and histological findings were compatible with CCAM type 2. A diagnosis of CCAM type 2 associated with IL-PS was established based on imaging and histological findings. The patient remained asymptomatic 1 year after surgery.

This case combines an unusual association of congenital malformations – IL-PS and CCAM – with an infrequent diagnosis in adulthood. Few cases are described in literature of CCAM and IL-PS association.[2,5–7] We found reports of presenting in adulthood were even scarcer.[6]

These lesions are usually diagnosed prenatally or in the first years of life. Both can have a wide spectrum of presentations ranging from asymptomatic to neonatal death depending on the size of the mass and consequent physiological impairment.[1,2] After the first year of life recurrent infections are the most common presentation for both pathologies.[1–4] It is important to note that potential for malignant transformation has been described in both entities, with stronger evidence in CCAM.[3] Imaging has an important part in diagnosis of these lesions. Before birth ultrasound has a key role and after birth CT scan is the gold standard. Morphological features, location and blood supply can help distinguish the different malformations but this differentiation can be particularly difficult. Final diagnosis is established by histological analysis.[1–3] CCAM is found in most cases to be an isolated finding, although type 2 has been described with other anomalies.[1,2] Whereas the existence of further congenital anomalies, such as lung, cardiac, diaphragm or chest wall anomalies, is more frequent in PS, particularly in cases of EL-PS, it only happens in 15% of IL-PS cases.[1,5] The mainstay of treatment for CCAM and PS is surgical resection, lobectomy is the usual procedure of choice.[1–3] The authors present this case because it is so rare. Although uncommon and infrequently diagnosed in adults, congenital malformations should be included in the differential diagnosis of repeated infections, particularly if they are on the same lobe.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Yellow bronchoscopy: A severe case of tracheal and bronchial Jaundice

Dear Editor,

Jaundice, which can be caused by a very wide variety of conditions, results from the increased levels of bilirubin in the blood. Due to the affinity of bilirubin to elastin, jaundice is best detected by examining the skin and sclera, but can also be found in others elastin-rich tissues such as skin, sclera, intima of the vessel wall and ligaments.

We present the case of a 68-year-old nonsmoker male who was referred to the emergency department for evaluation of 1-week history of jaundice, light-colored stools, dark urine, anorexia and weight loss. Blood work showed an elevated direct bilirubin 9.34 mg/dL (normal, 0–1 mg/dL) and he was diagnosed with a carcinoma of the pancreas head.

Bronchoscopy was performed during a pulmonary nodule investigation and revealed a nodule on the left side of the epiglottis and severe vocal cords and bronchial tree jaundice (Fig. 1).

Bronchoscopy is not a frequently performed exam in patients with such high levels of bilirubin in the blood. In the present case bronchoscopy was performed due to the nodule location, making it difficult to access through transthoracic lung biopsy and due to the need for pulmonary metastasis exclusion in order to get a correct staging.

The high amount of elastin present in the airways makes it possible to observe a fine yellow coloration characteristic of jaundice in the bronchial tree, as observed in these endoscopic images of rare beauty and iconographic interest.

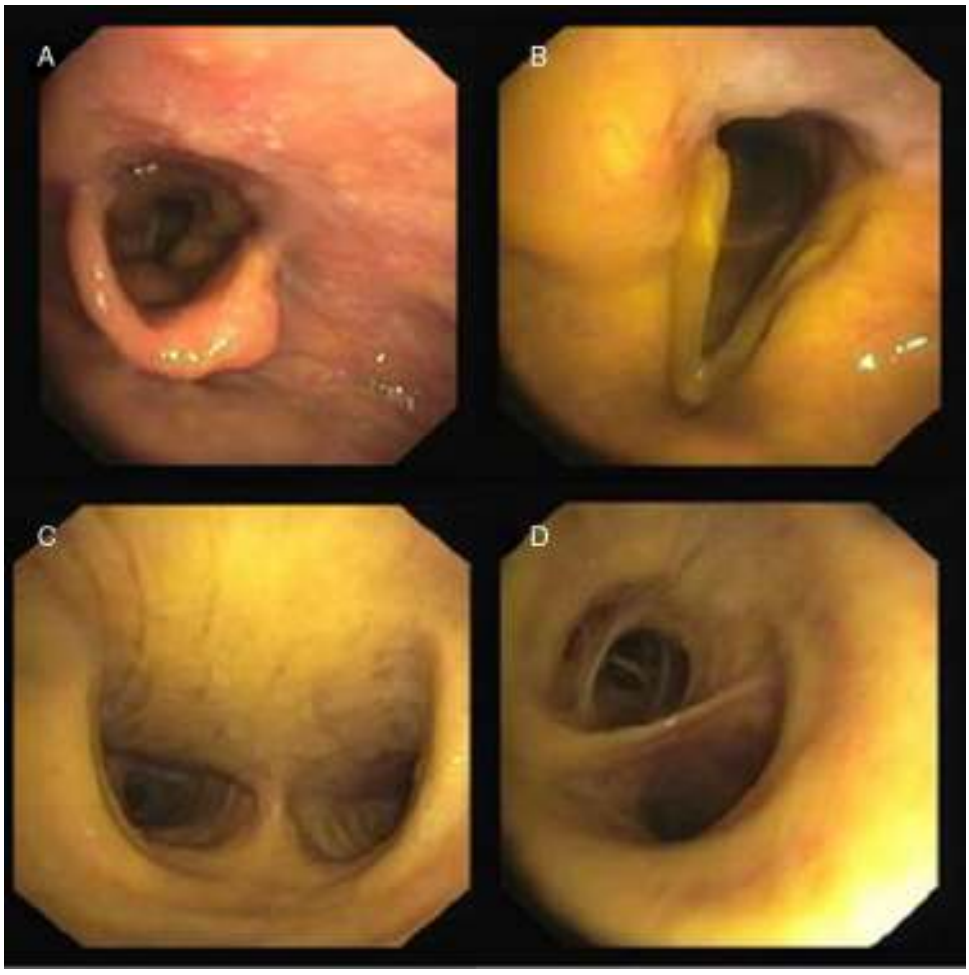

Fig. 1 (A) Epiglottis; (B) Vocal cords; (C) Carina; (D) Truncus intermedius.

Conflict of interest

The authors have no conflicts of interest to declare.

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Misconceptions in the assessment of cough peak flow measurements for extubation or decanulation protocols

Dear Editor,

Winck et al. proposed routine use of cough peak flow (CPF) measurements in the assessment of extubation and decanulation readiness.[1] However, intubated patients cannot cough because they cannot close their glottis to hold pressure, and the paper the authors cited suggesting that 90 L/m predicts successful extubation[2] is irrelevant for patients with neuromuscular respiratory muscle weakness since they can almost always be successfully extubated even when their unassisted CPF and vital capacities are unmeasurable.[3,4] The paper that the authors referred to that used 160 L/m as a reference reported the need for maximum unassisted or assisted CPF to reach 160 L/m after extubation.[5] Assisted CPFs are attained by patients air stacking to the maximum lung volumes that can be held by the glottis and then an abdominal thrust is applied to generate CPF.[6] It is the maximum flow that can be generated through the upper airways that is important and not whether the patient can generate that flow him/herself because the greater the flow, the greater the patency of the airways, and the more effective mechanical insufflation–exsufflation (MIE) will be in elimi-