ISSN No:-2456-2165

Multi-Infarct Dementia with Acute Intra Cerebral Hemorrhage and Chronic Subdural Hygromas: A Case Study

Kanaga K.*[1], Miriyam Claudish AJ.[2], Tharani S.[3], Karthika J.[4]
[1,2,3]M. Pharm, [4]Pharm.D, The Tamilnadu Dr. M. G. R. Medical University, Chennai, INDIA

Abstract:- Multi-infarct dementia (MID) is a prevalent cause of dementia in older adults. In the current report, we look at a small acute haematoma in the right parietal lobe, a small infarct in the right thalamus and occipital lobe, a multiple chronic infarct in both cerebral hemispheres and a mild subdural hygroma over bilateral cerebral convexity caused by Diabetes and Coronary Artery Disease. A 86-year-old man patient was diagnosed with Multi-infarct dementia, Acute ICH in the right parietal lobe, Chronic subdural hygroma and Vitamin D3 insufficiency. The patient was administered with Diuretics, Anti-convulsants, Anti-Depressants, Cholinesterase Inhibitors, Antibiotics, Psychostimulants, Nootropics and Anti-hyperlipidemic drugs. Early identification, proper Blood Pressure (BP) management and therapy are all required for the treatment of Multi-Infarct Dementia.

I. INTRODUCTION

In the ICD-11,Vascular Dementia is referred to as Dementia owing to cerebrovascular illness.[1]Vascular dementia (VaD) or Vascular cognitive impairment is a kind of Dementia is caused by the problem with blood flow to the brain due to cerebrovascular illness. Ischemia or decreased blood flow results in the tissue and cell destruction of afflicted region is known as an infarct. The three kinds of vascular dementia include subcortical vascular dementia, multi-infarct dementia and stroke-related dementia. Subcortical vascular dementia is caused by damage to small artery walls in the central nervous system. A series of mini-strokes affects many areas, resulting in multi-infarct dementia. The third type involves strokes, which may leads to more severe damage. [2] Multi-Infarct Dementia (MID) is a prevalent cause of dementia in the elderly. MID is caused by an ongoing sequence of strokes (disruptions in the circulation to the brain). A disruption in blood flow damages the brain tissue. Some of these strokes may have lack of obvious clinical signs. These events are referred by doctors as "silent strokes."[3] Cognitive impairment is typical in people with Intra Cerebral Haemorrhage (ICH), regardless of age. Pathology of the lung ICH is commonly related to Cerebrovascular Amyloid Angiopathy and Cognitive Impairment.[4]

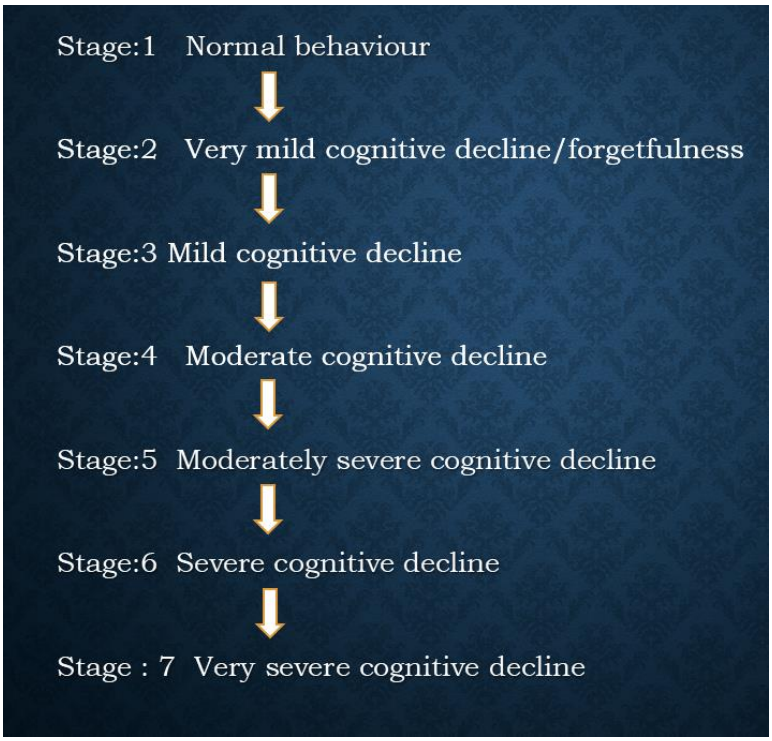

Fig. 1: Stages of Dementia with vascular involvement

The Stages of Dementia with vascular involvement have been described above.[5]

II. RISK FACTORS

Age factors, elevated blood pressure, cigarette usage, excessive cholesterol levels, diabetes type 2, arterial disease and cerebrovascular illness. In addition Geographic origin, genetic propensity and past strokes are all risk factors.6

Cerebral Amyloid Angiopathy, which includes the formation of beta-amyloid plaques in the walls of the cerebral arteries can occasionally leads to Vascular Dementia. While amyloid plaques are a common component of Alzheimer's Disease, Vascular Dementia may develop as a result. Cerebral amyloid angiopathy, on the other hand this can develop in persons who never had dementia. The amyloid beta buildup is common in older adults with normal cognitive function.7

The risk factors for multi-infarct dementia are same to those for stroke. They are as follows:

High levels of blood pressure, Diabetes, Cardiovascular disease, Congestive heart disease, problems in heart valves, inflammation of carotid arteries, Hyperlipidemia (high LDL cholesterol levels) and Smoking.8

III. SIGNS AND SYMPTOMS

People with vascular dementia experience increasing cognitive deterioration which is either immediately or subacutely as in moderate cognitive impairment and commonly stepwise, following several cerebrovascular episodes (strokes). Some patients may appear to recover in between quiet strokes and then deteriorate. A fast worsening condition may result in mortality due to a stroke, heart disease or infection.[9]

The signs and symptoms are cognitive, motor, behavioral in a considerable number of patients and emotional. These changes often takes place between 5-10 years to manifest. The signs are typically the same as in other dementias but they primarily include cognitive decline and memory impairment severe enough to interfere with daily activities, sometimes with the presence of focal neurologic signs and evidence of features consistent with cerebrovascular disease on brain imaging (CT or MRI).[10]

Multi-Infarct Dementia (MID) symptoms might appear unexpectedly.Confusion or difficulties with short-term memory, wandering or being disoriented in familiar surroundings, walking quickly, shuffling steps, inability to control one's bladder or bowels, laughing or sobbing inappropriately, difficulty adhering to instructions, problems with money counting and monetary transactions. Some persons with MID appears to have improved symptoms for brief period of time but later deteriorate after having further strokes.[8]

IV. CASE DESCRIPTION

An 86-year-old male patient was brought to the neurology department. He had complained of generalized weakness, memory impairment and involuntary micturition for the previous 20 days. While admitted, the patient was conscious oriented and afebrile. His vital signs such as saturated oxygen, pulse rate and respiratory rate were all normal. He had a history of type II Diabetes with irregular treatment and Coronary Artery Disease with no treatment. The patient's previous medication history included T. Metformin 500mg OD and T.Tamsulosin 2.5mg HS. The patient has no family history.

The patient's CT Brain Plain report suggested that he had Small acute haematoma in right parietal lobe, Small infarct right thalamus and occipital lobe , Multiple Chronic infarct in bilateral cerebral hemisphere and Mild subdural hygroma over bilateral cerebral convexity.The patient haematology study report suggested that the Prothrombin Time (PT) and International Normalised Ratio (INR) was found to be elevated. From the patient's demographics and imaging study report the patient was diagnosed to have Multiple Infarct Dementia, Acute ICH in right parietal lobe , Chronic subdural hygroma and Vitamin-D3 deficiency**.** Then the patient was treated with the following drugs for 5 days: Inj. Mannitol 100ml IV TDS, Inj. Levetiracetam 500mg IV BD (Anti- convulsant), Inj.Ondansetron 4mg IV BD (Anti-emetic), Inj.Methylcobalmin 1000mg IV OD (Vitamin injection), T.Atorvastatin 40mg HS (HMG CoA reductase inhibitor), Inj.Citicoline 500mg orally (Psychostimulant), T.Donepezil 5mg orally (Cholinesterase inhibitor) and T.Memantine 5mg orally (NMDA Receptor antagonists).On day 3, a STAT dose of Vitamin D3 was administered and the Inj.Levitiracetam 500mg IV BD(anti- convulsant) was replaced with T.Escitalopram(Anti-depressant) + T .Clonazepam HS (Anti-convulsants) and discontinued. On the day 4, T.Cefixime 200mg BD(Cephalosporin antibiotic) and T.Clindamycin 600 mg OD (lincomycin antibiotic) were added.Treatment at the time of discharge:T.Atorvastatin 20mg HS, T.Citicoline 500mg OD, T.Cefixime 200 mg BD,T.Pan-D 40 mg, T.Methylcobalmin 1500mcg OD,T.Thiamine 100mg OD for 5 days with Vitamin D3 sachet for weekly once. After the course of treatment, he was discharged home on the day 5 with only a marginal improvement.

ISSN No:-2456-2165

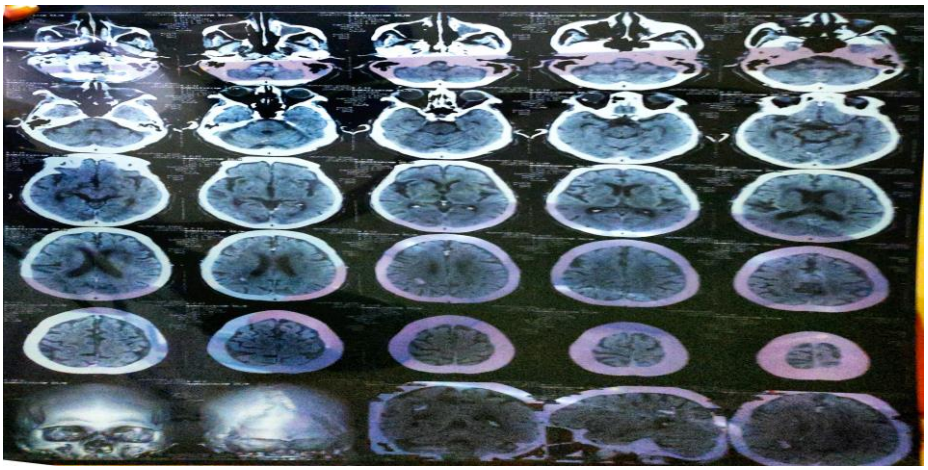

Fig. 2: Axial View Of MRI

V. DIAGNOSIS

Blood tests (for anaemia, vitamin insufficiency, thyrotoxicosis, infection and so on), X-rays of the chest, ECGs and neuroimaging, preferably a scan with functional or metabolic sensitivity beyond a standard CT or MRI are all suggested examinations for cognitive impairment. Single Photon Emission Computed Tomography (SPECT) and Positron Emission Tomography (PET) neuroimaging may be used in conjunction with mental status exams to confirm a diagnosis of multi-infarct dementia when accessible as a diagnostic tool.[11]

Diagnosis Criteria
(NINDS-AIREN criteria)

Most cases of acute post stroke vascular dementia connect the modified criteria for probable vascular dementia.

- When executive dysfunction results in a loss of functional independence, dementia with an acute onset is present.
- Neuroimaging demonstrates relevant cerebrovascular lesions.
- There is a clear temporal relationship between stroke and cognitive loss. This last criterion is typically not met in subacute vascular dementia.[12]

VI. TREATMENT

Research has been proposed to examine if a plant called ginkgo biloba EGb761 extract improves mental processes, routine tasks and quality of life quality in the treatment of vascular dementia.[13]

Multiple trials revealed Memantine, a Non-competitive N-methyl-D-aspartate (NMDA) receptor antagonist and the cholinesterase inhibitors galantamine, donepezil and rivastigmine to have a small benefit in the treatment of VaD.[14]

There is no current therapy to reverse the brain damage produced by a stroke. Treatment focuses on preventing future strokes by treating or avoiding the illnesses and medical conditions that put people at risk of stroke, such as hypertension, diabetes, high cholesterol and cardiovascular disease. Prevention includes eating a good diet, exercising, avoiding smoking, consuming alcohol in moderation and maintaining a healthy weight is the best treatment for MID.[15]

VII. DISCUSSION

While the association between ICH and cognition is clinically known, the available data do not appear to indicate a clear causal relationship between brain bleeding and cognitive decline. While there may be an ICH volume effect on cognition in the acute time following ICH, it appears that cognitive impairment is largely driven by the kind in degree of underlying illness in the long term. Future therapeutics focused at reducing ICH-related acute symptoms may improve patient outcomes.[16]

Cognitive impairment (CI) is a common complication of Intra Cerebral Haemorrhage (ICH). While a growing number of studies have investigated this link, several evidence gaps continue to exist.[17] According to the University of Manchester, the researchers want to test Amlodipine as a treatment for vascular dementia in humans. If it is successful, this will be the first clinically validated therapy for vascular dementia caused by small vessel disease and it may be used to prevent further progression in people who show early signs of the condition.

VIII. CONCLUSION

We present the case of, an 86-year-old male patient with multi-infarct vascular dementia. The patient was administered with Diuretics, Anti-convulsants, Anti-depressants, Cholinesterase Inhibitors, Antibiotics, Psychostimulants, Nootropics and Anti-Hyperlipidemic drugs. The patient condition was marginally improved over time and he was finally discharged.

REFERENCES

[1.] "ICD-11 for Mortality and Morbidity Statistics". *icd.who.int*. Retrieved 13 June 2023.
[2.] "Types of vascular dementia | Alzheimer's Society". www.alzheimers.org.uk. Retrieved 13 May 2023.
[3.] https://www.agingcare.com/articles/overview-symptoms-multi-infarct-dementia-110701.htm
[4.] Xiong L, Reijmer YD, Charidimou A, Cordonnier C, Viswanathan A. Intracerebral hemorrhage and cognitive impairment. Biochimica et Biophysica Acta

(BBA)-Molecular Basis of Disease. 2016 May 1;1862(5):939-44.
[5.] https://dementech.com/2022/06/28/what-are-the-7-stages-of-vascular-dementia Retrieved 13June 2023.
[6.] https://web.archive.org/web/20120120004037/http://www.dcmsonline.org/jax-medicine/2000journals/February2000/vascdement.htm Retrieved 13June 2023.
[7.] Vlassenko AG, Mintun MA, Xiong C, Sheline YI, Goate AM, Benzinger TL, Morris JC. Amyloid-beta plaque growth in cognitively normal adults: Longitudinal [11C] Pittsburgh compound B data. Annals of neurology. 2011 Nov;70(5):857-61.
[8.] https://my.clevelandclinic.org/health/diseases/6063-multi-infarct-dementia Retrieved 13June 2023.
[9.] "NINDS Multi-Infarct Dementia Information Page". *www.ninds.nih.gov*. Retrieved 13 May 2023.
[10.] *Encyclopedia of the Human Brain - Dementia Associated with Depression* Retrieved 13 May 2023.
[11.] Bonte FJ, Harris TS, Hynan LS, Bigio EH, White III CL. Tc-99m HMPAO SPECT in the differential diagnosis of the dementias with histopathologic confirmation. Clinical nuclear medicine. 2006 Jul 1;31(7):376-8.
[12.] Roman GC. Vascular dementia. InNeurology and clinical neuroscience 2007 Dec 1 (pp. 635-643). Elsevier.
[13.] Wang M, Peng H, Peng Z, Huang K, Li T, Li L, Wu X, Shi H. Efficacy and safety of ginkgo preparation in patients with vascular dementia: a protocol for systematic review and meta-analysis. Medicine. 2020 Sep 9;99(37).
[14.] Birks J, McGuinness B, Craig D. Rivastigmine for vascular cognitive impairment. Cochrane Database of Systematic Reviews. 2013(5).
[15.] https://www.brainfacts.org/diseases-and-disorders/neurological-disorders-az/diseases-a-to-z-from-ninds/multi-infarct-dementia Retrieved 13 May 2023.
[16.] Brouwers HB, Chang Y, Falcone GJ, Cai X, Ayres AM, Battey TW, Vashkevich A, McNamara KA, Valant V, Schwab K, Orzell SC. Predicting hematoma expansion after primary intracerebralhemorrhage. JAMANeurol71 (2): 158–164.
[17.] Potter T, Lioutas VA, Tano M, Pan A, Meeks J, Woo D, Seshadri S, Selim M, Vahidy F. Cognitive impairment after intracerebral hemorrhage: a systematic review of current evidence and knowledge gaps. Frontiers in neurology. 2021:1493.