Recurrent perioperative atrial fibrillation in a patient with aneurysmal subarachnoid haemorrhage: A case report and review of literature

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Electrocardiography (ECG) abnormalities, ranging from morphological waveform changes to potentially life-threatening arrhythmias, are known to complicate subarachnoid haemorrhage (SAH) in its acute phase; prevalence rate ~ 27–100%).[1-7] With emphasis on early neurosurgical intervention in aneurysmal SAH, more patients with abnormal ECGs are likely to present for surgery, making it imperative for the anaesthesiologist to be well-versed with this complication. Misinterpretation of the ECG to signify cardiac disorders like myocardial infarction can unnecessarily delay neurosurgery and, development of intra-operative arrhythmias can cause sudden clinical deterioration or even death,[8] thus jeopardising the cardiovascular stability vital to aneurysm surgeries. Atrial fibrillation (AF) is known to have life-threatening consequences like haemodynamic compromise, thromboembolism and ischaemic stroke, dilated cardiomyopathy and cardiac failure.[9,10] We report here the development of this serious arrhythmia in a patient with SAH undergoing early aneurysm clipping and discuss the relevant literature.

A 46-year-old female patient was admitted with headache and altered sensorium (Glasgow Coma Score ~ E3 V4 M6). Computerised tomography (CT) brain revealed SAH (Fischer grade III) with no midline shift and normal ventricles; digital subtraction angiography confirmed a 10 × 7 mm, anterior communicating artery aneurysm. Emergency craniotomy and aneurysm clipping was planned. Pre-operative evaluation revealed no evidence of prior hypertensive or cardiac illness, a blood pressure (BP) of 148/96 mmHg, a regular heart rate (HR) of 106/min, normal chest auscultation and no other systemic abnormality; investigations including chest X-ray, ECG and serum electrolytes were normal. Treatment with phenytoin and nimodipine infusion (2 mg/h) was initiated and surgery undertaken 3 h later. In the operating room, the patient developed AF with a fast ventricular rate (HR – 202/min, irregularly irregular) while her BP (104/62 mmHg) and oxygen saturation (99%) were normal. Except for hypokalaemia (serum potassium – 2.8 mmol/L), the arterial blood gas was normal (Hb – 12.7, pH – 7.45, PaO_2 – 135, $PaCO_2$ – 32.2, sodium – 133, bicarbonate – 24.2, base excess/deficit – −0.03, blood sugar – 118). She was administered intravenous (IV) potassium supplementation and metoprolol 2 mg in three doses, following which, there was a decrease in her HR (140–145/min, irregular) and BP (88/56 mmHg); dopamine infusion (6 μg/kg/min) and rapid IV fluids were given to normalise the BP (110/68 mmHg). Deferring surgery for further cardiac evaluation was not considered. The patient was anaesthetised using IV thiopentone in titrated doses (total 150 mg), vecuronium (0.1 mg/kg bolus, 0.8–1.0 μg/kg/min infusion), fentanyl (2 μg/kg bolus, 1 μg/kg repeated hourly), oxygen: nitrous oxide (50:50) and isoflurane (0.25–0.5%); ventilation was controlled, and mannitol (0.5 g/kg) was infused. Half an hour later, she had another episode of tachycardia (HR – 220/min, irregular) and hypotension (BP – 40/20 mmHg) with a normal central venous pressure (CVP) of 8 cm H_2O. Dopamine infusion was increased and the patient administered rapid IV fluids, adrenaline (1 ml, 1:10000) and preservative free lignocaine (60 mg); amiodarone infusion @ 0.5 mg/min was started. The patient's condition gradually stabilised at a BP of 100–110/70-80 mmHg, HR of 150–160/min, irregular, and a CVP of 9 cm H_2O; her heart rhythm also normalised by the end of surgery. Following adequate recovery from anaesthesia she was transferred to the intensive care unit for close monitoring. Post-operative echocardiography (ejection fraction > 60% and no regional wall motion abnormalities, cardiac hypertrophy or valve impairment), serum electrolytes and thyroid status were normal. Amiodarone and

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dopamine infusions were discontinued within 24 h. The next day the patient had another episode of AF (HR – 158–162/min, irregular), but without hypotension (BP – 115/84 mmHg). Oral diltiazem (30 mg, 8 hourly) was started and the AF resolved within 2 days; she was stable thereafter. However, on the sixth post-operative day she developed sudden neurological deterioration followed by respiratory insufficiency and haemodynamic impairment, for which, ventilator support and inotropes were started. Her condition worsened rapidly and she died the following day; CT scan could not be done.

In the absence of prior cardiac disease and presence of a normal echocardiogram and thyroid status, the recurrent episodes of peri-operative AF in this patient were most likely due to SAH and concomitant hypokalaemia. Cardiac repolarisation abnormalities involving the S-T segments, T, P and U waves and QTc intervals manifest in the ECGs of over 50% of patients within the first few days of SAH onset; a prolonged QTc interval, with a broad, slurred and inverted T wave is the most common abnormality.[1,3] SAH-induced cardiac rhythm disturbances are also known.[1-4,8,11] While the commoner sinus tachycardia and bradycardia and, premature atrial and ventricular beats are relatively benign and resolve spontaneously within 2–3 weeks, uncommon abnormalities (1–4% patients) like ventricular tachycardia, ventricular flutter and fibrillation, AF and flutter, Torsades de pointes, paroxysmal supraventricular tachycardia and atrio-ventricular blocks can lead to serious complications.[1,3] SAH-related ECG abnormalities are suggested to be independent predictors of mortality and disability;[1,3,5,6] a significant relationship was reported between arrhythmias and an increased risk of cardiovascular co-morbidity, prolonged hospital stay, poor functional outcome and high mortality due to cerebral infarction, re-bleeding and myocardial infarction.[1] Our patient died a week after surgery, presumably due to severe vasospasm-induced cerebral infarction; whether this mortality was related to SAH-induced arrhythmias cannot be ascertained. The aetiology of ECG changes is hypothesised variously as systemic and intra-myocardial catecholamine release causing sub-endocardial ischaemia and focal necrotic and haemorrhagic lesions, coronary vasospasm, reversible post-ischaemic 'stunned myocardium', injury of the arrhythmogenic cerebral insular cortex and, hypokalaemia secondary to repeated vomiting, raised plasma catecholamines and corticosteroids release; irreversible, fatal AF was reported in two SAH patients with hypokalaemia.[4] Development of arrhythmias in SAH is influenced by the presence of cerebral vasospasm, hypothalamic dysfunction, hyperglycaemia, hypoxia, electrolyte imbalance, presence of blood in the subarachnoid space, severely raised intra-cranial pressures and brain stem compression.[1,3,6,12]

Patients with aneurysmal SAH undergoing surgery in the acute phase are potentially at-risk for developing peri-operative arrhythmias; Andreoli *et al*., reported serious arrhythmias in 7 out of 70 patients during and after surgery undertaken within 24 h of SAH.[2] Supraventricular tachyarrhythmias like AF and atrial flutter are reported as less frequent manifestations in SAH (incidence of AF – 2–10%) by most authors;[2-4,8,11] in contrast, Frontera *et al*., observed AF to be the commonest arrhythmia in SAH (76% out of 4.3% clinically significant arrhythmias).[1] Pre-operative detection of AF warrants a thorough evaluation of its clinical manifestations and complications, and detection of other causes like raised intra-cranial pressure, coronary artery disease, myocarditis, thyroid abnormalities, electrolyte imbalance, acute lung disease, alcohol intake, pulmonary embolism, previous surgery, etc. Investigations include a 12-lead ECG recording, Holter monitoring, echocardiography, chest radiography, thyroid function tests and cardiac electrophysiological studies;[9] routine ECG monitoring is recommended in all SAH patients in the acute phase,[2] and the length of QTc interval can help detect predisposition to potentially lethal tachyarrhythmias in the presence of hypokalaemia.[7] Management of AF is directed towards correcting the precipitating factors and treatment by either, cardioversion with drugs like flecainide, dofetilide, propafenone, ibutilide and amiodarone or electrical shock with a direct current (DC) of 200 J or, controlling the ventricular rate with IV beta blockers (esmolol, metoprolol, propranolol) or non-dihydropyridine calcium channel blockers (diltiazem, verapamil); beta blockers may be preferable in SAH induced AF, which is adrenergically mediated.[9] The often aggressive pre-operative approach involving urgent angiography and surgery in aneurysmal SAH patients may not allow their proper evaluation and treatment, which happened in our case. It is recommended that patients with new onset AF, no acute cardiac illness and confirmed absence of structural heart disease on echocardiography may proceed with surgery following control of ventricular rate (<100/min) with IV beta blockers or diltiazem; pre-operative electrical cardioversion may be required if AF is associated with symptomatic hypotension, heart failure or myocardial ischaemia. Prophylactic anti-arrhythmics are seldom indicated in a first-detected episode of AF.[9] Surgery may have to be deferred in cases of poorly controlled ventricular rate, uncorrected symptoms or confirmed presence of heart disease.[13] Intraoperative management of AF includes maintenance of hemodynamic stability

with careful usage of anesthesia drugs, readiness with inotropes and CVP-guided fluid therapy and, aggressive correction of hypokalaemia, anaemia and respiratory insufficiency; measures deployed in aneurysm surgeries like induced hypo-/hypertension, hypervolaemia, high-dose mannitol, etc., can be detrimental in the presence of arrhythmias.[2] Acute intra-operative AF can be controlled with IV esmolol (0.5–1 mg/kg bolus, 60–200 μg/kg/min infusion), diltiazem (0.25 μg/kg bolus, 5–15 mg/h infusion) or verapamil (75–150 μg/kg bolus); amiodarone (150 mg loading dose, 0.5–1 mg/min infusion) is preferred in critically ill patients and those with accessory pathways or if conventional measures are ineffective;[9] digoxin is no longer preferred for rapid management of AF.[9] Post-operative management is directed towards prevention of recurrent AF and thromboembolic complications. AF recurrence or persistence, well-known in SAH patients, is initially managed by beta blockers or calcium channel blockers and by ibutilide, propafenone or amiodarone if it lasts beyond 24 h.[13] Elderly patients, those with prolonged AF and those requiring DC cardioversion, are at a high risk of thromboembolism and may require anti-coagulation with low molecular weight heparin or un-fractionated heparin or warfarin to achieve an international normalised ratio target of 2 to 3. Due to the high risk of re-bleeding in patients with vascular intra-cerebral lesions, anti-coagulation should be done cautiously under close coagulation profile monitoring.[14]

Thus, development of peri-operative arrhythmias is a real threat during the acute phase of aneurysmal SAH, and their appropriate handling is vital to a safe patient outcome. The post-operative mortality in our patient, presumed to be due to vasospasm-induced cerebral infarction, could also possibly be related to the occurrence of these arrhythmias, though this is largely speculative.

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