Necrotizing Pneumonia Due to *Bordetella bronchiseptica* in an Immunocompetent Woman

To the Editor: *Bordetella bronchiseptica* is a gram-negative pleomorphic coccobacillus found in the upper respiratory tracts of wild and domestic animals, often causing the condition known as kennel cough.[1] There are few published cases of respiratory disease caused by *B bronchiseptica* in humans although initially its ability to produce acute bronchitis[2] and even family outbreaks in children[1] has been described. Nevertheless, it causes pneumonia in immunocompetent subjects exceptionally[3] and does so with greater frequency in immunocompromised patients.[1]

We report the case of a woman with no risk factors for immunodepression suffering from severe pneumonia due to *B bronchiseptica.* To our knowledge, this is the first case in a patient with pulmonary emphysema in the Spanish literature.

The patient was a 68-year-old housewife with hypertension, irritable bowel syndrome, and pulmonary emphysema. She was an ex-smoker of 40 pack-years who had lived with a dog for the past 10 years and had been admitted to our hospital a year earlier with community acquired pneumonia of the middle lobe. Admission followed a week of dry cough, pleuritic pain on the right side, and increased baseline dyspnea, eventually occurring at rest. In the emergency department the patient's general condition appeared good and she had a low-grade fever. Breathing was normal and hemodynamic parameters were stable. Auscultation of the lungs revealed a slight increase in vocal resonance in the right upper field, with no adventitious sounds. The rest of the physical examination was normal. The hemogram showed leukocytosis with no left shift, biochemistry revealed mild hyponatremia, and there was a slight increase in D-dimer levels. All other hematology parameters were normal. Blood gas analysis with the patient breathing room air showed a pH of 7.49, $PaCO_2$ of 33.2 mm Hg, PaO_2 of 54 mm Hg, bicarbonate concentration of 25.2 mmol/L, and oxygen saturation of 90.2%. A chest x-ray showed an alveolar interstitial infiltrate with a loss of volume in the right upper lobe together with signs of pulmonary hyperinflation and reduced vasculature in both lung fields. Empirical treatment was initiated with 1 g/8 h of intravenous amoxicillin—clavulanic acid and intravenous levofloxacin was added at a dosage of 500 mg/12 h on the third day due to persistence of the fever. Computed tomography angiography ruled out

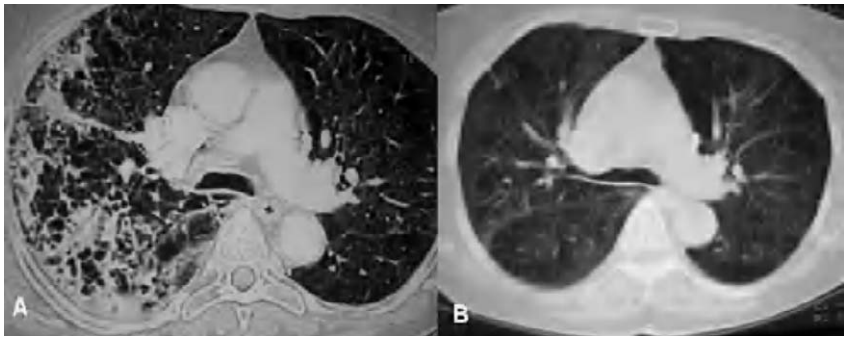

Figure. *A*: Computed tomography of the chest showing extensive alveolar infiltration with areas of necrosis and centrilobular emphysema in the right upper lobe. *B*: Computed tomography of the chest 45 days after admission.

pulmonary embolism but showed an alveolar infiltrate with areas of necrosis and septal thickening in the right upper lobe, centrilobular emphysema predominantly in the upper lobes, and involvement of multiple lymph nodes located in the right paratracheal, precarinal, subcarinal, and bilateral hilar regions and in the aortopulmonary window (Figure 1A). Fiberoptic bronchoscopy revealed no lesions indicating malignancy, although it did show changes indicative of chronic bronchitis. Bronchoalveolar lavage and protected brush catheter examination were performed, as well as fine-needle aspiration on the affected mediastinal lymph nodes. The bronchoalveolar lavage isolated 10^4 colony-forming units of *B bronchiseptica,* sensitive to amoxicillin—clavulanic acid, gentamicin, and tobramycin with intermediate sensitivity to ciprofloxacin, and resistant to cefotaxime and co-trimoxazole. The Ziehl stain and culture were negative for the remaining samples and cytology was negative for malignancy. The blood cultures and urine antigen assays for *Legionella pneumophila* and *Streptococcus pneumoniae,* and respiratory serology, including for *Aspergillus fumigatus,* were negative. Lung function tests showed a mixed breathing pattern that was predominantly moderately obstructive, with a positive bronchodilator test, and plethysmography revealed an increase in residual volume due to air trapping, with moderate reduction in pulmonary diffusion corrected for alveolar volume. Clinical symptoms and blood gas analysis results improved and the patient was released 12 days after admission under treatment with 875/125 mg/8 h of amoxicillin-clavulanic acid and 500 mg/24 h of levofloxacin, taken orally, for a further 21 days. In subsequent follow-up examinations, tomography showed slow but gradual improvement. Three months after admission, there was a slight increase in peripherally located residual densities associated with pleural and parenchymal fibrous tracks and extensive changes in the bullous emphysema in both hemithoraces (Figure 1B).

B bronchiseptica causes pneumonia in patients with immunodepression related to solid tumors, Hodgkin's disease, bone-marrow transplant, hemodialysis, cystic fibrosis, and, principally, acquired immune deficiency syndrome.[4] The ability of this bacterium to colonize and cause infection of the respiratory tract depends on many virulence factors, including adhesins such as filamentous and fimbrial hemagglutinins, and it can produce dermonecrotic toxins, tracheal cytotoxin, and hemolysins, thus allowing it to adhere to the epithelial cells and persist in the lower respiratory tract. Furthermore, it can inhibit white cell function and cause apoptosis of alveolar macrophages.[5] BvgAS, a 2-component signal transducing system that regulates the expression of all the proteic virulence factors, has been identified for *B bronchiseptica* and other members of the genus.[6] Persistence in the host despite the presence of specific antibodies has been shown in respiratory infection models in mice, indicating that the bacillus may persist within the cells.[5] Our patient presented recurring pneumonia—the second time with considerable parenchymal necrosis—possibly due to the aforementioned virulence factors or to repeated exposure to the reservoir of zoonoses. Treatment of these broncho-pulmonary infections is difficult and relapses have been described.[2] Prolonged treatment with antibiotics is recommended, especially in immunocompromised patients.

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