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Case Report

Hyperhomocysteinemia and Ischemic Brain Injury: A Rare Case of Cystic Encephalomalacia in an Adult

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ABSTRACT

Cystic Encephalomalacia refers to the destruction of brain tissue following cerebral infarction and ischemia, infection, head trauma, or other injury. Cystic encephalomalacia (CE) is a most rare disease in adults. Though multiple causative factors come into play, hyperhomocysteinemia is now regarded as a potent aetiology. Here, we describe a 48-year-old male with previous history of cerebrovascular accident (CVA) who was admitted with multiple episodes of seizures. MRI showed chronic infarcts with cystic encephalomalacia changes, and metabolic screening demonstrated elevated levels of homocysteine. This is a case showing the significance of metabolic factors in ischemic cerebral injury and early screening. Differential diagnosis of CE from other changes due to ischemia is of paramount importance to ensure proper treatment and prognosis.

Keywords: Cystic Encephalomalacia; Hyperhomocysteinemia; Ischemic cerebral injury; Homocysteine

INTRODUCTION

Cystic encephalomalacia frequently occurs in newborn who have experienced prenatal or perinatal hypoxic incidents. This condition is typically uncommon in adult population.[1] Cystic encephalomalacia refers to the presence of cystic degenerative cerebral spaces resulting from the injury.[2] Such injuries may be caused by head trauma,[2] infection,[3] [4] or ischaemia, infarction. [5] CE threatens patient health by causing brain tissue necrosis and cystic degeneration, leading to severe neurological symptoms. Linked to a notable increase in cerebrospinal fluid volume, is typically accompanied by liquefied residuals of necrosis, and hemosiderin. This condition may result in movement disorders, seizures, cognitive decline, memory loss, and social impairments.[6]

Mild hyperhomocysteinemia (hHcy) predisposes to stroke because it participates in atherosclerosis.[7] Its elevation is brought about by deficiency of vitamin B12, B6, and folate, which are frequent in developing Countries.[8] No clinical guidelines advocate Hcy treatment but controlling its level may be one of the proposed methods for preventing and treating stroke.[9] No specific treatment is available for cystic encephalomalacia.[10]

CASE HISTORY

A 48-year-old male with a history of an old CVA and left-sided hemiparesis (6 months prior) had been on Ayurvedic medicine and was brought to the casualty with symptoms of involuntary movements in all four limbs, frothing from the mouth, and loss of consciousness following seizure-like activity that lasted for 5 minutes. Postictal confusion, which lasted for 30 minutes, was present, along with weakness in the left lower limb. Patient was in altered sensorium, exhibiting irrelevant speech and agitation, and was not obeying commands. Spontaneous eye movements were observed. The patient was alert, with a GCS of 13/15 (E4V4M5), and had bilaterally small (2mm) and reactive pupils. Neurological examination revealed

bilateral extensor plantar responses (positive Babinski sign), left-sided hypertonia, and asymmetrical quadriparesis, with 0/5 power in both the left upper and lower limbs (complete paralysis) and decreased power in the right upper limb (3/5) and right lower limb (2/5). Early CT of the brain showed no acute infarct, bleed, or cerebral oedema. Two days later, a CT scan of the brain revealed hypodensity in the right fronto-parieto-temporal region, consistent with gliosis.

MRI findings revealed a subacute infarct with haemorrhagic transformation involving the left tail of the caudate, the fronto-parieto-temporal lobes, and the insular cortex. Additionally, chronic infarcts with cystic encephalomalacia changes (Figure-1) were observed in the right frontoparietal lobes, corona radiata, internal capsule, external capsule, insular cortex, right caudate nucleus, and the left head of the caudate nucleus, indicating sequelae of a prior cerebrovascular accident (CVA). T2/FLAIR hyperintensity with diffusion restriction in the tail of the left caudate nucleus, insular lobe, and fronto-parieto-temporal lobes was suggestive of an acute ischemic stroke. Blooming on SWAN MRI indicated haemorrhage or iron deposition. GyT1 hyperintensity with hemosiderin deposition (iron storage due to previous bleeding) in the right frontoparieto-temporal lobes suggested prior ischemic injury with cortical laminar necrosis.

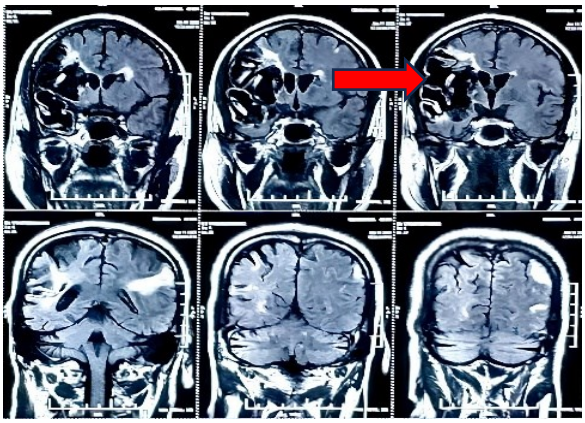

Figure-1 MRI: Chronic infarct with cystic encephalomalacia changes in right corona radiata, internal capsule, external capsule, insular lobe cortex, right caudate, head of left caudate nucleus and right fronto-parieto-temporal lobes as described-Sequelae of prior insult

MR angiography of the circle of Willis and neck vessels revealed non-visualization of the right internal carotid artery (ICA) from its origin and the middle cerebral artery (MCA), with collateral circulation via the anterior cerebral artery (ACA), which suggested an underlying thrombus and chronic small vessel ischemic changes. Bilateral carotid and vertebral artery Doppler imaging revealed absent color uptake and no spectral waveform in the right ICA, further supporting the presence of a thrombus. Electroencephalography (EEG) demonstrated low-voltage generalized slow waves, indicative of diffuse cerebral dysfunction. Coagulation parameters, including PTT, INR, and APTT, were within normal limits. Inflammatory markers revealed elevated CRP (45 mg/L). The lipid profile was within the normal range. Metabolic studies indicated severely elevated homocysteine levels (HCY: 40.8 µmol/L), which could suggest underlying metabolic dysfunction contributing to vascular pathology.

In this patient, hyperhomocysteinemia contributed to chronic ischemic changes and recurrent infarcts. Brain injury due to thrombus formation in the right ICA and MCA, along with an old right-sided CVA (right MCA territory infarct) as a complication of this brain injury, led to the development of cystic encephalomalacia. The patient presented with symptoms of generalized seizures and hemiparesis, along with right upper motor neuron (UMN) palsy as a complication of encephalomalacia (CE). He was treated with Clopidogrel (75 mg) & Aspirin (150 mg) to Prevent thrombus formation and reduce stroke recurrence, Heparin (50000 IU) – Prevent clot propagation in the right ICA and MCA, Levetiracetam (500 mg) – Control and prevent recurrent seizures, Mannitol (100 ml) – Reduce intracranial pressure and cerebral edema, Telmisartan (40 mg) & Amlodipine (5 mg), Bisoprolol (2.5 mg) – Optimize blood pressure control to reduce further ischemic risk, Cyanocobalamin, Folic Acid, Pyridoxine – Lower homocysteine levels to reduce vascular damage. Physiotherapy was initiated, and shunting was recommended. The patient showed symptomatic improvement and was discharged.

DISCUSSION

Increased homocysteine concentration (16 µmol) contributed predictive ability to stroke recurrence or death in addition to vascular risk factors (hypertension).[11] Herein, in this case, the patient exhibited a high level of HCY: 40 mc mol/L, which is the major risk factor for recurrent stroke and chronic cerebral ischemia. It is presently well established that vitamin B treatment lowers total Hcy levels, which results in a decreased stroke risk. A number of studies

and meta-analyses, such as the HOPE-2 trial, the French SuFolOM3 trial, a subgroup analysis of the Vitamin Intervention for Stroke Prevention trial excluding patients with renal failure, and a subgroup analysis of the VITATOPS trial excluding patients on antiplatelet therapy, all demonstrated a decreased risk of stroke. Levels of tHcy are a potentially modifiable risk factor for stroke.[9] Here this patient was given vitamin B supplements like, Neurobion Forte, Oxintoff plus, Homocheck.

Encephalomalacia refers to the destruction of brain tissue following cerebral infarction and ischemia, infection, head trauma, or other injury. These injuries most commonly are found in infants and children; but uncommon in adults. Most of symptoms will vary with the size and location with neurological and/or seizure disorders. With manifestations often hemiparesis and epilepsy.[10-14] Magnetic resonance imaging (MRI) is a radiation-free, non-invasive imaging method commonly used in the diagnosis of severe head injury due to its high specificity and sensitivity in the detection of CE of varying etiology. Based on the clinical information, the potential solitary or concomitant primary etiologies of CE were hypoxic ischemic injury (38%), traumatic brain injury/hemorrhage (28%), intracranial infection (26%), premature birth (10%), cerebral infarction (8%), genetic disorders (4%), and neonatal hypoglycemia (2%).[6] This patient has hypoxic ischemic injury and cerebral infarction as an etiology. Fatish Serhat erol et al reported a patient who developed epileptic seizures due to encephalomalacia cysts as a late complication of radiotherapy for basal cell carcinoma, illustrating the potential for cystic encephalomalacia to act as an epileptogenic focus.[15] Currently no specific treatment is available. The treatment modalities employed are surgical removal of the cysts, antiepileptics, shunting, rehabilitation and physical therapies. Treatment for a lifetime and case monitoring are required as in patients with epilepsy to enhance quality of life. [10,16] Here, in our patient CE is treated symptomatically with antiepileptics and root cause of CE was hyperhomocysteinemia for recurrent infarction was treated with vitamin B supplements, antiepileptics, anticoagulants and physiotherapy.

And as a complication of CE, generalized seizures seen following neurological deficits like weakness in left lower limb and altered sensorium, right UMN palsy, was treated with antiepileptic drug like Levetiracetam, Clobazam and antipsychotics like Haloperidol for postictal confusion.

CONCLUSION

This case emphasizes the role of hyperhomocysteinemia in recurrent ischemic infarcts, resulting in cystic encephalomalacia and extensive neurological impairment. Early identification and treatment of metabolic risk factors (hyperhomocysteinemia) are essential to avoid further progression. Though definitive therapy is not available, a multidisciplinary treatment with anticoagulants, antiepileptics, and physiotherapy can bring about betterment. This case highlights metabolic screening in stroke patients to avoid long-term complications.

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