

# Incidental diagnosis of two intracranial aneurysms following surgical evacuation of chronic subdural hematoma

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## ABSTRACT

The development of subarachnoid hemorrhage (SAH) following evacuation of chronic subdural hematoma (CSDH) is a very rare phenomenon. SAH in this context occurring secondary to intracranial aneurysm rupture is still rare. We report a case of an elderly lady who presented with right hemiplegia and altered sensorium. Computed tomography (CT) scan of the brain revealed a left fronto-temporoparietal CSDH with midline shift, which was promptly evacuated surgically via a single burr-hole. Postoperatively, her level of consciousness deteriorated and there was increased the amount of drain. Emergency CT of the brain revealed diffuse SAH. CT cerebral angiogram revealed one aneurysm each in the right internal carotid artery and anterior communicating artery. Meanwhile, her consciousness level improved on conservative management. The relatives were not keen for further follow-up.

**Key words:** Chronic subdural hematoma, intracranial aneurysm, intraoperative rupture, subarachnoid hemorrhage

## Introduction

Chronic subdural hematoma (CSDH) is one of the most common entities seen in routine neurosurgical practice. Though its surgical evacuation is relatively simple, many complications have been described, including the development of intracerebral hematomas. Subarachnoid hemorrhage (SAH) following evacuation of CSDH is a very rare entity, and only three cases have been reported in the English literature.<sup>[1-3]</sup> Of these, two were associated with intracranial aneurysm rupture and a single aneurysm was detected postoperatively in each of these cases. This is the first time in the literature that two intracranial aneurysms have been diagnosed following evacuation of CSDH though we are not sure which one had ruptured intra-operatively.

## Case Report

A 70-year-old lady, who had sustained an accidental fall at home, 1-week back with head hitting ground (which did not require hospitalization); presented with a history of reduced speech and activity of 1-week duration. She also developed weakness of the right side of the body over 1-week. There was no history of fever, seizures, vomiting or incontinence of bowel or bladder. She did not have any significant past history other than that of chronic obstructive airway disease, for which she was on oral Salbutamol. The Glasgow coma scale (GCS) score at presentation was E4V3M5, and her pupils were equal in size and reacting to light. She had right hemiplegia and right upper motor neuron type of facial palsy. Her vital signs were stable. Her routine blood investigations and coagulation profile were normal.

Emergency computed tomography (CT) scan of the brain was taken, which showed a left fronto-temporoparietal CSDH measuring 14 mm in maximum thickness and a midline shift of 8 mm to the right [Figure 1]. She underwent emergency single burr-hole evacuation of the subdural hematoma under general anesthesia. The burr was widened to a 2.5 cm craniectomy. Motor-oil colored fluid escaped following the opening of the dura, which was slowly evacuated. Wash was given inside the cavity with saline using an infant feeding tube, till the effluent was clear. Brain was lax, and a subdural drain was kept on closure. Loading dose of Phenytoin was given intra-operatively.

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One hour postoperatively, her GCS was found to be E4V3M4 (after recovery from anesthesia), and there was 450 ml of blood in the drain. But her pupils were equal in size and reacting to light, and vital signs were normal. An emergency CT brain was taken which showed diffuse SAH in the cortical sulci and posterior interhemispheric region; and a small left-sided extradural hematoma with minimal residual collection and pneumocephalus. There was no significant midline shift [Figure 2]. She was conservatively managed, and oral nimodipine and phenytoin were started. Her GCS improved and became normal by second postoperative day. The right hemiplegia and facial palsy also resolved. CT cerebral angiogram was taken, which revealed two intracranial aneurysms [Figure 3]. The larger one was saccular and arising from the junction of cavernous and supra-clinoid portions of right internal carotid artery, pointing anterolaterally, and measuring 4.4 mm × 3.5 mm × 3.1 mm. The other one was measuring 3.2 mm × 2.4 mm and was arising from anterior communicating artery. A1 segment of left anterior cerebral artery and bilateral posterior communicating arteries were hypoplastic.

The management options including surgical clipping of aneurysms were discussed with the relatives, but they were unwilling for further surgical intervention. At discharge from the hospital on seventh postoperative day, her GCS was E4V5M6, and there was no motor deficit.

## Discussion

Even though the most common cause of SAH is trauma, ruptured intracranial aneurysms account for 75-80% of nontraumatic SAH cases. Aneurysms may arise from a defect in the muscular layer of the arterial wall or secondary to atherosclerotic or hypertensive vascular disease. Risk factors for aneurysmal SAH include hypertension, substance abuse (alcohol, cocaine, and tobacco), oral contraceptive use, pregnancy and parturition, lumbar puncture or cerebral angiography in patients with unruptured aneurysms, and advanced age.

Chronic subdural hematoma is an abnormal collection of liquefied blood degradation underneath the dura mater that may result in brain tissue compression and subsequent neurological sequelae.<sup>[4]</sup> It is one of the most common neurosurgical conditions and are usually treated by simple burr-hole drainage. But, as for any surgery, the evacuation of CSDH is prone to develop complications; including recurrence, new hemorrhages (subdural, intracerebral), subdural empyema, tension pneumocephalus, seizures, and cerebral infarction.<sup>[5]</sup> SAH following evacuation of CSDH is a very rare complication, and three such cases have been reported.<sup>[1-3]</sup> Of these, only two cases were associated with aneurysm - one was arising at the basilar apex<sup>[2]</sup> and the other one at the anterior communicating artery.<sup>[3]</sup> To the best of our knowledge and as per PubMed search,

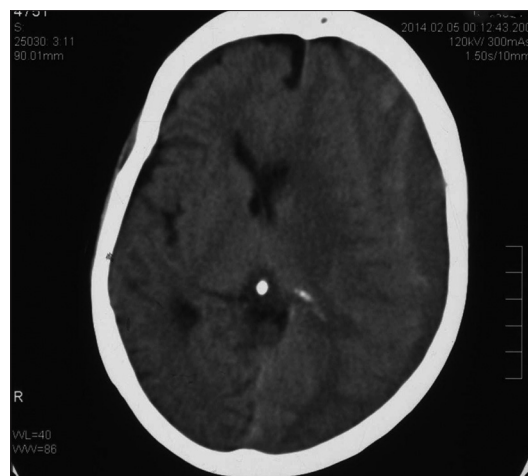


Figure 1: Preoperative plain computed tomography scan of the brain showing left fronto-temporoparietal chronic subdural hematoma with midline shift

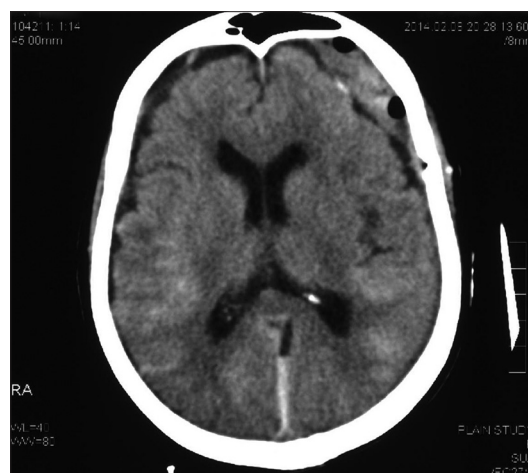


Figure 2: Postoperative computed tomography scan of the brain showing diffuse subarachnoid hemorrhage

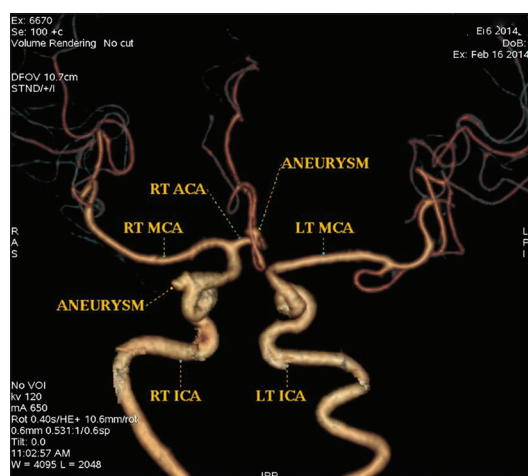


Figure 3: Computed tomography cerebral angiogram showing right internal carotid artery and anterior communicating artery aneurysms

no case has been reported till date showing the detection of two aneurysms following evacuation of CSDH.

The most insidious complication of treatment for CSDH is intracerebral hemorrhage, following removal of hematoma, with the incidence ranging between 0.7% and 5%. It was hypothesized that sudden restoration of normal pressure in regions of faulty cerebral vascular autoregulation due to subcortical swelling underlying surface compression, focal impedance of the venous drainage or ischemic loss of carbon dioxide reactivity might in turn lead to the vascular damage that results in intraparenchymal hematoma.<sup>[6]</sup>

The cause of SAH following CSDH evacuation was proposed to be due to mechanical traction on the wall of the aneurysmal sac or to variation in the transmural pressure gradient across the sac. The intracranial hypotension that developed following CSDH evacuation caused the aneurysmal bleed. But once the intracranial pressure (ICP) started rising after the development of SAH and subdural bleed, the sac of the aneurysm might have been held in tamponade; so that further bleeding was prevented.<sup>[2]</sup>

In the two other similar reports, the intra-operative findings that led the authors to think of some new pathology were brain swelling,<sup>[3]</sup> increased amount of drain (300 ml)<sup>[2]</sup> and deterioration in the patients' neurological status. In our case also, there was increased the amount of drain (450 ml) and worsening of neurological status. The worsening can be attributed to the sudden rise in ICP following SAH. The increased amount of drain could possibly be explained by the generalized hyperperfusion that might have caused fresh bleeding from the CSDH membranes or by the extension of SAH into the subdural space through a tear in the arachnoid which might have been preexisting. A drain amount of >300 ml over 1-h following CSDH evacuation should be looked with suspicion.

There was a poor patient outcome in the other two similar studies. But our patient made a successful recovery.

To conclude, aneurysmal SAH should also be added to the differential diagnosis of intracerebral hematomas when there is increased the amount of drain or worsening of neurological status or brain bulge following evacuation of a CSDH.

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