be achievable. Our study was insuffciently powered to do this.

Rheumatology key message

- Despite its major functional effect on ERAP2 expression, rs2248374 shows no association with AS.

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Relapsing polychondritis-associated meningitis and encephalitis: response to infliximab

Sir, Relapsing polychondritis (RPC) is an uncommon systemic inflammatory disorder of unknown origin. A minority of patients with RPC develop neurological involvement (3%) [1]. The prognosis of patients with RPC complicated by meningoencephalitis (ME) is poor. The reported mortality of RPC-associated meningitis is 12% (3 out of 25 patients), and that of patients with RPC-associated encephalitis is 36.4% (4 out of 11 patients) [2–8]. Therapy with infliximab has been effective in several cases of resistant RPC. Nevertheless, the effects of anti-TNF-α therapy on RPC-associated meningitis and encephalitis have not previously been described. We report a patient with RPC and recurrent episodes of ME refractory to therapy with high-dose glucocorticoids and CYC, who had a satisfactory and long-lasting response to therapy with infliximab.

A 57-year-old male who immigrated 8 years ago from Ecuador presented with fever (up to 39°C), severe headache and two generalized seizures. During the previous 4 years, he had had one episode of erythema nodosum, and several episodes of symmetrical polyarthritis (hands and wrists), auricular chondritis, painful red eyes and dizziness, with the diagnoses of scleritis, cochlear dysfunction and neural deafness.

On admission, he was febrile (38.5°C) and confused with positive meningeal signs and a normal CT scan of the brain. Lumbar puncture (LP) disclosed 700 cells/ml, 98% lymphocytes, glucose 50 mg/dl and proteins 75 mg/dl. Cerebrospinal fluid (CSF) studies were negative for bacteria, virus, fungi and parasites or abnormal cells. Peripheral blood leucocytosis (20 × 10⁹/ml, 90% neutrophils) and elevated acute-phase reactant proteins were observed. Kidney and liver function, ANAs, ANCAs, RF, urinalysis and serological tests for HIV, hepatitis B virus, hepatitis C virus, CMV, EBV, treponema, rickettsias, borrelia, coxiella, brucella and echinococcus were all normal or negative. MRI of the brain showed small T2 gadolinium-enhanced lesions in the periventricular white matter of both cerebral hemispheres (Fig. 1b). The patient improved with high-dose i.v. methylprednisolone and was discharged.

During the following 20 months, he had seven admissions for ME with negative CSF studies. These episodes occurred while the patient was not taking any medications previously associated with ME, including non-steroidal anti-inflammatory agents. Although these episodes of ME improved with high-dose i.v. glucocorticoids plus...
CYC, maintenance of remission (fever, auricular chondritis, polyarthritis and ME), could not be accomplished with daily doses of prednisone under 25 mg in association with low-dose CYC (3-5 mg/kg daily) or MTX (up to 20 mg weekly). The patient rapidly improved after therapy with infliximab plus prednisone (10 mg daily), and has a normal quality of life 20 months after commencing therapy with infliximab (Figs 1a and 2). The timing of infliximab infusions (3 mg/kg i.v.) has been tailored to the clinical response, from bimonthly infusions to monthly infusions (months 9-17). The timing of infliximab infusions has recently been increased to every 6 weeks.

Only 3% of patients with RPC develop neurological involvement [1], which has been related to meningeal inflammation [4] and vasculitis of the CNS [5]. A characteristic pattern of CNS involvement has not been established by MRI. Diffuse hyperintense lesions in the basal ganglia, and the periventricular and subcortical white matter have been the most common MR findings [3, 4, 6]. Medications like non-steroidal anti-inflammatory agents have been associated with ME [9, 10]. Nevertheless, our patient had episodes of ME while free of medications other than prednisone, which has not been associated with ME. Therefore, a role of medications to the recurrence of neurological symptoms seems unlikely.

Our patient presented several previously observed unique features of RPC: resistance to therapy with prednisone doses under 25 mg daily, CYC (i.v. bolus or p.o.), or MTX (20 mg weekly IM) [1]; two episodes of ME with polymorphonuclear pleocytosis, simulating bacterial meningitis [3]; the development of a reversible subacute dementia [4]; the first report of a patient with RPC-associated ME with a rapid and long-term improvement, 20 months after the commencement of therapy with infliximab.

RPC-associated meningitis and encephalitis, although rare, are relapsing conditions with elevated morbidity.

**Fig. 1** MRI of the brain. (a) MRI of the brain showing small T2 gadolinium-enhanced lesions in the periventricular and subcortical white matter of both cerebral hemispheres. (b) MRI of the brain after treatment. Improvement of the gadolinium-enhanced lesions.

**Fig. 2** MRI of the brain after treatment with infliximab. Improvement of the gadolinium-enhanced lesions.
and mortality. High-dose glucocorticoids plus CYC is the first-line therapy. Infliximab can be effective in resistant cases of relapsing polychondritis associated with CNS involvement.

**Rheumatology key message**

- Infliximab is effective for resistant relapsing polychondritis-associated ME.

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**Antonio Garcia-Egido**, Christian Gutierrez, Carmen De la Fuente and Francisco Gomez

1. Department of Internal Medicine, Unit of Rheumatology, Hospital Universitario Puerto Real and 2Casines Primary Care Center, Puerto Real, Cadiz, Spain.

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Correspondence to: Francisco Gomez, Av. de la Paz 16 (Santa Ana), 11500 El Puerto Santa Maria, Cadiz, Spain. E-mail: francisco.gomez@uca.es

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**Cervical syphilitic spondylodiscitis associated with neurosyphilis**

Sir, Syphilis is a venereal disease caused by *Treponema pallidum*, family Spirochaetes. Clinical manifestations of acquired syphilis are in several stages. The primary stage is characterized by chancre, which if left untreated may lead to the secondary stage, often within 8 weeks after transmission, characterized by rash, fever, lymphadenopathy, mucocutaneous lesions, condyloma lata, alopecia and/or headaches. Late syphilis, the tertiary stage, develops in about one-third of untreated cases and is characterized by neurosyphilis, cardiovascular and/or gummatous lesions [1, 2]. Late manifestations are rare because of effective treatment. Musculoskeletal manifestations can be associated with congenital, secondary and tertiary syphilis and mimic a wide variety of diseases, especially granulomatous diseases. Neurosyphilis can be asymptomatic or present as meningitis and cranial nerve palsies (facial and auditory nerves). Parenchymatous neurosyphilis with paresis or tabes dorsalis is rare [3]. We describe the first case of cervical syphilitic spondylodiscitis associated with neurosyphilis. The patient gave his informed consent for the report according to the Declaration of Helsinki.

A 61-year-old Caucasian man presented a 2-month history of fatigue, night sweats and cervical pain. Two months before admission, he suddenly had bilateral deafness associated with a rash on his chest that lasted 2 weeks. At admission, physical examination revealed cervical and axillary lymphadenopathy (lymph nodes 2 cm); neurological examination was normal and cervical stiffness was absent. Laboratory tests revealed elevated white blood cell count, 11 400/mm³; CRP level 37 mg/l; hepatic cytolysis and cholestasis; and hypergammaglobulinaemia, 24 g/l. Microbiological tests, including blood and urine culture and tests for HIV, tuberculosis, viral hepatitis C and B and toxoplasmosis, gave negative results, but cervical spine CT scan and MRI findings revealed typical C2–C3 spondylodiscitis (Fig. 1A and B). Abdominal echography results were normal. Hepatic histological examination revealed chronic hepatitis with mild sclerosing cholangitis. The audiogram showed bilateral sensorineural hearing loss of 40 dBel. Cerebral MRI findings were normal. Lumbar puncture revealed lymphocytic meningitis, with 86 lymphocytes/mm³, and increased protein level, 0.92 g/l, but normal glucose level and negative bacteriology results. PET/CT revealed abnormal fluorodeoxyglucose uptake in cervical lymph nodes. Lymph node biopsy revealed non-specific inflammatory changes. C2–C3 biopsy confirmed the diagnosis of chronic spondylodiscitis, with no bacteria in culture. Syphilis serology gave positive results on *T. pallidum* haemagglutination assay (TPHA), 1/163 840; and Venereal Disease Research Laboratory (VDRL) test, 1/256; and in cerebral spinal fluid (CSF): TPHA, 1/2560; VDRL, 1/2. The patient received i.v. penicillin G (24 MUI/d) for 3 weeks, then three