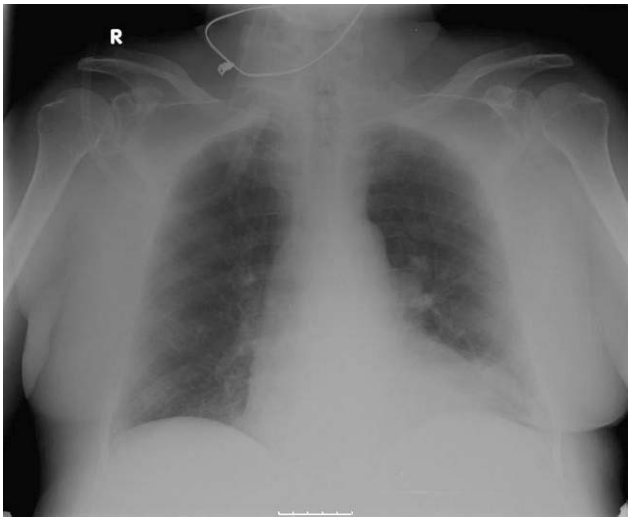

Figure. Posteroanterior chest x-ray showing signs of precapillary pulmonary hypertension and increased linear markings (tram trackings).

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Factors That Modify C-Reactive Protein Production in Patients With Community-Acquired Pneumonia

Factores que modifican la Producción de Proteína C Reactiva en Pacientes con Neumonía Adquirida en la Comunidad

To the Editor:

Although a number of studies have assessed the value of C-reactive protein in managing patients with community-acquired pneumonia, their results have been inconsistent. Furthermore, to differentiate between community-acquired pneumonia and other diseases, different C-reactive protein values have been proposed as the best cutoff point: 50 mg/L, 100 mg/L, and 125 mg/L.[1] Some studies, moreover, have linked high admission levels of C-reactive protein with the etiology or prognosis in patients with community-acquired pneumonia, whereas others have failed to confirm this association.[1-3] Although most guidelines currently used in managing community-acquired pneumonia do not include C-reactive protein assessment as a tool to assist clinical decision making, some guidelines, including those of the British Thoracic Society, recommend that further prospective studies be conducted in order to further investigate the potential value of C-reactive protein levels in the management of patients with community-acquired pneumonia.[1,4]

Given this background, we conducted a study designed to determine whether C-reactive protein admission values might be affected by specific patient characteristics. Accordingly, we analyzed the influence of age, sex, comorbidity, the number of days since the onset of infection, and previous treatment with antibiotics on C-reactive protein levels in a cohort of 161 patients attended to consecutively in the emergency department of our hospital. The sample, which consisted of 112 men (69.6%), had a mean (SD) age of 63.1 (18.5) years and included 84 patients (52.2%) with chronic diseases. Disease severity was measured using the Pneumonia Severity Index, with patients categorized as follows: 31 patients (19.2%) in class I, 72 patients (44.7%) in classes II and III, 45 patients (27.9%) in class IV, and 13 patients (8.0%) in class V. Hospitalization was necessary for 78 patients (48.4%), and 17 patients (10.5%) died while in hospital. The cause of the pneumonia was identified in 65 cases (40.3%). The most frequently encountered microorganisms were *Streptococcus pneumoniae* (42 patients, 26%), *Chlamydia pneumoniae* (8 patients, 4.9%), *Legionella pneumophila* (6 patients, 3.7%), and *Mycoplasma pneumoniae* (3 patients, 1.8%). Serum samples were analyzed for C-reactive protein in a turbidimetric assay (Tinaquant; Roche Diagnostics, Mannheim, Germany) following the manufacturer's instructions and for a C-reactive protein detection range of 1 mg/L to 560 mg/L.

The assay revealed that 155 patients (96.2%) had C-reactive protein levels above 100 mg/L on admission. The Table summarizes the associations between patient C-reactive protein values and the variables analyzed. Sex, the presence of comorbidities, the number of days since onset, and previous treatment with antibiotics had no bearing on C-reactive protein levels on admission. C-reactive protein levels were, however, lower in elderly patients. No relationship was found between C-reactive protein levels and either death during hospitalization (217.6 [159.4] mg/L) or recovery (286.4 [159.8] mg/L) (*P* not significant).

The results of our study indicate that age may have a bearing on C-reactive protein levels. Our results are, furthermore, consistent with previous sepsis model analyses that found that the production of tumor necrosis factor-α and interleukin 1β—both considered to be stimulants of C-reactive protein production[5]—was lower in elderly patients. For this reason we are of the opinion that future research into the value of C-reactive protein in respiratory infections should take into account patient age, and should assess the usefulness replicated C-reactive protein measurement.[6]

Table
Influence of Characteristics of Patients With Community-acquired Pneumonia on C-Reactive Protein (CRP) Values

	Yes		No		P
	No. of Patients	CPR Levels,[a] mg/L	No. of Patients	CRP Levels,[a] mg/L	
Age >65 y	87	240.4 (120.2)	74	336.0 (181.6)	<.001
Men	112	285.1 (164.4)	49	282.5 (145.5)	.924
Comorbidities[b]	84	283.0 (159.8)	77	285.7 (157.9)	.917
Symptoms >1 day	134	291.4 (166.2)	27	248.9 (107.5)	.205
Previous antibiotic treatment[b]	34	283.4 (183.8)	127	284.6 (151.7)	.969

[a] C-reactive protein values are expressed as means (SD).
[b] Heart failure, respiratory failure, liver failure, kidney insufficiency, or immunodeficiency.
[c] Duration >1 day.

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Adalimumab in the Treatment of Parotid Sarcoidosis

Adalimumab en el tratamiento de la sarcoidosis parotídea

To the Editor:

Adalimumab (brand name Humira®), is a biological blocker specific to the tumoural necrosis action factor which causes a fast decrease in the reactants of acute stage inflammation. Its accepted indications in the technical specifications to date are rheumatoid arthritis, psoriatic arthritis and ankylosing spondylitis.[1-3] We present a case of systemic sarcoidosis with outbreak of acute parotiditis which does not respond to regular treatment, but presents important clinical improvement with this drug.

A female aged 40 with no known medical allergies or harmful habits, with pathologic background of high blood pressure under hydrochlorothiazide treatment, with a pacemaker since 2000 for sinus node dysfunction and 3 miscarriages. In February 2001, she developed erythema nodosum in the lower extremities together with an outbreak of bilateral parotiditis which responded to treatment with 30mg of orally administered prednisone. In February 2002 she presented heart failure due to possible myocardial sarcoidosis and the echocardiogram displayed a 32% ejection fraction and moderate aortic insufficiency. She responded appropriately to diuretic treatment (furosemide, 40mg/12 h) and angiotensin-converting enzyme inhibitors (enalapril, 20mg/day).

In April 2008 she presented a new outbreak of bilateral parotiditis with fever, arthralgia and dyspnoea. She received treatment with prednisone at a dose of 30mg/day for 2 months, with no improvement in the parotiditis. Given the lack of response, in June 2008, treatment was started with infliximab at a dose of 300mg/day (a monthly dose), reaching a maximum dose of 400mg, with no clinical response. Given the poor results obtained to that point from treatments, we considered the option of trying adalimumab (Humira®). In September 2008, adalimumab was commenced at a dosage of 40mg once a fortnight for 4 months, with which the patient presented a good clinical response with disappearance of the clinical signs of parotiditis.

Despite the scarce experience in the use of this drug for sarcoidosis that does not respond to conventional treatment, adalimumab could be considered a therapeutic alternative for this type of patients.

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