

Antibodies in Neurosarcoidosis

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Citation

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Abstract

A 33-year-old African American woman was admitted to our hospital with bilateral facial nerve palsy. One week prior to presentation, the patient experienced numbness and paresthesia of her face. Paresthesia was located over her upper lip, and around the eyes and frontal area. Three days before presentation, the patient developed facial droop, difficulty closing eyes, inability to smile, loss of taste, and ocular dryness. The patient's past medical history was unremarkable. She was a life long nonsmoker. Physical examination at admission revealed normal vital signs. Neurological examination showed bilateral facial nerve palsy and decreased sensation to touch and pain in the ophthalmic and maxillary distributions of the trigeminal nerve. Mental status and the remaining neurological examination were normal. Skin, chest, cardiac, and abdominal examinations were unremarkable. Cerebral magnetic resonance imaging (MRI) showed no meningeal signal enhancement, intracranial mass, cerebral or brain stem signal abnormality, or vascular disease. Lumbar puncture with cerebrospinal fluid (CSF) sampling was done. The CSF opening pressure was normal. CSF analysis showed a white cell count of 66 /uL, lymphocytes 92%, glucose of 46 mg/dl (normal range 40-70mg/dl), protein of 70 mg/dl (normal range 15-55 mg/dl), negative CSF Venereal Disease Research Laboratory value (VDRL), negative CSF Lyme antibody, and negative Herpes virus polymerase chain reaction. Other laboratory findings included a negative human immunodeficiency virus antibody, erythrocyte sedimentation rate of 30 mm/min, negative anti-nuclear antibody, angiotensin-converting enzyme of 32 U/L, and mildly elevated hepatic aminotransferases. Computed tomographies scan of the chest showed right hilar and right paratracheal lymphadenopathies. Bronchoscopy with transbronchial lung biopsy as well as fine needle aspiration of the lymphadenopathy was performed. Fine needle aspiration cytology and flow cytometry were negative for malignancy. The transbronchial biopsy showed granulomatous pneumonitis consistent with a diagnosis of sarcoidosis.

Tissue cultures and special stains for mycobacterial and fungal infections were negative. The patient was started on oral prednisone 80 mg daily. The facial palsy improved on oral prednisone. Pulmonary function testing was not possible due to her bilateral facial paralysis (the patient was unable to make a seal around the mouthpiece). The finding of noncaseating pulmonary granulomas of unknown cause, facial nerve palsy, and mediastinal lymphadenopathy is characteristic of sarcoidosis. Interestingly, the CSF anti-myelin-associated glycoprotein antibody titer by enzyme-linked immunosorbent assay (ELISA) was 1:1600 (Titers >1500 are considered positive).

Sarcoidosis is systemic disorder of unknown cause that is characterized by noncaseating granuloma formation in affected organs. Nervous system involvement is also commonly seen in sarcoidosis. Abnormal antibody production may contribute to the disease process and nervous system injury in sarcoidosis. Autoantibodies to brain endothelial cells have been described in patients with neurosarcoidosis¹. Local synthesis of Kveim-specific IgG antibody in the cerebrospinal fluid of patients with neurosarcoidosis has also been detected². Myelin oligodendrocyte glycoprotein (MOG) is a specific central nervous system myelin glycoprotein that is exclusively seen on the external surface of the myelin sheath and oligodendrocyte membrane. Antibodies to MOG have been implicated in the immunopathogenesis of multiple sclerosis. Anti-MOG antibodies in neurosarcoidosis have been reported in the literature. As MOG antigen can activate both encephalogenic T-cells and demyelinating antibodies in experimental animals, it may be important in the pathogenesis of neurosarcoidosis³. Anti-myelin-associated glycoprotein (MAG) antibody is another antibody associated with neurological disorders. Elevated anti-MAG antibody titers can be detected in autoimmune demyelinating sensorimotor neuropathies⁴. Elevated anti-MAG antibody in neurosarcoidosis has not been reported before. Further

studies are necessary to define the role of anti-MAG antibody and other antibodies in the pathogenesis of neurosarcoidosis.

References

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