 2, in February 2025, a chest computed tomography scan showed that the trachea was centered, the thoracic cage was symmetrical, and the right diaphragm was partially protruded, with intact diaphragm continuity (Red arrow).

power of the right lung was slightly weakened, while the respiratory sounds of the left lung were normal, and no obvious dry or wet rales were heard. The abdomen was soft, and there was no edema in the lower extremities. The patient was eventually diagnosed with right-sided localized DE.

The patient was then treated with 0.5 g of nemonoxacin injection per dayfor anti-infection, cough suppression, and asthma relief. After consultation with the surgical department, considering that the patient's current symptoms of chest tightness and shortness of breath had improved, and there were no respiratory or digestive system disorders, conservative treatment was temporarily adopted, and surgery was not required. The patient improved and was discharged. During the 6-month outpatient follow-up, the patient's general condition was good, and the symptoms of chest tightness and shortness of breath were stable A re-examination of the chest CT showed that the position of the right diaphragm was relatively fixed and did not further elevate.

Discussion

The diaphragm is the primary muscular structure separating the thoracic and abdominal cavities. Under normal conditions, the dome of each hemidiaphragm typically projects anteriorly to the level of the 5th or 6th rib and posteriorly to the 10th rib. DE occurs significantly more frequently on the left side than the right (8:1 to 9:1), likely due to the presence of the liver and associated portal structures providing protective support on the right side.[1]

Diaphragmatic elevation refers to a condition where the diaphragm becomes partially or completely raised into the thoracic cavity due to hypoplasia, paralysis, or external factors. This abnormal positioning can affect the normal anatomy and function of both thoracic and abdominal organs, potentially leading to migration of abdominal contents into the

thoracic cavity.[6] As the diaphragm receives both motor and sensory innervation from the phrenic nerve, any damage to this nerve may result in weakened diaphragmatic contraction and subsequent elevation.[7] However, unilateral impairment typically does not cause significant respiratory dysfunction in healthy individuals, as each hemidiaphragm is independently innervated by its own phrenic nerve (C3-C5). The unaffected hemidiaphragm and accessory respiratory muscles can often compensate for unilateral paralysis.[8] This compensation may be compromised in patients with comorbidities or pre-existing pulmonary conditions. Notably, infection represents a key predisposing factor for DE.

In our case series, we observed unilateral DE following both COVID-19 and influenza virus infections, a phenomenon also documented with other viral illnesses. Existing research has established associations between unilateral diaphragmatic paralysis and various viral infections including herpes zoster, Zika virus, poliovirus, among others. Notably, a Zika virus case series reported four infants with diaphragmatic weakness who ultimately died of respiratory failure.[9] Becroft documented a congenital DE case linked to cytomegalovirus infection,[10] while Mitsiakos et al reported a female neonate with congenital DE associated with parvovirus B19 infection.[11] Emerging evidence further supports the link between COVID-19 and DE caused by phrenic-nerve injury.[12,13]

Our report presents two distinct cases: one following SARS-CoV-2 infection and another after influenza A virus infection. Laboratory tests in both patients revealed only restrictive ventilatory dysfunction of varying degree; an elevated C-reactive protein attributable to fever was present solely in the second case. These findings indicate that the respiratory infection itself was mild and that virus-induced DE is the principal cause of impaired respiratory function. Since the pandemic onset, neurological manifestations of COVID-19 have been frequently reported, including Guillain-Barré syndrome and myasthenia.[14] Pioneering studies of Wuhan cohorts highlighted the prevalent neurological symptoms affecting muscular function and motor control in COVID-19 patients.[15–18] Subsequent reports have increasingly recognized SARS-CoV-2's neurotropism, including cranial nerve involvement, suggesting a capacity for systemic neural invasion.[19,20] Mechanistically, upregulated ACE-2 expression in infected tissues may facilitate viral neuronal entry and diaphragmatic injury.[21,22] Additionally, SARS-CoV-2-induced immune activation can mediate both direct and immune-mediated muscle damage,[23] exacerbating respiratory dysfunction. Our findings suggest these neurological manifestations may result from direct viral neuroinvasion. Abdeldayem et al's study of 1527 COVID-19 patients identified 23 cases of unilateral diaphragmatic paralysis, with 21 achieving complete recovery,[24] indicating a potentially reversible neuropathic process similar to Bell's palsy.[25] However, not all cases resolve spontaneously; chronic diaphragmatic dysfunction may necessitate surgical intervention. While Ribet et al advocate early surgical correction for all diagnosed eventrations,[26] others reserve surgery for cases with mediastinal shift or respiratory failure.[27]

Notably, this report documents the first known progression from congenital localized to complete DE following COVID-19 infection. This novel association enhances our understanding of SARS-CoV-2's pathogenic mechanisms and warrants further investigation. Clinically, our findings emphasize the importance of monitoring diaphragmatic function in COVID-19 patients, particularly those with congenital diaphragmatic abnormalities, to guide timely intervention and improve outcomes. This insight provides new diagnostic and therapeutic considerations for optimizing management and reducing complication risks in this patient population.

Currently, there are no reported cases of DE caused by influenza A virus infection, and the underlying mechanisms remain unclear. Based on available literature, influenza virus may potentially invade the central nervous system through peripheral nerves or induce cytokine storms in the CNS by compromising the blood-brain barrier.[28] While cytokines play crucial roles in viral clearance and intercellular communication within the immune system, excessive cytokine production can lead to severe immunopathological damage.[29] Supporting this, Daiva et al[30] found significantly elevated serum levels of interleukin-6 (IL-6), IL-10, and tumor necrosis factor-alpha (TNF-α) in influenza patients compared to uninfected controls or those without neurological complications. These cytokines not only mediate local inflammatory responses but may also adversely affect neural tissues, potentially representing a key mechanism of influenza-associated neural injury.

Building upon these findings, we hypothesize that influenza A virus may similarly damage the phrenic nerve. Additionally, the virus may infect respiratory epithelial cells, triggering widespread inflammation that leads to alveolar structural damage, epithelial cell desquamation, and impaired gas exchange, ultimately resulting in respiratory failure. To compensate, respiratory muscles (including the diaphragm) undergo sustained overuse, maintaining prolonged tonic contraction that may lead to diaphragmatic fatigue and dysfunction, manifesting as diaphragmatic elevation. The

inflammatory response and systemic stress induced by influenza infection may cause microvascular spasm and constriction, compromising blood supply to the phrenic nerve. This ischemia deprives neural tissue of oxygen and essential nutrients (eg, glucose, amino acids) while allowing metabolic waste accumulation, creating a state of cellular "starvation" and "intoxication". Progressive neural dysfunction may impair phrenic nerve conduction, reduce diaphragmatic motility and causing elevation.

Persistent post-influenza cough represents another indirect factor contributing to diaphragmatic elevation. Violent coughing alters thoracoabdominal pressure gradients, inducing diaphragmatic spasms during repetitive contractions. Reduced diaphragmatic tone, combined with sustained increases in intra-abdominal pressure, may lead to diaphragmatic displacement. In severe cases, cough-induced strain may even cause diaphragmatic rupture and hernia formation.[31,32]

While COVID-19-associated phrenic-nerve injury leading to progression from localized to complete DE has not been previously reported, our study also reveals a potential association between influenza A virus and DE, a novel observation not previously documented in the literature. This discovery advances our understanding of the pathogenic mechanisms underlying influenza-related diaphragmatic dysfunction. For clinicians managing influenza patients with prolonged cough and dyspnea, these findings suggest expanding diagnostic considerations beyond primary respiratory symptoms. A more comprehensive approach incorporating diaphragmatic evaluation may improve rapid clinical assessment and treatment optimization, ultimately reducing adverse outcomes. However, both cases involved elderly patients and only a single instance per virus; extrapolation of these findings and management principles to younger populations requires further study. Whether to intervene early in asymptomatic, localized DE accompanying SARS-CoV-2 infection remains an open question which should be addressed in future prospective studies.

Conclusion

The presented reports demonstrate that SARS-CoV-2 and influenza can result in DE via phrenic-nerve injury. When post-viral cough, chest tightness, or abdominal discomfort persist, clinicians should screen for diaphragmatic dysfunction early, confirm it with imaging, and institute tailored therapy—antivirals, respiratory rehabilitation, or plication—before irreversible deterioration occurs.

Ethics Statements

The study was conducted in accordance with the Declaration of Helsinki. All procedures performed in the study involving human participants were in conformity to the ethical standards of the Ethics Committee of the Affiliated People's Hospital of Ningbo University. Ethics approval was not required by the local ethics committee, as this is a case report with anonymized details. Written informed consent for publication of the clinical details and clinical images was obtained from the patient.

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Disclosure

The authors report no conflicts of interest in this work.

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