Case Report

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A Case of Aneurysmal Sub Arachnoid Hemorrhage Complicated by Nstemi and Acute Pulmonary Edema

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Abstract

Aneurysmal SAH is a life threatening event which can cause permanent disability, the mortality and morbidity will be increased with associated complications like ACS and Acute pulmonary edema, which are rare complications of aneurysmal SAH. The combination of these three conditions imposes important treatment dilemmas. These patients tend to have electrocardiographic changes along with elevated cardiac biomarkers and neurogenic stress cardiomyopathy and pulmonary edema.

Keywords: Aneurysmal; Sub Arachnoid; Hemorrhage; Nstemi; Acute Pulmonary Edema.

INTRODUCTION

Aneurysmal SAH is condition which can cause severe neurological disability and is highly fatal, can be complicated by acute pulmonary edema and ACS both of them directly indicate severity of haemorrhage, as well as overall prognosis.

Patient can develop Takot subo like cardiomyopathy and neurogenic pulmonary oedema as result of neurologically induced over stimulation of sympathetic nervous system via brain heart connection and may complicate aneurysmal SAH.

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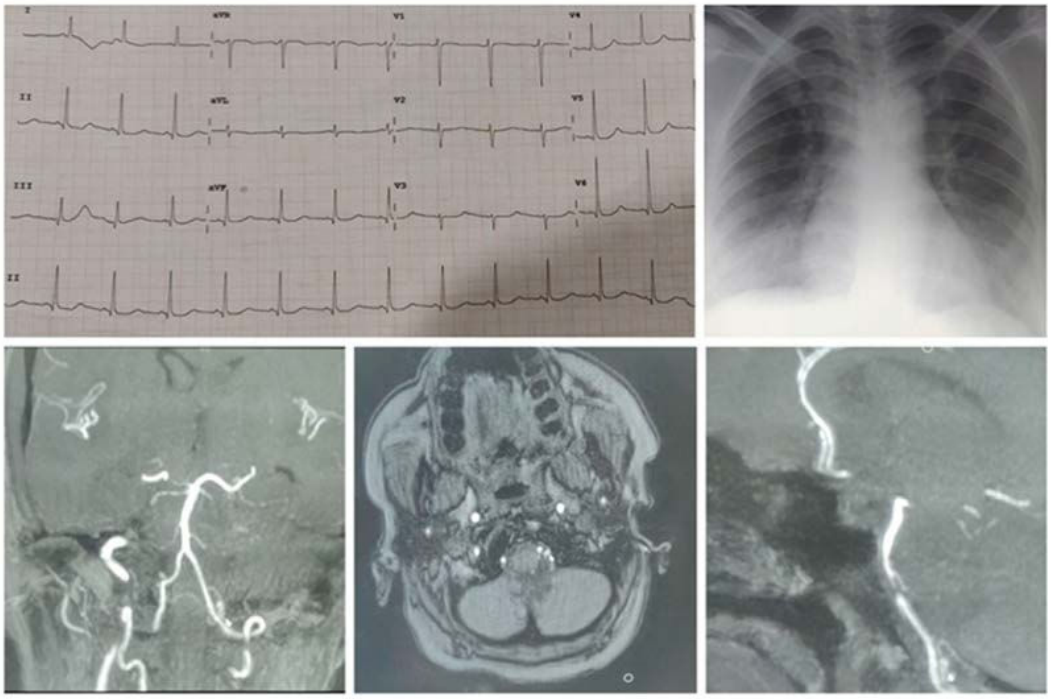
CASE PRESENTATION

A 38 year old female with no known comorbidities presented to emergency department with complaints of sweating, vomiting, neck pain since 1 day. Her last known normal was 1 day prior to arrival. In emergency department she had sudden onset of pounding headache, she never had a headache like this before. She denied loss of consciousness, loss of sensation, loss of motor function, difficulty speaking, facial drooping, loss or change in vision. Prior to arrival in emergency department she presented in another facility with complaints of sweating, vomiting and neck pain, there initially evaluated symptomatic care given, ECG performed which showed ST depression in lead V1 to V6, II, III and avF with elevated cardiac biomarker troponin I.

In our ED patient developed sudden onset of severe headache with two episodes of projectile vomiting, non-bilious, non-bloody. Initial vitals HR-100 beats per minute, BP-180/100 mmHg, Spo2 – 90% on room air, RR-22 cycles per minute, Temp-98F, HGT- 158mg/dl. On examination HEENT -normal , RS- B/L equal air entry, B/L crepts present, CVS S1 S2 heard, abdomino pelvic examination and CNS examination were unremarkable, GCS: 15/15, pupils: B/L equally reactive to light. B/L peripheral pulse felt.

Immediately IV analgesia, PPI, anti-emetic given. Arterial blood gas analysis done Ph-7.5/ pCo2-19/po2-53/Hco3-20/lac-2.8mmol/L. Started on O2 support 6L by face mask. Chest X ray done – showed acute pulmonary edema. While in ED patient underwent non contrast CT scan of her brain. Imaging revealed diffuse SAH along B/L cerebral hemispheres, B/L sylvian fissures and basal cisterns with ventricular extensions. A stroke protocol was initiated, she had initial NIHSS of 0 upon arrival to ED.

Initial blood investigations indicated leukocytosis with normal creatinine and electrolytes. She was started on Labetalol IV infusion for fine tuning of blood pressure. On physical examination patient was diaphoretic and appeared to be lethargic but was able to answer questions. Patient was oriented to time place and person and able to provide accurate history. There were no signs of dysarthria, she had normal comprehension and fluency. Her pupils were equal, round and reactive to light and accommodation. She was able to track objects and had no signs of nystagmus. Interventional radiology and neurosurgery consultation sought, case taken up for DSA. Found to have left ruptured PICA aneurysm followed by surgical procedure, suboccipital craniectomy and clipping of aneurysm with placement of EVD was done. Patient was transferred to neurosurgery intensive care unit for observation and further management.



DISCUSSION

SAH is bleeding from the sub-arachnoid space which lies between the arachnoid and pia matter. The annual incidence of non-traumatic SAH varies from 2-22 per 100000.[1] The incidence in women is almost twice as high as in men. Most cases of Rupture of SAH caused by rupture of aneurysms or AV malformations. Approximately 15-20% cases don't have vascular lesions.[2]

The usual presenting complaints of SAH are Headache, neck pain, vomiting, loss of conciousness. Headache is sudden onset of severe headache described as a thunder clap type, often patients describe it as worst headache of life. These patients are hypertensive on presentation, may show signs of Meningismus.

Physicians should have high clinical suspicion in patients presenting with sudden onset of severe

headache, A non-contrast Head CT should be done to rule out SAH. If Head CT is negative, lumbar puncture should be performed, it likely shows elevated CSF opening pressure, elevated RBC count, that doesn't diminish from CSF fluid tube 1 to tube 4 and xanthochromia.[3]

The severity of SAH is assessed by Hunt & Hess grading system, one of the widely used grading system across the world.

The risk of vasospasm based on haemorrhage pattern in Head CT is assessed by Fischer scale Aneurysmal SAH associated with cardiac and pulmonary manifestation are highly fatal.

The overall prognosis depends on the degree of delayed cerebral ischemia, volume of initial bleed and rebleeding.[4]

In Our case patient had Regional wall motion abnormality in 2D ECHO due to neurologically induced over stimulation of sympathetic nervous system via brain heart connection and may complicate SAH.

This patient also had Pulmonary Edema secondary to SAH which resolved after Neurosurgical intervention.

Takotsubo like cardiomyopathy increases the mortality rate of SAH.

Similarly, Garg and Bar states that various systemic complications occur as a result of aneurysmal sub arachnoid haemorrhage, complications include electrocadiographic changes, troponin I elevation, neurogenic pulmonary edema, anemia, hyponatremia, neurogenic stunned myocardium.[5]

Electrocardiographic changes occurred during the acute stage on 50-100% of SAH cases,[6-8] the most common abnormalities are non-specific ST deviations, T wave inversions and prolonged QT interval, although ECG abnormalities and elevated Troponin and abnormal left ventricular wall motion in patient with SAH are well known phenomenon, the associated with ST elevation MI is rare. Approximately 20% of sub arachnoid haemorrhage cases with elevated troponin I found to have increased risk of hypotension, pulmonary edema, left ventricular dysfunction, and delayed cerebral ischaemia.[9-11]

This unfortunate combination may prompt the cardiologist to perform PCI and administer antithrombotic drugs. The unprotected aneurysm make the neurologist reluctant to administer anti thrombotic agents.

Ideally treatment of an unprotected aneurysm with concomitant MI would consist of immediate coiling or clipping of aneurysm followed by PCI.

Clinical Hunt And Hess/WFNS (the latter is more objective)

I - asymptomatic or mild headache/GCS 15, no motor deficit.

II - moderate-severe headache, meningism and no weakness/GCS 13-14, no motor deficit.

III - mild alteration in mental status, GCS 13-14, motor deficit.

IV - depressed LOC and/or hemiparesis/GCS 7-12+/- motor deficit.

V - posturing or comatose/ GCS 3-6, motor deficit present or absent.

CT - Fisher

I - no Blood.

II - diffuse deposition of SAH without clots or layers of blood > 1mm.

III - localized clots and/or vertical layers of blood 1mm or > thickness.

IV - diffuse or no subarachnoid blood but intracerebral or intraventricular clots.

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