

showed features of tuberculous peritonitis like mesenteric thickening giving a stellate appearance, omental thickening, free and loculated ascites and matting of bowel loops. There were also calcified periportal and peripancreatic lymph nodes. Following aspiration of the abscesses the patient responded well to the antituberculous chemotherapy. So even though amoebic and pyogenic liver abscess are more common, the possibility of tuberculous liver abscess should always be considered if the patient is not responding to the antibiotics, and also has findings suggestive of active or healed tuberculosis like calcified periportal and peripancreatic lymph nodes and lung lesions as in our case. Delay in diagnosis may lead to complications, multiple operations and morbidity as rupture of abscess occurred in our case⁷.

It is concluded that in a case of multiple liver abscesses, the possibility of tuberculous liver abscess should be considered especially if the patient is not responding to the conventional treatment for pyogenic

and amoebic abscess and features of active or healed tuberculosis are present in the abdomen or at any other site. Delay in the diagnosis may result in complications like rupture of abscess.

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Unilateral Ocular Myasthenia Gravis following Acute Sinusitis-A Case Report.

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Abstract: Myasthenia is an autoimmune disease in which IgG antibodies directed against the acetylcholine receptors at the neuromuscular junction prevent normal muscle contraction and lead to muscle weakness. Myasthenic muscle weakness of acute onset can occur following identifiable stimuli. We describe a case of a 40 years old lady with sinusitis and who subsequently underwent a sinus endoscopy, and following this within days developed sudden onset isolated, unilateral, painless ptosis. She was then diagnosed as ocular myasthenia and treated with pyridostigmine to which she responded well.

INTRODUCTION

Myasthenia gravis is caused by antibodies to post synaptic acetylcholine receptor and evidence indicates that AchR abs must be present in circulation prior to development of a 40 years old lady with sinusitis and who subsequently underwent a sinus endoscopy, within days developed sudden onset isolated, unilateral, painless ptosis. Such rapid evolution of myasthenia following an identifiable stimulus has been previously described¹. It is speculated that the remote effects of autoinflammation secondary to tissue microtrauma leads to sudden increase in muscle permeability and greater exposure of receptors to antibodies, with resulting acute impairment of neuromuscular transmission¹.

CASE REPORT

A 40 years lady had complaints of cough, cold and headaches of a few days duration, was diagnosed as a case of right sided frontomaxillary sinusitis and treated by an ENT specialist with antibiotics and a subsequently a sinus endoscopy. She eventually improved and became asymptomatic.

However, within 7-10 days of endoscopic procedure, she complained of sudden onset drooping of the right eyelid, which characteristically worsened in the evenings. She had no headaches, periorbital pains, dimness or double vision, numbness on the face or limb weakness. She was not on any medications and had no history of recent vaccination.

Then she was seen at our clinic and examination revealed a definite right sided ptosis with evidence of fatigability. There were no signs of local inflammation, fever or tenderness. The rest of her gross neurological and general physical examination was normal. A probable

diagnosis of ocular myasthenia was made and she was referred for a neurologist opinion.

Subsequent investigations revealed a normal MRI Brain including both orbits, normal S. TSH and Blood sugars. She underwent a repetitive nerve stimulation test which was noncontributory. This was followed by a tensilon (edrophonium) test. Edrophonium is a short acting anticholinesterase that prolongs the presence of neurotransmitter acetylcholine at the neuromuscular junction. This revealed a striking improvement in her ptosis and reported as strongly positive. She was then started on oral pyridostigmine following which her ptosis settled. Three months after initial diagnosis, patient is symptom free.

DISCUSSION

Myasthenia is an autoimmune disease in which IgG antibodies directed against the acetylcholine receptors at the neuromuscular junction prevent normal muscle contraction and lead to muscle weakness². A long acting oral anticholinesterase such as pyridostigmine is the first line of treatment. Immunosuppression with prednisone is used when symptom improvement is inadequate³. This case demonstrates the importance of considering neurotransmitter diseases in the approach to new onset focal weakness that involves the ocular or bulbar musculature.

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