MASSIVE CEREBRAL INFARCTION FOLLOWING EVACUATION OF INTRACEREBRAL HEMATOMA

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ABSTRACT

Rarely primary ICH and infarction can occur simultaneously and development of cerebral infarction soon after intracerebral hemorrhage is still rarer. Uncontrolled atherosclerotic risk factors including hypertension and hyperlipidemia together with antithrombotic therapy are the major reasons for the simultaneous ischemic and hemorrhagic lesions. We report a 67 year male presented with history of sudden onset of left hemiplegia. An urgent CT scan brain showed a large right basal ganglionic hematoma with mass effect and midline shift. The patient underwent evacuation of hematoma. After initial improvement the patient again deteriorated on day 3. A follow up CT scan showed hemorrghic infarct at the site of surgery with mass effect and midline shift.

Keywords: intracranial hematoma, cerebral infarction, hypertension

Rarely primary ICH and infarction can occur simultaneously (1-4) and development of cerebral infarction soon after intracerebral hemorrhage is still rarer. (5) A 67 year male presented with history of sudden onset of left upper and lower limbs weakness associated with left sided facial weakness. The patient was a known hypertensive on irregular medication for last five years. There was no history of trauma, seizures or fever. On examination in the emergency room the patient was afebrile, his blood pressure was 190/100 mmHg, pulse rate was 90/minute. On chest examination he had bilateral conducted sounds, air entry was equal. Other general and systemic examination was unremarkable. He was in altered sensorium (GCS-E1, V1 and M4). Pupils were bilateral equal and reacting to light. He had left hemiplegia of grade 0/5 with left upper motor neuron type of facial palsy. He was moving right upper and lower limbs. The patient intubated in emergency room and an urgent CT scan was performed. CT scan brain showed a large right basal ganglionic hematoma with mass effect and midline shift (Fig. 1). After explaining the poor prognosis to the relatives the patient was taken for evacuation of intracerebral hematoma. A right fronto-temporal craniotomy was performed and the ICH was evacuated. There was thick clotted blood. After evacuation of the hematoma the brain became lax and pulsatile. Dural could be closed easily. Following surgery the patient was kept on elective ventilation. Next day morning the patient was opening eyes to call and localizing with right side. On day 3 post-surgery the patient became dull and started decerebrating. A CT scan brain was repeated and it showed hemorrghic infarct at the site of surgery with mass effect and midline shift (Fig. 2). The patient was started on anti-edema measures. His coagulation profile was within normal limits. The patient did not respond to the management and succumb to his illness.

Intracerebral haemorrhage (ICH) is the disabling form of stroke and most of the ICH (> 75%) are primary or spontaneous (due to hypertensive arteriopathy and cerebral amyloid angiopathy). (6) Primary ICH and infarction (especially lacunar infarcts) have the same underlying pathogenesis i.e. arteriosclerotic changes in the small perforating arteries. (3,7,8) Uncontrolled atherosclerotic risk fac-

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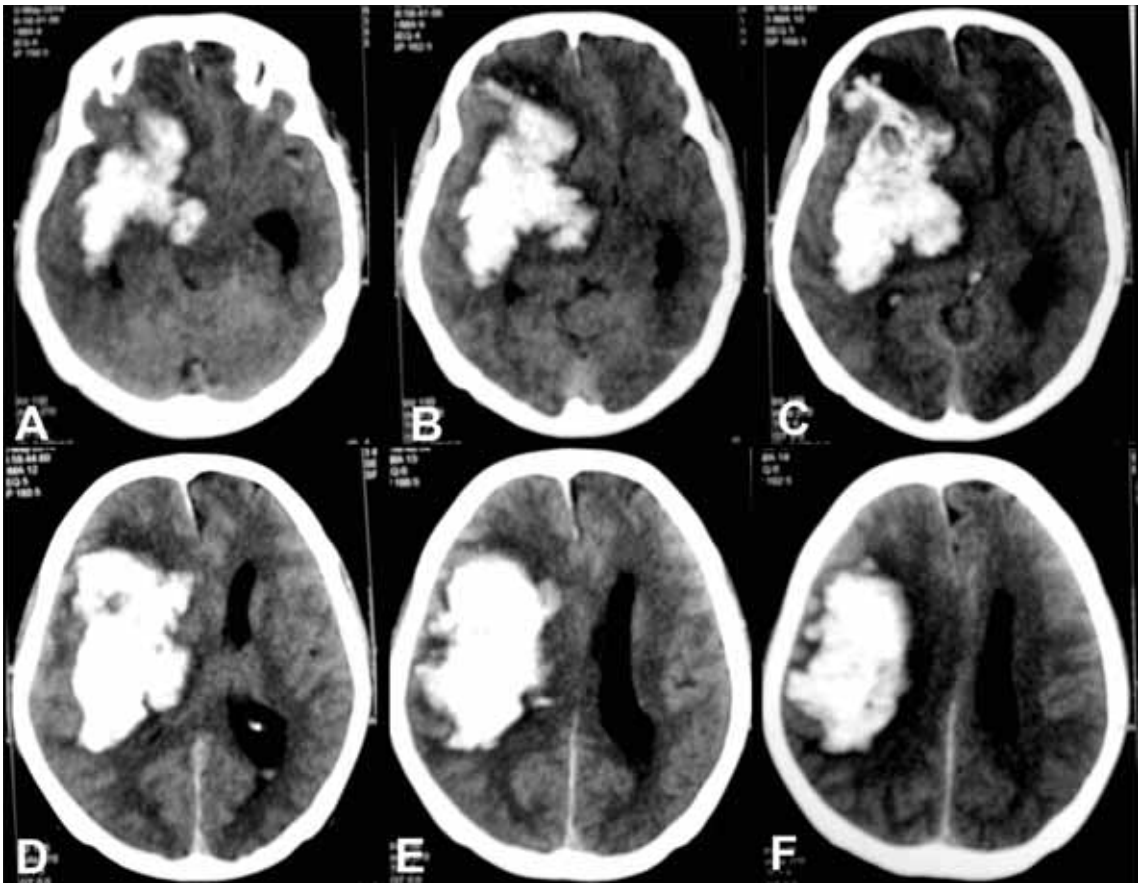

FIGURE 1. *Plain CT axial images showing large intraxial collection of blood attenuation (70-80 HU) in the right capsulo-ganglionic region involving the thalamus, fronto-parietal and temporal lobes causing mild midline shift of 10 mm to right and mild obstructive hydrocephalus*

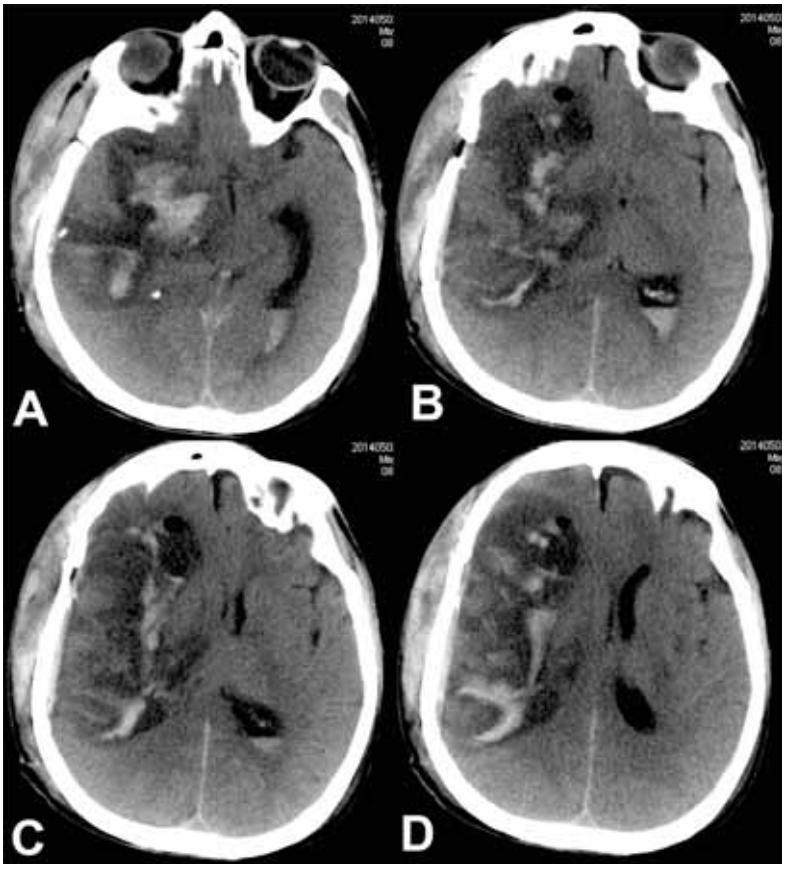

FIGURE 2. *Post-operative plain CT axial images showing near total evacuation of hematoma with wedge shaped hypodense area involving perisylvian cortex and fronto-temporal cortex corresponding to MCA territory suggestive of infarct*

tors including hypertension and hyperlipidemia together with antithrombotic therapy have been recognized as the major reasons for the simultaneous ischemic and hemorrhagic lesions. (4) ICH may produce infarcts through mechanical compression of cerebral vessels, hemodynamic instability, inflammation and concomitant small-vessel pathology in a high risk group of patients. (5) In addition aggressive blood pressure lowering may also cause acute ischemic infarcts after ICH. (3,9) Subclinical acute ischemic lesions have been increasingly described on brain magnetic resonance imaging following spontaneous intracerebral hemorrhage. (6) It has been increasingly recognized that diffusion weighted imaging (DWI) is superior in detecting small foci of acute ischemic changes. (6,9,10)

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