sis and the prompt implementation of vital support and antibiotic treatment, our patient had a good outcome.

Conclusions

Due to its rarity and initially unspecific symptoms, patients with mTSS are at risk of misdiagnosis. Even though molecular methods are not available in many laboratories, its implementation should be considered since they represent a possibility for faster diagnostics, and to obtain more information for the clinical and epidemiological surveillance of mTSS/TSS-associated SA lineages.

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Anisocoria and optic neuritis associated with *Mycoplasma pneumoniae* infection

Anisocoria y neuritis óptica en infección por Mycoplasma pneumoniae

Although the most common clinical form of infection by *Mycoplasma pneumoniae* (*M. pneumoniae*) is respiratory, this microorganism can cause extrapulmonary manifestations, the most serious of which is neurological[1]. We report here a case with anisocoria and optic neuritis.

This was a 32-year-old man with no previous medical history who had a two-week history of productive cough, dyspnoea, low-grade fever, nasal congestion and left hearing loss. Chest X-ray (Fig. 1, upper) revealed an infiltrate in the right upper lobe. *Legionella pneumophila* and pneumococcal urinary antigen tests were negative and treatment was started with amoxicillin/clavulanic acid 875/125 mg/8 h.

Two days later, the patient had not improved and attended Accident & Emergency; he had tachypnoea, with a baseline saturation of 88%, which rose to 95% with nasal cannula at 2 lpm, HR 46 bpm, blood pressure 124/68 and rhonchi in the upper right lung field.

Tests revealed leucocytosis with neutrophilia, coagulopathy (INR 1.3) and C-reactive protein of 103 mg/l, and a repeat chest X-ray showed no changes. Treatment was escalated to ceftriaxone 2 g/24 h with levofloxacin 500 mg/24 h and the patient was admitted to internal medicine.

On day three of admission he developed anisocoria, with greater mydriasis in his left eye. CT scan of the brain showed no acute intracranial findings. Pilocarpine eye drop test showed involvement of the left third cranial nerve.

CT angiogram of the head ruled out vascular injury and MRI ruled out cavernous sinus pathology, showing right maxillary sinusitis and left ethmoid sinus retention cyst (Fig. 1, lower). After 24 h, the anisocoria resolved spontaneously.

Multiplex PCR was carried out on nasopharyngeal exudate and sputum, which was positive for *M. pneumoniae* and negative for coronavirus, MERS-CoV, rhinovirus/enterovirus, influenza and parainfluenza virus, metapneumovirus, adenovirus and respiratory syncytial virus. Serum was also positive for *M. pneumoniae* IgM. HIV, syphilis and hepatotropic virus serologies were all negative. Azithromycin 500 mg/24 h for seven days was prescribed.

Four days later, the patient consulted once more due to loss of visual acuity in his left eye. Visual field testing revealed a diffuse loss of sensitivity with a superior altitudinal visual field defect in the left eye and visual acuity of 0.5. Lumbar puncture ruled out infection of the central nervous system. As parainfectious retrobulbar optic neuritis was suspected, he was started on corticosteroid therapy and made a full recovery.

At subsequent check-ups, the electroencephalogram, cervical spine magnetic resonance imaging and brain magnetic resonance angiography were normal, and the follow-up lumbar puncture showed no oligoclonal bands. Blood aquaporin 4 antibodies and myelin oligodendrocyte glycoprotein antibodies were negative, but he had a mild sustained IgG2 deficiency. After seven weeks, he continued to be IgM positive, with IgG seroconversion against *M. pneumoniae* (signal 1.41).

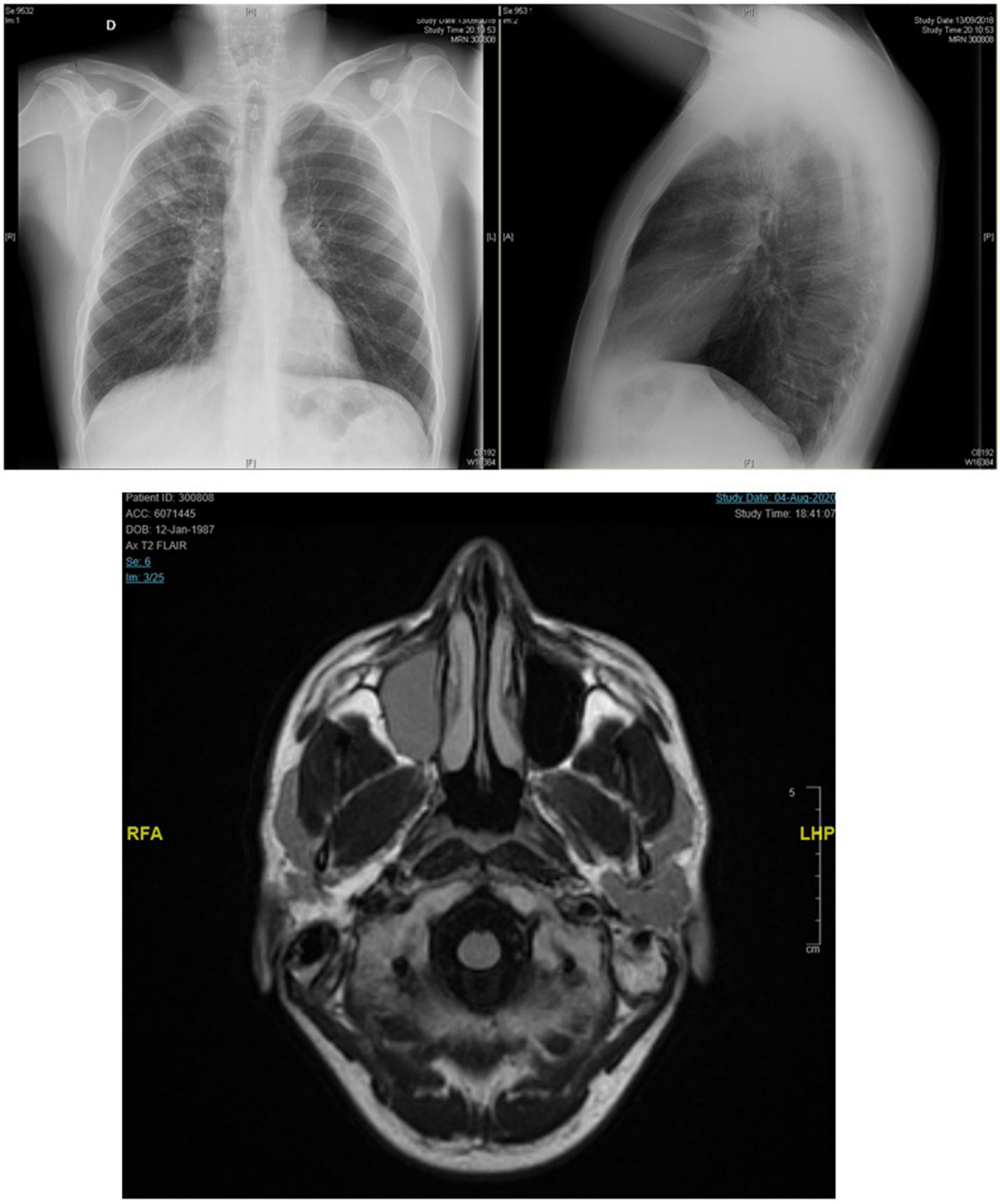

Fig. 1. Chest X-ray (upper image) and brain MRI of the patient (lower image).

The incidence of central nervous system complications due to *Mycoplasma* spp. has not been established; it ranges from 1% to 7% in hospitalised patients, with the mortality rate as high as 10%[1]. Over half of cases are found in patients between 6 and 21 years of age[2], but it also occurs in adults. Parainfectious neuritis usually occurs bilaterally and is more active in children[3], and up to 20% may have no prior respiratory infection[4].

There are several mechanisms involved in the neurological complications, none of which is exclusive. There may be direct cell damage following haematogenous spread of the microorganism reaching the central nervous system. When the bacteria damages cells, the innate immune response is activated, attracting different cytokines such as IL 18, which activates helper T cells 1 and 2, or IL 8, which attracts neutrophils. It can be mediated by immune complexes due to antigenic elements, which bind to the patient's macrophages and monocytes, initiating immune reactions due to the antigenic similarities between *M. pneumoniae* and brain parenchyma antigens[5]. Vascular damage is also possible[6].

The incidence of *Mycoplasma* spp. infections is increasing in patients with hypogammaglobulinaemia, which indicates compromise of humoral immunity in the pathogenesis of these cases[7]. Antibodies inhibit the growth of *M. pneumoniae in vitro*, preventing proliferation on the surface of colonised mucosa[8], without affecting neutrophil opsonisation[9].

Our patient had optic neuritis as a rare complication in *M. pneumoniae* infections. It is likely that partial IgG2 deficiency facilitated infection and cell mimicry led to the neurological symptoms. We need to be aware of the association between hypogammaglobuli-

naemia and the risk of *M. pneumoniae* infection, as these patients can suffer from more severe and prolonged illnesses and develop more complications[10].

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Infective endocarditis caused by *Streptococcus cristatus*

Endocarditis infecciosa causada por Streptococcus cristatus

A 76-year-old patient presented at our hospital with intermittent fever of up to 40 °C lasting three weeks, coinciding with stem cell inoculation in both hips. The patient had required aortic valve replacement six years earlier and, due to liver cirrhosis with hepatocarcinoma in segment 5, had also required partial hepatectomy and cholecystectomy five months earlier. Twenty days before, the patient had a dental filling for which he did not receive antibiotic prophylaxis. On examination, the patient had a fever of 39.1 °C with a pansystolic murmur, and a blood test showed 12,900 leukocytes, 90% of which were neutrophils. Suspecting endocarditis, a blood culture was obtained and the patient was admitted for study and antibiotic treatment with vancomycin and cefepime (1 g/12 h and 2 g/24 h intravenously [IV], respectively).

A transthoracic echocardiogram was performed, in which thickened aortic leaflets were observed, and also a transoesophageal echocardiogram, in which no clear images of endocarditis were observed. The blood culture was positive after 20 h of incubation, showing Gram-positive cocci in chains in the Gram stain. The blood culture was inoculated on CNA agars (Becton Dickinson, New Jersey, USA), TSA agars with 5% sheep blood (BD™) and chocolate agar. At 24 h, growth was observed, with its identification and antibiotic sensitivity provided by means of the SMIC-ID-11 (BD™) panel in the BD Phoenix™ AP system. The strain was identified as *Streptococcus cristatus* (*S. cristatus*), which was sensitive to penicillin (MIC ≤0.03 mg/l), vancomycin (MIC = 1 mg/l), teicoplanin (MIC ≤1 mg/l) and clindamycin (MIC ≤0.03 mg/l). In a subsequent blood culture, *S. cristatus* was identified again, changing the antibiotic treatment to ceftriaxone (2 g/24 h) while waiting to perform CT + PET-CT. After administration of IV contrast and 18-FDG, lesions compatible with small infarcts in the lower pole of the spleen and left kidney were found, as well as increased metabolic activity in the periprosthetic aortic valve, which did not prevent us from ruling out endocarditis (Fig. 1).

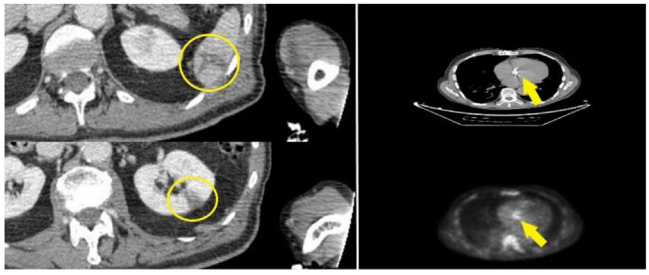

Figure 1. Joint CT and PET-CT study performed following endocarditis protocol after inconclusive result by transoesophageal echocardiogram by administration of intravenous contrast and 18-FDG, with imaging from the cervical region up to and including the pelvis. Hypodense lesions of triangular morphology and new appearance in the lower pole of the spleen and interpolar region of the left kidney, compatible with small infarcts (yellow circles). Focal increase in metabolic activity (yellow line) on the prosthetic aortic valve annulus's anterior region makes it impossible to rule out endocarditis without significant morphological findings.

The condition was considered as probable endocarditis (one major criterion and three minor criteria) and the patient completed six weeks of IV treatment. The patient was followed up through outpatient visits, confirming clinical improvement and normality in subsequent echocardiograms. To confirm the identification, the strain was reseeded to sequence the 16S ribosomal RNA gene. A 637 bp sequence was obtained that was entered into BLAST®, and was identified as *S. cristatus* with an identification percentage of 99.37% (see sequencing protocol and sequence in Appendix B: Supplementary material attached).

S. cristatus was isolated for the first time from the human oral cavity, belonging to the *mitis*group.[1] A 2014 study showed that *S. cristatus, Streptococcus oligofermentans* and *Streptococcus sinensis*