

A Case Series on Unilateral Elevation of Diaphragm

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ABSTRACT

We report a case series of seven patients aged 24 to 78 years who presented with unilateral elevation of the diaphragm secondary to diverse underlying mechanisms. The diagnoses included sarcoidosis-related pulmonary inflammation, gallbladder adenomyomatosis with sepsis and metabolic derangement, pulmonary tuberculosis with Chilaiditi's syndrome, acute pulmonary embolism with diaphragmatic eventration, superior vena cava syndrome due to non-small cell lung carcinoma, cryptogenic organizing pneumonia with an incidental thymic cyst and pyogenic liver abscess. This series emphasizes that unilateral diaphragmatic elevation should not be attributed only to phrenic nerve injury or primary diaphragmatic paralysis; pulmonary, abdominal, vascular, mediastinal and inflammatory causes should also be systematically evaluated. Early identification and treatment of the primary disorder resulted in clinical improvement and radiological normalization in most reversible cases.

Keywords: unilateral, diaphragm, elevation, copd.

INTRODUCTION

The normal position of the diaphragm, comprising the right and left hemidiaphragms, shows a slight asymmetry, with the right hemidiaphragm typically positioned one intercostal space higher

than the left on an erect frontal chest X-ray (1). When this difference exceeds 3 cm, or when the left hemidiaphragm lies higher than the right, the condition is termed *unilateral elevation of the diaphragm*. This finding warrants further evaluation, as it represents an abnormal presentation associated with multiple potential etiologies.

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Unilateral elevation of the diaphragm may be classified according to physiopathological mechanisms rather than considered as a single diagnostic entity. The major categories include: (i) neuromuscular causes, such as phrenic nerve palsy, cervical spine disease, neuropathy, or primary diaphragmatic paralysis; (ii) pulmonary and pleural causes, including lower-lobe collapse, fibrosis, tuberculosis, organizing pneumonia, pleural effusion, or chronic obstructive pulmonary disease-related diaphragmatic dysfunction; (iii) subdiaphragmatic and abdominal causes, such as liver abscess, hepatobiliary inflammation, abdominal masses, colonic interposition, or subphrenic collections producing mechanical elevation or inflammatory restriction; (iv) mediastinal and thoracic mass lesions causing compression or traction; and (v) vascular or postoperative causes, including pulmonary embolism, eventration, or structural weakness after surgery. This mechanism-based classification improves clinical application because it directs the physician toward targeted respiratory, abdominal, vascular and neurological evaluation rather than a simple list of differential diagnoses (2-4).

We present a series of seven cases in which different physiopathological mechanisms were identified as causes of unilateral diaphragmatic elevation. This case series provides practical insights for primary care physicians, who are often the first point of contact for patients presenting with breathlessness, chest discomfort, abdominal symptoms, sepsis, or unexplained radiographic abnormalities. Unilateral elevation of the diaphragm is frequently overlooked or misattributed to benign anatomical variation, leading to delays in appropriate referral and treatment. By highlighting a structured mechanism-based approach and a spectrum of pulmonary, abdominal, vascular, mediastinal, and inflammatory etiologies, this series equips primary care providers with a broader and more clinically applicable diagnostic framework. □

CASE SERIES

In this series of seven cases, diaphragmatic elevation was identified as a secondary radiological sign linked to diverse underlying conditions,

with patients ranging in age from 24 to 78 years (mean 51 years) and a slight male predominance (four males, three females). The etiologies included pulmonary inflammatory and infectious diseases (sarcoidosis, tuberculosis, cryptogenic organizing pneumonia), abdominal pathologies causing mechanical displacement (liver abscess, gallbladder adenomyomatosis, Chilaiditi's syndrome), mediastinal masses (non-small cell lung carcinoma, thymic cyst) and vascular compromise (pulmonary embolism). Comorbidities such as hypertension, diabetes, chronic obstructive pulmonary disease and prior pulmonary infections were present in several patients, potentially influencing both presentation and recovery (Table 1).

Across the seven cases, chest X-ray consistently revealed unilateral diaphragmatic elevation, more often on the right side, sometimes accompanied by ipsilateral basal opacities, volume loss, or mediastinal shift. In several patients, X-ray also showed associated features such as blunting of the costophrenic angle, raised gastric bubble, or overlying bowel gas suggestive of Chilaiditi's sign. High-resolution computed tomography (HRCT) provided detailed characterization of the underlying pathology – identifying basal fibrosis, consolidations, cavitary lesions, mediastinal masses, pleural effusions, or eventration – and confirmed the absence or presence of diaphragmatic thickening or defect; it also helped differentiate between pulmonary, abdominal and mediastinal causes by visualizing contiguous structures, thereby guiding targeted management.

In five reversible cases (Cases 1, 2, 3, 6 and 7), timely treatment of the primary cause through antibiotics, corticosteroids, drainage, bronchodilators, or supportive care led to symptom resolution and improvement or normalization of diaphragmatic position. In the malignant case (Case 5), only partial relief was achieved, while the acute vascular event (Case 4) had a fatal outcome despite anticoagulation and supportive care. The representative images selected for publication are directly related to the case series and demonstrate the principal imaging patterns observed across the cases (Figures 1-4). □

TABLE 1. Characteristics of study patient

Case	Age	Gender	Comorbidities	Key investigation findings	Diagnosis	Treatment
1	24	Male	None	CXR: left diaphragmatic elevation, B/L hilar lymphadenopathy; HRCT: calcified nodules, cicatricial atelectasis; serum ACE↑; PFT: restrictive pattern	Sarcoidosis with pulmonary inflammation	Oral prednisone taper
2	51	Female	Type 2 DM, hypertension	Labs: AKI, hyponatremia, hypokalemia; CXR: right diaphragmatic elevation; USG: gallbladder wall thickening with adenomyomatosis; ABG: metabolic + respiratory acidosis	Pre-renal AKI due to severe dehydration, sepsis, gallbladder adenomyomatosis, secondary hypoparathyroidism	IV fluids, antibiotics, supportive care
3	60	Male	Type 2 DM	CXR: right diaphragmatic elevation, Chilaiditi's sign; HRCT: tree-in-bud nodules, fibrotic streaks; sputum AFB +; Mantoux +	Pulmonary tuberculosis with Chilaiditi's syndrome	Anti-tubercular therapy
4	60	Female	Femoral neck fracture (immobilization)	CXR: right diaphragmatic elevation, mediastinal shift; D-dimer↑; HRCT: eventration with lung collapse; ABG: acute respiratory alkalosis	Acute pulmonary embolism with diaphragmatic eventration	Anticoagulation (Enoxaparin), supportive care (fatal outcome)
5	78	Male	COPD, chronic smoker	CXR & CECT: anterior mediastinal mass obliterating SVC, right diaphragmatic elevation; polycythemia	SVC syndrome from NSCLC (Grade II squamous cell carcinoma)	SVC stenting, symptomatic management
6	54	Female	H/o pulmonary TB	CXR: left diaphragmatic elevation; CECT: cryptogenic organizing pneumonia, thymic cyst; USG: mild pleural effusion	Cryptogenic organizing pneumonia with incidental thymic cyst	Antibiotics, steroids, bronchodilators, conservative cyst management
7	30	Male	None	CXR: right diaphragmatic elevation; CECT: large segment VIII liver abscess compressing diaphragm; culture: <i>Streptococcus milleri</i>	Pyogenic liver abscess with diaphragmatic elevation	Intravenous antibiotics, percutaneous drainage



FIGURE 1. Case 1 chest radiographs: (A) pre-treatment posteroanterior chest X-ray showing left hemidiaphragm elevation with crowding of ribs, mild mediastinal shift and bilateral hilar lymphadenopathy; (B) follow-up chest X-ray after treatment showing improvement and near-normalization of the left hemidiaphragm position

DISCUSSION

This case series is one of the first to comprehensively describe the diverse etiologies of unilateral diaphragmatic elevation, supported by

clinical details and imaging across multiple conditions. The spectrum of causes encountered in the series highlights that unilateral hemidiaphragm elevation is not a diagnosis in itself but a

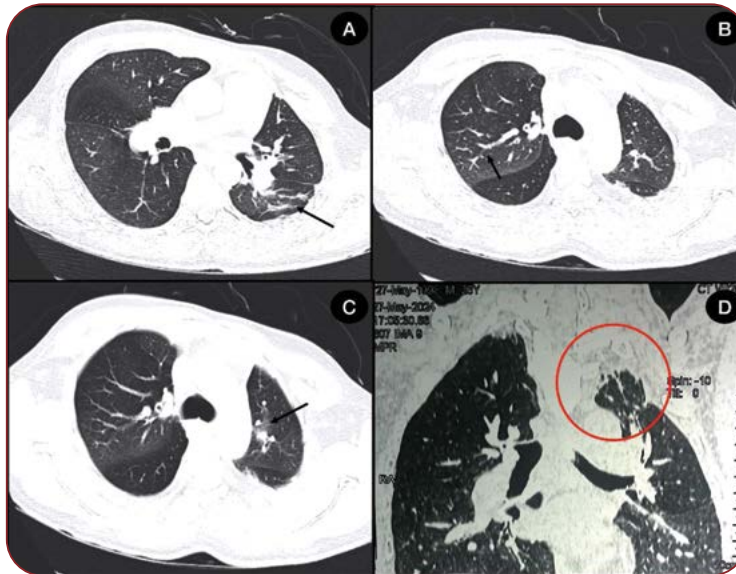


FIGURE 2. Case 1 HRCT chest images showing associated parenchymal findings rather than the diaphragm level: (A) fibrotic streak in the superior segment of the left lower lobe (black arrow); (B) fibrotic streaks in the apical segment of the right upper lobe (black arrow); (C) fibrotic streaks in the apico-posterior segment of the left upper lobe (black arrow); and (D) cicatricial atelectasis of the anterior segment of the left upper lobe (red circle)

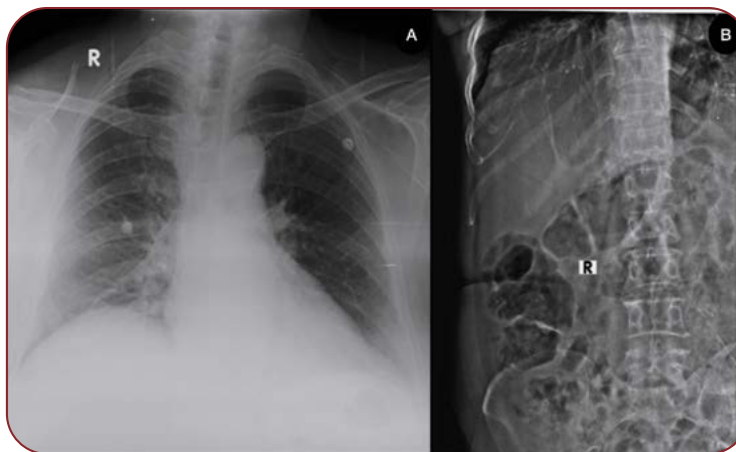


FIGURE 3. Case 3 radiographs: (A) chest X-ray (PA view) showing right hemidiaphragm elevation; and (B) erect abdominal radiograph demonstrating colonic interposition beneath the right hemidiaphragm, supporting Chilaiditi's sign/syndrome in the clinical context

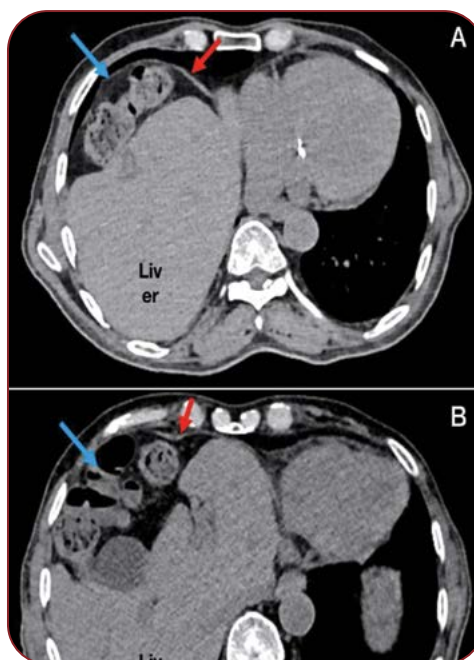


FIGURE 4. Case 3 computed tomography abdomen, axial views (A, B), showing colonic hepato-diaphragmatic interposition (blue arrows) with elevation of the right diaphragmatic dome/eventration (red arrows).

radiological sign with varied underlying systemic, infectious, neuromuscular and abdominal origins.

The first case involved a 24-year-old male with sarcoidosis, a systemic granulomatous disorder of unknown etiology characterized by non-caseating granulomas. Although sarcoidosis rarely affects respiratory muscles, previous studies by Schreiber *et al* (5) and Buerschen *et al* (6) have described isolated instances of unilateral diaphragmatic elevation associated with it. Diagnosis may be challenging because respiratory muscle weakness is often mild, making markers such as serum IL-2 receptor and neopterin helpful. Importantly, surgical plication should be avoided unless sarcoidosis is definitively excluded (5).

Gallbladder and hepatobiliary conditions represented an important abdominal mechanism of right hemidiaphragm elevation. In the present series, gallbladder adenomyomatosis with sepsis and metabolic derangement was associated with right diaphragmatic elevation, while previous literature has described acute cholecystitis as a similar mechanism through focal peritonitis or subphrenic inflammation that restricts diaphragmatic movement. Our findings are consistent with Yee *et al* (7), who reported right hemidiaphragm elevation as an important radiographic clue in gallbladder inflammation. Ultrasonography is therefore essential when right-sided diaphragmatic elevation is accompanied by abdominal pain, sepsis, or abnormal hepatobiliary findings (7).

Diaphragmatic eventration represented another etiology, as seen in a 60-year-old male with tuberculosis. Eventration involves congenital or acquired thinning of diaphragmatic muscle without structural discontinuity (8). The past literature, including works by Morrison and Calvino *et al* (9, 10), notes traumatic, idiopathic and congenital origins, though tuberculosis-associated cases have also been reported. Studies by Ogbole (11) and Singh *et al* (12) suggest a possible association between diaphragmatic contour abnormalities and previous tuberculosis, likely related to chronic structural changes. Paudel K *et al* (13) found that, in a 72-year-old woman, chest X-ray demonstrated elevated right hemidiaphragm as well as a radio-opaque lesion on the right hemithorax. The patient had a history of tuberculosis infection 25 years ago and the present CT scan showed multiple calcified lesions in the hemithorax. These lesions, which were situated near the phrenic nerve, could be a contributing factor to diaphragmatic paralysis. Cases of diaphragmatic eventration in individuals with a history of tuberculosis have been documented in a case report on a 63-year-old male patient by Ogunkoya JO *et al* (14), indicating a possible connection between the two conditions.

Acute pulmonary embolism (PE) was identified in two patients, consistent with earlier reports by Zubairi *et al* and other authors showing elevated diaphragms in a subset of PE cases

(15, 16). The mechanism may involve lower-lobe infarction or diaphragmatic dysfunction due to vascular compromise. As chest radiographs lack specificity, confirmation via fluoroscopic sniff test strengthens diagnostic accuracy (17).

Diaphragmatic dysfunction secondary to chronic obstructive pulmonary disease (COPD) also emerged as an etiology. In one patient, non-small cell lung cancer (NSCLC) was additionally identified on histopathology. Chronic obstructive pulmonary disease is known to cause diaphragm muscle atrophy through systemic inflammation, oxidative stress, hyperinflation and activation of proteolytic pathways such as NF- κ B. Imaging modalities, including ultrasound, CT, electromyography (EMG) and nerve stimulation, play an essential role in evaluating diaphragmatic performance in COPD and guiding rehabilitation and weaning strategies (18, 19).

Abdominal conditions, particularly liver abscess, were also implicated. In a 30-year-old male, ultrasonography confirmed a large hepatic abscess corresponding to right hemidiaphragm elevation on chest radiograph. Prior literature, such as the case reports by Wang *et al* (20), describes similar presentations and potential complications including pleural extension or empyema when abscesses are subdiaphragmatic.

Mediastinal and thoracic pathology should also be considered. In the present series, a patient with non-small cell lung carcinoma developed superior vena cava syndrome with right diaphragmatic elevation, while another patient had cryptogenic organizing pneumonia with an incidental thymic cyst. These cases emphasize that mass effect, traction, basal lung disease and associated pleural or mediastinal abnormalities may all contribute to hemidiaphragm elevation and should be evaluated with cross-sectional imaging when chest radiography is inconclusive.

Overall, this case series shows the importance of considering a broad differential diagnosis for unilateral diaphragm elevation in a primary care setup. Early recognition of these conditions can significantly improve patient outcomes, as timely evaluation and targeted management have been shown to restore normal diaphragmatic position in most cases. Therefore, this case series enhan-

ces diagnostic vigilance in primary care settings and promotes more accurate triage, reducing the risk of missed or delayed diagnoses. □

CONCLUSION

Unilateral elevation of the diaphragm can result from various local pathologies involving the lungs, liver, or gallbladder. Once this radiological finding is identified, a thorough evaluation for these underlying conditions is essential. Most importantly, true pathological elevation must be distinguished from normal hemidiaphragm variation. This radiological sign should

not be overlooked, as it carries a wide range of diagnostic possibilities allowing for improved outcomes of patients. □

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