

Case Report

Mesial temporal sclerosis

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ABSTRACT

Mesial temporal sclerosis is closely related to temporal lobe epilepsy, a type of partial (focal) epilepsy in which the seizure initiation point can be identified within the temporal lobe of the brain. Mesial temporal sclerosis is the loss of neurons and scarring of the deepest portion of the temporal lobe and is associated with certain brain injuries. We present a case of a 40-year old male who was presented with generalized seizures and was diagnosed with mesial temporal sclerosis.

Keywords: Generalised seizures, Mesial temporal sclerosis

INTRODUCTION

The etiology of MTS is still not fully understood. However, there is now considerable evidence, from both animal and clinical studies, that has shown that MTS is not only the cause of seizures, but is also the result of seizures.¹ Animal studies suggest that the mechanism by which seizures can cause MTS is through the release of excitatory amino acids, primarily glutamate.²

These excitatory amino acids lead to prolonged neuronal depolarization and result in the entry of cytotoxic amounts of calcium, which can eventually lead to sclerosis.

Clinical studies also suggest that prolonged seizures are a risk factor for MTS and that a period of time is required for the lesions to fully develop.^{3,4} Brain damage from traumatic injury, infection, a brain tumor, a lack of oxygen to the brain, or uncontrolled seizures is thought to cause the scar tissue to form, particularly in the hippocampus, a region of the temporal lobe.

The region begins to atrophy; neurons die and scar tissue forms. This damage is thought to be a significant cause of temporal lobe epilepsy.

CASE REPORT

Study presents a case of 40 year old male who visited with chief complains of multiple episode of seizures since 3 days with irrelevant talks on 26 June 2016.

Patient presented with multiple episode of seizure which was generalized tonic clonic, with-uprolling of eyes, with tongue bite, weakness of upper and lower limb following episode of seizure, urinary incontinence during seizure. Patient also gives history of post ictal confusion, with memory loss of seizure; patient also has history of irrelevant talks post seizure. Patient has aura before the episode of seizures.

There were no history of fever, vomiting, neck rigidity, co morbidities, fall, trauma and any surgery in past or any addiction. Patient has been hospitalised for seizure 1 year before also for similar complains patient didn't take any medication for the same. Patient's seizures have increased in frequency with hampering of daily activities for which patient got hospitalized. On examination patient conscious oriented cooperative in time place and person; with vitals being stable with blood pressure of 100/70 and a pulse rate of 88/min, noicterus, edema, pallor, cyanosis or lymphadenopathy.

On systemic examination it was found that Per abdomen was soft, non-tender, bowel sounds heard, no oragnomegaly. Respiratory-b/l conducted sounds. Cardiovascular-s1s2+. In Central nervous system there were no signs of neck rigidity.

Laboratory investigations

All lab investigations were within normal limits, with no electrolyte abnormality; with no metabolic cause and EEG done was found to be normal.

MRI brain was s/o reduction in volume of right hippocampus with flattening, loss of interdigitation and mild dilation of the adjacent temporal horn of the lateral ventricle findings s/o rt mesial temporal sclerosis. Patient is currently on tablet levipril 500 mg bd and is seizure free since 1month.

DISCUSSION

The left and right medial temporal lobes of the brain assist in memory formation. They include the hippocampus and the amygdala. Sclerosis is abnormal hardening of tissue. Mesial temporal sclerosis is the scarring of the medial (middle or on the centerline of the body) temporal lobes of the brain. Scarring of the hippocampus is the most common form; this condition is called hippocampal sclerosis.

Often mesial temporal sclerosis is found concurrent with temporal lobe epilepsy or pathology. While MTS is the most common symptomatic pathologic entity in adult and adolescent patients with seizures of temporal lobe origin, it is considered to be a rare pathologic finding among infants and young children.¹⁻³

Clinical presentation of MTS in adults and adolescents is characterized by auras that often involve visceral sensation, motionless stare, loss of awareness, and oral alimentary automatism.⁵ Infants typically do not have any identifiable auras, present with seizures that are of longer duration (>1 minute), and have more prominent convulsive activity. Moreover, infants predominately present with behavioral arrest with possible impairment of consciousness. Their automatism is mostly discrete and predominantly orofacial.³

MRI is the radiological investigation of choice for the evaluation of patients with MTS, since it can identify structural abnormalities.⁶ However, other MR techniques, such as magnetic resonance spectroscopy (MRS) and functional MRI (fMRI) are also being increasingly used in the diagnosis of MTS.

MR spectroscopy can detect metabolic abnormalities, while fMRI allows for the noninvasive evaluation and localization of cognitive, motor, and sensory deficits that are associated with MTS. Also, intraoperative MRI-based image-guided systems are now used as an adjunct in the

surgical treatment of MTS for better anatomic localization.⁷

Along with MRI, the use of PET scans with FDG is also well-established for the diagnosis of MTS. In the interictal state, the epileptogenic temporal lobe indicates decreased glucose metabolism in approximately 80% of these patients. Moreover, PET scans may be a reliable indicator of clinical outcome postsurgery; the presence of a hypometabolic temporal lobe is a favorable prognostic indicator.⁸

Antiepileptic drug treatment for patients with MTS is effective in the suppression of secondarily generalized seizures. However, 50% of patients continue to have partial attacks. With combined use of MRI, PET, and video-EEG, we can properly select patients who can benefit from anterior temporal lobe resection. In such patients, complete seizure control is achieved in >80% of cases.⁴ Patients who had an earlier age of onset and a low number of previously used antiepileptic agents have an overall better outcome after surgery.⁹

Surgical treatments for mesial temporal sclerosis

Mesial temporal sclerosis treatments generally consist of managing the epilepsy symptom by using either anticonvulsant medication or, if medication is not effective, surgery. Surgical treatments for intractable epilepsy include a temporal lobectomy or vagal nerve stimulation.

Temporal lobectomy is a surgical procedure to remove the brain tissue in the temporal lobe that is causing the seizures. An amygdalo-hippocampectomy is the surgical removal of the amygdala and the hippocampus after other treatments have failed to provide relief. Neuroimaging allows our expert brain surgeons to carefully target the damaged tissue and allow as much healthy brain tissue to remain as possible.⁹ Vagal Nerve Stimulation is a minimally invasive mesial temporal sclerosis treatment that employs an implanted stimulator to deliver electric impulses to an electrode on the vagal nerve in the neck via a lead wire implanted under the skin. These impulses modulate the brain's circuitry.

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