Empyema *Necessitatis* Caused by *Corynebacterium jeikeium:* 19th Century Questions, 21st Century Answers☆

Empiema necessitatis *por* Corynebacterium jeikeium: *preguntas del s.* XIX, *respuestas del s.* XXI

To the Editor,

Empyema necessitatis is a rare complication of pleural infections treated late or inadequately. It is characterized by pus spreading from the pleural cavity through the underlying tissues to form an abscess in the chest wall that sometimes even forms a skin fistula.[1,2] Most cases are caused by *Mycobacterium tuberculosis* infection, although it has also been described, albeit less frequently, in association with other microorganisms such as *Streptococcus pneumoniae*, *Staphylococcus*, Gram-negative bacilli and polymicrobial infections.[1] We report a case of empyema necessitatis caused by *Corynebacterium jeikeium*. Treatment was complex but exclusively medical, and to our knowledge this is the first case with these characteristics described in the literature.

Our patient was an 84-year-old man with a history of pulmonary tuberculosis (TB) at the age of 20 years, treated with collapse therapy. He consulted due to a painful swelling in the right lower hemithorax in the upper quadrant of the abdomen, accompanied by dyspnea, non-productive cough, and fever of 38.2 °C. Of note on examination was an erythematous, swollen, painful area in the lower axillary mid-line, measuring 8 cm (Fig. 1A). Signs of right pleural effusion were detected on auscultation. Chest X-ray showed right pleural effusion encapsulated with extensive fibrothorax and loss of volume with an image of a calcified outer surface, measuring about 14×8 cm, and bronchiectasis. Chest CT revealed a pleural collection with a calcified outer shell, air-fluid level, and a soft tissue lesion in the lower chest wall measuring 8.2×7 cm, containing fluid. The lesion extended through the intercostal space to the right subphrenic region (Fig. 1B). Communication was identified between the most posterior and anterior area of the fibrothorax and the described mass. Fiberoptic bronchoscopy (FB) ruled out bronchopleural fistula. Both collections were drained by incision and evacuation of the purulent material from the abscess in the chest wall, and a 22F chest tube was placed, with total drainage of 2400 ml of pus. Urokinase was instilled every day and the abscess was packed for healing by secondary intention. Serial cultures of pleural fluid and FB specimens were negative for mycobacteria, and aerobic and anaerobic bacteria. The patient was treated with amoxicillin-clavulanic acid, initially intravenously and then orally for a total of 3 months, after which he remained asymptomatic. Radiological monitoring showed resolution of the pleural effusion, disappearance of the fibrothorax, and cure of the skin abscess. Seven months later, the patient presented spontaneously due to general malaise and recurrence of the skin abscess, although it was smaller in size than the initial lesion. X-ray confirmed relapse of the empyema. Thoracentesis retrieved pus with similar characteristics and an aerobic, Gram-positive, catalase-positive bacteria identified as *C. jeikeium* was grown on culture. Susceptibility testing showed a multiresistance pattern, with susceptibility to erythromycin, vancomycin, and clindamycin only. A Veress needle was used to drain 800 ml from the pleural cavity and local abscess care was given. After outpatient treatment with clarithromycin 1 g/24 h and clindamycin 300 mg/8 h, the patient's clinical and radiological situation had resolved and hospital admission was not required.

Empyema necessitatis is a rare complication that occurs when pleural empyema spreads to form an abscess in the chest wall, penetrating the adjacent tissues and forming a skin fistula. It occurs most frequently in the subcutaneous tissue of the chest wall, between the clavicular and the anterior axillary mid-lines,[3] although it has also been described in the esophagus, paravertebral soft tissue, retroperitoneum, pericardium, and groin. Empyema necessitatis was already a rare complication (10%) in the days before antibiotics. Up to 75% of the cases were caused by *M. tuberculosis*,[1] although it only grows in 10%–40% of cultures.[1] Other causes described less frequently are pyogenic infections (*Staphylococcus aureus*, *S. pneumoniae*, Gram-negative bacilli), actinomycosis, cancers,[1,2,4] or complications after chest injuries,[3] pneumonectomy, or thoracoplasty.[5] Clinical and radiological suspicion are essential. CT is useful for confirming the diagnosis and for evaluating communication of the collection with the pleural space through the chest wall and revealing, as in our case, an extrapleural mass in the chest wall, in addition to the findings in the pleural cavity. Chest ultrasound is also useful for evaluating both the lesions and their progress, while avoiding unnecessary irradiation. *C. jeikeium* is a Gram-positive aerobic bacteria that on rare occasions causes opportunistic infections.[6] Cases of bacteremia, septicemia, endocarditis, and osteomyelitis have been described, but this is the first report of this microorganism causing empyema necessitatis.

Although the standard treatment of a chronic chest cavity infection, isolated from the rest of thorax, and with no chance of

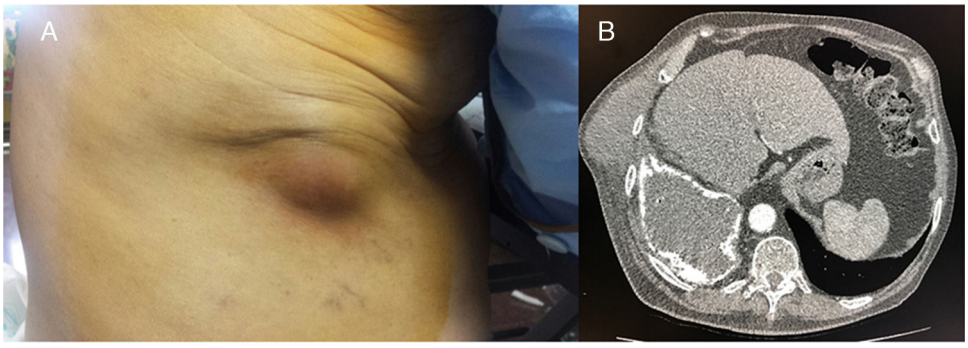

Fig. 1. (A) Painful swelling in chest wall. (B) Computed axial tomography showing pleural effusion with calcified surface and rupture with fistulous tract between both collections.

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becoming occupied by lung parenchyma, should be open thoracostomy with intensive draining and healing by second intention, we treated our patient conservatively, given his advanced age and underlying disease.

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Bacteremic Pneumococcal Pneumonia in Adults☆

Neumonía neumocócica bacteriémica en el adulto

To the Editor,

Streptococcus pneumoniae is one of the major microorganisms involved in community-acquired respiratory infections, including pneumonia, which may occur with pneumococcal bacteremia.[1] The incidence of bacteremic pneumococcal infection is higher in certain groups of the population, particularly the elderly and patients with underlying debilitating diseases. This severe invasive presentation is associated with high mortality.[2] The inclusion of antipneumococcal vaccination in European immunization programs and the selective pressure of antibiotics on the circulating strains have produced changes in the incidence and epidemiology of the disease.[3] Recent years have seen a re-emergence of strains of *S. pneumoniae* resistant to penicillin and other antimicrobial agents, making these infections difficult to treat and complicating the prognosis. The main limitation of the 23-valent polysaccharide vaccine (PPSV23) is that it has shown no significant effect on the risk of developing bacteremic pneumococcal pneumonia (BPP).[4] The impact of the 13-valent conjugate vaccine (PVC13) on bacteremia and other invasive forms of pneumococcal disease in adults is still under evaluation.[4] In this paper, we describe the epidemiological characteristics and predictors of mortality in patients with a diagnosis of BPP, and the antimicrobial resistance profile and distribution of serotypes of the isolated strains. All patients older than 14 years diagnosed with pneumonia and positive blood culture for *S. pneumoniae* during the period 2011–2016 were selected. In total, 159 episodes of BPP in 159 patients were included. Distribution by sex was 100 (62.9%) men and 59 (37.1%) women. Mean age was 75 years (IQR: 59–85); 73% patients showed a minimum inhibitory concentration (MIC) ≤0.06 for penicillin and 94% showed an MIC ≤1 for cefotaxime. As for other antimicrobials, 8% were resistant to levofloxacin, 27% to erythromycin, and 20% to clindamycin. Twenty-eight percent of the clindamycin-resistant strains showed an inducible resistance pattern. Serotypes were determined in 158 strains, and up to 33 different serotypes were identified. The most frequent were serotypes 3 (16%), 19A (11%), 14 (8%), 22F (7%), 19F (5%), 6C (4%) and 12F (4%).

The group of strains with serotype 19A was of particular interest, as they showed a high rate of resistance to each of the antibiotics studied: 67% had a MIC >2 for penicillin, 70% had an MIC >1 for cefotaxime, and 33%, 28% and 31% of the strains were resistant to erythromycin, clindamycin, and levofloxacin, respectively. Mean incidence of BPP was 7.8 cases per 100 000 inhabitants/year (range: 3.9–10.6) (Fig. 1). In total, 72% of patients had an underlying disease, such as diabetes, heart disease, cancer, respiratory disease, immunosuppression, among others. Overall in-hospital mortality was 23%. Risk factors significantly associated with mortality were age ≥65 years (OR: 3.13; 95% CI: 1.21–8.07; *P*=0.02), heart disease (OR: 2.61; 95% CI: 1.09–6.25; *P*=0.03), and cancer (OR: 3.13; 95% CI: 1.35–7.25; *P*=0.01). Despite the aging of the population and the increase in life expectancy of patients with debilitating diseases, we did not observe a significant variation in the incidence of BPP between the beginning and the end of the study period. The number of strains in which penicillin resistance was detected (27%) is similar to figures published in the literature.[5] In our population we are unaware of vaccination rates with PCV13 and PPV23, but PPV23 vaccination in the adult population (>60 years) in Spain ranges between 52.5% and 66%.[6,7] Despite this, serotype 19A, included in the PPV23 and PCV13 vaccines, was the second most common, and the one that was most frequently associated with multiresistance. In our series, we found no association between mortality and the different serotypes, due probably to a type II error. BPP mortality ranges from 13% to 16%, and is higher in certain population groups, such as the elderly, who present mortality rates of 22%–51%.[8] The high mortality rate found in our study (23%) may be due to the high proportion of elderly patients with comorbidities in our series.

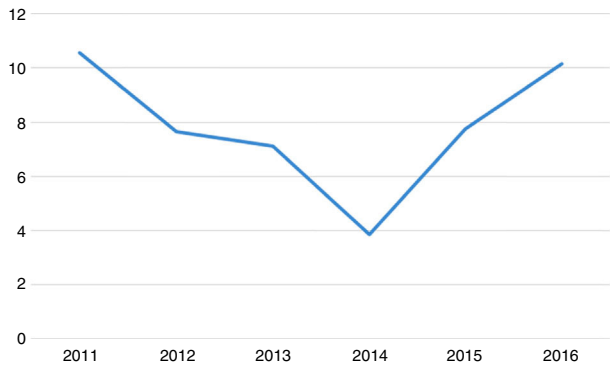

Fig. 1. Annual BPP incidence rate expressed per 100 000 inhabitants/year.

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