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Case Report

Basilar artery thrombosis due to hyperhomocysteinemia treated conservatively: a therapeutic success

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ABSTRACT

Basilar artery thrombosis is a rare and potentially fatal cause of posterior circulation stroke. Among the various etiologies, hyperhomocysteinemia is crucial and often under diagnosed, especially in developing countries. Authors describe the case of a 15-year male who presented with 1-day history of headache, vomiting and altered mental status. Non-contrast CT Head revealed multiple acute infarcts in posterior circulation of brain. Laboratory investigations revealed hyperhomocysteinemia. Contrast enhanced MRI Brain showed acute infarcts in bilateral cerebellar hemispheres, pons, midbrain, medulla and vermis. MR venography was suggestive of Basilar Artery thrombosis. Thrombolysis was not done due to delayed presentation; hence authors resorted to conservative management with folic acid and vitamin B supplementation. The patient showed gradual clinical improvement and had complete clinical recovery during follow up visit. A favorable outcome with conservative management of basilar artery thrombosis is rarely reported in literature.

Keywords: Basilar infarct, Basilar stroke, Hyperhomocysteinemia, Posterior circulation stroke, Young stroke

INTRODUCTION

Basilar artery is a major vessel contributing to the posterior cerebral circulation. The vertebrobasilar system formed by vertebral and basilar arteries perfuses the posterior part of Circle of Willis. It provides oxygenated blood primarily to the cerebellum, brainstem, thalamus, occipital and medial temporal lobes of brain. Posterior circulation stroke, which accounts for 15-20% of all strokes, carries a poor prognosis.[1-3] Hyperhomocystenemia being a major modifiable risk factor of stroke has a strong correlation with cerebral ischemia in both young and elderly population. It has been postulated that high degree of oxidative stress occurs in hyperhomocysteinemia leading to its ischemic potential.[4-6] Serum Homocysteine level more than 15 micromol/litre has been found to be an independent risk factor for ischemic stroke.[7] Elevated serum homocysteine can lead to basilar artery thrombosis which comprises about 1% of all strokes. Early recognition of the condition and prompt thrombolysis in the window period forms the mainstay of the treatment. In case of late presentation, conservative management with vitamin B and folic acid supplementation can be beneficial, though complete recovery with this mode of treatment is rarely reported. Authors describe basilar artery thrombosis in a 15-year-old boy who was managed successfully with conservative management.

CASE REPORT

A 15 years boy, strict vegetarian by diet, from the Indian state of Uttar Pradesh presented to the emergency room (ER) in an unconscious state. His parents told us that the boy, previously healthy, had intense headache (occipital), multiple episodes of vomiting 1 day back which was quickly followed by loss of consciousness. There was no history of fever, seizures and head injury. Patient was

initially admitted in a primary health centre where he received only intravenous fluids and was referred to us for further evaluation and management. He had no significant family history or any drug history. On examination, the patient was in altered mentation with GCS score of 10 (E2V4M4), having normal to increased reflexes in all four limbs, bilateral extensor plantar response and mid dilated sluggish reacting pupils on both sides. He had a National Institute of Health Stroke Scale (NIHSS) score of 24. Signs of meningeal irritation were absent. Blood pressure recorded in right upper extremity was 160/80 mmHg. He had a normal pulse rate (84/min), oral temperature and respiration (oxygen saturation 96% on room air). Cardiac murmurs were absent. Authors ordered an urgent non-contrast CT of the brain in the ER which revealed multiple ischemic infarcts in cerebellum and brainstem suggestive of posterior circulation ischemic stroke (Figure 1). Thrombolysis or endovascular procedures could not be performed due to late presentation (beyond 24 hours) and conservative management with intravenous mannitol (1g/kg - 8 hourly) and aspirin was started.

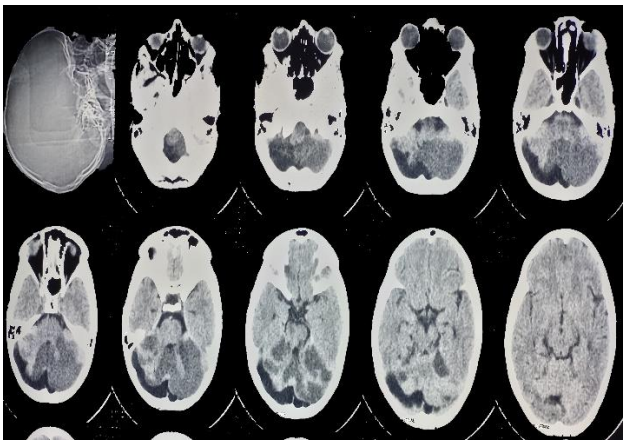

Figure 1: Multiple infarcts in brainstem occipital lobes and cerebellum (posterior circulation stroke).

Considering the young age of the patient, a thorough search for possible etiology of ischemic event was planned. Initial routine laboratory investigations (complete blood count including differentials and platelet count, prothrombin time, electrolytes, blood sugar) were normal. Doppler ultrasonography of carotid arteries didn't reveal any significant stenosis. 2-dimensional echocardiogram revealed normal valvular morphology, ventricular function (ejection fraction of 56%) with no evidence of vegetations or thrombus. Protein-C (80IU/dl) and Protein-S (32mg/L) levels were normal. Serum homocysteine was raised (17 micromol/litre). Serum vitamin B12 (370 pmol/L) and folate (5.5 nmol/L) were deficient. Antinuclear antibody (ANA) and Antiphospholipid antibody testing were noncontributory. Gadolinium enhanced MRI Brain with angiography, done on 3rd day of hospitalization, revealed acute infarcts in bilateral cerebellar hemispheres, pons, midbrain, medulla and vermis with nonvisualization of basilar artery suggestive of thrombosis (Figure 2). In view of elevated homocysteine levels and deficient vitamin B12 and folate levels, patient was put on intravenous methylcobalamin, folic acid and vitamin B6 supplementation. He continued receiving antiplatelet and other supportive treatment (nasogastric feeding, limb and chest physiotherapy) during hospital stay. The patient showed signs of gradual neurological recovery after 2 weeks manifested as spontaneous eye opening (E4V4M4) and limb movement (power in all four extremities - 3/5 MRC scale).He was discharged on day 24 with a moderate disability (modified Rankin Scale score of 3) and a prescription of oral aspirin, vitamin B6, B12 and folic acid .On his follow up visit after 6 months of discharge, patient had no significant disability (mRS score -1), normal power (MRC 5/5) and sensorium (GCS 15). Investigations showed a decline in serum homocysteine (6.8micromol/L) and concurrent increase in vitamin B12 (980 pmol/L) and folic acid (20nmol/L) levels.

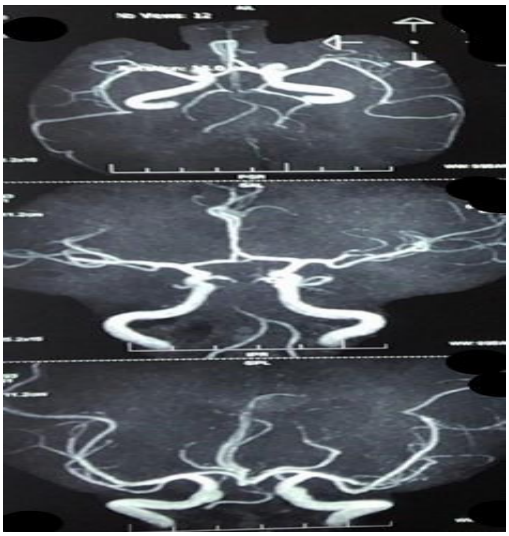

Figure 2: Magnetic resonance angiogram of brain revealing non visualization of basilar artery suggestive of thrombosis.

DISCUSSION

Basilar artery thrombosis constituting about 1% to 4% of all ischemic strokes poses a great diagnostic and therapeutic challenge to the treating physicians. The posterior circulation strokes have longer prodrome and the most common symptoms include nausea, vertigo, headache and neck pain. Frequent causes include embolism, atherosclerosis, arterial dissection and less commonly prothrombotic states (Protein-C, Protein-S deficiency and hyperhomocysteinemia), meningitis, arteritis and neurosyphilis. Etiology cannot be identified in many posterior circulation strokes, thus remaining obscure. Despite advancements in imaging techniques and treatment modalities, the morbidity and mortality of basilar artery thrombosis is high. Epidemiological data estimate mortality rates ranging from 45% to 86%. Intravenous thrombolysis/endovascular therapy is

considered when patients present within 4.5 hours. A better outcome was observed in basilar artery occlusion in children as compared to adults regardless of intra-arterial treatments in one study.[8] There is paucity of existing literature showing outcomes of conservative treatment in patients of basilar artery thrombosis. Since the patient presented beyond the critical window period, a logical decision of non-surgical management was taken by our team. Authors did thorough investigations to exclude all possible etiologies of young stroke in our patient. Apart from hyperhomocysteinemia, no other abnormalities were found that could explain hypercoagulabilty and stroke in this case. Hyperhomocysteinemia has a multifactorial origin that includes genetic, nutritional, pharmacological, and pathological factors.

A proatherogenic effect rather than a prothrombotic effect can explain the pathogenic role of hyperhomocysteinemia in large vessel disease based on the higher association of the former with large artery disease as compared to small artery disease in one study.[9] Various potential mechanisms have been proposed - these include direct endothelial injury, mitogenic effect on smooth muscle cells, impaired endogenous fibrinolysis, endothelial nitrous oxide response, and alteration in arachnoidic acid metabolism.[10-12] Hyperhomocysteinemia by inducing an elastolytic process in the arterial wall leads to loss of elastin and eventual stiffening of arteries that results in hypertension.[11] The treatment of hyperhomocysteinemia entails utilization of cofactors in homocysteine metabolism - vitamin B6 in transsulfuration pathway and vitamin B12 and folic acid in remythlation pathway. Thus, supplementation of these cofactors forms the mainstay of treatment.

The patient in this study was treated for hyperhomocysteinemia, in accordance with HOPE-2 Study (Heart Outcomes Prevention Evaluation-2 Study) with antiplatelet agents and vitamin supplementation.[13] The clinical response to vitamin supplementation combined with improving laboratory parameters on follow up (declining homocysteine and rising B12 levels) further confirmed our diagnosis. This is one of the rare reported cases of basilar artery occlusion due to hyperhomocyteinemia which improved solely on conservative management.

This case emphasizes two vital deductions - First, a high index of suspicion should be sought for basilar artery thrombosis in patients with fluctuating level of consciousness with neurological deficit with prodromal symptoms (nausea, vomiting, headache) and Second, a pro-thrombotic state must be ruled out in young vegetarian patients, especially hyperhomocysteinemia.

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