

Letter to Editor

A Rare Stroke due to a Rare Mechanism- Embolic Recurrent Artery of Heubner Infarct**Anandhu Suresh¹, Sajeesh Rajendran², Somarajan Anandan³, Sourav Asha Somarajan¹***From, ¹Resident, ³Consultant Neurologist, Department of Neurology, St Joseph Hospital, Anchal, Kerala, ²Consultant Neurologist, Mar Sleevea Medicity, Palai, Kerala.*

Dear Editor,

Recurrent artery of Heubner (RAH), also known as the medial striate artery or long central artery, is the largest perforating branch from the proximal anterior cerebral artery (ACA) and is the only perforator routinely seen on cerebral angiography. It was first described by the German pediatrician Johann Otto Leonhard Heubner in 1872. Neurosurgical procedures such as aneurysm treatment on the anterior part of the circle of Willis can result in damage of the RAH leading to neurological deficits [1]. Strokes involving the Recurrent Artery of Heubner are quite rare and are often asymptomatic. As it is a penetrating vessel, stroke due to RAH involvement is either due to arteriosclerosis or branch atheromatous disease. Embolic stroke in the territory of RAH is extremely rare. Here we describe a case of stroke due to RAH involvement, probably due to embolism.

A 73-year-old male with hypertension and dyslipidemia presented to the Emergency Department with generalized tiredness for three days, followed by mild dysarthria and difficulty in walking for two days. Relatives also noticed a delay in answering questions and reduced speech. He had urgency of micturition. There was no h/o any comprehension difficulty or naming difficulty. His past medical history was significant for pituitary adenoma resection in 2004. On initial assessment, his vital parameters were stable. Neurological examination revealed mild dysarthria with preserved comprehension and naming, left-sided upper motor neuron facial palsy, and left hemiparesis involving both upper and lower limbs with muscle power of 4/5 (MRC grading). Sensory examination was normal, and the left plantar response was extensor. There were no cerebellar signs. Biochemical evaluation showed random blood sugar of 229 mg% with HbA1c of 6.8%.

His serum sodium was 135.8 mEq/L and potassium was

3.9 mEq/L. His serum bicarbonate was low (19 mmol/L). His thyroid function including free T4 was normal. His fasting cortisol was low normal (7.1 microgram/dL) and fasting DHEA-S was low 15.5 microgram/dL (Normal 33.6-249) suggesting mild adrenal insufficiency. We did not do cosyntropin stimulation test due to local unavailability. MRI brain demonstrated an acute infarct involving head of caudate nucleus, anterior limb of internal capsule and anterior lentiform nucleus (Figure 1,2 and 3).

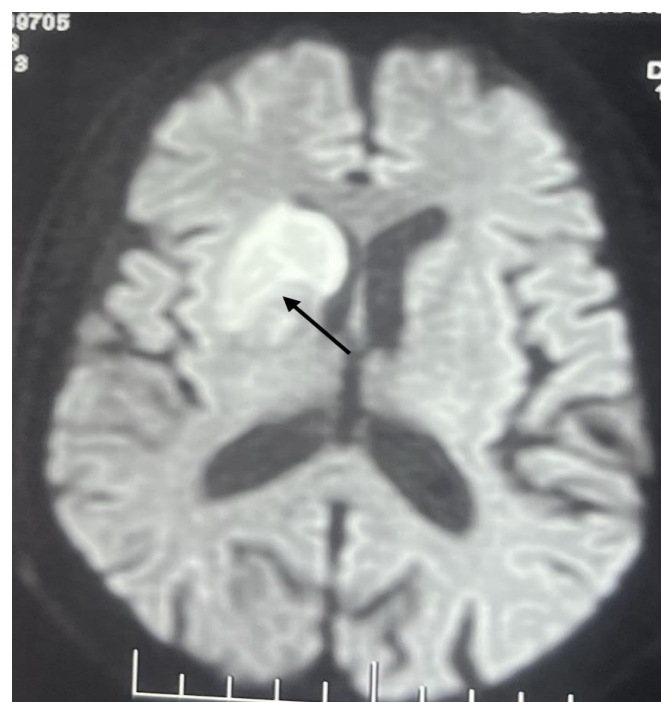


Figure 1: Axial diffusion weighted image showing diffusion restriction involving head of caudate nucleus, anterior limb of internal capsule and anterior lentiform nucleus. (ice cream cone shaped infarct)

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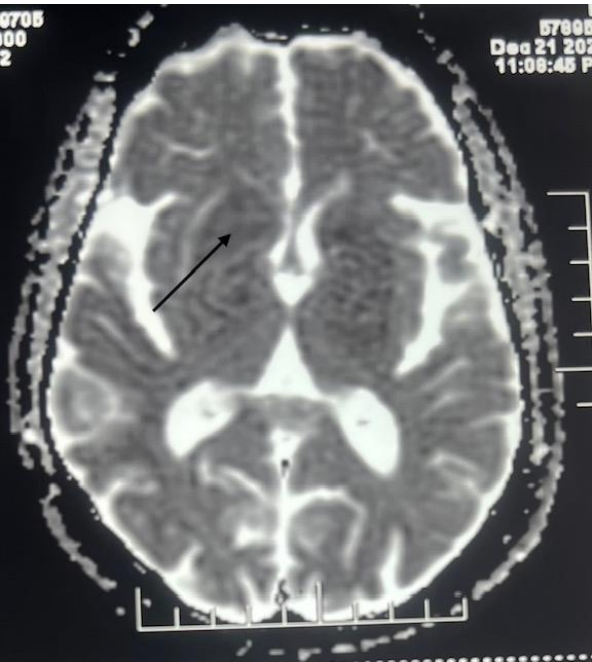


Figure 2: Axial apparent diffusion coefficient image showing corresponding hypointensity.

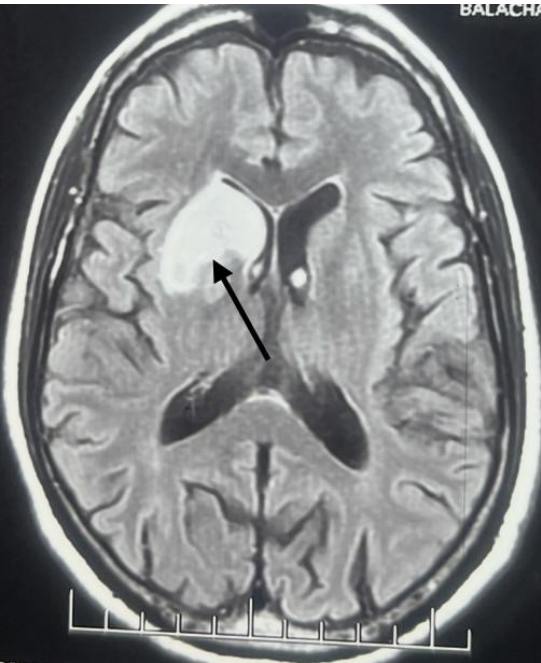


Figure 3: Axial FLAIR image showing hyperintensity of head of caudate nucleus and anterior lentiform nucleus and anterior limb of internal capsule.

MR angiography of the brain and neck vessels was normal indicating absence of large-vessel occlusion or significant stenosis, favouring perforator artery infarct Figure [4]. There were few acute infarcts involving right precentral gyrus, right post central gyrus and right superior parietal lobule favouring an embolic mechanism. His routine blood tests were normal. Echocardiogram showed left ventricular hypertrophy and 72-hour Holter study showed few runs of supraventricular tachycardia. He was treated with dual antiplatelets, atorvastatin and physiotherapy with which his weakness improved.



Figure 4: MR angiography showing normal intracranial arteries

Typically, the RAH is the largest of the perforating medial lenticulostriate arteries arising from the ACA. In approximately 25% of individuals, there is a single Heubner's artery; often, there are two, three, or even four recurrent arteries. The artery primarily supplies blood to the head of the caudate nucleus, the medial portion of the globus pallidus, the anterior crus of the internal capsule, the anterior hypothalamus, the nucleus accumbens, parts of the uncinate fasciculus, the diagonal band of Broca, and the basal nucleus of Meynert [2]. (Table 1)

RA originate from the A2 in 57%, at the level of the ACom in 35%, and from the A1in 8%. The recurrent artery of Heubner originated within 4 mm of the ACom in 95% of cases [3].

Table 1: Structures supplied by RAH

S. No.	Structures Supplied by RAH
1	Head of caudate nucleus
2	Medial portion of globus pallidus
3	Anterior limb of internal capsule
4	Anterior hypothalamus
5	Nucleus accumbens
6	Part of uncinate fasciculus
7	Diagonal band of Broca
8	Basal nucleus of Meynert

Typical RAH occlusion cause faciobrachial monoparesis. Other symptoms can include behavioral changes (abulia, agitation, delirium), contralateral hemiparesis, dysarthria and hemichorea. Apathy and decreased drive are often present, presumably resulting from infarction of the nucleus accumbens. Most common behavioral abnormality is abulia and slowness of activity is frequent. Sensory functions are often preserved. Bilateral involvement can result in akinetic mutism. Sometimes it can be silent [4]. Etiology of RAH infarct can be due to parent vessel atherosclerosis, embolism or lipohyalinosis. It can be

involved due to vasospasm, dissection or head trauma [5]. It can also be injured during Acom aneurysm surgery [6]. In a series of caudate infarcts, small-artery disease was diagnosed in 14 patients (59%), cardiac embolism in 5 patients (20%), and large-artery disease in 2 patients (8%), and 2 patients (8%) had mixed etiology [7].

RAH infarct often has an ice-cream cone appearance with caudate head forming the scoop of ice cream and anterior limb of internal capsule and anterior lentiform nucleus involvement forming the cone. In our case multiple discrete cortical and subcortical infarcts favoured embolism as the mechanism of infarct. Even though a definite cardiac source was not obtained and embolism is still possible from the heart or aortic arch.

In conclusion RAH infarct should be suspected when a patient present with faciobrachial monoparesis with apathy and it can be confirmed with an MRI brain showing ice-cream cone appearance at the level of caudate head.

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