A CEREBELLITIS IN A TWENTY YEAR-OLD: A POSSIBLE ROLE FOR MYCOPLASMA PNEUMONIAE

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Introduction:

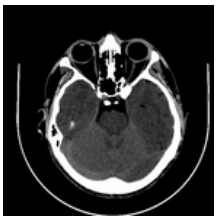
Cerebellitis is an inflammation of the cerebellum. It's a syndrome that occurs mostly in children and often presents as a post-infectious disorder. Mycoplasma pneumoniae is a bacterium associated with respiratory infections, but it is also described in the literature as a pathological agent of cerebellitis, either as a direct agent or by autoimmune mechanism. We hereby present a case of a young man with cerebellitis and a positive serology for M. pneumoniae. Numerous tests were performed to certify the link between the infection and the neurological syndrome. In this particular instance, the diagnostic procedures did not prove to be of any significance. However, the patient exhibited a complete restoration of their condition following the administration of empirical therapy. This case suggests that Mycoplasma infections should be considered in the differential diagnosis of encephalitis, particularly in cases affecting the cerebellum, and that the autoimmune mechanism should also be taken into consideration.

Case report:

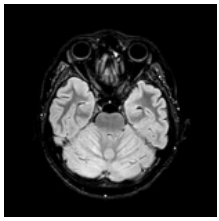
A previously healthy 20-year-old man presented to the ER with a high fever, headache and cough. A head CT scan was performed, which came back negative, and he was later dismissed. Four days later, he suddenly developed postural instability, dysarthria and dysphagia, so he was readmitted to the ER. This time, the head CT scan revealed bilateral cerebellar hyperdensity, initially hypothesised to be of a flogistic-infectious nature. Blood tests revealed leukocytosis. During the neurological examination, the patient presented with moderate dysarthria and truncal and limb ataxia. A lumbar puncture was performed and turbid CSF was extracted. Initial analysis of the fluid showed 400 cells, a protein level of 190 mg/dL and a glucose level of 47 mg/dL. The patient was admitted to the Neurology Unit. His previous medical history was unremarkable. In agreement with the infectious disease specialist, the patient was started on ampicillin (3 g every six hours), acyclovir (10 mg/kg every eight hours) and desametasone (4 mg four times per day). A head MRI scan confirmed the suspicion of cerebellitis, showing weak hyperintensity in T2/FLAIR and modest diffusion restriction of the cerebellum. During the hospitalisation, the remaining CSF test results arrived: cytomorphological analysis showed numerous lymphocytes; FilmArray, virological (PCR) and cultural analysis were negative; and isoelectric focusing and OCBs were also not relevant. Due to the negative results, ampicillin was suspended, as was acyclovir after 14 days of treatment.

Autoimmune screening was performed and resulted positive for ANA with a titer of 1:320. Serology testing was performed for HIV, hepatitis, tuberculosis, rubella, herpes virus and atypical bacteria, and the results were positive for Mycoplasma IgM antibodies, with IgG levels slightly below the threshold. One week later, the mycoplasma serology test was repeated with increased levels of both IgM and IgG. After discussing the case with the infectious diseases specialist and the microbiologist, it was decided to also test for Mycoplasma antibodies in the CSF, but this was not significant.

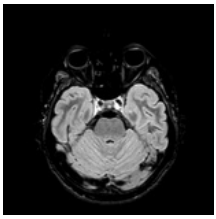
A review of the literature was therefore performed, revealing a link between Mycoplasma pneumoniae infections and antibodies mediating autoimmune encephalitis. For this reason, it was decided to switch to high-dose steroid therapy (500 mg per day for five days) and search for the antibodies most frequently associated with this condition, including onconeural and autoimmune encephalitis-related antibodies, as well as anti-GAD and anti-ganglioside antibodies. These tests were negative, but they were all performed after the corticosteroid therapy had been initiated. Meanwhile, the patient showed progressive improvement until complete restoration of his previous condition. Complete resolution of the inflammation was confirmed by a new MRI scan that showed no abnormalities. The patient was dismissed at home.



the first CT performed at the ER



the first MRI with cerebellar hyperdensity in T2/FLAIR sequences



the second MRI with the complete resolution of the inflammation

Conclusions:

Our case illustrates cerebellitis in an otherwise healthy patient with a recent upper respiratory tract infection and positive serology for M. pneumoniae. This bacterium is associated with respiratory infections, but it is also described in the literature as a pathological agent of cerebellitis, either as a direct agent or by autoimmune mechanism. Serology on CSF was negative, so the autoimmune mechanism seemed more likely. The antibodies most frequently associated with it were negative, but they were performed after corticosteroid treatment. We must also consider that to date not all autoimmune mechanisms and antibodies involved in all types of encephalitis are known.

References:

1. Komatsu H, Kuroki S, Shimizu Y, Takada H, Takeuchi Y - Mycoplasma pneumoniae meningoencephalitis and cerebellitis with antiganglioside antibodies - Pediatric Neurology - 1998 - 18(2)
2. Randeep Guleria 1, Nazima Nisar, Tirlok Chand Chawla, Nihar Ranjan Biswas - Mycoplasma pneumoniae and central nervous system complications: a review - The journal of laboratory and clinical medicine - 2005
3. Jay Desai 1, Wendy G Mitchell - Acute cerebellar ataxia, acute cerebellitis, and opsoclonus-myoclonus syndrome - Journal of child neurology - 2012 - 27(11)

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