A Study on Clinical Profile of Tumefactive Demyelination

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Abstract: The term tumefactive is used in literature to describe large demyelinative lesion that are frequently misdiagnosed as brain tumours.Tumefactive demyelinative lesions (TDL) can mimic intracranial neoplasm and pose diagnostic dilemma both in clinical presentation and conventional MR imaging features.The aim of our study was to study the clinical and radiological profile of TDL and their response to steroids

Keywords: TDL,brain tumour, ADEM, MR Spectroscopy, steroids

I. Introduction

TDL represent a separate entity intermediate between classic MS and ADEM. These lesions represent an ill defined sub group of demyelinating disease with atypical clinical presentation and imaging findings. On imaging TDLs and high grade intracranial neoplasm can both show contrast enhancement, perilesional edema, varying degrees of mass effect and central necrosis. Supratentorial TDLs can be confused with astrocytoma, oligodendroglioma and those involving the corpus callosum can mimic butterfly lesions like glioblastomamultiforme and lymphomas. TDLs can be confused with high grade glial neoplasms on histopathological evaluation because of the presence of hypercellularity and atypical reactive astrocytes with mitotic figures. However MR Spectroscopy can help differentiate the two. MRS in neoplasm shows elevated choline peak indicating increased cell turnover. In demyelination there is reduced N acetyl aspartate/creatinine ratio and presence of lipid and lactate peaks. These indicate neuronal destruction and high inflammatory activity respectively. We present ten cases of TDLs masquerading as brain tumour and the role of MRSpectroscopy in establishing the diagnosis

II. Materials And Methods

The study was conducted in the Department of Neurology from January 2017 to January 2018. About 10 patients were diagnosed as cases of TDL. Investigation done were complete Hemogram, ESR, Chest X-ray, CSF Analysis, MRI Brain and Spine with contrast, MR Spectroscopy, VEP and Nerve Conduction Study.

III. Result And Discussion

All the ten patients were within the spectrum of monophasic demyelinating illness. Onset was acute to subacute with features of raised intracranial lesion like headache, vomiting, followed by neurological deficits. Five patients presented with left hemiparesis, three patients with rt hemiparesis and two with paraparesis. One patient had peripheral demyelination along with left hemiparesis and one patient had optic neuritis along with paraparesis. Investigation showed CSF analysis negative for OCB, raised protein and few lymphocytes. MR Imaging revealed more than 2 cm T1 hypo intense, T2 hyperintense supratentorial lesions with contrast enhancement of the rim enhancement type. Mass effect and vasogenic edema were present. One had involvement of the corpus callosum mimicking a butterfly glioma and two had extension into the spinal cord. MRSpectroscopy showed reduced N acetyl aspartate/creatinine ratio and presence of lipid peak. All patients showed good response to steroids.
IV. Conclusion

TDL especially single dominant ones need to be differentiated from neoplasms as they respond well to treatment and also to avoid unnecessary biopsy and resection. MR Spectroscopy plays an important role in establishing diagnosis.

References