CLINICO-RADIOLOGICAL SPECTRUM OF MOG ANTOBODY ASSOCIATED DISORDER (MOGAD) IN PEDIATRIC POPULATION.

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INTRODUCTION

- MOG antibody associated disorder (MOGAD) is an antibody mediated CNS inflammatory –demyelinating disorder of CNS.
- Clinical presentation: optic neuritis, transverse myelitis, acute demyelinating encephalomyelitis and acute cortical encephalitis.
- Myelin oligodendrocyte glycoprotein (MOG) is present on the surface of myelin sheath and is produced by oligodendrocytes (the myelin-forming cells) of the CNS.
- Symptoms: fever, headache, nausea, vomiting, malaise, altered mental status, vision impairment, ataxia, hemiparesis, hemisensory loss (as per location of lesion).
- MRI Optic nerves : anterior optic pathway lesion with optic nerve head swelling and injury of the retrobulbar nerve segment.
- MRI Spinal cord: LETM–characteristic extensive involvement of the spinal cord.
- Localization in a medullary cone is believed to be highly specific for MOGAD diagnosis.
- T2-weighted and FLAIR hyperintense lesions,: bilateral, poorly delineated, blurred and extensive lesions involving juxtacortical white matter, deep grey matter and rarely cortical grey matter.
- FLAMES (FLAIR-hyperintense lesions in anti-MOG-associated encephalitis with seizures) has cortical lesions, also sometimes associated with FLAIR-variable unilateral enhancement of the leptomeninges (FUEL)
- Immunotherapy (steroids, IVIG, plasmapheresis) is the cornerstone of treatment

OBJECTIVES

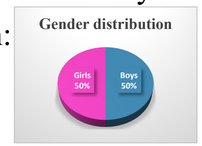
To study the clinical and radiological spectrum of MOG antibody associated disorder.

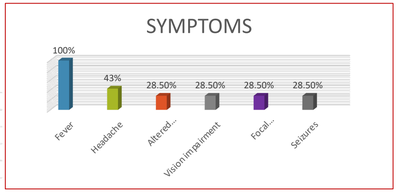
MATERIAL AND METHODS

- A retrospective study of 14 patients of age <16 years , diagnosed with MOGAD based on serology.
- The demographic profile, age of onset of symptoms, clinical features, CSF studies, radiological findings, treatment and response to treatment and number of relapses were evaluated.
- All patients were treated with Inj. Methylprednisolone and oral steroids.
- The patients with no or minimal response, were given inj. IVIG.

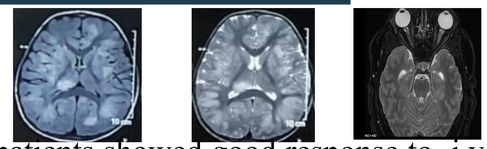
RESULTS

- Mean age of onset : 6.2 ± 2.4 years
- Gender distribution:





- Relapse was seen in 21.4% of patients (3/14) . 2/3 had 4 relapses and 1/3 patient had 1 relapse (presentation :Focal neurological deficit>> seizures)
- MRI showed extensive lesion involving cortical and sub cortical white matter in ADEM and optic nerve swelling in case of Optic neuritis



10/14 patients showed good response to i.v. steroids and 4/14 patients with minimal response to steroids required IVIG.

Relapses: treated with steroid and IVIG.

Multiple relapses: started on azathioprine.

DISCUSSION

	Our study	Hennes et al 2017	Hacochen et al 2018	Fadda et al 2018	Duigan et al 2018	Armangue et al 2020	Serin et al 2020
No. of patients	14	65	102	99	76	116	9
Mean age of onset (yrs)	6.2	7	7	10	-	6.2	7.6j
ADEM	43%	52%	52%	36%	60%	40%	67%
ON	29%	28%	40%	29%	37%	20%	22%
TM	21%	11%	-	10%	4%	11%	-
Acute Encephalitis	7%	-	-	-	-	19%	11%
Relapses	21.4%	34%	100%	16%	49%	28%	44%

Serin et a showed subcortical White matter involvement as most common finding on MRI . Treatment with immunosuppressants showed good response.

CONCLUSION

MOGAD is an immune-mediated encephalitis. MRI shows demyelination in variable areas. There is good response to treatment with immune-suppressants.

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