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Editorial article

Obstructive Sleep Apnoea and Tau: A Good Night's sleep may detangle you from Alzheimer's!!

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Obstructive Sleep Apnoea (OSA):

Obstructive Sleep Apnoea (OSA) is a form of age-related sleep disordered breathing (SDB)[1].The etiology being in spite of continuous struggle to breathe, there occurs a recurrent partial or complete collapse of the upper respiratory tract while asleep.[2] The clinical features of OSA includes loud snoring, insomnia, choking or gasping during sleep, morning headaches, and daytime somnolence. Obstructive Sleep Apnoea is associated with obesity which is a critically emerging public health issue. The prevalence of OSA differs with age and sex. It is more prevalent in older men. About 20 to 50% of older adults suffer from OSA. [1]

Sleep Disordered Breathing (SDB) and mild cognitive impairment (MCI):

Cognitive derangement and dementia are the common occurrence in aged populations suffering from OSA. Studies show that the SDB (Sleep Disordered Breathing) may upraise the risk of mild cognitive impairment (MCI) or dementia over a period of 5 years. Patients with Sleep apnoea had an upsurge of 170% more risk of dementia when compared with controls shorn of sleep apnoea after obtaining age and sex matching [1].

Obstructive Sleep Apnoea and Alzheimer's disease (AD):

The most prevalent neuropsychiatric ailments in the older adult populations are depression and cognitive impairment. Research points out that a higher prevalence rate of depression in patients with OSA. Evidence from cognitive studies indicates that OSA is associated with diminished attention span, vigil, verbal episodic memory and decision-making skills. When compared with age, sex-matched healthy controls, old aged adults with OSA may posses a higher peril for cognitive impairment / Alzheimer's disease(AD). OSA is known to play a major role in disparaging the neural and vascular organizations of the Brain. Recent structural neuro- imaging studies postulate that, in individuals with OSA, there are evidence of atrophy in hippocampus, damages to microvasculature, along with derangement of integrity, abnormalities and hyperintensities in the white matter. There is also evidence of loss of gray matter. [2]

The amyloid cascade hypothesis:

Recent research discloses the most persuasive theoretical prototype that explains the etiological roots of OSA resulting in AD. The amyloid cascade hypothesis demonstrates the central etio pathological event that results in the in development of AD. It theorizes that the Root cause of neurodegeneration and dementia in brain eventually occurs due to the disparity amongst the process of synthesis and clearance of a neuro peptide called amyloid-β (Aβ) peptide. During the early phases of AD (preclinical AD), the soluble form of amyloid-β (Aβ) peptide is converted into an insoluble form.

The insoluble Aβ peptide is then amassed into solid plaques called amyloid plaques. This results in drop in the levels of soluble Aβ peptides in cerebrospinal fluid (CSF). While estimated during sleep, the levels of Soluble Aβ in the interstitial fluid (ISF) has been shown to decrease and to increase during wakefulness.[3]

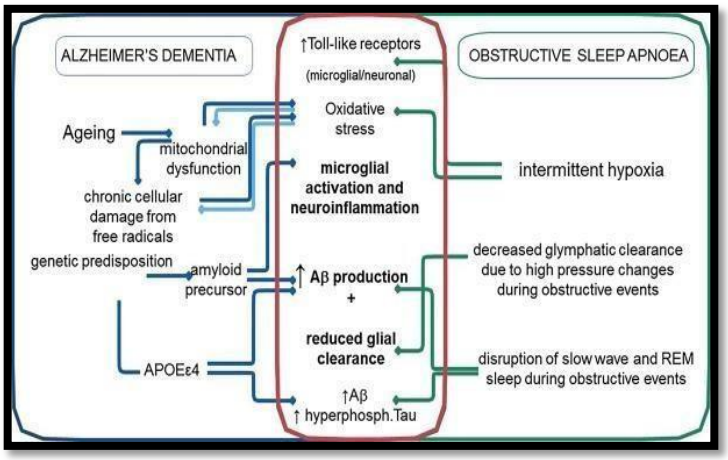
Tau Kinetics in Alzheimer's disease:

'Tau' is a protein which is present in the brains of normal healthy people. Under certain disease conditions, such as AD these tau proteins clumps together to create tangles. These tangles damage the adjacent neural tissue that leads to cognitive impairment. It is considered as Alzheimer's disease biomarker.[4] In early stages of AD, The Tau Tangles, gets deposited in the neurons of medial temporal lobe, *trans*-entorhinal cortex, amygdala and hippocampus. It does spreads to the neocortical association areas in the later stages. [3] As tau tangles mushroom and more areas become affected, people increasingly struggle to think clearly. [4,5]

Obstructive Sleep Apnoea and Glymphatic clearance:

OSA leads to diminished communication amongst interstitial fluid and cerebrospinal fluid in the brain. In OSA patients, the intrathoracic and intracranial pressure inclined to raise during the forced respiratory exertion in opposition to a closed airway. During this process, an abrupt reversal of pressures befalls at the last stage of apnoea, ensuing repetitive fluctuations of high-pressure differences. This hampers the glymphatic circulation from ISF into CSF consequential to retention of metabolites. The concentrations of Aβ, tau and resultant metabolites in the ISF is elevated. In patients with OSA complicated with chronic hypertension, vascular stiffness occurs in conjunction with cerebral amyloid angiopathy. Consequently, the resultant efficiency of glymphatic clearance diminishes leading to fast progression in accretion of amyloid plaque deposition which is identified as the central etiopathology in AD. [3]

Role of Obstructive Sleep Apnoea in Alzheimer's disease:



Conferring to a preliminary study, patients with OSA were scanned using PET (Positron emission tomography) revealed that they may have elevated levels of Tau protein tangles deposited in the region of brain called entorhinal cortex in the temporal lobe. This region is primarily concerned with memory functions, navigation management and time perception. Study found that the accumulation of Tau in the entorhinal cortex of patients with apnea, was about 4.5 % higher in than controls without apnea. [6]

Significant role of sleep disruptions has always been associated with Alzheimer's disease and its progression for decades. Long since it was hypothesized by the scientists that sleep disorders are associated with Alzheimer's disease with regards to the deposition of Alzheimer's protein called amyloid beta (Aβ) peptides in the brain. But the recent scientific study data suggests that sleep disruption grounds the accumulation of detrimental protein tau to form tangles that intensifies swiftly and spreads into the neighbouring areas of brain in no time leading to structural and functional derangements resulting in AD.[4,5]

Good night's sleep:

Trying to get a good night's sleep is essential in conserving the brain health. To recover the brain from the stresses caused all through the day, a good night sleep is more than necessary. Data suggests that the real-life factor, 'sleep' has a noteworthy role in the prevention, development, progression and spread of neural diseases such as AD. It may also delay and hold back the disease process.[4,5]

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