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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**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. On admission, physical examination revealed cervical and axillary lymphadenopathy (lymph nodes 1–2 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
i.m. injections of benzathine–penicillin (2.4 MU). Two months after the end of the treatment, hearing was improved, and cytolysis, cholestasis and cervical pain disappeared. The lumbar puncture revealed 9 cells/mm³. Cervical MRI revealed marked improvement (Fig. 1C and D).

Syphilis remains a significant health concern in Europe. The incidence was low between 1950 and 1990, but increased in high-income settings from the beginning of the 21st century [1]. Twelve million people are infected every year [4]. The only definite criterion for diagnosis of active syphilis is the detection of T. pallidum on darkfield microscopy, direct fluorescent antibody tests or PCR of diseased lesion samples [5]. However, a presumptive diagnosis is possible with serological tests for syphilis: non-treponemal tests such as VDRL, micro-precipitation reaction (MPR) and rapid plasma reagin (RPR) tests, and treponemal tests such as TPHA, and fluorescent treponemal antibody with absorption (FTA-abs) tests [4]. The diagnosis of neurosyphilis is based on three elements in CSF: level of albumin > 0.4 g/l, number of cells >5/mm³ and a positive VDRL test result [6]. Our patient had all these elements. Positive CSF test results for TPHA alone do not confirm the diagnosis of neurosyphilis because of the possibility of passive diffusion of T. Pallidum.

Treatment is with penicillin and depends on the stage of syphilis. According to guidelines [7], musculoskeletal manifestations in the secondary disease stage could be treated with an i.m. injection of benzathine–penicillin and neurosyphilis with i.v. penicillin. Our patient received 3 weekly injections of benzathine–penicillin after classical treatment for neurosyphilis.

Cases of musculoskeletal localizations are rare in acquired syphilis (<1%), but are more frequent in congenital syphilis. The predominant localizations are the skull, sternum and long bones [8]. A few cases of syphilitic spondylitis, mostly lumbar, have been described [9–11], but none is recent. In all cases, the diagnosis was difficult but treatment was efficient. No case of
syphilitic spondylodiscitis has been reported. All other symptoms for our patient were due to syphilis, as confirmed by improvement with treatment. Deafness was due to neurosyphilis or labyrinthitis. A recent study showed that 10% of patients with secondary syphilis had hepatic involvement [12]. Acquired syphilis must be considered in case of spondylodiscitis associated with hearing loss.

**Rheumatology key message**

- Acquired syphilis must be considered in cases of osseous involvement associated with hearing loss.

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**Cryptococcal meningitis in an HIV-negative patient with rheumatoid arthritis treated with rituximab**

Sir, mAb agents, such as infliximab and rituximab, are increasingly prescribed for treatment of RA. We present a rare case of cryptococcal meningitis in a patient with severe RA after treatment with rituximab and, some significant time before that, infliximab.

A retired 70-year-old man with a 12-year history of RA was admitted with a 1-month history of reduced mobility, confusion, decreased appetite and weight loss. He had previously received infliximab over an 8-year period—a total of 39 infusions—the last dose being administered 7 months before admission. He subsequently received two doses of rituximab 4 months before admission. Other medications included prednisone 10 mg daily and MTX 7.5 mg weekly.

During admission he became increasingly confused, pyrexial and developed a left-sided facial palsies with slurred speech. A CT scan showed ischaemic changes and old left basal ganglia infarct. He was started on aspirin for presumed cerebrovascular accident.

Over the following 2 weeks, he remained confused and developed a left-sided motor neurone VIth nerve palsy, dysarthria, globally reduced power and worsening of visual acuity. Repeat CT head showed no new features. Lumbar puncture (LP) examination demonstrated raised leucocytes at 60/mm3 with a lymphocytic predominance. Cerebrospinal fluid (CSF) glucose was <2.6 mmol/l, protein (0.47 g/l). He was commenced on acyclovir and the last dose being administered 7 months before admission. He subsequently received a total of 39 infusions—the last dose being administered 7 months before admission. He subsequently received two doses of rituximab 4 months before admission. Other medications included prednisone 10 mg daily and MTX 7.5 mg weekly.

During admission he became increasingly confused, pyrexial and developed a left-sided facial palsy with slurred speech. A CT scan showed ischaemic changes and old left basal ganglia infarct. He was started on aspirin for presumed cerebrovascular accident.

Over the following 2 weeks, he remained confused and developed bilateral lower motor neurone VIth nerve palsy, dysarthria, globally reduced power and worsening of visual acuity. Repeat CT head showed no new features. Lumbar puncture (LP) examination demonstrated raised leucocytes at 60/mm3 with a lymphocytic predominance of 99%, low-glucose CSF:serum ratio (17%) and raised protein (0.47 g/l). He was commenced on acyclovir and transferred to an infectious diseases unit.

Repeat LP showed opening pressure (OP) 19 cm, lymphocytes 48/mm3, with Cryptococcal antigen (CRAG) positive at titre of 1:2048. Cryptococcus neoformans was subsequently cultured and sequenced as var neoformans using ITS1 and D2 primers. Sensitivity testing showed minimum inhibitory concentrations (MICs) as follows: flucytosine 8 mg/l (break point <1 mg/l), fluconazole 32 mg/l (<8 mg/l), amphotericin 0.03 mg/l (<0.125 mg/l), itraconazole 0.25 mg/l (<0.125 mg/l) and voriconazole 0.5 mg/l (<0.125 mg/l). MRI brain showed non-specific white matter changes and old left basal ganglia infarct. HIV test was negative and full blood count was normal with no neutropenia (initial neutrophils 4.8×109/l) throughout admission. Serum CRAG was initially positive (1:4096), but repeated blood cultures failed to grow Cryptococcus.