

image analysis is important, in order to avoid an incorrect diagnosis of enterovesical fistula with vesicoureteral reflux.

Ureterocolic fistulas are rare and have a variety of causes, most being urological<sup>(1)</sup> or iatrogenic in origin, with an inflammatory, neoplastic, or idiopathic etiology. The recent increase in the number of ureteroscopic and laparoscopic procedures has greatly increased the incidence of ureterocolic fistulas<sup>(2)</sup>, because surgical manipulation generates inflammation that affects the ureter and leads to their formation<sup>(3)</sup>. Anatomically, most cases involve the right ureter, in its upper and middle thirds, rarely occurring on the left<sup>(1,4,5)</sup>.

The most common symptom of ureterocolic fistula is non-specific abdominal pain, typically on the flanks. Peritoneal irritation with psoas muscle involvement can lead to Morton's triad, consisting of low back pain, thigh adduction, and lower limb flexion. As a rule, there are no digestive symptoms, although some patients present with pneumaturia or fecaluria<sup>(6)</sup>.

Ureterocolic fistula can be diagnosed on contrast-enhanced examinations, such as X-rays obtained after a barium enema and retrograde cystourethrography<sup>(7)</sup>. Obtaining X-ray images after administration of a barium enema is the most reliable method of identifying the fistulous tract<sup>(8)</sup>. Although retrograde voiding cystourethrography can allow visualization of the fistulous tract, it can be difficult to identify the ureteral meatus with the method, because of the surrounding edema attributed to the inflammatory process and because of obstruction of the ureteral pathway by the fistula. Computed tomography is the most sensitive method for identifying pneumaturia and the fistulous tract.

The treatment of ureterocolic fistula consists in the surgical removal of the fistula. The technique employed varies depending on which portion of the ureter is affected, as well as on whether there is any accompanying renal dysfunction.

In the case reported here, the emergence of the ureterocolic fistula was iatrogenic, being attributed to previous surgical manipulation. A contrast-enhanced imaging examination was essential because it allowed the fistula to be corrected during the surgical procedure that was being planned, as well as allowing the patient to be referred to the nephrology department for the clinical monitoring of any renal dysfunction that might develop.

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#### Lymphocytic interstitial pneumonia and pulmonary amyloidosis in Sjögren's syndrome

Dear Editor,

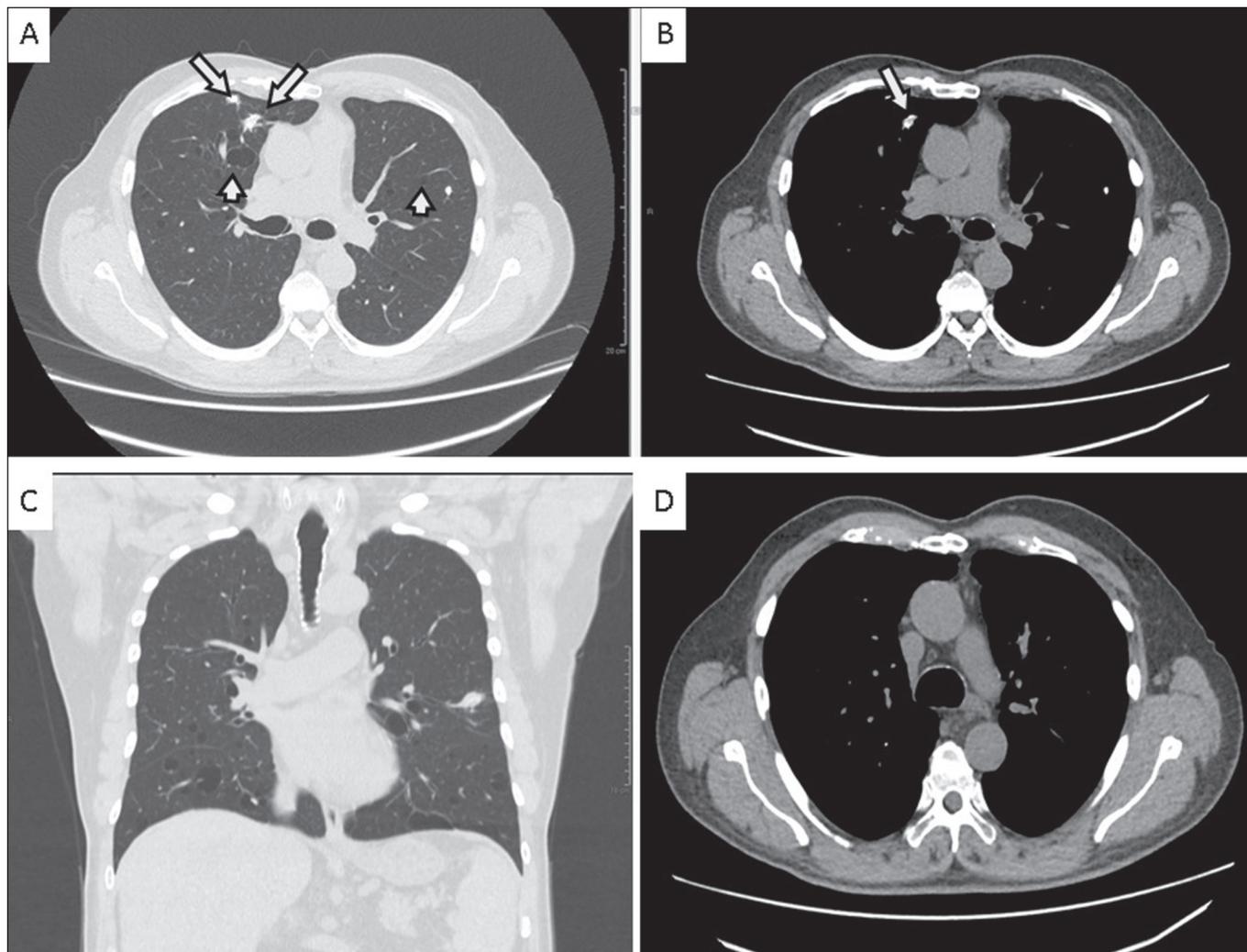
A 72-year-old male patient who was a former light smoker presented with a complaint of dyspnea. In 2014, he had been diagnosed with Sjögren's syndrome during investigation of thrombocytopenia identified on a routine laboratory test. An X-ray performed prior to transurethral resection of the prostate showed pulmonary nodules. Further evaluation with computed tomography (CT) of the chest revealed multiple thin-walled pulmonary cysts in the peribronchovascular and subpleural regions of both lungs, predominantly in the middle and lower lung fields, together with solid, irregular, partially calcified nodules, some in close proximity to the cysts (Figure 1). A biopsy of the largest nodule revealed fragments of lung parenchyma with lymphocytic infiltrate and proteinaceous fibrin filling the alveolar spaces, with degenerated red blood cells (ghost cells), sometimes forming hyaline membranes. Complementary analysis of the material showed a light chain amyloidosis (kappa) peptide profile.

Sjögren's syndrome is an autoimmune disease in which lymphocytes attack the glands that generate saliva and tears<sup>(1)</sup>. Many patients with Sjögren's syndrome develop interstitial lung diseases such as lymphocytic interstitial pneumonia (LIP), amyloidosis, follicular bronchiolitis, and even lymphoma<sup>(1,2)</sup>. On CT, LIP can manifest as ground-glass opacity or consolidations, as well as

septal thickening, centrilobular nodules, and cysts<sup>(3)</sup>. Cysts are believed to be formed by air trapping caused by a check-valve mechanism, with airway dilation distal to bronchiolar obstruction caused by lymphocytic infiltrate, and can be the only residual findings in chronic cases<sup>(3,4)</sup>.

Amyloidosis occurs due to excessive formation and deposition of certain proteins in an abnormal fibrillar pattern, resulting in malfunction of the affected organ<sup>(3,4)</sup>. Pulmonary nodular amyloidosis typically manifests as multiple nodules, of varying attenuation, which can cavitate<sup>(3,4)</sup>. Some are associated with mucosa-associated lymphoid tissue lymphoma<sup>(3,4)</sup>. In the clinical context of Sjögren's syndrome, calcification within a nodule is more consistent with amyloid nodules<sup>(2)</sup>. More rarely, amyloidosis can also lead to the formation of pulmonary cysts, of varying sizes<sup>(3,4)</sup>. The mechanism of cyst formation is uncertain and is believed to involve a check-valve mechanism secondary to narrowing of the airways, caused by the accumulation of inflammatory or amyloid cells or by capillary rupture due to amyloid deposition with alveolar destruction and cyst formation<sup>(3)</sup>. In alveolar-septal amyloidosis, the CT findings include septal thickening and ground-glass opacity, whereas CT shows circumferential thickening of the tracheobronchial wall in the more common form of the disease<sup>(3–5)</sup>.

The prognosis for patients with amyloidosis and LIP varies, the condition resolving in some patients, whereas it progresses to pulmonary fibrosis and respiratory failure in others<sup>(3)</sup>. Although there is no cure, corticosteroids can be used



**Figure 1.** **A,B:** Axial CT images of the chest at the level of the main bronchi, with lung and mediastinal window settings (**A** and **B**, respectively), showing thin-walled cysts in the peribronchovascular and subpleural regions (short arrows), as well as irregular, calcified pulmonary nodules (long arrows). Note also the preservation of the thickness of the bronchial wall. **C:** Coronal CT scan of the chest, with lung window settings, showing sparse nodules and cystic predominance in the middle and lower lung fields. **D:** Axial CT scan of the chest, with mediastinal window settings, highlighting the absence of mediastinal lymphadenopathy and the preservation of the thickness of the tracheal wall.

for symptom relief<sup>(3)</sup>. In rare cases, such as the one presented here, Sjögren’s syndrome, amyloidosis, and LIP can coexist. In patients with Sjögren’s syndrome, distinguishing cystic amyloidosis from LIP with amyloidosis is a diagnostic challenge. The diagnosis of LIP with amyloidosis should be ruled out before attributing the cysts exclusively to amyloidosis, because the CT finding of multiple calcified nodules and cysts in patients with Sjögren’s syndrome typically suggests a diagnosis of pulmonary amyloidosis and lymphoproliferative disease, which should be born in mind by the attending physician.

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