

The results of the treatment of 19739 cases of TB were evaluated in both periods, the median of therapeutic success (88% versus 60.5%,  $P=0.01$ ), the median of failure (0.3% versus 0.85%,  $P=0.02$ ) and the median of dropouts (7.1% versus 34.25%,  $P=0.01$ ) were significantly different. No difference was found in the deceased (3.1% versus 3.7%,  $P=0.73$ ). The results of the patients' treatment are shown in [Table 1](#).

Our study showed that between 2002 and 2008, when basic food baskets were distributed to TB patients, the success rate of treatment was higher than in the period from 2009 to 2014, when there were no food baskets. With the removal of such incentives to patients, there was a considerable increase in treatment withdrawal. Similar to our study, a study conducted in Afghanistan found that the treatment success increased from the moment that the WFP introduced food assistance to TB patients.<sup>4</sup> In Brazil, the provision of basic food baskets significantly increased adherence to TB treatment.<sup>11</sup> Other studies have also shown the positive impact of incentives on patients' adherence to TB treatment,<sup>12,13</sup> taking into account the socio-economic difficulties experienced by these vulnerable groups.<sup>3</sup>

We assume that the incentive impact assessment in the outcome of treatment in our study was limited to the analysis of grouped data and that the sociodemographic and socioeconomic data of each patient would need to be assessed for a more complete analysis. However, our analysis strongly suggests the importance of incentives in patients' adherence to treatment, and therapeutic success against TB.

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## S. aureus Pneumonia and Sternoclavicular Septic Arthritis: An Unusual Complication\*



### Neumonía por S. aureus y artritis séptica esternoclavicular, una complicación insólita

To the Editor,

A rare complication of community-acquired pneumonia (CAP) is septic arthritis (SA). This infection is exceptional, and does not usually occur simultaneously with the acute process.<sup>1,2</sup> It is most commonly caused by *Streptococcus pneumoniae*.<sup>1,3</sup> The joints most often affected in these cases are the knee, the hip and the shoulder.<sup>1,3</sup> Most SA are caused by *Staphylococcus aureus* and *Streptococcus*, and develop in patients with predisposing risk factors.<sup>1–6</sup> We report a case of pneumonia due to *S. aureus*, accompanied simultaneously with a sternoclavicular septic arthritis (SSA) due

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to bacteremia in a young woman without known risk factors. This is the first report of this association in the literature.

A 60-year-old woman, non-smoker, with no history of immunosuppression or risk factors for lung disease attended the emergency room with pleuritic pain and pain in the left shoulder. 38 °C fever, cough without expectoration and intense pain in the mid-clavicular region and left shoulder. Chest radiograph revealed mild infiltrate in the left lower lobe. Signs of consolidation and egophony in the left lower field were detected on examination. A very painful non-fluctuating swelling with no signs of inflammation in the skin was observed in the upper left sternoclavicular region. Fiberoptic bronchoscopy was performed, revealing signs of inflammation, and samples were sent for pathology and microbiology studies. Blood cultures were negative. No valvular lesions or vegetations suggestive of endocarditis were observed on echocardiogram. Empirical treatment with ceftriaxone and levofloxacin was started, with little clinical response, while morphine was required for pain control. Chest computed tomography (CT) showed inflammatory changes in the left pectoral muscle and a pseudonodular image measuring 3.3×2.3 cm in the space between the anterior portion of the first and second left

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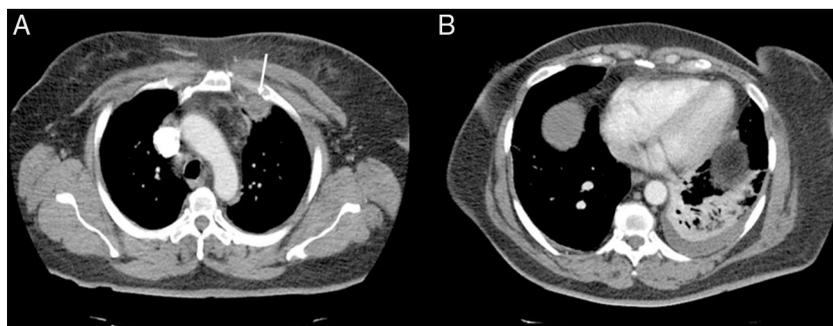


Fig. 1. Pseudonodular image in the space between the first and second left costal arch (arrow), and simultaneous consolidation in the left lower lobe.

costal arch, and pulmonary consolidation with pleural effusion in the left lower lobe (Fig. 1). Findings were confirmed on both ultrasonography of the neck and bone scintigraphy. Ultrasound-guided fine needle aspiration and biopsy was performed, from which *S. aureus* was isolated. The strain was resistant to ampicillin, and susceptible to erythromycin, gentamicin, clindamycin, ciprofloxacin, levofloxacin, and cotrimoxazole. The same microorganism was isolated from the bronchoscopy samples. During admission, intravenous ciprofloxacin and amoxicillin–clavulanic acid were administered, in line with susceptibility results, and improvement was observed in clinical symptoms, radiological signs, and acute phase reactants. Drainage was not required. Treatment continued on an outpatient basis for another 40 days, with complete resolution of the syndrome.

SSA is exceptional and accounts for only 1%–9%<sup>2,4</sup> of SA, and generally occurs in patients with debilitating risk factors and immunosuppression.<sup>1–6</sup> It is also unusual to see the simultaneous development of SA in the acute period of an episode of pneumonia, as it tends to occur later.<sup>1,2</sup> In our patient, the SSA was attributed to the bacteremic pneumonia, as the same microorganism was isolated. *S. aureus* pneumonia in a patient without risk factors is in itself exceptional. The clinical picture of SSA, in contrast to our case, is generally insidious, and presents with fever, pain in the shoulder, and edema and erythema in the sternoclavicular joint.<sup>1,2,4–6</sup> The most widely used diagnostic test is ultrasound, although CT can identify the degree of bone destruction, and scintigraphy is used to delimit the inflammatory area and guide the biopsy and aspiration procedure. The definitive diagnosis depends on isolation of the microorganism. This will indicate the appropriate antibiotic therapy, which should continue for at least 4 weeks in the absence of

complications.<sup>1–3,5,6</sup> Surgical treatment is recommended in case of extensive osteomyelitis, abscesses, empyema, or mediastinitis.<sup>1,4,5</sup> In conclusion, pneumonia can unusually cause SA, and exceptionally SAA, and these entities may go unnoticed in the clinical context. As this process is potentially disabling and possibly fatal, etiologic diagnosis should not be delayed.

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## Pediatric Interstitial Lung Disease: An Ongoing Challenge<sup>☆</sup>



### La enfermedad pulmonar intersticial en el niño. Todavía hoy un reto diagnóstico

To the Editor,

Surfactant protein C deficiency causes interstitial lung disease (ILD) of varying severity. Diagnosis in children is complex, due to the rarity and heterogeneous clinical manifestations of this

entity.<sup>1,2</sup> We report a case of this disease that was initially incorrectly diagnosed.

Our patient was a boy, born at term, with no significant history. At the age of 14–15 months, he was admitted twice due to bronchiolitis caused by syncytial respiratory virus and bronchitis due to adenovirus. After the second admission, persistent tachypnea, respiratory failure, and bilateral infiltrates on chest X-ray were observed. Further examinations ruled out malformations, immunodeficiencies, pulmonary hypertension, and other infections. Bronchoalveolar lavage was performed: cell count was normal, with no eosinophilia, and negative Gram stain and microbiological cultures. Lung computed tomography (CT) showed a diffuse ground glass pattern with hilar lymphadenopathies. Lung biopsy was performed by thoracoscopy. The specimen was sent to a reference laboratory, and the report described “changes associated with bronchiolitis obliterans”. The patient was discharged at the

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