CASE REPORT

Decompressive Craniectomy for Bilateral Anterior Cerebral Artery Infarction: Case report and brief literature review

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Abstract

Decompressive craniectomy for "malignant" bilateral anterior cerebral artery infarction (BACAI) has not been reported in the literature. Here we present such a case in relation to its presentation, surgical management, outcome and anatomical basis.

Bifronto-temporal decompressive craniectomy was performed on a patient diagnosed with acute infarction in bilateral anterior cerebral artery (ACA) territories and rapidly deteriorating having clinical and radiological signs of tonsillar herniation. Patient sustained several motor disabilities at sixth week following surgery, however, post-operative CT showed reversing of the "conning" with spaced basal cisterns which was compatible with clinical improvement.

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Introduction

Simultaneous bilateral anterior cerebral artery infarction (BACAI) is rare, and only few case reports are available in scholarly articles. Decompressive craniectomy for "malignant" BACAI has not been reported in the literature. Here we present such a case in relation to its presentation, surgical management, outcome and anatomical basis.

Case report

A 53-year woman admitted to a peripheral unit with abnormal behavior and poor response for two hours. She had a history of faintishness for two weeks, and an abnormal gait for two days. She presented with both urinary and foecal incontinence on the day of admission. She gave neither a history of fever nor seizures. She had hypertension and diabetes optimally controlled with treatment. On admission she was afebrile, Glasgow coma score (GCS) was 10 (Eye 4, Verbal 1, Motor 5), blood pressure was 135/90 mmHg and capillary blood sugar (CBS) was 424 mg/dl.

She was transferred to a tertiary care centre for further evaluation and management. At the emergency treatment unit her vitals were stable. Initial computed tomography (CT) scans of the brain revealed hyperdense anterior cerebral artery sign with acute infarction in the bilateral anterior cerebral artery (ACA) territories (Figure 1 and 2). Urine for ketone bodies was positive but there was no acidosis in the arterial blood gas analysis. Initial full blood count revealed neutrophil leukocytosis

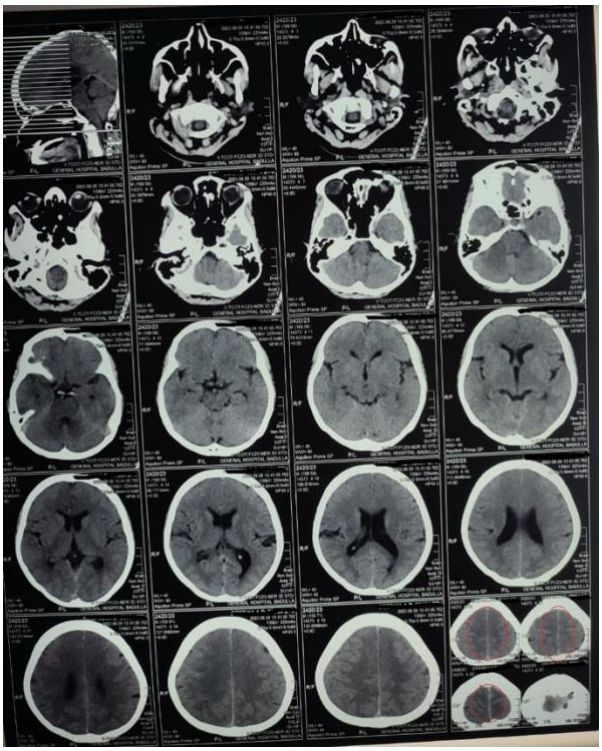

Figure 1: CT scan showing hypodense areas suggestive of bilateral anterior cerebral infarction

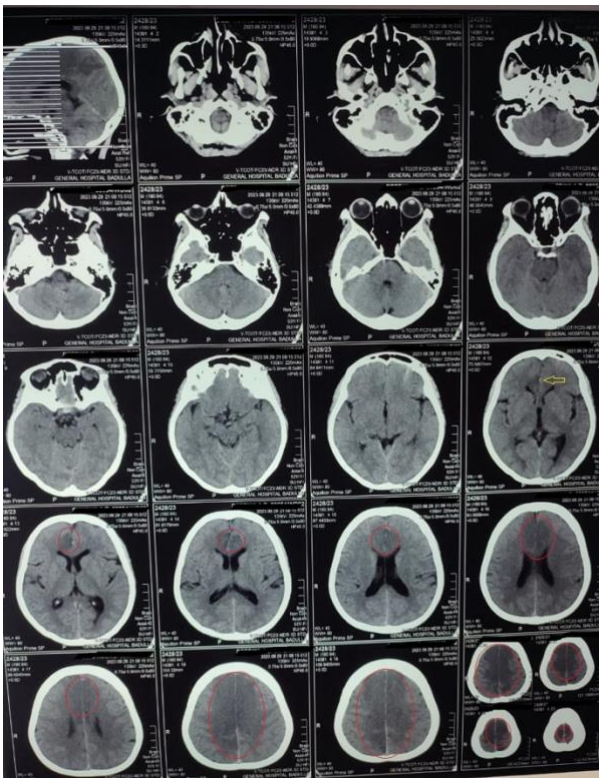

Figure 2: CT scan showing hypodense areas suggestive of bilateral anterior cerebral infarction (circle) and hyperdense anterior cerebral artery sign (arrow)

(WBC 13.38 X 10^9/L, N 86.1%), haemoglobin of 12.6% and platelets of 152x 10^9/L. Her renal functions, serum electrolytes and liver functions were within normal ranges and coagulation profile was normal. Her ECG was regular, 2D echocardiography was normal and carotid duplex scan was normal.

She was managed with general stroke care including nasogastric feeding, fluid balance with indwelling urinary catheter, glycemic control with soluble insulin, regular Aspirin, Atorvastatin and antihypertensives. Subsequent CTs showed marked infarctions in previously described territories.

During the hospital stay from the day 3 to day 4, she rapidly deteriorated with GCS of 3 and unequal pupils, without prior seizure or aspiration. Repeat CT revealed extensive involvement of infarction compared to previous CTs with marked cerebral oedema, obliteration of basal cisterns and tonsillar herniation of 5 mm below the foramen magnum (Figure 3). Patient was immediately intubated, paralyzed and ventilated with head elevated to 30 degrees. 3% saline bolus was given to reduce the cerebral oedema. Then she was referred to the neurosurgical unit for opinion on surgical management of raised intracranial pressure.

After discussing with family members regarding possible poor outcome even after the surgical management, they requested to proceed with surgical decompression, accepting any possible poor outcome. Patient underwent bifronto-temporal decompressive craniectomy with dural opening, decompressing both frontal and temporal lobes. Patient was weaned on post-operative day 2 and tracheotomy was performed to manage ventilator associated pneumonia. The post-operative CT showed reversal of conning and spaced basal cisterns with extracranial herniation of oedematous brain (Figure4).

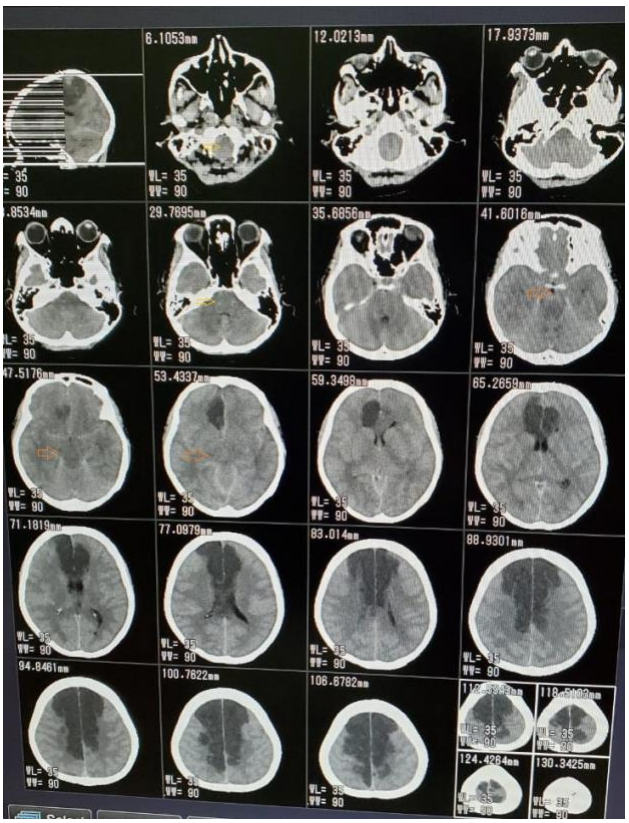

Figure 3: Marked infarction with obliteration of basal cisterns (orange arrow) and tonsillar herniation (yellow arrow)

The patient had post-op stroke care at neurosurgical unit for 10 days. After weaning the tracheostomy she was discharged for home based rehabilitation.

At six weeks following surgery the patient had akinetic mutism, bilateral lower limb and left upper limb weakness, and fecal incontinence. She was nursed with nasogastric tube feeding and indwelling urinary catheter.

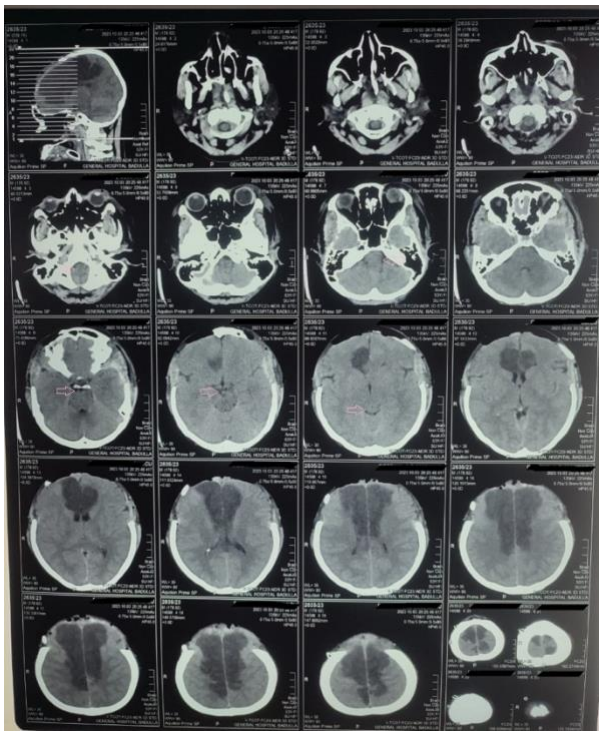

Figure 4: The post-operative CT showing reversal of conning and spaced basal cisterns (arrow) with extracranial herniation of oedematous brain

Discussion

The anterior cerebral artery (ACA) is the major vessel supplying the frontal pole, interhemispheric region and medial superolateral region of each cerebral hemisphere. Its perforators supply the anterior limb of the internal capsule, head of the caudate nucleus and associated deep white matter. The ACA is described in five segments; precommunicating segment (A1), postcommunicating infracallosal segment (A2), precallosal segment (A3), supracallosal segment (A4) and postcallosal segment (A5).

ACA infarctions are rare representing 0.3-4.4% of all cerebral infarctions in series reports (1). Anastomostic complex may account for the low rate of infarctions in this territory.

Simultaneous bilateral ACA infarctions are even rarer, and only few case reports are available in scholarly articles (2-17). In one study, 27 patients with ACA territory infarctions were reported out of 1490 patients with cerebral infarctions of which there were only two with bilateral ACA infarctions (18).

Bilateral ACA infarction may be the result of either an occlusion of a single artery supplying both ACA territories, or simultaneous occlusion of both vessels supplying respective ACA territories (Figure 5). This may be at pre-communicating level, communicating level or post-communicating level of the ACA vasculature.

These includes,

1. aplastic/hypoplastic A1 with dominant A1 occlusion
2. spasm of anterior communicating artery complex following aneurysmal subarachnoid haemorrhage (SAH)
3. azygos A2 occlusion
4. bihemispheric A2 occlusion

When the right and left A2 segments fuse, a single A2 segment is formed which is referred to as an azygos ACA. When one of the A2 segment is hypoplastic or terminates early, the contralateral ACA can divide and supply both hemispheres. This is referred to as a bihemispheric ACA (19).

In a study of anterior cerebral artery variations detected by MR angiography by Akira Uchino et al, 5.6% of unilateral A1 segment aplasia, and 2% of unpaired A2 segments were found among 891 cases (20).

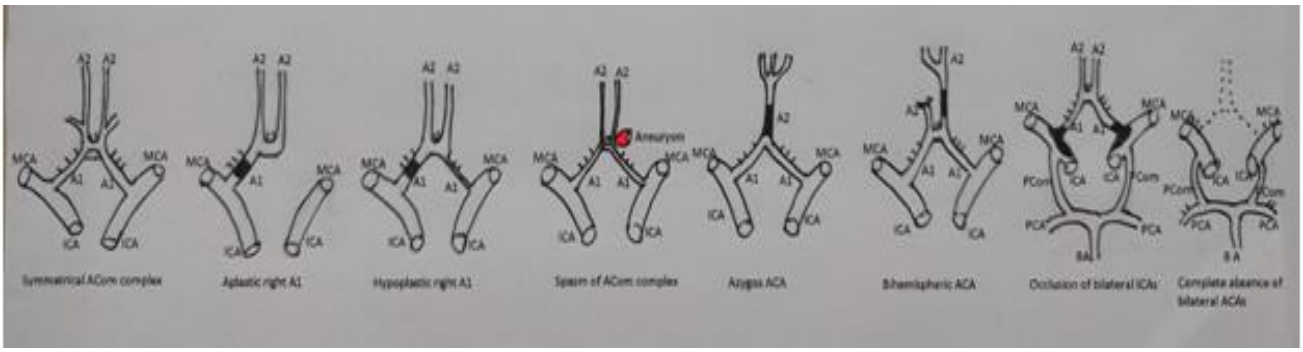

Figure 5: Anatomical basis of bilateral anterior cerebral artery infarction. ACA – anterior cerebral artery, ACom- Anterior communicating artery.

Recurrent ACA territory infarctions caused by the bilateral absence of complete ACAs have been reported (21). The posterior cerebral artery (PCA) and the middle cerebral artery (MCA) were compensating for blood supply insufficiency in the ACA.

Sudden bilateral ACA infarctions due to occlusion of bilateral internal carotid artery (ICA) demonstrated by time-of-flight magnetic resonance angiography in a 38 male smoker has been reported by M. Krisnan (10). Subsequent CT angiogram showed recanalized middle cerebral arteries, and interval CT angiogram after one month showed a hypoplastic left A1.

The main risk factors for ACA infarctions are hypertension, diabetes mellitus, dislipidaemia, cigarette smoking, atrial fibrillation (AF) and myocardial infarctions (22). The stroke mechanism includes cardiogenic embolism, ICA-ACA embolism and artherosclerosis. In the latter group, detailed stroke mechanism includes local branch occlusion, in situ thrombotic occlusion, artery to artery embolism and a combination. (23). Cardiac embolism from different sources includes AF, intra cardiac thrombus, valvular disease and tumours.

Some reports suggest cardioembolism as the most frequent aetiopathogenic mechanism of ACA infarctions (24).

It is interesting that acute isolated ACA infarctions due to atherosclerotic aetiology more commonly involve the left ACA (25). Furthermore, cardiac emboli tend to have a predilection for left ICA and its branches especially in the setting of AF (26). Bovine aortic arch configuration is associated with left hemispheric laterality of cardioembolic strokes (27). Other reported mechanisms of ACA infarctions include, arterial dissection (28, 29), vasculitis, coagulopathic status and vasospasm. Kauret et al., reported a case of bilateral anterior cerebral artery infarction due to dissection of the cervical segment of the left internal carotid artery induced by frequent neck massages in a patient with hypoplastic right A1 segment (11).

Vasospasm that occurs as a complication of SAH caused by rupture of ACom aneurysm can result in bilateral ACA infarctions. Fatal bilateral ACA infarction as a consequence of associated SAH and vasospasm in a patient with pituitary apoplexy has been reported by SadeepMohodra (30). Bilateral ACA infarction following a viper bite was reported

by Shoskote et al., proposing vessel occlusive thrombi due to disseminated intravascular coagulation, thrombosis favoring venom composition or vasculitis as hypotheses for this occurrence (31).

As the ACA supplies the medial surface of the frontal lobe with its pericallosal branch; the head of the caudate nucleus via the recurrent artery of Heubner; deep white matter (which contains fronto-striatal projection and reciprocal connection between the frontal cortex and thalamus) via perforator arteries, bilateral ACA infarction may present with bilateral motor weakness, incontinence, hypobulia/apathy, primitive reflexes and basal ganglia symptoms including parkinsonism gait, tremor and facial dystonia(32).

Our patient had bilateral lower limb with left upper limb weakness, dual incontinence and poor response to commands. With the progression of the cerebral oedema her conscious level reduced further with anisocoria. A. Ferbert et al presented two cases of bilateral ACA territory infractions whose initial diagnosis was basilar artery occlusion (12). Both had tetraparesis, in one it was asymmetrical. Both had their eyes opened but did not respond to command except that after a delay they followed with their eyes a smoothly moving object. Computed tomography (CT) revealed a fresh bilateral ACA infarction. They concluded that bilateral ACA territory infraction should be considered in the differential diagnosis of basilar artery occlusion. M. Macedo et al reported a similar case of extensive bilateral ACA stroke mimicking basilar artery occlusion, in which a 68-year man admitted with 12 hours evolution of left hemiparesis who got worse with GCS of 11 with lower limb predominant tetraparesis and bilateral Babinsky sign. The brain MRI showed extensive and symmetrical bilateral ACA infarcts and the presence of intraluminal thrombi within the azygos artery (13).

Imaging following bilateral ACA infarction shows infarctions in bilateral ACA territory which includes paramedianfrontoparietal cerebral cortex, anterior corpus callosum, anterior limb of the internal capsule and inferior portion of the caudate head.

In our patient initial CT showed hyperdense ACA sign (HDACAS) with acute infarction of above-mentioned regions. Subsequent imaging showed progression of infarction with marked cerebral oedema and tonsillar herniation. The HDACAS can be visualized in 46% of ACA infractions and visible more frequently when the CT is performed early (< 2.5 h after the onset) and is associated with high NHISS score (33). However, Sanjith et al., has reported that initial CT scan can miss the diagnosis in 74 % cases of acute isolated ACA infarctions (25)

In our patient, intracranial atherosclerosis is the more likely mechanism of the stroke, given her underlying hypertension and diabetes. We found no evidence to suggest cardioembolism, extracranial ICA-ACA embolism or SAH that could lead to vasospasm. We also believe that CT angiography/ digital subtraction angiography should have been performed to see any vascular anomaly, but it was not readily available prior to emergency decompressive craniectomy which we performed soon after the referral to us and the

family did not want to investigate further following the surgery.

Decompressive craniectomy for malignant middle cerebral artery infarction is well established. Decompressive hemicraniectomy for fatal unilateral ACA infarction has been reported in a single case report by Stefanie Leistner (34) whereas bifrontotemporal decompression for bilateral "malignant" ACA infarction has not been reported in the literature.

Our patient underwent bilateral frontal and temporal decompressive craniectomy under general anaesthesia with head fixed on three-point Mayfield head fixator device. Lateral part of both sphenoid lesser wings trimmed, and bilateral frontal and temporal lobes were decompressed after dural opening. Post-operative CT showed reversing of the "conning" with spaced basal cisterns which was compatible with clinical improvement of the patient.

However, at six weeks following the surgery patient had akinetic mutism, bilateral lower limb and left upper limb weakness, fecal incontinence and cared with nasogastric tube feeding and indwelling urinary catheter (modified Rankin scale -5). The mortality and the outcome cannot be compared due to the unavailability of the similar literature.

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