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Commentary

Utility of a Novel Clinical Triad (Chandra's TRIAD) for Early Diagnosis of Autoimmune Encephalitis (AIE) in Patients with Progressive Cognitive Decline

Thomas Gregor Issac*

Department of Geriatrics Psychiatry Unit, National Institute of Mental Health and Neurosciences, Bengaluru, India

ABSTRACT

Autoimmune Encephalitis (AIE) often presents with cognitive and behavioural symptoms often mimicking progressive degenerative dementias. Early identification of these potentially reversible conditions can improve patient outcomes, reduce caregiver burden and promote cost effectiveness. The clinical trial of symptoms namely panic attacks, catatonia and day time sleepiness is often associated with anti N-methyl D-aspartate (NMDA) receptor mediated encephalitis and the former two symptoms along with seizures is suggestive of anti-Voltage Gated Potassium Channel (VGKC) receptor mediated encephalitis. This triad serves as a pragmatic tool to easily identify AIE and also helps to distinguish between the two conditions.

Keywords : Autoimmune encephalitis; Dementia; Anti-N-methyl D-aspartate receptor; Anti-voltage gated Potassium channel receptor

DESCRIPTION

Autoimmune Encephalitis (AIE) is a rare yet important neuropsychiatric condition which often masquerades as progressive cognitive decline [1]. This leads the clinician to often consider a degenerative etiology in most cases of progressive cognitive decline resulting in delay in appropriate management strategies and worsening of clinical condition leading to irreversible atrophy of the brain parenchyma, lesser response to treatment when diagnosed in later stages along with significant financial and caregiver burden [1,2]. The specific antibody panel required for AIE is often expensive and available only in tertiary or specialized centres especially in developing countries leading to underdiagnosis of the same. It is therefore pertinent that a high index of suspicion for diagnosing AIE is maintained and some clinically unique criteria to be considered which helps the clinician to establish a possible diagnosis of the same and facilitate early referral or initiation of appropriate immunomodulators thereby preventing further neuronal excitotoxic damage [3]. It is in this regard that some preliminary work initiated by the team headed by Chandra, et al. becomes relevant [4].

There is often a plethora of symptoms associated with AIE, the limbic region is most commonly affected, the most consistent yet common symptoms which was associated with AIE was planned to be identified in the study by Chandra, et al. which looked retrospectively in the great deal the symptomatology in various reversible dementias especially AIE [4]. The study shows the pattern in 35 patients recruited over a period of 5 years who were diagnosed to have features of AIE. One patient was excluded due to presence of co-morbid retroviral infection. In rest of the 34 patients, majority of the patients had anti- anti N-Methyl D- Aspartate (NMDA) antibodies (67.6%) and the rest had anti- Voltage Gated Potassium Channel (VGKC) antibodies (32.4%). The clinical presentations noted these patients and was subtyped, compared and analysed. The various symptoms which were identified were features of panic attacks, catatonia, occasional seizures and daytime sleepiness. These symptoms were assessed using not just subjective reports but various scales as well. DSM V criteria was used for identification of catatonia and when panic symptoms were reported, the Panic Disorder Severity Scale (PDSS) was utilized in addition to the Epworth Sleepiness Scale for assessing day time sleepiness [4].

The triad of symptoms which were identified in patients and of statistical significance were presence of Panic attacks, excessive daytime sleepiness and presence of catatonic symptoms (predominantly in anti (NMDA)-Receptor AIE) or seizures (predominantly in anti (VGKC)- Receptor AIE).This also

Correspondence to: Dr. Thomas Gregor Issac, Senior Resident, Geriatric Psychiatry Unit ,National Institute of Mental Health and Neurosciences (NIMHANS), Bengaluru, India, E-mail: thomasgregorissac@gmail.com

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corroborates to the anatomical substrates identified in AIE which are the limbic regions which account for panic symptoms and the hypothalamic structures with regards to excessive sleepiness and also the involvement of frontal subcortex and cortical structures which are implicated in catatonic symptoms and grey matter involvement resulting in seizures [5]. The authors have also implicated the role of these features to be consistent and early presentations in the patients with seropositivity detected after detailed evaluation.

Therefore, this novel clinical triad could be named as Chandra's Triad which could be of significant utility in aiding early diagnosis of AIE and could reduce delay in appropriate management thus reducing long term morbidity and cognitive decline in specific cases of neurocognitive decline which are potentially reversible and treatable but mostly remain misdiagnosed, underdiagnosed or sometimes even undiagnosed.

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