Case of Alzheimer’s disease with dysregulation of calcium & haemoglobin levels
– a Case Report

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Abstract: Alzheimer's disease (AD), the most well-known cause of dementia, is a perpetual neurodegenerative disease described by intellectual and behavioural impedance. This disease might be showed as a dynamic visuospatial disintegration and transient memory loss, gradually leading to uncoordinated bodily function and eventually to death. The patient we report here is a 71 year old female with loss of ability in finding directions and comprehension shape of materials. There was fronto temporal involvement in her CT scan investigations. Likewise, biochemical investigations demonstrated calcium and haemoglobin dysregulation. This case report demonstrates the importance of dysregulation of calcium and haemoglobin in confronting Alzheimer’s.

Keywords: Alzheimer’s disease (AD), dementia, visuospatial disintegration, transient memory loss

INTRODUCTION:
Alzheimer’s disease (AD), the most widely recognized dynamic neurodegenerative disease results from irreversible loss of neurons, especially in the cortex and hippocampus. A noteworthy focus of AD research has been to comprehend the genetic etiology of AD and its relationship to AD neuropathology. The key neuropathological features of AD are abundant neurofibrillary tangles composed of hyperphosphorylated tau protein and senile plaques made of β-amyloid (Aβ). The accumulation of Aβ is viewed as a central component in the pathogenesis of AD and has been associated with the 3 autosomal dominant, deterministic genes known to be involved in Early Onset Alzheimer’s Disease (EOAD), Presenilin 1 (PSEN1), Presenilin 2 (PSEN2), and Amyloid Precursor Protein (APP). A fourth gene, apolipoprotein E (APOE), has been affirmed as a risk factor for late onset AD (LOAD).

Alzheimer’s disease (AD), results from irreversible loss of neuronal cells and synaptic degeneration in influenced regions of the brain, first in the hippocampus and entorhinal cortex, and later in the temporal and parietal lobes, and additionally in the frontal and occipital lobes [1]. The AD is characterized by two major brain lesions, referred to as neurofibrillary tangles (NFT) of hyperphosphorylated tau protein and senile plaques made of β-amyloid (Aβ).

The amyloid-forming protein, named β-amyloid (Aβ), is a peptide of 40–43 residues in length, which is produced by proteolytic cleavages of the longer amyloid precursor protein (APP). The plaque centre is encompassed by dystrophic neuritis, activated microglia and reactive astrocytes, indicating that amyloid deposition gives rise to inflammatory responses. Aβ depositions likewise occur as diffuse plaques (detected only by immunohistochemical methods) and can also be found in the walls of small cerebral blood vessels. The neurofibrillary tangles (NFT) are composed of unusually phosphorylated tau, a microtubule binding protein. The hyperphosphorylated tau assembles in paired helical filaments (PHF) and accumulates in the cytoplasmic compartment of the neurons [2-9].

CASE PRESENTATION:
A 71-year-old Indian housewife female with no formal education, presented to hospital was diagnosed with Alzheimer’s disease. The patient complained of inability to perform her daily work satisfactorily and experienced continuous neglect in finding items and issues in navigating to required
fact that the direct mechanistic connection between calcium dysregulation and AD pathology is still under scrutiny, one may infer that calcium-intervened pathogenesis influencing numerous cell frameworks. The study by Mattson MP et al.; showed that perturbed synaptic ER calcium homeostasis result in neuronal dysfunction and death in Alzheimer’s disease (AD) by aberrant proteolytic processing of the beta-amyloid precursor protein (APP). This study demonstrate that PS1 mutations cause abnormalities in ER calcium homeostasis leading to neuronal degeneration promoted by increasing levels of the neurotoxic forms of beta-amyloid (Aβ) and by decreasing the levels of the neuroprotective secreted form of APP (sAPP alpha). Eventually, these facilitate Aβ and tau deposition, loss of synapses, and ultimately, loss of memory [11–13]. In this way, considering calcium dyshomeostasis as a fundamental part of AD-connected synaptic pathology may yield new insights into the cellular mechanisms of cognitive deficits and offer novel therapeutic interventions.

Second, dysregulated hemoglobin levels may be a risk factor for AD with subjective decay and expanded mortality in the elderly subjects [14]. R.C. Shah, et al.; reported that hemoglobin concentrations in elderly subjects are associated with a lower level of cognitive function, particularly in semantic memory and perputal speed [15]. Cross sectional study by Denny SD et al.; and Chaves PH et al.; provide evidence in support of the hypothesis that dysregulated hemoglobin levels might be an independent risk factor for functional and cognitive impairment in elderly subjects [16, 17].

Lastly, the CT-Scan contemplated from patient indicated atrophic changes conspicuous in frontal and temporal lobes. Extreme atrophic change with prominent sulci and ventricles was seen in alzheimer patient with widening of occipital horn of lateral ventricle.

Table 1: Electrolytic profile of Alzheimeric patient

<table>
<thead>
<tr>
<th>S.No</th>
<th>Parameters</th>
<th>Results</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Na+</td>
<td>135.0 mmol/L</td>
<td>136.0 mmol/L</td>
</tr>
<tr>
<td>2.</td>
<td>K+</td>
<td>4.3 mmol/L</td>
<td>3.5 mmol/L</td>
</tr>
<tr>
<td>3.</td>
<td>Ca++</td>
<td>1.20 mmol/L</td>
<td>1.05 mmol/L</td>
</tr>
</tbody>
</table>

Table 2: Hematological profile of Alzheimeric patient

<table>
<thead>
<tr>
<th>S.No</th>
<th>Parameters</th>
<th>Results</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Haemoglobin (HGB)</td>
<td>7.9 g/dl</td>
<td>11.5</td>
</tr>
<tr>
<td>2.</td>
<td>HCT</td>
<td>28.9 %</td>
<td>35.0</td>
</tr>
<tr>
<td>3.</td>
<td>Mean Corpuscular Hemoglobin (MCH)</td>
<td>19.3 pg</td>
<td>25.0</td>
</tr>
<tr>
<td>4.</td>
<td>Mean Corpuscular Hemoglobin</td>
<td>27.3 g/dl</td>
<td>30.0</td>
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CONCLUSION:
In conclusion, we recently diagnosed Alzheimer’s patient with the help of CT scan examination and dysregulated biochemical parameters. This case report emphasizes the importance of dysregulation in ionized calcium and hemoglobin in AD diagnosis.

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COMPETING INTERESTS:
The authors declare that they have no competing interests.

CONSENT:
Written informed consent was obtained from the patient.

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