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Neuropsychiatric patterns in cerebral amyloid angiopathy and psychiatric presentations in old age

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Cerebral Amyloid Angiopathy (CAA) is diagnosed in various settings including stroke units, memory clinics and geriatric psychiatry. CAA is also observed in community dwelling populations. Clinical presentations including neuropsychiatric presentations were described in the last two decades. Various neuropsychological manifestations have been described which include impairment in perceptual speed, episodic memory, semantic memory, attention and executive function and global cognitive impairment. Neuropsychological manifestations included a new manifestation of high impulsivity, in addition to organic personality change, and depression. This study focuses on neuropsychological impairments and psychiatric manifestations observed in CAA patients and discusses the possibility of a neuropsychological profile for CAA.

Cerebral Amyloid Angiopathy (CAA) is a neurovascular disease characterised by b-amyloid fibrils deposited in the walls of cerebral blood vessels. 1. A contemporary systematic overview collectively studied the results of four population groups based on pathological studies concerning the link between the presence of CAA (of any severity) and dementia, and found that 55-59% of patients living with dementia also suffered from CAA, while a CAA prevalence of only 28–38% was determined in patients without dementia. 2. CAA micro bleeds are believed to occur in greater than 30% of adults over the age of 70, and are potentially even more prevalent given the amount of symptomatic individuals with micro bleeds. 3. CAA is diagnosed in various settings including stroke units, memory clinics and geriatric psychiatry. CAA is observed in community dwelling populations as well. 4. Risk factors in the development of CAA include age. 5. A genetic facto, apolipoprotein E alleles. 6. Clinical presentations including neuropsychiatric presentations have been described in the last two decades. These include symptomatic intracerebral haemorrhage, cognitive impairment and dementia, rapidly progressive cognitive and neurological decline and transient neurological symptoms.

Various neuropsychological manifestations have been described which include impairment in perceptual speed, episodic memory, semantic memory, attention and executive function and global cognitive impairment. Cognitive disorders in CAA patients were frequent, in numerous domains. Patients exhibited significant deficits in language, processing speed and executive and memory functions compared to the control group, but were not different on attention and praxis domains. Studies showed that naming was the most impaired process, as in the deep group, followed by executive and processing speed. This pattern of frontal cognitive dysfunction was already highlighted in the CAA population.

In clinical settings Modified Boston criteria allow for probable or possible CAA diagnosis or exclusion of diagnosis of CAA. The Modified Boston criteria were proposed in 2010 in order to incorporate cortical

superficial siderosis into the radiological diagnosis of probable CAA. They comprise of combined clinical, imaging and pathological parameters, and are based upon the original Boston criteria. The Modified Boston criteria for diagnosis of 'probable CAA' was pathologically validated in 2010, and compared to the Boston criteria had an increase in sensitivity (95%, 95% confidence interval (CI) 76% to 99%) with only a modest decrease in specificity (81%, 95% CI 62% to 93%).

Intracerebral haemorrhage from CAA is most common in the frontal lobes. CAA pathology might cause cortical atrophy, an effect that might at least partly be mediated by vascular dysfunction. CAA is mainly a neurological illness, but may present with psychiatric symptoms. Currently there are no studies that address the psychiatric manifestations of CAA in a systematic way. We report a case series describing four cases with neuropsychiatric manifestations presented to a geriatric psychiatric unit.

Case 1: A 77 year old male with a background of paroxysmal atrial fibrillation had presented with headache, drowsiness and left visual inattention. His Computerised Tomographic (CT) head scan showed a right temporal lobe acute intra parenchymal haemorrhage with 4mm of midline shift. A Magnetic Resonance Image (MRI) brain scan two months later showed a right temporal lobe residual haematoma with a background evidence of amyloid angiopathy on susceptibility weighted imaging (SWI) with confluent hyper intensity in the periventricular white matter of both occipital lobes on T2/FLAIR. On follow up he developed temporal lobe focal seizures. There was also evidence of cognitive decline [Addenbrooke's Cognitive Examination (ACE III) - 71/100]. He was referred to a psychiatrist for depression, anhedonia and poor sleep with little response to Citalopram. There were no frank delusions or hallucinations and past psychiatric history was unremarkable. As there was only partial control of seizures on Leviteracetam and the potential for this agent to contribute to low mood this was cross-titrated with carbamazepine and eventually stopped. Additionally, Citalopram was switched to Mirtazapine. Following this depression markedly improved and seizures abated.

Biography

Raghavakurup Radhakrishnan has completed his MBBS, DPM,DNB (Psychiatry); and he is a MRCPsych Consultant Psychiatrist in Old age Waitemata District Health Board, New Zealand. He has also published few articles in his area of interest which is Psychiatry.