A Prospective Study on Clinical Profile of Cans Vacuities

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Abstract: Vasculitis that affects the CNS is one of the most formidable diagnostic and therapeutic challenge for physicians for several reasons. The clinical manifestation of CNS Vasculitis are highly variable. CNS is a common target for many forms of Systemic Vasculitis. Specific Non invasive tests are lacking and material for pathophysiological investigation is limited. Correct diagnosis requires a high degree of suspicion along with knowledge of other diseases that may masquerade as Vasculitis.

Clinical patterns that might facilitate recognition have been proposed, though therapeutic trials have been few. The aim of our study was to categorize patients identified with CNS vasculitis according to clinical classification described by SCOLDING et al 1997 and to follow their prognosis and response to treatment.

Key Words: CNS Vasculitis, SCOLDING et al, Type 1 presentation, Type 2 presentation, Type 3 presentation, steroids.

Date of Submission: 02-05-2018 Date of acceptance: 18-05-2018

I. Introduction

The pathophysiology of neurological vasculitis is relatively well understood. In both Primary and Secondary CNS vasculitis the neurological features arise principally through ischemia and infarction. Focal or multifocal infarction or diffuse ischaemia affecting any part of the brain occurring acutely, subacutely, recurrently or chronically explain the protean manifestation, wide variation in disease activity, course and severity and the absence of a pathognomic or even typical clinical picture. In 1997 SCOLDING et al described three clinical patterns of presentation in CNS vasculitis patients which help in diagnosis:

1) Acute or subacute encephalopathy with headache and an acute confusional state progressing to drowsiness and coma.
2) Superficially resembling Atypical MS in phenotype with relapsing remitting course and features such as optic neuropathy and brain stem episodes but also accompanied by other features less common in MS, severe and persisting headache, encephalopathic episodes or hemispheric stroke like episodes.
3) Intracranial mass lesion with headache, drowsiness, focal signs and increased intracranial pressure.

II. Materials and Methods

The study was conducted in the Department of Neurology Coimbatore Medical College Hospital from June 2015 to June 2017. About 20 patients diagnosed as CNS Vasculitis were selected. The study included 18 female patients and 2 male patients. Patients with both Primary and Secondary CNS Vasculitis were included in the study. Investigations included Hb%, ESR, Peripheral smear, CRP, Mx, CSF Analysis for Biochemical parameters, serology, cell count, Immunological investigation, ANA Profile, ACL-LAC Assay, Imaging; CT, MRI.

Brain with MRA, Doppler study of intracranial and extracranial vessels. Arterial biopsy was not obtained in any of the patients.

Identified cases of vasculitis were classified according to Clinical Pattern of Presentation SCOLDING et al 1997 and their response to steroids studied.

III. Results and Discussion

In our study of 20 cases of vasculitis Type 1 presentation was seen in 5 patients 25%. Type 2 presentation in 11 patients 55% and Type 3 presentation in 4 patients 20%. Type 2 presentation was the most common presentation. Type 1 and Type 3 patterns of presentation had radiological evidence of vessel involvement. Two cases had large vessel vasculitis. Six cases involved medium sized vessels. One case presented as polyangitis overlap syndrome with involvement of both large and small vessel. Type 2 presentation had no radiological evidence of vessel involvement due to small vessel involvement. Response to steroids; Type 1 and Type 3 patterns of presentation (9 patients) showed improvement with steroids. Type 2 pattern (11 patients) showed little response to steroids or remained static.
IV. Conclusion

Categorisation of patients based on clinical features correlates to some extent with the possible type of vessel involvement. Steroids do make a significant impact in recovery. Type 1 and Type 3 patterns improved with steroids. Type 2 pattern showed poor response to steroids.

References