Discussion

Mediastinal parathyroid cysts are relatively rare. These unusual lesions can present with locoregional clinical manifestations and, when they are active from a biochemical point of view, can also be associated with systemic symptoms. In 1925, de Quervain published the first case of satisfactory surgical resection of a parathyroid cyst in the mediastinal compartment,[1] although to date fewer than 150 cases have been described among published studies.

The non-specific symptoms are related with the appearance of a space-occupying lesion, such as the one described in this report. The cysts can cause dyspnea as a result of marked tracheal deviation or stenosis, or dysphagia due to esophageal compression or deviation.

The differential diagnosis is usually established by radiological examinations combined with laboratory tests, which show an increase in the serum calcium and/or PTH concentration.

In the context of a state of hyperparathyroidism, a functioning mediastinal cyst must be considered, so its resection should be performed with simultaneous cervical examination, which is easy to perform for cervical cysts infiltrating or located in the anterior mediastinum. For parathyroid cysts in the posterior mediastinal compartment that require thoracotomy, simultaneous cervicotomy appears to be overtreatment, and should only be considered after demonstrating persistent hyperparathyroidism after surgery.

No deaths have been documented among published studies, although the resection of giant cysts can rarely be associated with postoperative complications.[2] VATS has been described for the successful management of these cystic lesions, although all cases refer to small cysts located in the prevascular and anterior mediastinum.[3–5]

The exceptional nature of this report resides in the large size of the parathyroid cyst, which ruled out a VATS approach. However, it was successfully resolved using a thoracotomic approach without any postoperative morbidity, which resulted in complete clinical resolution of the presenting symptoms.

Funding

This study was carried out as part of research grant "Dottorato di Ricerca – XXVI ciclo – Scienze Chirurgiche – Alma Mater Studiorum Università degli Studi di Bologna".

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Respiratory Infection Caused by *Bordetella hinzii*☆

Infección respiratoria por Bordetella hinzii

To the Editor,

Bordetella hinzii is a species isolated in poultry with respiratory disease, and has occasionally been described in humans. We present the case of an 85-year-old woman with respiratory symptoms consisting of cough, expectoration and dyspnoea on moderate exertion of several days duration. Sputum culture was requested from her primary care centre. It was inoculated in blood agar, chocolate agar and MacConkey agar (Oxoid®). The sample quality was considered optimal after Gram staining, with abundant polymorphonuclear leukocytes and Gram negative bacilli found on examination. After 24 h incubation at 35 °C, profuse growth of two types of colonies was detected on all three media. Two colony morphologies were observed on the MacConkey agar: a pinkish, mucoid colony, and a second colourless colony. Identification performed using the MALDI-TOF–MS biotyper 3 system classified them as *Klebsiella oxytoca* and *B. hinzii*, the latter with an index of 2.241, which is considered optimal for genus and species. Molecular confirmation was carried out by sequencing 500 base pairs of the 16S rRNA gene[1] and comparing the sequence obtained with those banked in GenBank, using the NCBI BLASTn algorithm. Ninety-nine percent homology with other existing *B. hinzii* strains was obtained, including *B. hinzii* strain LMG 13501.[1] The antibiotic treatment prescribed in the emergency department at discharge was amoxicillin–clavulanate 2000/62.5 mg every 12 h. Antibiotic susceptibility testing performed using the WalkAway® system microdilution panel (Siemens) found sensitivity to the following antibiotics: amoxicillin–clavulanate (MIC≤8/4 μg/ml), azithromycin (MIC=1.5 μg/ml), piperacillin–tazobactam (MIC≤ 8 μg/ml), gentamicin (MIC=4 μg/ml) and levofloxacin (MIC≤ 1 μg/ml); intermediate sensitivity to: ampicillin–amoxicillin (MIC=16 μg/ml) and ciprofloxacin (MIC=2 μg/ml), and resistance to cefuroxime (MIC>16 μg/ml), tobramycin (MIC>8 μg/ml) and trimetoprim–sulfametoxazol (MIC>4/76 μg/ml). Since *B. hinzii* was first described in 1994,[1] isolates have been reported in respiratory samples from three patients. In one patient with cystic fibrosis, *B. hinzii* was isolated sequentially in sputum for a period of 3 years,[2] together with *Staphylococcus aureus* in all samples except two, where it was the only microorganism isolated. It was also isolated in the bronchoalveolar lavage of a patient with acquired immunodeficiency virus (HIV), together with *Nocardia asteroides*.[3] In this case, there were symptoms of respiratory infection, as in our patient. The aetiological role of *B. hinzii* may be questioned in our case, as *K. oxytoca* was cultured in par-

☆ Please cite this article as: Palacián Ruiz MP, et al. Infección respiratoria por *Bordetella hinzii*. Arch Bronconeumol. 2013;49:409–10.

allel in the respiratory sample, but in the literature it appears as the only microorganism isolated in culture that causes the disease.[4] *B. hinzii* also appears as a causal agent of bacteraemia in three cases described in the literature, with immunosuppression the common factor in two of them: one patient with myelodysplastic syndrome and another patient with HIV.[4] Similarly, *B. hinzii* also appeared as a causal agent of chronic cholangitis in a transplant patient on immunosuppressant treatment,[5] so it is considered that it may have a potentially pathogenic role in immunocompromised persons. There were no findings in our patient to suggest immunosuppression. As in some previous publications,[2,3] our patient had no known avian exposure, suggesting that the organism was obtained from another unidentified source. *B. hinzii* is usually resistant to, or has intermediate resistance to ampicillin, cefuroxime, ceftriaxone, cefotaxime, ciprofloxacin and tobramycin, and is sensitive to imipenem, meropenem, gentamicin, amikacin and trimetoprim–sulfametoxazol, which was similar to the antimicrobial sensitivity of our isolate, except as regards trimetoprim–sulfametoxazol. Molecular identification using MALDI-TOF–MS and 16S sequencing, as was performed here, provides the correct microbiological diagnosis.[6] The use of rapid techniques that increase the reliability and speed of identification of this microorganism could lead to clarifying its role as a coloniser and human pathogen.

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Bilateral Pulmonary Sequestration in an Adult: Case Report and Review of the Literature☆

Secuestro pulmonar bilateral en el adulto: aportación de un caso y revisión de la literatura

To the Editor,

We present the case of a 64-year-old man, ex-smoker, with clinical indicators of chronic bronchitis. He had been on home oxygen for more than 10 years and was on treatment for sleep apnoea-hypopnoea syndrome (SAHS) with nocturnal continuous positive airway pressure (CPAP). He was admitted in 1990 for pneumonia in the left lower lobe (LLL), secondary to symptoms of near-drowning in seawater. During the follow-up, triangular consolidation persisted in the LLL, but both computed tomography (CT) and fibrobronchoscopy failed to provide a diagnosis. CT-guided transthoracic needle aspiration was performed, with cytology negative for malignancy and a negative microbiological study. The patient was admitted on several occasions in recent years for exacerbation of COPD. A new chest CT scan was requested in 2004 due to consolidation in the LLL, and he was diagnosed with possible left intralobar pulmonary sequestration (PS). The patient refused any further studies. In 2009, he was admitted for pneumonia. The chest CT scan (Fig. 1) showed a 6–7-cm complex formation, in a medial location in the LLL, supplied by a large vessel originating in the inferior thoracic artery with drainage to pulmonary veins, corresponding to an intralobar PS with probable superinfection; in a symmetrical location, contralateral, there was another abnormal vessel, also originating in the aorta, corresponding to another intralobar PS in the right region. After improvement with treatment, he was discharged for follow-up at the clinic, but did not attend his check-ups. Most intralobar PS are unilateral, and bilateral PS are very rare.[1] For Kohler, PS was first described by Rokitanski and Rektorzik in 1861, but it was not until 1946 when Pryce[2] made it known as a clinical entity. In 1972, Felson et al.[3] reported a case of bilateral PS confirmed by pathological examination. In 1977, Karp et al.[1] described another case in a 13-year-old girl who presented a mass in the LLL, discovered after a chest radiograph. CT scanning and pulmonary arteriography were carried out, showing a left intralobar PS, already suspected, and a right PS that was not previously observed. A few cases have been published since then, similar in that they concern paediatric or adolescent patients. In 2009, Yamamura et al.[4] explained the surgical procedure performed on a 44-year-old patient who presented bilateral intra- and extralobar PS. Wei and Li[5] analysed 2625 cases of PS in 2011, among which only 3 were bilateral and 2 of these intralobar. The case presented is doubly unique in that this is an older patient with bilateral intralobar PS. He presented recurrent pneumonia with a changing aspect in the LLL, in which diagnostic tests were inconclusive. The possi-

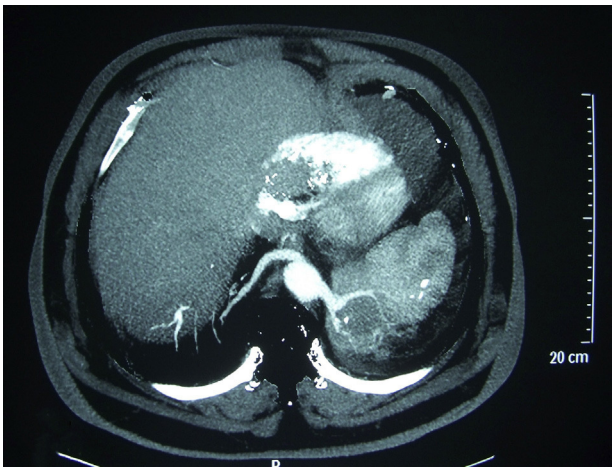

Fig. 1. CT angiography. The feeding arteries of both sequestrations, subsidiaries of the thoracic artery, can be seen.

☆ Please cite this article as: Fontalba Navas M, et al. Secuestro pulmonar bilateral en el adulto: aportación de un caso y revisión de la literatura. Arch Bronconeumol.2013;49:410–11.