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Artery of percheron infarct - a rare thalamic stroke

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Abstract :
Thalamic infarcts can occur in about 15 of all stroke patients mostly presenting as sensory disturbances in one half of the body with or without hemiparesis. Large thalamic infarcts causing coma and mortality are rare. We report a rare case of stroke in young presenting as coma initially and subsequently found to have bilateral medial thalamic infarct due to Artery of Percheron occlusion on MRI of the brain. A 28 year old male, non-hypertensive, non- diabetic with no prior heart disease admitted in medical ward with altered sensorium for few hours with no previous episodes or illness. On examination he had no focal deficit except for altered sensorium. Even though there are numerous causes for coma, early imaging in this patient revealed the cause of coma as stroke due to bilateral thalamic infarct. The patient regained the consciousness one day later and found to have other features of bilateral thalamic infarct such as vertical gaze palsy, loss of accommodation , memory disturbances . On further investigations he was confirmed to have artery of percheron infarct as well as hyperhomocysteinemia.The patient improved in about a week and discharged with recent memory disturbance and vertical gaze palsy. This patient is presented for the rare cause of stroke-hyperhomocysteinemia with thalamic artery of percheron infarct and emphasized the need for early imaging.

INTRODUCTION:

Stroke is a major cause of death and disability worldwide. Strokes occur either in anterior circulation or posterior circulation. Posterior circulation supplies approximately one-fifth of the total brain. These areas include cerebellum, brainstem ,occipital lobes, medial temporal lobes and thalamus. Posterior circulation is formed by 1 basilar artery, 2 vertebral arteries and 2 posterior cerebral arteries[1]. Thalamic stroke can occur in isolation or in combination with other areas of involvement. Isolated involvement of thalamus is not very common compared to other areas of involvement.

Thalamic stroke can manifest in different ways ranging from numbness or severe thalamic pain and even coma[2]. Herewith, we are reporting a young patient

CLINICAL FEATURES:

A 28 year old male, construction worker, non-hypertensive, non- diabetic with no prior heart disease or transient ischemic attack was admitted in medical ward with history of altered sensorium for few hours with no previous episodes or illness. On that day he had giddiness and slept for few hours and couldn't be woken up from sleep. There is no history of weakness of face or limbs. There is no history of fever, headache, seizure, drug or substance intake prior to the onset of illness.He is an occasional alcoholic and regular smoker. No history of similar illness in the family.On examination patient was afebrile, not icteric, he was in altered sensorium, moves all 4 limbs to stimulus and no facial weakness. All deep tendon reflexes were normal. Plantar reflex was flexor. Fundus examination was normal. No other focal deficit seen. Vital signs were stable.No signs of meningeal irritation seen. Other systems were clinically normal. On regaining consciousness next day, he had recent memory impairment, loss of accommodation reflex and vertical gaze palsy present. Motor, sensory and cerebellar systems were normal. INVESTIGATIONS Routine biochemical investigations were done. . His complete hemogram, blood sugar, renal parameters,lipid profile, serurm electrolytes were normal. His blood Venereal disease research laboratory, Human immunodeficiency virus 1&2 , Hepatitis B surfaceAntigen, Anti Hepatitis C virus – were all negative.Urgent computed tomography of brain was done which showed bilateral medial thalamic hypodensities. On further investigations,serum protein C and protein S levels were normal.serum homocysteine elevated [41µmol/l].His Electrocardiogram , echocardiogram, carotid & vertebral doppler were normal. Cerebrospinal fluid-biochemical analysis and cells are normal, Japanese encephalitis and varicella antibodies were negative. He was confirmed to have bilateral medial thalamic infarct due to artery of percheron occlusion in magnetic resonance imaging of brain .

The patient was treated medically with antiplatelets, lipid lowering drugs and folic acid and vitamins. He improved in about a week and was discharged with minimal memory disturbance and vertical gaze palsy.

DISCUSSION:

Thalamic infarcts can occur in about 15% of all stroke patients mostly presenting as sensory disturbances in one half of the body with or without hemiparesis. Large thalamic infarcts alone causing coma is rare. Unlike other regions, thalamus has got different regions and multiple different nuclei and connected to different areas of brain through extensive connections with afferent and efferent fibres[3]. Thalamus subserves different functions ranging from sensory function to maintaining arousal state. Hence a thalamic lesion can have different and varied manifestations ranging from a common and classical hemisensory loss to comatose state. The risk factors for thalamic vascular syndromes are similar to other areas of stroke. As like other areas of stroke the incidence of thalamic stroke increases in old age[4]. The thalamus is supplied by multiple small vessels originating from the posterior communincating arteries and P1 and P2 segments of the Posterior cerebral arteries. The paramedian thalamic vessels arise separately in each basilar communicating arteries or from a vascular arcade connecting basilar communicating arteries . It often arise from 5 a single pedicle that originates in one of the basilar communicating arteries . Therefore, unilateral posterior cerebral artery occlusions may result in bilateral paramedian thalamic infarcts causing [6,7]

Transient loss of consciousness or somnolence; occasionally akinetic mutism Behavioral changes (confusion, agitation, aggression, disorientation, apathy, manic delirium, a frontal lobe-like syndrome) Recent memory loss (with anterograde and retrograde components); persistent memory loss is observed only with damage of the dominant anterior nucleus or mamillothalamic tract. Vertical gaze and convergence defects Contralateral hemiataxia, asterixis, or motor weakness The common clinical triad of artery of percheron of infarct – altered sensorium , memory disturbance and vertical gaze palsy classicaly present in our patient. On investigations, he had moderate elevation of serum homocysteine level apart from smoking history as an important predisposing factor for the development of stroke in this young patient.

CONCLUSION :

In this patient, even though there was very minimal neurological deficit at presentation and since there are numerous causes for coma, early imaging revealed the cause of coma as stroke due to bilateral medial thalamic infarct . This patient is presented for its interesting association of hyperhomocysteinemia with thalamic artery of percheron infarct. This also highlighted the association of hyperhomocysteinemia alone as an obvious predisposing factor in this young patient causing stroke. This presentation emphasized the need for early imaging even though very minimal clinical signs present.

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