



Imaging of Pulmonary Manifestations in Chronic Kidney Disease: A Review

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Abstract

Keywords

- ▶ chronic kidney disease
- ▶ pulmonary
- ▶ pulmonary edema
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- ▶ CT
- ▶ pulmonary infection

Lungs and kidneys share a symbiotic relationship in maintaining homeostasis of body. Hence, derangement of one system is bound to affect the functioning of the other. The thoracic manifestations of chronic renal failure present a wide spectrum ranging from problems related to fluid and salt balance, calcium–phosphate metabolism, compromised immunity, and additional issues related to different modes of dialysis. In most of the cases, chest radiograph coupled with ultrasound and computed tomography (CT) are sufficient to offer a definitive diagnosis. This review aims to summarize the imaging features of thoracic manifestations of chronic kidney disease (CKD) with emphasis on imaging-based discriminating features.

Lungs and kidneys share a close symbiotic relation, and often complement each other's function in maintaining homeostasis. Derangement of one system inevitably effects the functioning of the other, and vice versa. Renal disease is rapidly emerging as a public health crisis, with about 843.6 million cases of chronic kidney disease (CKD) documented globally in 2019.¹

The radiological manifestations of thoracic complications of CKD present an interesting spectrum of protean features, encompassing problems related to disturbance of fluid and solute balance, calcium–phosphate homeostasis, compromised immunity, and additional complications related to dialysis treatment. In most of the cases, clinical details along with chest radiograph and ultrasound, complemented with computed tomography (CT), are sufficient to arrive at a definitive diagnosis, without the need for invasive sampling. Occasional use of nuclear medicine techniques is deemed necessary, mainly in equivocal cases, as a problem-solving tool. This review aims to revisit the thoracic complications of

CKD patients, with emphasis on imaging-based key differentiating features.

For the ease of tabulation, thoracic manifestations of CKD can be broadly divided into pulmonary, pleural/pericardial, and thoracic cage–related changes and are enlisted as in ▶**Table 1** although entities can have manifestations in multiple compartments with predominance in one or the other.

The discussion shall proceed with issues related to volume redistribution followed by those due to compromised immunity, imbalance in calcium–phosphate metabolism, uremia-induced specific conditions including lung dysfunction and, finally, dialysis-related complications including iatrogenic causes. Although a broad spectrum of other entities can also involve lung and kidneys simultaneously, detailed discussions of thoracic imaging manifestations of such “pulmonary-renal syndromes” or thoracic complications of nephrotic syndrome are beyond the scope of the present article and, hence, have been omitted.

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Table 1 Thoracic complications of chronic kidney disease

	Manifestations	Pathophysiology
Parenchymal	Pulmonary edema	Fluid and salt imbalance
	Infection	Compromised immunity
	Metastatic pulmonary calcification	Calcium–phosphate dysmetabolism
	Uremic pneumonitis	Diffuse alveolar damage
	Diffuse alveolar hemorrhage	Pulmonary-renal syndrome
	Basal atelectasis (following CAPD)	Raised intra-abdominal pressure in CAPD
Pleural/pericardial	Pulmonary edema	Fluid and salt imbalance
	Infection (tubercular) Parapneumonic effusion (bacterial)	Compromised immunity
	Uremic pleuropericarditis	Uremia-induced injury
	Pleural effusion (following CAPD)	Diaphragmatic leak/pleuroperitoneal fistula
Thoracic cage	Subperiosteal bone resorption Rugger jersey spine	Renal osteodystrophy
	Glenohumeral joint arthropathy, periarthrits	Dialysis-related amyloidosis

Abbreviation: CAPD, continuous ambulatory peritoneal dialysis.

Parenchymal Manifestations

Pulmonary Edema

In patients with CKD stage V requiring dialysis, pulmonary edema may arise due to a complex interplay of several factors affecting the hemodynamics. Although the pathophysiology is poorly understood, proposed mechanism of pulmonary edema in CKD is depicted in ►Fig. 1.²

Chest Radiograph

Chest radiograph (CXR) offers an inexpensive and accurate imaging modality for early assessment of pulmonary ede-

ma and allows easy follow-up after treatment. It was initially proposed by Milne et al³ that cardiogenic pulmonary edema due to left ventricular failure (LVF) could be differentiated from renal edema on the basis of the CXR. They proposed that renal edema typically presents with central batwing distribution representative of alveolar edema, and when present, this pattern identifies renal edema with an accuracy of about 80% (►Fig. 2A). However, subsequent studies showed that not all renal edemas present with the “typical batwing distribution,” nor is this pattern specific to pulmonary edema of renal origin.^{4,5} In fact, studies with CT have shown that pulmonary edema

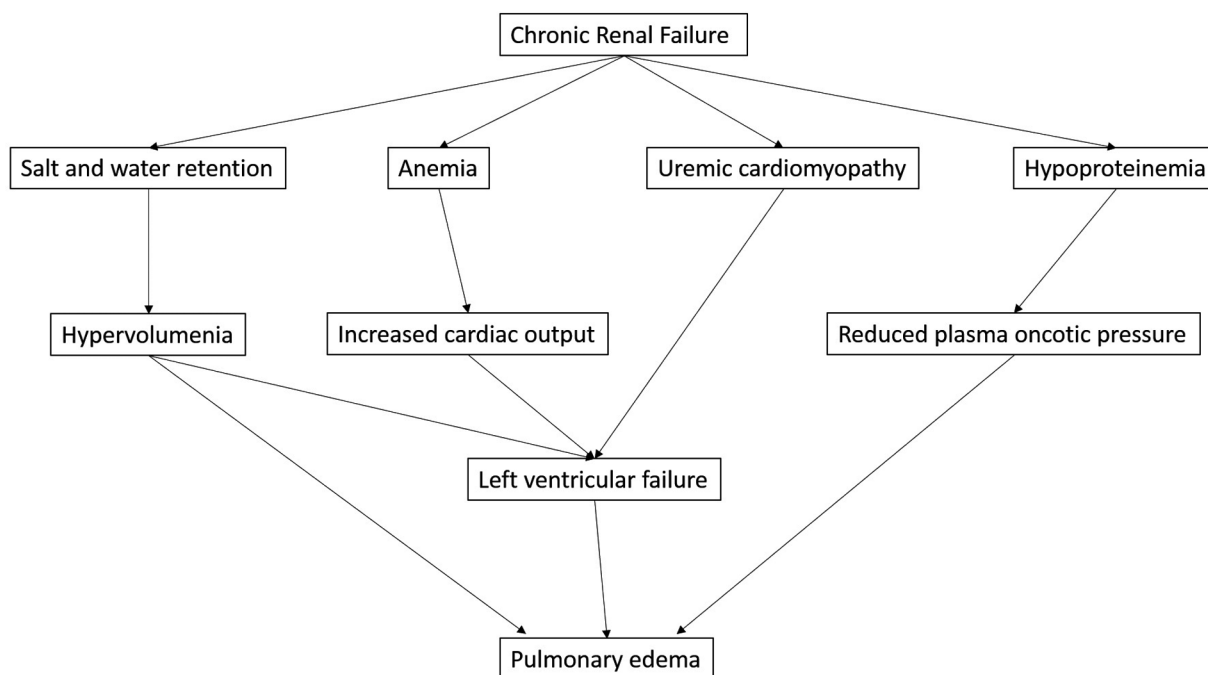


Fig. 1 Flowchart showing the pathophysiology of the proposed mechanism of pulmonary edema in chronic kidney disease (CKD).

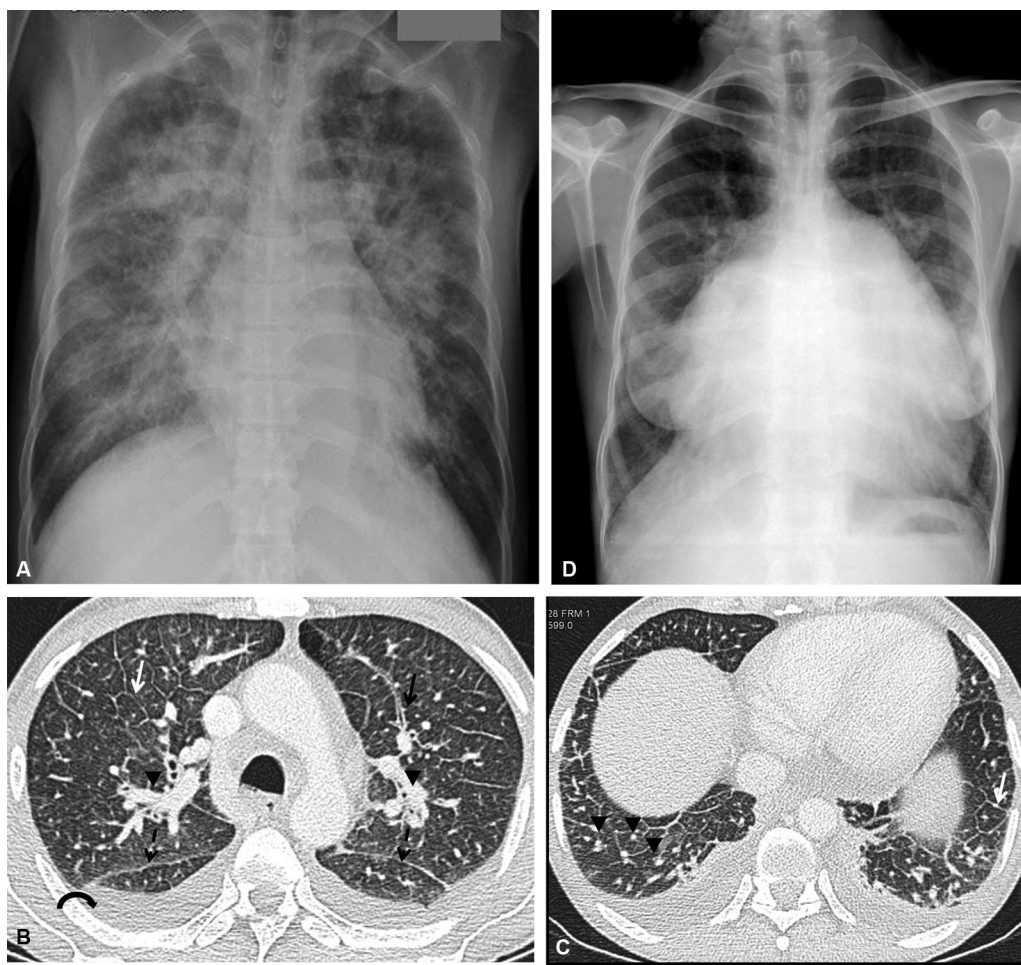


Fig. 2 (A) Central batwing distribution of pulmonary edema in renal failure. Frontal chest radiograph (CXR) in 27-year-old man with chronic kidney disease (CKD) on dialysis shows bilateral perihilar ill-defined fluffy alveolar opacities with relative peripheral sparing and normal cardiac size. (B,C) Computed tomography (CT) features of pulmonary edema (different patient). Axial high-resolution CT (HRCT) chest images in the lung window of a 45-year-old lady with CKD shows bilateral diffuse smooth interlobular septal (white arrows) and peribronchovascular thickening (black arrow), interlobar fissural thickening (black dashed arrow), and pleural effusion (black curved arrow). The prominent centrilobular artery and vascular engorgement (arrowheads in C) were noted. (D) Pulmonary edema of cardiogenic origin (different patient). Frontal chest radiograph of a 37-year-old lady with cardiogenic pulmonary edema shows cephalization of pulmonary vasculature, peribronchial cuffing, and alveolar opacities in bilateral middle to lower zones. Note the markedly enlarged cardiac silhouette.

of various etiology can have a wide range of distribution of pulmonary opacities.⁶

Computed Tomography

High-resolution CT (HRCT) is more sensitive than CXR for detecting pulmonary edema. Manifestations can include diffuse or focal ground glass opacities, with or without smooth interlobular septal thickening or peribronchovascular interstitial thickening. Centrilobular ground glass nodules may also be seen. Dilated pulmonary veins or prominent centrilobular artery branches may be seen representative of vascular engorgement. There may be thickening of interlobar fissure either due to subpleural edema or secondary to pleural effusion.⁷ (►Fig. 2B and C)

Distribution of pulmonary edema in renal failure is usually bilateral; however, occasional cases of unilateral pulmonary edema have been described usually when associated with cardiac dysfunction (mitral valve insufficiency), or a structural abnormality of underlying lung or its vasculature.^{8,9}

In clinical practice, distinction between pulmonary edema of renal or cardiogenic origin is difficult and often inconsequential, given the multifactorial etiology of its pathogenesis in chronic renal failure, more so in severe cases of pulmonary edema (►Table 2).

In addition, Slanetz et al, in their retrospective study of 46 patients with clinical features of congestive heart failure reported two new CT features: enlarged mediastinal lymph nodes (LNs) in 55% and hazy mediastinal fat in 33% of cases¹⁰ in addition to the above-mentioned usual features of pulmonary edema. The enlarged LNs were most commonly located in the subcarinal, paratracheal, and hilar region. The underlying pathogenesis most likely represents diffuse intrathoracic edema, which, in chronic or severe cases, may spill over to cause mediastinal congestion presenting as lymphadenopathy and heterogeneous mediastinal fat. These findings show regression after appropriate treatment.^{10,11} However, nonresolving mediastinal lymphadenopathy even after adequate therapy and short-axis

Table 2 Imaging pointers to suggest renal versus cardiac origin of pulmonary edema

Imaging features	Pulmonary edema of renal origin	Pulmonary edema of cardiac origin
Redistribution of pulmonary blood flow	Balanced (homogeneous) flow	Temporal progression from initial cephalization or inversion from the base to the apex
Regional distribution of pulmonary opacities (not very specific)	Perihilar without gravitational preference ^a	Gravitational (basal) predominance, homogenous axial distribution along with a perihilar component
Cardiac silhouette	Usually normal; may be enlarged if there is concomitant cardiac failure	Usually enlarged

^aThis imaging feature is not very specific for pulmonary edema of renal origin as discussed earlier in the text.

diameter of LN greater than 2 cm are suspicious for alternate diagnosis and should be investigated.

CXRs acquired before and after dialysis offer an accurate estimation of systemic and pulmonary hydration status.⁵ Measurement of the mediastinal vascular pedicle width and caliber of the azygous vein at the arch level is representative of the systemic hydration status (►Fig. 3).^{12,13} Change of the vascular pedicle width by more or less of 5 mm is suggestive of a variation of the systemic fluid volume of approximately 1 L.¹³ In addition, a comparison of the baseline radiographs (predialysis) with subsequent films allows identification of patients with significant weight increment (4%).⁵ CXR also allows evaluation of fluid accumulation in third spaces such as pleural and pericardial effusion and chest wall soft-tissue edema (►Fig. 3). In cases of inadvertent excessive fluid subtraction during dialysis, CXR may reveal a pattern known as “empty lung” characterized by reduction of the vascular pedicle width compared to the baseline, collapse of the azygous vein, and pulmonary oligemia.¹⁴

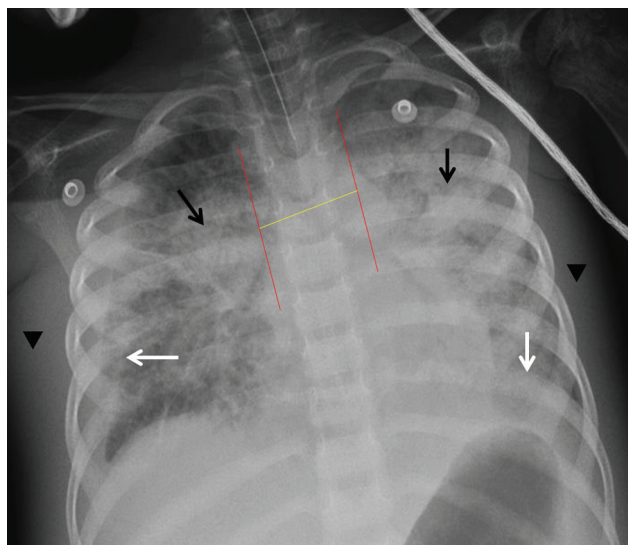


Fig. 3 Engorged vascular pedicle width in systemic overhydration. Frontal chest radiograph of a child with chronic kidney disease (CKD) secondary to nephrotic syndrome and pulmonary edema shows enlarged vascular pedicle (within red lines) with bilateral perihilar alveolar opacities, consolidation (black arrows), and pleural effusion (white arrows). Chest wall soft-tissue edema bilaterally (black arrow-head) is noted.

Ultrasound

Ultrasound, as a modality, has recently gained a lot of interest in evaluation of lung pathologies. Body ultrasound, which includes measurement of the inferior vena cava (IVC) diameter and its inspiratory collapse on M mode and lung ultrasound (LUS), forms a part of the “5B” approach used for comprehensive assessment of the volume status in addition to clinical evaluation. The IVC collapsibility index can predict the volume status, but it only reflects the intravascular volume (preload) and not actual tissue hydration. Moreover, right-sided heart failure, diastolic dysfunction, and tricuspid regurgitation are important limiting factors.¹⁵

LUS evaluates the extravascular lung water (EVLW), which is a surrogate marker of congestion of pulmonary interstitium.¹⁵ Evaluation of B-lines or lung comets is a pivotal part in the assessment of the lung water. With respect to LUS, B-lines refer to reverberation artifacts, which are vertical, originating from the pleural line, sharp, laserlike hyperechoic, extending till the edge of the screen without fading, and show sliding motion in tandem with respiration (►Fig. 4A and B).

While different authors have used different approaches to perform LUS, the “28-rib interspaces technique” uses a quantitative and very comprehensive protocol for scanning to cover bilateral anterior and lateral chest at the 28-rib intercostal scanning sites.^{16,17} It involves scanning the second to fourth intercostal spaces (left side) and the second to fifth intercostal spaces (right side) at the parasternal and midclavicular line (anterior chest), and at the anterior and midaxillary line (lateral chest; ►Fig. 4C–E). At every site, the number of B lines are counted (0–10). No detectable B lines are given a score of 0, while a complete white out screen is ascribed a value of 10. The total sum of B lines gives a score that gives an estimate of EVLW. The amount of EVLW is considered to be mild or absent when the total number of comets are ≤ 15 , moderate for 16 to 30, and serious when B lines are in excess of 30.¹⁸

However, LUS has its own limitations, which include operator dependency and technical issues related to image acquisition in morbidly obese patients, with subcutaneous emphysema or overlying dressing. Also, it has relatively limited specificity as a similar pattern can be seen in any diffuse interstitial pneumonia, acute respiratory distress syndrome (ARDS), as well as fibrotic interstitial lung diseases.¹⁹

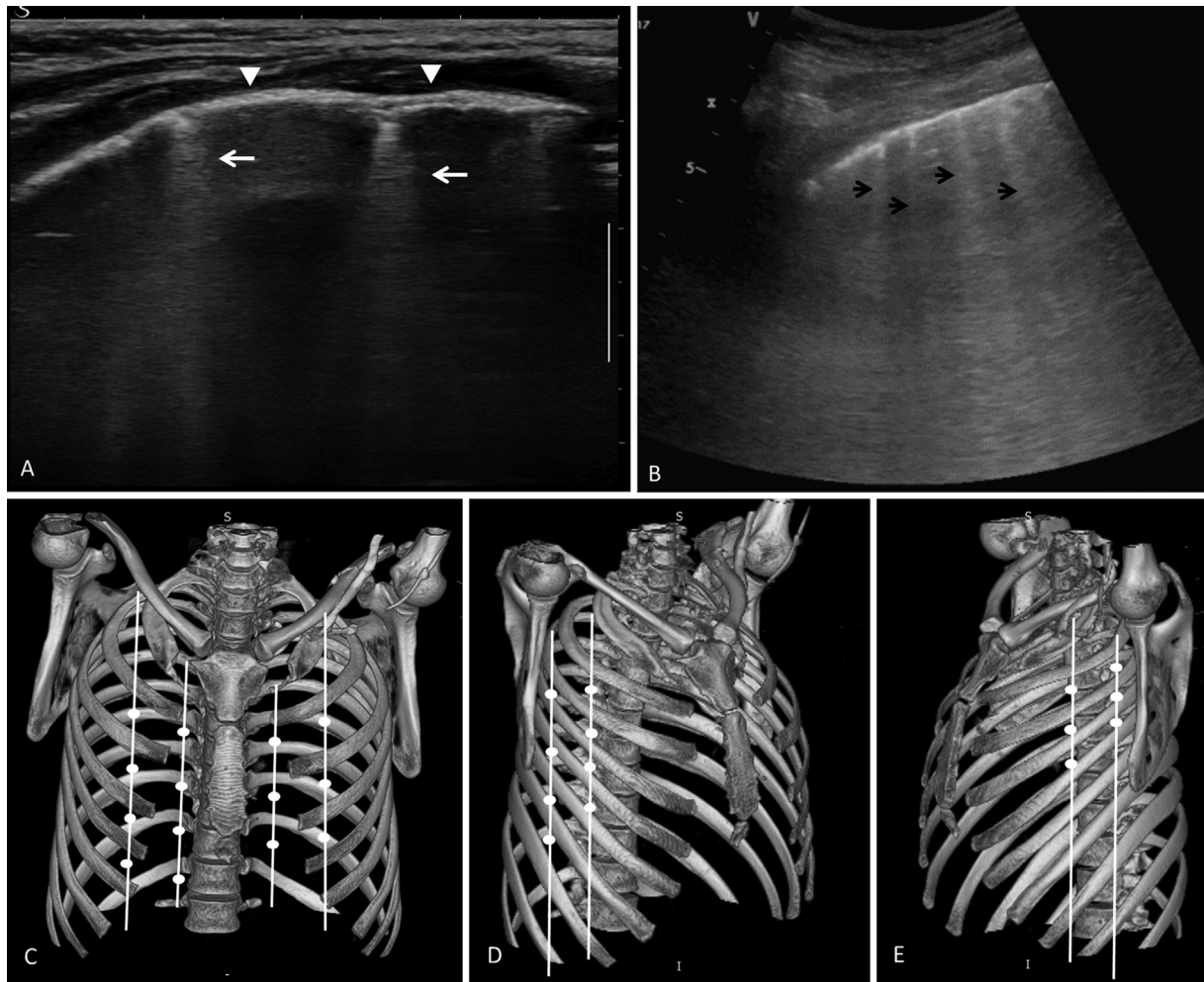


Fig. 4 Lung ultrasound to assess pulmonary edema. (A) Intercostal approach in a normal subject using a linear probe shows B-lines as vertical, sharp hyperechoic reverberation artifacts (*white arrows*) originating from the pleural line (*arrowhead*) and extending till the edge of the screen without fading and showing sliding motion. (B) Intercostal approach using a curvilinear probe in a different patient with pulmonary edema shows multiple B-lines (*black arrows*). Three-dimensional (3D) volume-rendered computed tomography (CT) image of the chest showing the landmarks of “28-rib interspaces technique” for lung ultrasonography (LUS). (C) Anteroposterior view showing bilateral parasternal and midclavicular lines (*white lines*) with the second to fifth intercostal spaces on the right and to the fourth on the left side (*white dots*) as scanning points. (D) Right and (E) left lateral views showing anterior and midaxillary lines with similar scanning points making a total of 28.

Pulmonary Infections

Pneumonia is a leading cause of infectious morbidity and mortality in patients with CKD and end stage renal disease (ESRD) receiving dialysis.²⁰ Pneumonia-related mortality is approximately 14- to 16-fold higher in patients on dialysis, as compared to the general population. Not only is there an increased incidence but they are also susceptible to increased severity of pneumonia, as evidenced by higher rate and duration of hospitalization when compared to the general population.²¹

CKD is an inherently immune-compromised state due to depression of cell-mediated immunity and impaired phagocytic mechanism and, hence, infections tend to occur more often than the general population.²² Associated comorbidities including advanced age, high prevalence of diabetes mellitus (DM), and frequent exposure to potential infectious risk factors during the normal course of dialysis therapy add to the predisposition.²⁰

Streptococcus pneumoniae is the most common etiologic pathogen, followed by *Haemophilus influenzae* and *Legionella pneumophila*. Associated DM and cardiovascular disease increase the susceptibility to pneumococcal disease. Of particular interest, CKD patients are less likely to present with expected classical clinical and laboratory manifestations during an episode of bacterial pneumonia. This may often lead to underestimation of diagnosis and severity of pneumonia. Consequently, fewer microbiologic investigations are performed and hence the chances to isolate a causative pathogen in this population are lower.²¹

Patients who are on routine hemodialysis therapy represent a distinct category vulnerable to health care-associated pneumonia, with high prevalence of multidrug-resistant organisms. Slinin et al, in their retrospective review, analyzed the microbiological etiology of pneumonia in patients on chronic hemodialysis. *S. pneumoniae* was the most commonly found bacteria followed by *Pseudomonas aeruginosa*, *Klebsiella*

species, and *H. influenzae*. *Staphylococcus* species are uncommonly documented.²³ Similar results were reported by Viasus et al who also documented *S. pneumoniae* as the most common pathogen in this subgroup of patients, and relatively low frequency of gram-negative bacilli and *S. aureus*.²¹ However, a subsequent study from Taiwan showed higher prevalence of *S. aureus* pneumonia in patients with estimated glomerular filtration rate (eGFR) less than 55 mL/min/1.73 m.^{2,24} The radiological manifestations are similar to the patterns observed in general population (►Fig. 5A).

Tuberculosis is an emerging public health concern in CKD patients, with a 6.9- to 52.5-fold higher risk in this subgroup as compared to the general population.²⁵ This particularly applies to high TB burden countries like India and China, which also account for a vast majority of CKD patients worldwide. Clinical manifestation of TB in CKD is often atypical and insidious, mimicking uremia with resultant delay in diagnosis. Extrapulmonary disease predominates in about 60 to 80% of cases along with miliary TB²⁶ (►Fig. 5B). One study reported an incidence of 11% of TB in CKD patients on maintenance

dialysis. Extrapulmonary TB was present in 78% of patients, while pulmonary TB was seen in 21.7% cases, of which pleural effusion was the commonest manifestation²⁷ (►Fig. 5C). Similar findings were also reported by Taskapan et al who found pleural effusion to be the most frequent presentation of pulmonary TB occurring in CKD patients²⁸ (►Fig. 5C). Of the extrapulmonary sites, tubercular lymphadenitis and peritonitis are the most often reported localizations.^{29,30} A persistent, unilateral effusion particularly if showing loculation, internal septation, or pleural thickening should raise a suspicion of TB. Also, as mentioned earlier, the diagnosis of mediastinal nodal TB can be challenging in these patients as these LNs may also enlarge due to fluid overload. Particularly, as these patients undergo noncontrast CT examination, the differentiation becomes difficult.

Fungal infections are now increasingly being recognized in patients with renal dysfunction, those receiving dialysis as well as renal transplant recipients (►Fig. 5D). Dialysis-dependent patients are at an increased risk of fungal infection-related mortality as compared to the general population.

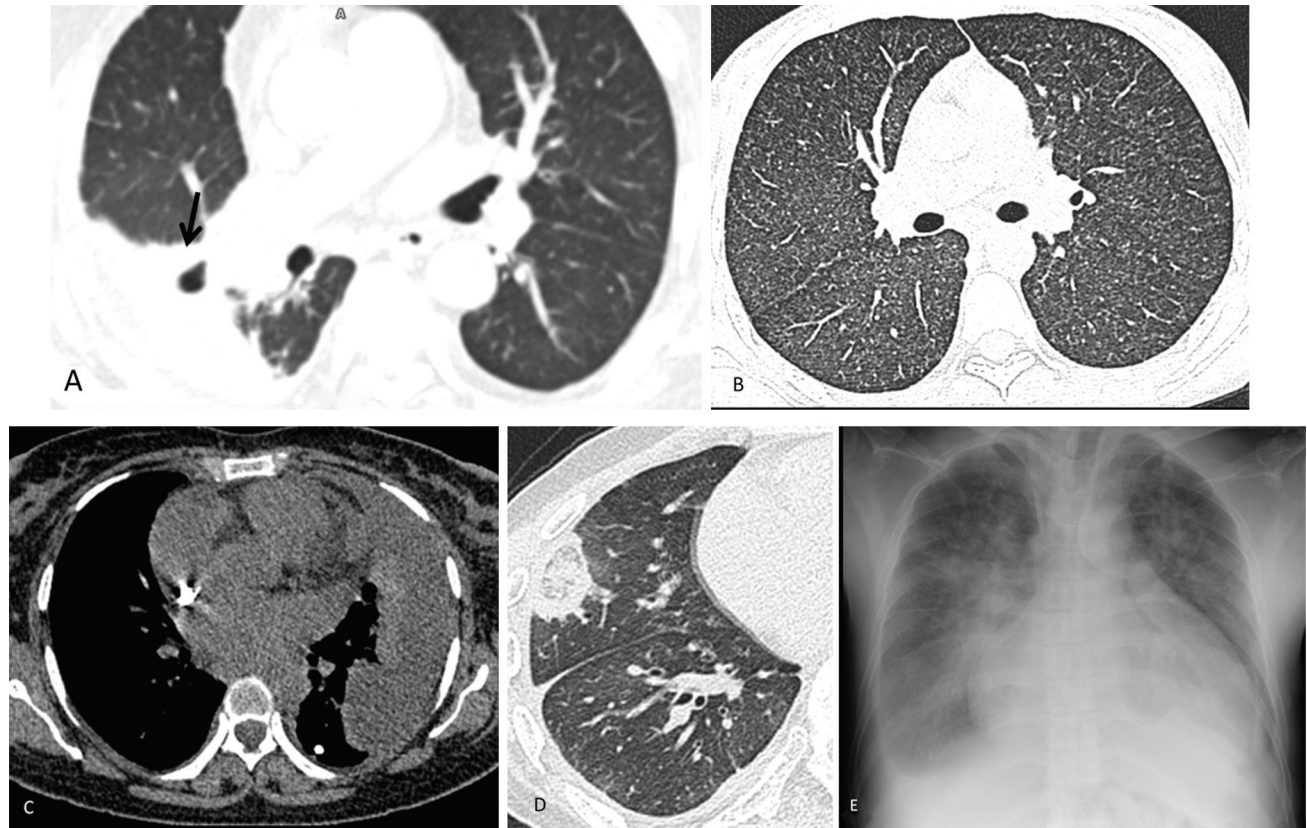


Fig. 5 Pulmonary infections in chronic kidney disease (CKD; different patients). (A) Bacterial pneumonia. Axial noncontrast computed tomography (CT) image (lung window) in a 57-year-old patient with CKD, fever, and cough for 10 days shows focal consolidation in the right lower lobe with cavitation. The patient responded well after 2 weeks of antibiotic therapy. (B) Miliary tuberculosis (TB) in a 36-year-old man with CKD. Axial CT image (lung window) shows randomly distributed miliary nodules in bilateral lungs. (C) Tubercular pleural effusion in a 35-year-old lady with CKD. Axial noncontrast CT image (mediastinal window) shows left-sided loculated pleural effusion. Thoracocentesis revealed exudative pleural effusion and positive for *Mycobacterium tuberculosis* on polymerase chain reaction (PCR). (D) Fungal infection in a 46-year-old patient with CKD who presented with cough, chest pain, dyspnea, and high-grade fever. Axial CT of the chest (lung window) shows focal lesion in the right upper lobe with internal ground glass opacity and peripheral consolidation. Mild associated fissural effusion noted. CT-guided biopsy from the lesion revealed mucormycosis. (E) A 57-year-old man with CKD stage V and reverse transcriptase PCR (RT-PCR) positive for severe acute respiratory syndrome (SARS) coronavirus on nasopharyngeal swab. Frontal chest radiograph (CXR) shows cardiomegaly with bilateral central perihilar air space opacities and bilateral pleural effusion. Central venous catheter in the right internal jugular vein can be noted.

Analysis of the 1999 United States Renal Data System (USRDS) database revealed *Candida* as the dominant etiology of fungal infection (79%) followed by cryptococcosis (6%), coccidioidomycosis (4%), and *Aspergillus* (3.8%). Mucormycosis was reported in 29 (1.5%) patients³¹ (►Fig. 5D).

CKD has also been identified as a risk factor for severe coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2; ►Fig. 5E). A recent meta-analysis to evaluate the severity and mortality in COVID-19 patients with underlying renal and liver dysfunction revealed 1% overall prevalence of CKD in COVID-19 patients with severity of 83.93% (47/56) and mortality of 53.33% (8/15) in CKD.³²

Altered Calcium and Phosphorus Metabolism

Metastatic Pulmonary Calcification

This is a rare entity encountered in patients with CKD on long-term dialysis but has been reported in about 60 to 80% of autopsies of patients who have been on dialysis.³³ The underlying pathogenesis is a derangement of calcium–phosphate metabolism, with precipitation of calcium salts in otherwise normal tissues. Not uncommonly, it may also be seen in

primary hyperparathyroidism, hypervitaminosis D, sarcoidosis, milk alkali syndrome, and other hypercalcemic states.

The majority of the patients are asymptomatic, but some can present with dyspnea and cough. Tissue deposition of calcium, particularly in pulmonary alveolar interstitium and bronchial and vascular walls with concomitant fibrosis, impairs the diffusion capacity of the lungs (manifested as reduced DL_{CO} on pulmonary function test [PFT]).³⁴ The insidiously progressive pulmonary fibrosis may eventually lead to cor pulmonale and respiratory failure in few patients.³⁴

The sensitivity of CXR for the detection of small pulmonary calcification is poor, being positive in less than 15% as compared to autopsy results. Dual energy digital CXR offers an improvement over the single energy routine CXR and is reported to be more sensitive and accurate in detecting pulmonary calcification.^{35,36} But this technique has not gained widespread use due to limited availability and obvious advantages of CT for evaluation of ancillary pulmonary changes.

The imaging findings are often nonspecific and can present as confluent or patchy airspace opacities, diffuse reticular opacities consistent with an interstitial disease, low-density

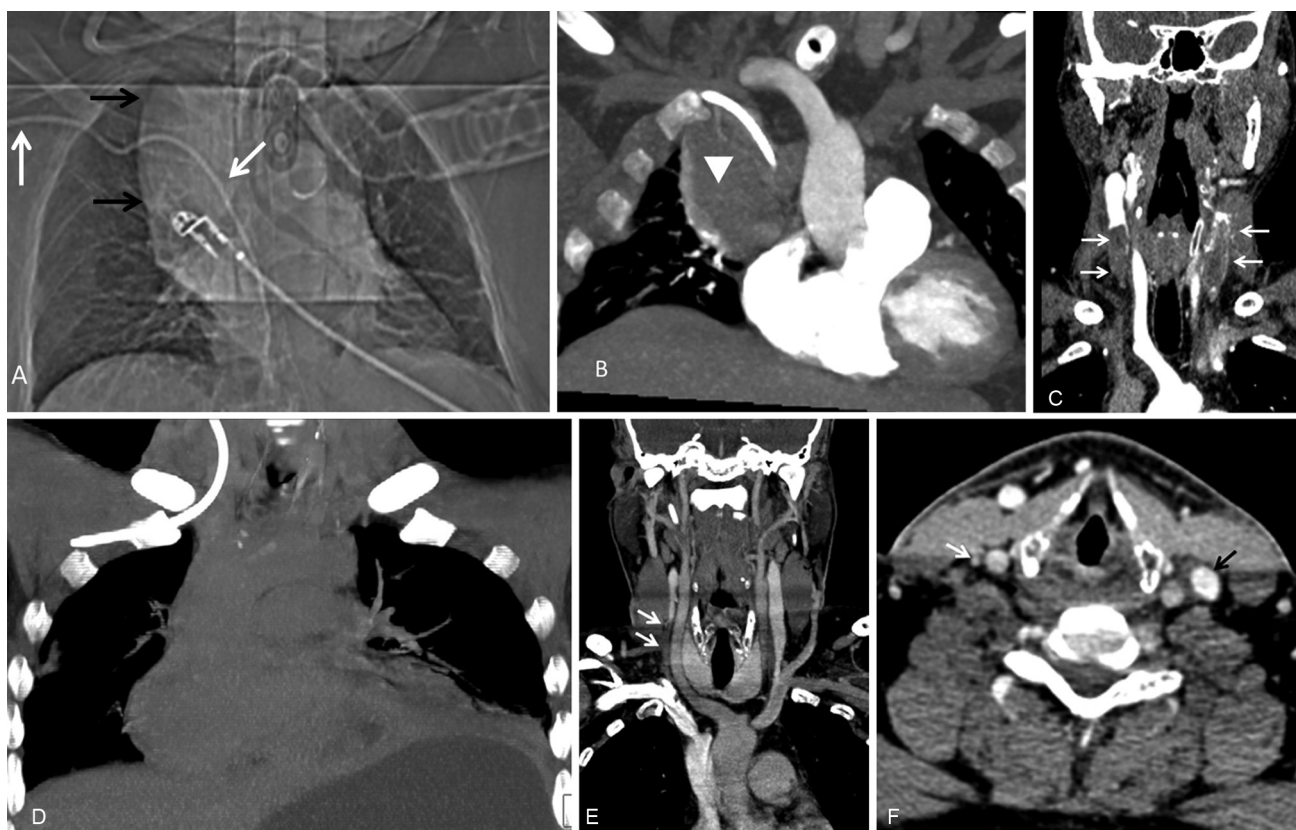


Fig. 6 Hemodialysis catheter-related complications (different patients). Mediastinal hematoma post right subclavian vein catheter insertion. (A) Topographic computed tomography (CT) image shows superior mediastinal widening with marked thickening of the right paratracheal stripe (black arrows) and subclavian vein catheter in situ (white arrows). (B) Coronal image of CT angiography of the chest reveals large hematoma extending from the thoracic inlet along the subclavian vein catheter and superior vena cava till the cavoatrial junction (arrowheads). (C) Catheter-related bilateral internal jugular vein (IJV) thrombosis in a 28-year-old man with chronic kidney disease (CKD). (C) Coronal CT angiography shows partial intraluminal thrombus in the bilateral IJV following catheterization (white arrows). (D) Malpositioned catheter. Coronal maximum intensity projection (MIP) image in a 43-year-old man with CKD shows malpositioned central venous catheter inserted via the right IJV with its tip in the right subclavian vein. Catheter-related venous stenosis in 30-year-old man with CKD. (E) Coronal and (F) axial contrast-enhanced CT (CECT) image shows stenosis of the right IJV (white arrow) secondary to previous catheterization. Normal IJV opacification on the left side (black arrow).

apical opacities, and discrete or confluent calcified nodules.³⁷ Cardiac size and pulmonary vascular markings are normal.³⁸ There is an upper lobe predominance of calcification, presumably because of the presence of high ventilation-to-perfusion ratio leading to local hypercapnia, which in turn creates local tissue alkalinity. This in turn promotes precipitation of calcium salts, especially in a background of hypercalcemia and hyperphosphatemia.⁵

It is essential to remember that the initial phases of the disease are associated with minimal calcium deposition, which are often difficult to recognize on CXR, especially with the high kilovoltage and low-contrast technique that is commonly in use.³⁹ Hence, there is considerable overlap with the imaging features of pulmonary edema, pneumonia, pulmonary alveolar hemorrhage, or infarct, which are other common entities encountered in these patient population, making CXR unreliable for diagnosis.³⁶

With time, they tend to become denser and more calcified; if left untreated, they can mimic other causes of pulmonary calcification including previous tubercular or fungal infection, especially in endemic areas.³⁶

CT, especially HRCT, has better sensitivity and specificity than CXR in early detection of pulmonary calcification⁴⁰ and is widely used for detection of metastatic pulmonary calcification (MPC; ▶Fig. 7). While calcification is rarely picked up on radiographs, it is seen in about 60% of cases on HRCT.³⁷

Although pathologically the calcium deposition is interstitial, the disease can mimic an alveolar pattern on CT similar to CXR. The various patterns described on HRCT are (1) multiple diffuse or focal nodules, predominantly centrilobular; (2) diffuse or patchy ground glass opacity (GGO); (3) dense consolidation, sometimes in lobar distribution; and (4) any combination of the above patterns. Dense consolidations usually result from profuse interstitial calcifications⁴¹ (▶Fig. 7). Interlobular septal thickening is characteristically absent despite the purely interstitial nature of the disease, which is attributed to preferential calcium deposition in the alveolar septa, pulmonary arterioles, and bronchioles.³⁷

A frequent association is calcification in the vessels of the chest wall.^{36,37} This combination of parenchymal and chest wall vascular wall calcification is considered to be diagnostic for MPC. CT may additionally reveal calcification of the myocardium, bronchial walls, superior vena cava, and the dura of the dorsal spine.³⁶

Magnetic resonance (MR) imaging features of pulmonary metastatic calcification have also been described and produce hyperintense signal on T1-weighted (T1W) images as compared to muscle due to marked T1 shortening.⁴²

^{99m}Tc methylene diphosphate (MDP) bone scan is the most sensitive and specific study for early detection of pulmonary calcification and is often used as a problem-solving tool.⁴³ In an appropriate clinical setting, pulmonary uptake of radionuclide even in the presence of equivocal findings on HRCT is adequate for diagnosis.⁴⁴

Treatment is mainly aimed at correcting the disturbances in calcium–phosphate metabolism in the body. Renal transplant may offer remission in some patients, while in others it progresses even after transplant.

Uremic Pneumonitis

Uremic pneumonitis is a poorly understood entity that was chiefly described in the predialysis era. While some considered it a continuum of cardiac dysfunction in renal failure patients, others went on to show, by experimental animal studies, that uremic pneumonitis, per se, was a distinct entity representing a manifestation of terminal uremia.^{45–48}

Uremic lung may be considered a form of diffuse alveolar damage (DAD) microscopically presenting with fibrin-rich edema fluid in alveoli, with frequent red blood cells and formation of hyaline membranes.^{45,46} CXR shows bilateral symmetrical perihilar opacities with relative sparing of lung bases and peripheral areas, a pattern similar to the “batwing or butterfly wing pattern” described in pulmonary edema⁴⁹ (▶Fig. 8). However, prompt and significant clearance of pulmonary opacities following dialysis in the absence of any detectable cardiovascular dysfunction suggests underlying uremia as the possible etiology.⁴⁹

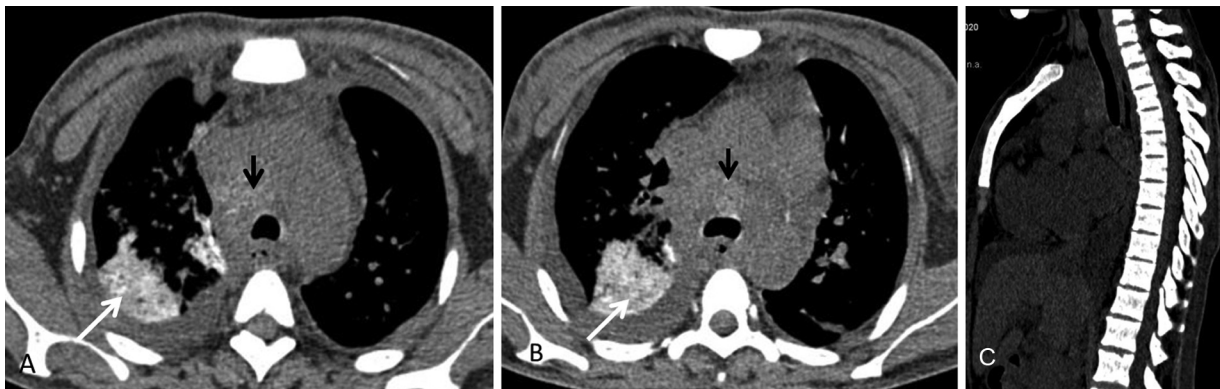


Fig. 7 Metastatic pulmonary calcification in a 34-year-old chronic kidney disease (CKD) stage 5 patient on dialysis for 1.5 years. (A,B) Axial and (C) sagittal noncontrast computed tomography (CT) images reveal dense consolidation in the right upper lobe with parenchymal calcification (white arrows) and mediastinal lymphadenopathy with fine calcification (black arrows). Bilateral mild pleural effusion and diffuse sclerosis of vertebrae and sternum (C) were noted, suggestive of renal osteodystrophy.

With the widespread availability and improvement in the dialysis technology in the present era, this entity has more or less become obsolete.

► **Table 3** summarizes the key imaging features to differentiate the common clinical entities that predominantly present with parenchymal manifestations in CKD including diffuse alveolar hemorrhage (► **Fig. 9**).

Lung Dysfunction in Chronic Kidney Disease

Advanced CKD is associated with impairment of lung function. With progressive decline in GFR, there is increasing pulmonary edema and respiratory muscle dysfunction, due to a complex interplay of fluid retention, cardiovascular, metabolic, and endocrine changes. In a study by Mukai et al, it was found that both restrictive and obstructive lung dysfunction increased with decreasing GFR, with a high prevalence (64%) of restrictive lung dysfunction in patients with comorbidities such as protein energy wasting, inflammation, and cardiovascular disease. Restrictive lung dysfunction was found to be an independent predictor of increased mortality in CKD patients.⁵⁰

Diffusion capacity of the lung for carbon monoxide (DL_{CO}) is also found to be reduced in patients with CKD, which persists even after successful renal transplant. The decline was significantly lower in those receiving continuous ambulatory peritoneal dialysis (CAPD). Subclinical pulmonary edema, common in these patients, is hypothesized to be the underlying cause. In addition, residual volume is also reduced in transplant recipients, likely due to gradual progression of subclinical pulmonary edema to fibrosis prior to transplant.³⁴

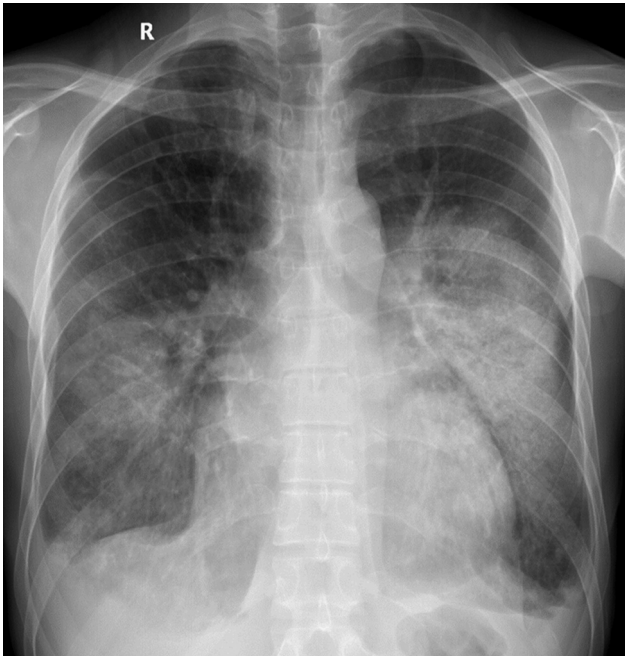


Fig. 8 A 27-year-old man with severe uremia and uremic lung. Frontal chest radiograph (CXR) shows bilateral symmetrical perihilar opacities with relative peripheral and basal sparing and bilateral mild pleural effusion. Normal cardiac silhouette was noted. Baseline echocardiography was normal. Postdialysis, there was prompt clearance of bilateral pulmonary opacities.

Pleural/Pericardial Manifestations

Uremic Pleuropericarditis

The causes of pleural/pericardial effusion in CKD include fluid overload, uremic pleuritis, parapneumonic effusion, tubercular effusion, and those on CAPD. Of these, on thoracentesis, effusions due to fluid overload and secondary to CAPD are transudative, while the rest are exudative. Uremic pleuropericarditis is present in about 20 to 40% of patients with CKD on autopsy.⁵¹ It is a fibrinous pleuritis that develops without associated pneumonitis in uremic patients.⁵¹ Patients may be asymptomatic initially or present with dyspnea, fever pleuritic chest pain, and audible friction rub on auscultation.²

The effusion is typically exudative and serosanguinous or hemorrhagic with sterile cultures. They tend to be unilateral and may sometimes be quite large (► **Fig. 10**). Notably, uremic pleural effusion is unrelated to the severity of uremia and can occur at any time during the course of renal dysfunction.⁵² The diagnosis of uremic pleuritis is one of exclusion as a number of other concomitant conditions can also produce pleural effusion in patients with CKD such as congestive heart failure, hypoproteinemia, autoimmune disease, infection, hemothorax, malignancy, and pulmonary embolism.⁵

Ultrasound usually demonstrates multiple thin internal septa, fibrous bands without vascularity, and debris. The presence of persistent exudative pleural effusion poses a diagnostic dilemma in these patients, especially in TB-endemic regions as both tubercular and uremic pleurisy present similarly. More often than not, such patients are empirically started on antitubercular therapy (ATT).

Studies have shown that uremia is still the most common cause of exudative pleural effusion in CKD even in high TB burden countries.⁵²⁻⁵⁴

Multiplex polymerase chain reaction (PCR; 100% sensitivity and 100% specificity) coupled with thoracoscopy for pleural nodularity (83% sensitivity and 100% specificity) is found to have very high accuracy for diagnosis of tubercular pleural effusion in the diagnostic workup of pleural effusions developing in CKD.⁵⁴ The effusion usually responds to intensified dialysis or renal replacement therapy (within 4-6 weeks), or may subsequently evolve into fibrothorax.

► **Table 4** summarizes the key imaging differentiating features of various conditions with predominant pleural manifestations in CKD.

Uremic pericarditis is believed to be caused by a mechanism similar to that in uremic pleuritis⁵⁵ (► **Fig. 10**). It usually develops as dry fibrinous pericarditis with characteristic auscultatory findings of pericardial friction with fever, pain, and electrocardiographic (ECG) changes.⁵ In some patients, a variable amount of hemorrhagic or exudative effusion may develop and can be picked up on ultrasound.²

Issues Related to Dialysis

Patients with ESRD who are dependent on dialysis are ever increasing in number. Two different approaches are available for the same: peritoneal dialysis or hemodialysis.

Table 3 Key imaging features to differentiate entities with parenchymal manifestations in CKD

Key imaging features	Pulmonary edema	Pneumonia	Noncardiogenic pulmonary edema	Metastatic pulmonary calcification	Diffuse alveolar hemorrhage
Distribution	<ul style="list-style-type: none"> Usually bilateral, symmetrical; occasionally unilateral Central perihilar Apicobasal gradient 	Unilateral, focal	<ul style="list-style-type: none"> Bilateral, patchy, nonhomogeneous Peripheral predominant Apicobasal and anteroposterior gradient 	<ul style="list-style-type: none"> Bi- or unilateral Symmetrical/focal Upper lobe predominance 	<ul style="list-style-type: none"> Uni- or bilateral, focal/diffuse Symmetrical/asymmetrical Central predominance, relative sparing of lung apices and periphery
Vascular engorgement	+ (Increased vascular pedicle width, prominent pulmonary veins and artery)	-	Normal or narrowed	Characteristic calcification of chest wall vessels	-
Cardiac silhouette	Enlarged usually; may be normal	Normal	Normal	Normal	Normal
Pleural abnormality	Pleural effusion; thickening of the interlobar fissure	±	Usually not seen	Not seen	Not seen
Interstitial edema	+ (Smooth septal thickening, peribronchial and perivascular cuffing)	-	-	Interlobular septal thickening characteristically absent	-
Parenchymal changes	Predominant GGO; may be centrilobular	Consolidation mainly, GGO ±	GGO; consolidation (exudative phase) Reticular opacities superimposed on GGO (intermediate/proliferative phase) Persistent reticulations, GGO, cysts, and traction bronchiectasis (late/fibrotic phase)	GGO; consolidation	GGO; consolidation
Resolution	Prompt resolution after therapy	Temporal evolution as per antibiotic response	Slow resolution >2 wk	<ul style="list-style-type: none"> Stable on CXR; no response to standard antibiotic therapy May or may not respond to renal replacement therapy 	Within 2–3 d with reticulation and interlobular septal thickening, may progress to fibrosis

Abbreviations: CKD, chronic kidney disease; CXR, chest radiograph; GGO, ground glass opacity.

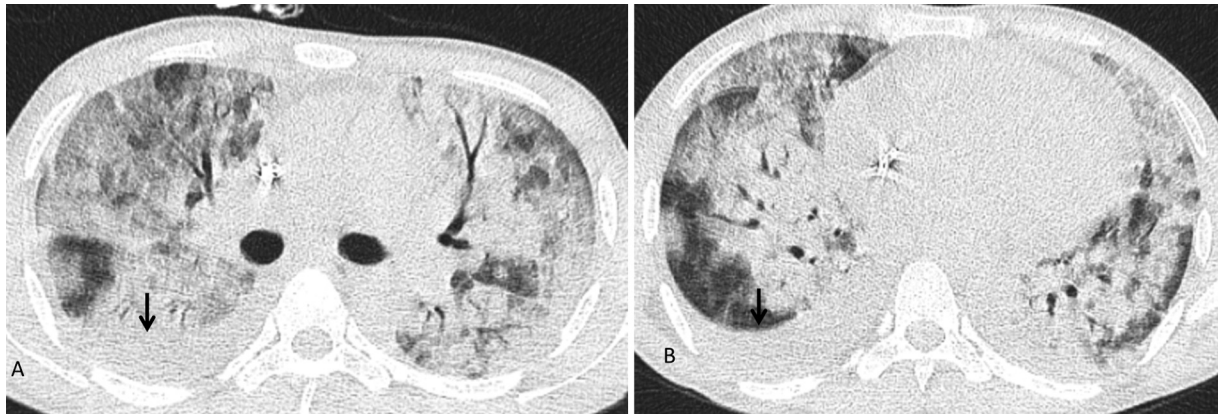


Fig. 9 Diffuse alveolar hemorrhage in a 34-year-old patient with chronic kidney disease (CKD) presenting with sudden onset cough, dyspnea, and concomitant drop in hemoglobin. (A,B) Axial computed tomography (CT) images in the lung window reveal multifocal and bilateral ground glass opacity (GGO), consolidation with air bronchogram, and relative peripheral sparing. Mild right pleural effusion is seen (*black arrows*).

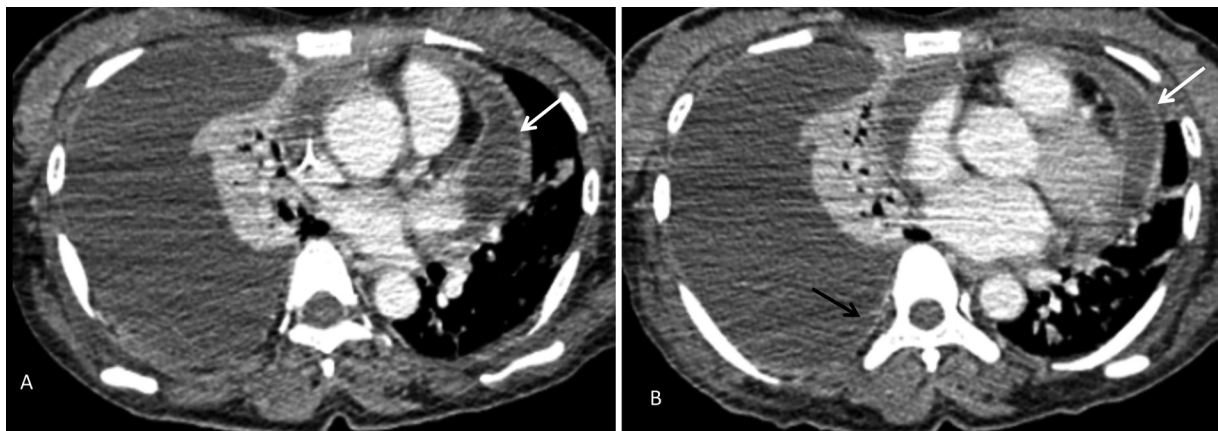


Fig. 10 Uremic pleuropericarditis in a 30-year-old man with chronic kidney disease (CKD). (A,B) Axial contrast-enhanced computed tomography (CECT) image in mediastinal window shows unilateral large right pleural effusion with mild pleural thickening (*black arrow*) and passive atelectasis of the right lung. Associated pericardial effusion with thickened, enhancing pericardium is also noted (*white arrows*).

Peritoneal Dialysis

Peritoneal dialysis involves exchange of fluids and salts via the peritoneal membrane. CAPD causes a temporary rise in intra-abdominal pressure, which in turn leads to elevation and reduced motility of the diaphragm. This is known to cause intraprocedural restrictive functional deficit, hypoxemia, and rise in pulmonary arterial pressure. It is not uncommon to find basal plate atelectasis, small pneumoperitoneum, and pleural effusion, usually on the right side in patients undergoing CAPD.⁵⁶

The pleural effusion that develops following CAPD may be due to focal breach in the diaphragm or due to the presence of the pleuroperitoneal fistula. The latter can be demonstrated by using CT peritoneography, which is considered the reference standard, MR peritoneography showing passage of an intra-abdominally injected contrast medium into the thoracic cavity, or by peritoneal scintigraphy demonstrating supradiaphragmatic radioactivity.^{57–59} Coronal and sagittal reformatted images are especially helpful to assess the lateral and posterior recesses of the peritoneal cavity.

Biochemical analysis of the pleural fluid (transudate) too demonstrates similar constitution as the peritoneal

dialysate, characterized by high glucose concentration with normal blood glucose concentration. It tends to persist even after removal of peritoneal fluid due to the presence of a check valve mechanism at the level of diaphragmatic leak.⁶⁰

Pulmonary infections also tend to be more common in patients undergoing CAPD, probably due to their compromised immunity compounded with reduced ventilation and frequent atelectasis observed in their lung bases.²² In addition, patients on CAPD are also more susceptible to develop peritoneal tuberculosis.^{25,30} In fact, tuberculosis overall is far more common in patients on chronic dialysis as compared to the general population.

Hemodialysis

The most common problems in relation to hemodialysis are iatrogenic and include malposition or malfunctioning of catheters or complications related to vascular access. The common complications related to all central venous catheters include pneumothorax, hemothorax, mediastinal hematoma (► **Fig. 6A, B**), vein thrombosis (► **Fig. 6C**), puncture site infection, catheter-related sepsis and catheter breakage.

Table 4 Key differentiating features of entities with predominant pleural manifestations in CKD

Features	Pleural effusion in fluid overload	Uremic pleuritis	Parapneumonic effusion	Tubercular pleural effusion
Nature	Transudate	Exudate	Exudate	Exudate
Appearance	Serous	Serosanguineous or hemorrhagic	Serous	Serous
Pleural fluid culture	Sterile	Sterile	Positive in case of empyema	Positive in case of empyema
Pleural fluid cytology	Lymphocyte predominant	Lymphocyte predominant	Neutrophil predominant	Lymphocyte predominant
Laterality	Usually bilateral; may be unilateral	Usually unilateral	Unilateral	Unilateral
Imaging				
• Cardiac size	Enlarged cardiac size and vascular engorgement	Normal cardiac size	Normal cardiac size	Normal cardiac size
• Parenchymal changes	May show associated features of pulmonary edema	No associated parenchymal changes	Parenchymal infiltration present	Parenchymal changes less frequently; extrapulmonary manifestations are common

Abbreviation: CKD, chronic kidney disease.

Compression of the catheter between the first rib and the clavicle is also applicable to this group of patients.^{61,62}

Catheter malposition is a commonly encountered complication (►Fig. 6D). Malpositioned catheter usually leads to catheter dysfunction as adequate flow cannot be generated. Localization is hence essential to be confirmed by CXR before final suturing of the catheter. The catheter may sometimes

inadvertently migrate into the azygous vein and mimic venous thrombosis.⁶³

A dialysis catheter-induced stenosis (►Fig. 6E, F) or thrombosis (►Fig. 6C) of veins is a common complication seen in up to 41% of hemodialysis patients.⁶⁴ Venous stenosis is more common in catheters inserted via the subclavian vein than via the internal jugular vein (IJV) as the twisted course

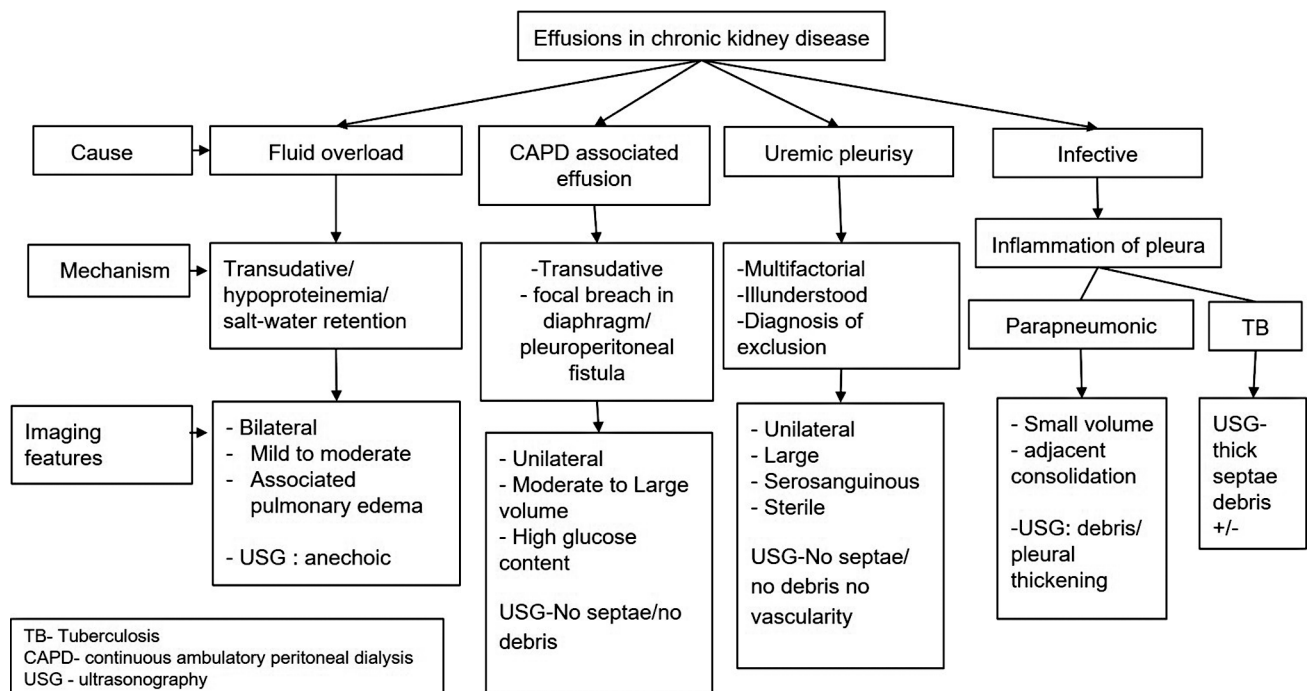


Fig. 11 Algorithm for assessment of pleural effusions in chronic kidney disease (CKD).

of the catheter in the body poses a risk factor for venous stenosis as opposed to a relatively straight course of the IJV.⁶⁵

Dialysis catheters, once inserted, act as a foreign body leading to venous thrombosis. Catheter-associated thrombosis is a risk factor for pulmonary embolism, seen in about 15 to 25% of patients.⁶⁶ Ultrasound with Doppler or invasive phlebography is required to confirm the diagnosis.

An imaging algorithm for effusions in CKD is shown in **Fig. 11**.

Thoracic Manifestations of Renal Osteodystrophy

CKD patients including those on long-term hemodialysis may present with specific bony lesions (typically in hands) that may sometimes be evident on CXR. These include subperiosteal resorption of distal epiphysis or conoid tubercle of the clavicle, subperiosteal erosion of the head and neck of the humerus suggestive of renal osteodystrophy, as well as glenohumeral joint involvement and peri-arthritis in dialysis-related amyloidosis.

To conclude, thoracic complications of CKD and subsequent dialysis-related complications are variable in their presentation. The intricate association between respiratory and renal functioning in maintaining physiological homeostasis underlines the importance of being aware of these changes. Radiology plays a crucial role in early identification of complications that mainly revolve around fluid and solute imbalance, calcium-phosphate metabolism, depressed immunity, and procedure-related in dialysis-dependent. Early institution of therapy and response to treatment can also be adequately assessed which can positively impact the quality of life for these patients.

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Conflict of Interest

None declared.

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