

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

	Yes		No		P
	No. of Patients	CPR Levels, <sup>a</sup> mg/L	No. of Patients	CPR Levels, <sup>a</sup> 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 <sup>b</sup>	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 <sup>b</sup>	34	283.4 (183.8)	127	284.6 (151.7)	.969

<sup>a</sup>C-reactive protein values are expressed as means (SD).

<sup>b</sup>Heart failure, respiratory failure, liver failure, kidney insufficiency, or immunodeficiency.

<sup>c</sup>Duration >1 day.

## References

- Macfarlane JT, Boswell T, Douglas G, Finch R, Holmes B, Honeybourne D, et al. BTS guidelines of the management of community-acquired pneumonia in adults – 2004 update [accessed 01/04/09]. Available from: <http://www.brit-thoracic.org/guidelines>
- Hedlund JU, Hansson LO. Procalcitonin and C-reactive levels in community-acquired pneumonia: correlation with etiology and prognosis. *Infection*. 2000;28:68-73.
- Menéndez R, Cavalcanti M, Reyes R, Mensa J, Martínez R, Marcos MA, et al. Markers of treatment failure in hospitalised community acquired pneumonia. *Thorax*. 2008;63:447-52.
- Alfageme L, Aspa J, Bello S, Blanquer J, Blanquer R, Borderías L, et al. Normativas para el diagnóstico y el tratamiento de la neumonía adquirida en la comunidad. Sociedad Española de Neumología y Cirugía Torácica (SEPAR). *Arch Bronconeumol*. 2005;41:272-89.
- Bruunsgaard H, Pedersen AN, Schroll M, Skinhoj P, Pedersen BK. Impaired production of proinflammatory cytokines in response to lipopolysaccharide (LPS) stimulation in elderly humans. *Clin Exp Immunol*. 1999;118:235-41.
- Chalmers JD, Singanayagam A, Hill AT. C-reactive protein is an independent predictor of severity in community-acquired pneumonia. *Am J Med*. 2008;121:219-25.

Pamela Cabezas,<sup>a</sup> Agustín Ruiz-González,<sup>a,b,\*</sup> and Miquel Falguera<sup>a</sup>

<sup>a</sup>Biomedics Research Institute (IRBLLEIDA), Hospital Universitario Arnau de Vilanova, Lleida, Spain

<sup>b</sup>Ciber de enfermedades respiratoria (CIBERES, 06/06/08). Spain

\* Corresponding author.

E-mail address: aruiz@arnau.scs.es (A. Ruiz-González)

## 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.<sup>1-3</sup> 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.

## References

- Abbott Laboratories. Highlights of prescribing information. Humira Adalimumab Medication [consultado 30/03/2009]. Available at: <http://www.rxabbott.com/pdf/humira.pdf>.
- Callejas-Rubio JL, Ortego-Centeno N, López-Pérez L, Benticuaga MN. Treatment of therapy-resistant sarcoidosis with adalimumab. *Clin Rheumatol*. 2006;25:596-7.
- Wells AU. Infliximab in extrapulmonary sarcoidosis: tantalising but inconclusive. *Eur Respir J*. 2008;31:1148-9.

Marta Cufi-Benet,<sup>a,\*</sup> Carles Sabadell,<sup>b</sup> and Oriol Codina<sup>c</sup>

<sup>a</sup>Medicina Familiar y Comunitaria, Hospital de Figueres, Figueres, Girona, Spain

<sup>b</sup>Servicio de Neumología, Hospital de Figueres, Figueres, Girona, Spain

<sup>c</sup>Servicio de Reumatología, Hospital de Figueres, Figueres, Girona, Spain

\* Corresponding author.

E-mail address: mcufi@hotmail.com (M. Cufi-Benet)