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Artery of Percheron infarct - A missed opportunity of stroke thrombolysis

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ABSTRACT

Acute ischemic infarction in the territory of the artery of Percheron (AOP). AOP is a relatively uncommon neurovascular anatomical variant of the posterior circulation. It is a single arterial trunk supplying bilateral paramedian thalami and the rostral midbrain, a crucial structure responsible for regulation of alertness, consciousness, and sleep. The clinical presentation may deviate from the typical sensorimotor deficits seen with classical stroke syndromes. Without a high index of suspicion, AOP infarcts can be missed clinically and radiologically, thereby missing the crucial window of thrombolytic therapy. We discuss a case of a 60-year-old male, who was presented with acute onset altered sensorium due to AOP infarction and missed the opportunity for intravenous thrombolysis due to delayed diagnosis.

Key words: *Artery of Percheron, Bilateral thalamic infarction, Ischemic stroke, Thrombolysis*

Acute ischemic infarction in the territory of the artery of Percheron (AOP) is uncommon, incidence varying from 0.1% to 2% of all ischemic strokes and 4–18% of all thalamic strokes [1]. Clinical presentation differs from classic stroke syndromes and resembles encephalopathic states. Due to its rarity and diagnostic difficulty on routine stroke protocol imaging, the AOP infarction can be missed easily. The blood supply to the thalamus arises from the perforating arteries of the posterior communicating artery and the posterior cerebral artery (PCA). The AOP was first described in 1973 by the French medical scientist Gerard Percheron [1]. According to him, there are four normal variants of the neurovascular anatomy of the thalami and midbrain .

The four variants are described as follows: Type I which is the most common where, each perforating artery arises from each left and right PCA; Type II a being the less common where, perforating arteries arise directly from the proximal segment of one of the PCAs; Type II b bilateral perforating thalamic arteries arise from a single arterial trunk called the AOP, which arises from the P1 segment of one PCA; and Type III where, several small perforating branches arising from single arterial arc bridging the P1 segments of both PCAs. We discussed a case of a 60-year-old male, who was presented with acute onset altered sensorium due to AOP infarction and missed the opportunity for intravenous thrombolysis due to delayed diagnosis [3-8].

CASE REPORT

A 60-year-old male with diabetes mellitus, hypertension, and ischemic heart disease for 10 years on regular medications, was presented to the casualty with sudden onset of unresponsiveness. He had a history of regular alcohol intake for >20 years.

On arrival to the emergency department, his Glasgow coma scale (GCS) was E1V2M1 (4/15), pulse was 58 min, blood pressure was 120/70 mmHg, temperature was normal, respiratory rate 20 breaths/min, and random blood sugar level was 105 mg/dl. On neurological examination, the pupils were bilaterally equal and normally reacting to light. There were no limb movements to deep painful stimuli. Bilateral plantar reflexes were extensor and his electrocardiogram was normal. He was intubated in the casualty. The differential diagnosis considered were cerebral infarction or hemorrhage.

A plain computerized tomography (CT) brain revealed global cortical atrophy (Fig. 1a). He was not thrombolyzed as his clinical and radiological findings were not consistent with any usual stroke syndromes. The patient was extubated within 12 h, with GCS of E3M6V4. He was confused, developing intermittent episodes of drowsiness, had dysarthria and an upward gaze palsy with no nystagmus. His power was 4/5 in all four limbs with bilateral positive Babinski sign.

Magnetic resonance imaging (MRI) brain was ordered the next morning which revealed, bilateral symmetric FLAIR and T2 weighted hyperintensities in paramedian thalami, which restricted on diffusion. Magnetic resonance angiography (MRA) and magnetic resonance venography were normal, and the radiological differentials considered were AOP infarction, Wernicke's encephalopathy or viral encephalitis (Fig. 1b and c). Table 1 shows the findings of variants of the thalamic blood supply in patient (Fig. 2)

Other investigations, including biochemical profile (bilirubin total - 0.4, direct - 0.1, liver enzymes - SGOT-32, SGPT - 38,

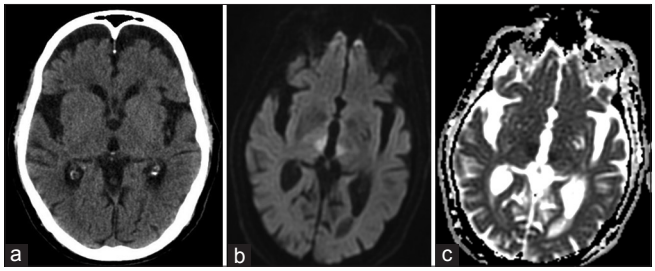

Figure 1: (a) Computerized tomography brain showing cortical atrophy; magnetic resonance imaging (MRI) brain showing, (b) MRI brain showing bilateral symmetric hyperintensities in paramedian thalami in diffusion weighted image, and (c) MRI brain showing bilateral symmetric hyperintensities in paramedian thalami with restricted diffusion in apparent diffusion coefficient

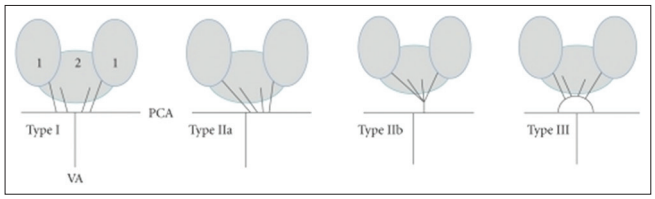

Figure 2: Variants of thalamic blood supply 1: Thalamus and 2: Midbrain; VA: Vertebral artery; and PCA: Posterior communicating artery

lipid profile - normal, serum creatinine - 0.5, blood urea - 36, serum sodium - 140, potassium - 4, and chloride - 92), hemogram (hemoglobin - 13.7 g%, total counts - 7800/mm^3, and platelet count - 3 lakh/mm^3), thyroid function test, urine analysis, cerebrospinal fluid analysis, serum ammonia, serum thiamine, vasculitis profile, chest radiograph, two-dimensional echo, and Doppler neck vessels were normal. The flavivirus workup was not carried out due to the absence of viral prodrome.

As the patient's relatives were unwilling for endovascular intervention, digital subtraction angiography was withheld. The patient was conservatively managed with antihypertensives, insulin, antiplatelets, statins, and intravenous thiamine. During his stay in hospital, the patient continued to be restless and irritable requiring treatment with benzodiazepines and antipsychotics. Electroencephalograph demonstrated a diffuse slowing without any epileptiform discharges. During his 17-day hospital stay, he showed progressive signs of improvement with appropriate nursing care and physiotherapy. At the time of discharge, his GCS was E4M6V4 and he was able to sit and walk with support. He was able to feed himself and was continent. He continued to have intermittent episodes of confusion, restlessness, and irritability. There was a marginal improvement in behavioral function at follow-up in 3 weeks.

DISCUSSION

AOP infarction on CT brain is seen as areas of hypodensity in bilateral paramedian thalami with or without rostral midbrain involvement. However, CT scan can also be normal and these infarcts can be easily missed, as in our case, unless suspected by the casualty physicians and an urgent MRI brain obtained. An acute infarct would restrict on diffusion weighted sequences in the MRI brain in the AOP territory. The AOP is too small to be seen by angiography computed tomography angiography or MRA [9]. With a brain MRI showing an acute stroke, intravenous thrombolysis can be performed, if the patient is within the therapeutic window, that is, 4.5 h of symptom onset [10].

Table 1: Symptoms of artery of percheron stroke

Vertical gaze palsy, bilateral internuclear ophthalmoplegia, pupillary abnormalities, and photophobia (65% of patients)
Severe impairment of anterograde and retrograde memory with confabulation and marked disorientation in time (58% of patients)
Acute disturbance in the state of consciousness, somnolence, frequently progressing to profound coma which can last several hours or days (42% of patients)
Lack of spontaneous thinking, loss of initiative, inappropriate social behavior, impulsivity, even aggressiveness, and emotional blunting

In comparison to ischemic lesions of other cortical/subcortical structures, thalamic stroke has a lower mortality rate and a much better prognosis for recovery of motor deficits. By contrast, the neuropsychological deficits in functions of memory, cognition, emotional response, and behavior tend to persist. These can subsequently interfere with the social and professional lifestyle of the patient after recovery from the acute state. Thus, a high index of suspicion is warranted to treat this stroke, using intravenous thrombolysis in the stipulated time window to prevent these neurological sequelae.

CONCLUSION

The clinical presentation of AOP infarction is usually confusing to the casualty physicians and evades the clear-cut definition of a "measurable neurologic deficit" often leading to the missed window of intravenous thrombolysis. The diagnosis of AOP infarct may be missed on normal CT brain and MRI is a better modality for identification of this uncommon stroke syndrome. Therefore, though rare, if suspected timely in cases presenting with sudden onset altered sensorium, thalamic strokes due to AOP infarcts may be offered thrombolysis in the crucial time window, leading to more favorable neurological outcomes.

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