

## Smoking and Subarachnoid Haemorrhage: A Case Study

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### Abstract

Intracranial haemorrhage is the deadly disease which may result from rupture of aneurysm or arterio-venous malformation and rebleeding is the life threatening complication. A forty years old female presented with three episodes of bleeding in one and half month, still she survived and returned home without any focal neurological deficit. She was not hypertensive, and had no history of diabetes or bleeding disorder, but unfortunately she was a smoker, which is an important risk factor, specially in female having intracranial aneurysm. As conventional angiogram is hazardous, MRA (Magnetic Resonance Angiogram) of brain was done which failed to reveal any aneurysm but C T angiogram showed two aneurysms involving both anterior and middle cerebral arteries.

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### Introduction:

Subarachnoid haemorrhage (SAH) is the fourth most cerebrovascular disorder following atherothrombosis, embolism and primary intracerebral haemorrhage but one that is often disastrous.<sup>1</sup> Most subarachnoid haemorrhages are caused by ruptured saccular aneurysms. Other causes include trauma, arterio-venous malformation (AVM), vasculitides, intracranial arterial dissections, amyloid angiopathy, bleeding diathesis, illicit drug use etc. Smoking and hypertension are two preventable risk factors. SAH is a medical emergency and needs intensive care at ICU (Intensive care unit). Removal of the aneurysm is the definitive treatment. Symptomatic treatment includes maintenance of blood pressure, fluid and electrolyte balance, analgesic etc.

### Case Presentation:

A lady of forty years of age, smoker, coming from a

lower middle class family, presented with sudden onset of severe headache with vomiting for fifteen days. She was afebrile, had no chest, bladder or bowel symptoms. On admission, her pulse rate was 76 beats/minute and blood pressure (B.P) was 100/70 mm of Hg. Patient was conscious, but irritable. She had no focal neurological deficit. Her neck was stiff and Kernig's sign was positive. CT scan of brain revealed features of raised intracranial pressure but had no bleeding inside (Fig:1). Cerebrospinal fluid (CSF) study showed plenty RBC (red blood cell) and otherwise normal. At that time, her platelet count was 350 lac/cmm, Prothrombin time (PT) and activated partial thromboplastin time (aPTT) was normal. We treated her with measures to reduce raised intracranial pressure like mannitol. As the patient improved we discharged her with the advice to report with MRA of brain. At home she again started smoking and on the third day during smoking suddenly she fell down and again developed severe headache and vomiting. On admission, she was conscious but drowsy. Her B.P was 100/70 mm of Hg, pulse rate was 76 beats/minute. Pupils were normal in size and reaction. She had no limb weakness, reflexes were normal, planter was flexor bilaterally. This time CT brain revealed frontal haemorrhage with perilesional oedema (Fig:2). We were treating her accordingly but on 13th day she suddenly became unconscious with G.C.S.(Glasgow Coma Scale) only 3; B.P was 130/100 mm of Hg, pupil was dilated with sluggish reaction, planter reflex was extensor bilaterally. This time CT scan revealed enlargement of the size of the previous haemorrhage with ventricular extension (Fig:3). She gradually improved on treatment, became conscious on 4th day, started taking oral food and gained sphincter control. We sent her for MRA of brain. Surprisingly MRA did not reveal any aneurysm or AVM (Fig: 4). Then we did a CT

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angiogram of brain which showed aneurysm of both anterior and middle cerebral arteries (Fig: 5).

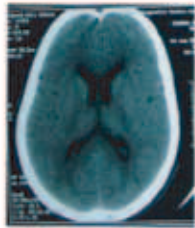


Fig 1: CT scan of brain shows no haemorrhage

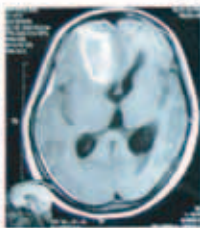


Fig 2: CT scan of brain shows frontal haemorrhage.

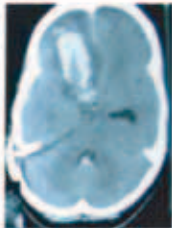


Fig 3: CT scan of brain shows haemorrhage with ventricular extension.



Fig 4: MRA of brain showing no aneurysm

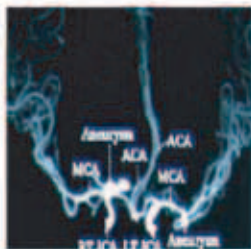


Fig 5: CT angiogram of brain showing aneurysm of anterior and middle cerebral arteries.

## Discussion:

The prevalence of intracranial saccular aneurysm is 5% and 20-30% of patients have multiple aneurysm.<sup>1</sup> The mean age of onset is 55 years.<sup>2</sup> Among the preventable risk factors cigarette is the commonest. A case control study of 432 adults revealed current cigarette smokers have increased risk having a clear dose dependent effect and the risk appears to be more prominent in case of woman having aneurysm.<sup>3-6</sup> A systematic review of 14 longitudinal and 23 case control studies that included 3936 patients with SAH, showed that current smoking was a significant risk factor.<sup>4</sup>

An analysis of data from Asia Pacific Cohort studies Collaboration (APCSC) involving 26 cohorts with 306,620 participants and 236 subarachnoid haemorrhage (SAH) events showed that the risk for SAH was significantly associated with current smoking.<sup>6</sup>

Hypertension is another major risk factor. Moderate to heavy alcohol consumption increases the risk of SAH. First degree relatives of patients with SAH have a 3 to 5 fold increased risk of SAH.<sup>7</sup> The genetic susceptibility to SAH appears to be heterogeneous. Some are autosomal dominant, others are autosomal recessive or multifactorial in inheritance. Elastin gene may be responsible on chromosome 7q for development of familial and sporadic SAH.<sup>8-10</sup> A polymorphism affecting the platelet adhesive glycoprotein GP11a HPA

-1 is associated with increased risk of thrombosis and decreased risk of SAH.<sup>11</sup> Oestrogen deficiency leads to increased risk in menopausal woman and oestrogen replacement therapy may lead to reduced risk to develop SAH.<sup>12</sup> Antithrombotic therapy did not show increased risk in present study but anticoagulant therapy increases the severity of SAH. The symptoms of SAH begin abruptly with sudden severe headache in 97% cases. The onset may or may not be associated with loss of consciousness, seizure or meningismus. Thirty to fifty percent patients may have a warning leak manifested only by sudden severe headache that precedes a major headache by 6-20 days.<sup>13</sup> So in case of sudden severe headache SAH should be excluded. In a prospective study, 148 patients presenting with sudden severe headache 25% had SAH and headache was the only symptom in 12% cases.<sup>14</sup> SAH is associated with high mortality. They usually last for few seconds but rebleeding is common. Rebleeding is highest in first 24 hrs after SAH and may correspond with maximal aneurysm diameter and Hunt Hess grade scale.<sup>1</sup> In 50 patients having SAH 5 rebled in the first week (all fatal), 8 in the second week (fatal), 6 in the third and fourth week (4 fatal), 2 in the next 4 weeks (2 fatal) making a total of 21 recurrences (16 fatal) in 8 weeks.<sup>1</sup>

Vasospasm may lead to infarction. Hydrocephalus can occur in patients having intraventricular extension of haemorrhage. Raised intracranial pressure may lead to impairment of consciousness. Seizure, hyponatremia and cardiac abnormalities may also complicate subarachnoid haemorrhage.

A non contrast head CT is the mainstay of diagnosis with or without lumbar puncture. Clot is demonstrated in 92% cases in first 24 hrs.<sup>15</sup> Cerebral angiography should be considered if diagnostic doubt persists. Brain M.R.I. in proton density and flair sequences may be as sensitive as CT brain.<sup>16</sup> No cause of haemorrhage can be found in 14-22% cases. Angiogram should be repeated if the initial one is negative. CT angiography and MRA are noninvasive tests and useful for screening. Conventional angiography or digital subtraction angiogram are the gold standard relative to CT angiography 83-98% and transcranial dopplar can again improve diagnosis further. But still small aneurysm can be missed.

Regarding treatment, removal of aneurysm is the definitive treatment. Symptomatic treatment includes management of blood pressure, fluid and electrolyte balance, analgesic and laxative etc.

## Conclusion:

Subarachnoid haemorrhage is the dangerous disease but

with proper diagnosis and treatment (including the neurosurgical intervention if required) improved prognosis can be expected.

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