Cardiac Manifestations of Subarachnoid Haemorrhage

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Abstract
Background: To evaluate the cardiac manifestations of aneurysmal subarachnoid haemorrhage (SAH) in terms of its effects on haemodynamics and changes in electrocardiography, echocardiography and cardiac troponin I.

Methods: In this cross-sectional study 96 patients, with a primary diagnosis of aneurysmal subarachnoid haemorrhage (SAH), were included. Cardiac abnormalities were defined as hypotension, pulmonary edema, ECG changes, echocardiographic wall motion abnormalities or reduction in ejection fraction below 50 percent and cardiac troponin I level above upper range of normal.

Results: Mean age ±SD was 45.9±22.2. Males were 57.6%. On presentation to the emergency department pulse was 77.2±13 beats/min, systolic blood pressure 126.7±18 mmHg and diastolic blood pressure 64.7±10.2 mmHg. Eleven patients (11.1%) developed pulmonary edema during ICU stay while 14.6% developed hypotension. ECG was abnormal in 75.7%. Various abnormalities on ECG included symmetrical T wave inversion (35.5%), hyperacute T wave (13.1%), ST segment elevation (13.1%), ST segment depression (32.2%), long QT (34.3%), prominent U wave (14.1%), Sinus bradycardia (46.4%) and Sinus tachycardia (30.3%). Echocardiography showed wall motion abnormalities in 17.2% and ejection fraction was less than 50 percent in 12.1%. Cardiac troponin I was elevated in 15.6%.

Conclusion: Patients with aneurysmal subarachnoid haemorrhage frequently develop cardiac injury which manifests as hemodynamic derangement, electrocardiographic changes and regional or global left ventricular dysfunction on echocardiography.

Key Words: Subarachnoid haemorrhage, Cardiac manifestations

Introduction
Subarachnoid haemorrhage (SAH) is a catastrophic neurological event which carries a high morbidity and mortality throughout the world. Considerable variation in the annual incidence of aneurysmal SAH exists in different regions of the world. It occurs primarily during young to mid adulthood in both sexes. Mortality rates vary widely. The most common cause of SAH is rupture of a congenital aneurysm in a blood vessel at the base of the brain. A less common source of hemorrhage is the rupture of an aneurysm of traumatic or infectious origin or rupture of an arteriovenous malformation.1-2

In addition to the usual clinical signs and symptoms of SAH, which include abrupt onset of severe headache, nuchal rigidity, nausea, vomiting and alteration in consciousness, electrocardiographic (ECG) abnormalities often occur. These abnormalities include both morphological waveform changes in the 12-lead ECG and arrhythmias. Although such cardiac manifestations can be associated with a wide variety of neurological events, including cerebrovascular accident, head injury, meningitis, and tumours, the highest prevalence and most pronounced changes occur after aneurysmal SAH.3

ECG changes occur during the acute stage of SAH, with the most common abnormalities involving the ST segment, T wave, and QT interval. In most cases, these abnormalities are clinically inconsequential and are attributed to neurally mediated electrophysiological effects. Some SAH patients, however, show evidence of structural cardiac damage. Plasma levels of the creatine kinase myocardial isoenzyme (CK-MB) are mildly elevated in 20% to 50% of patients, and a characteristic form of myocardial pathology, contraction band necrosis, is commonly found at autopsy. A reversible form of neurogenic myocardial "stunning," presumably related to myocardial catecholamine toxicity and contraction band necrosis, may occur after SAH.4-6

Neurogenic ECG changes after SAH are usually regarded as asymptomatic. The potential impact of neurogenic cardiac injury on left ventricular hemodynamic performance after SAH has received little attention but may have important implications because approximately 30% of patients develop delayed cerebral ischemia related to vasospasm. Clinical studies in humans indicate that vasospasm is
associated with a loss of autoregulation and experimental studies have shown that cerebral blood flow (CBF) in ischemic areas can vary passively with changes in blood pressure and cardiac output. Accordingly, hypovolemia has been implicated as a risk factor for symptomatic vasospasm and augmentation of blood pressure and cardiac output can reverse ischemic deficits in affected patients. 7-10

Patients and Methods
This descriptive study was conducted in the Department of Neurology, Lady Reading Hospital Peshawar from January 2008 to Dec 2011. Aneurysmal subarachnoid hemorrhage was diagnosed when a patient had sudden onset of severe headache and evidence of blood in brain ventricles on CT scan or, in case of negative CT scanning, by evidence of xanthochromia in the cerebrospinal fluid (performed 12 hours after the ictus). Aneurysm as source of bleeding was verified by MR cerebral angiography or CT angiography. Cardiac abnormalities were defined as hypotension, pulmonary edema, ECG changes, echocardiographic wall motion abnormalities or reduction in ejection fraction below 50 percent and cardiac troponin I level above upper range of normal.

Pulmonary edema was defined by the presence of characteristic diffuse infiltrates on chest radiography and reduced oxygenation requiring at least 40% supplemental oxygen. Hypotension was defined as a systolic blood pressure less than 100 mm Hg requiring treatment with intravenous pressors. Noncardiac causes of pulmonary infiltrates, hypoxemia, or hypotension were excluded.

Twelve Lead ECG was obtained and systematically analysed. Two-dimensional color-flow Doppler Transthoracic echocardiogram was performed. Left ventricular ejection fraction was measured using modified Simpson’s method. Two investigators (independent of each other and blinded from the clinical data of the patient) analyzed the echocardiographic images for the assessment of global and regional left ventricular function. Troponin I was measured on the first day of admission and was repeated on third day if the initial result was negative.

Patients having histories of hypertension, ischaemic heart disease, congestive heart failure, hypertrophic cardiomyopathy, aortic stenosis, abnormal serum values for potassium or total calcium, those treated with digitalis, diuretics, beta-blockers or other antihypertensive drugs, traumatic subarachnoid hemorrhage, hypertensive cerebral bleed and brain tumours were excluded to avoid misinterpretation of ECG.

Results
A total of 104 patients were screened in which 8 were excluded from the study due to history of pre-existing heart disease to avoid electrocardiographic, cardiac enzyme and echocardiographic abnormalities related to nonneurogenic mechanisms. Ultimately 96 patients were included in the study. Among these 57.6% were male and 39.4% were female. Mean age ±SD was 45.9±22 with a range from 10 to 80 years. On presentation to the emergency department, pulse was 77.2±13 beats/min, systolic blood pressure 126.7±18.3 mmHg and diastolic blood pressure 64.7±10.2 mmHg. Eleven patients (11.1%) developed pulmonary edema during ICU stay while 14.6% developed hypotension (Table 1).

ECG was abnormal in 75.7%. T wave inversion (35.5%) was the commonest. Echocardiography showed wall motion abnormalities in 17.2%. Ejection fraction was less than 50 in 12.1%. Cardiac troponin I was elevated in 15.6% (Table 2).

Table 1: Subarachnoid haemorrhage—Clinical characteristics

<table>
<thead>
<tr>
<th>Clinical characteristics</th>
<th>No(%)</th>
<th>Mean±SD</th>
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<tbody>
<tr>
<td>Age ± SD</td>
<td></td>
<td>45.9±22.2</td>
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<tr>
<td>Male</td>
<td>57(57.6)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>39(39.4)</td>
<td></td>
</tr>
<tr>
<td>Pulse(beats/min)</td>
<td></td>
<td>77.2±13.6</td>
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<tr>
<td>Systolic blood pressure(mmHg)</td>
<td>126.7±18.3</td>
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</tr>
<tr>
<td>Diastolic blood pressure(mmHg)</td>
<td>64.9±10.2</td>
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<tr>
<td>Pulmonary edema</td>
<td>11(11.1)</td>
<td></td>
</tr>
<tr>
<td>Hypotension</td>
<td>14(14.6)</td>
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Discussion
Patients with aneurysmal SAH manifest a spectrum of cardiac abnormalities which include haemodynamic derangements, electrocardiographic abnormalities and echocardiographic regional or global LV dysfunction. 11,12 An abnormally prolonged QTc interval with large T waves is a common finding in patients with aneurysmal SAH. Q-T interval , in the absence of conduction disturbances, is an indirect measure of the duration of cardiac repolarization. The prolongation may be due to a disturbed regional myocardial sympathetic activity, which may be part of a general disorder of the sympathetic nervous activity following aneurysmal SAH. The pattern of broad, slurred, inverted T waves associated with long QTc intervals is
commonly termed “cerebral,” “neurogenic,” or “giant” T wave. The U wave, which is often 1 mm or greater in amplitude in patients with SAH, can be mistakenly interpreted as part of a notched T wave if the U wave occurs early during repolarization. S-T segment depression is another ECG change often reported in SAH patients. ECG changes that occur during cardiac repolarization, such as abnormalities in the ST segment and the T wave, must be interpreted in the context of the patient’s neurological abnormalities. Neurologically mediated ECG changes are often misdiagnosed as myocardial ischemia or infarction, resulting in inadvertent or delayed treatment of the primary problem.

Theories about the underlying causes of ECG abnormalities in SAH are controversial. Cardiac injury due to elevated myocardial wall stress associated with tachycardia and hypertension has also been suggested as a causative factor. Coronary vasospasm and reversible post ischemic “stunned myocardium” may influence the development of ECG changes in patients with aneurysmal SAH. Evidence indicates a neurogenic etiology for ECG abnormalities in SAH. Animal studies suggest interesting links between brain structures and the heart. Unilateral alteration of sympathetic tone to the heart, is also partially held responsible. Injury to the insula, an area of the cortex thought to be involved in arrhythmogenesis, might be implicated in both abnormal cardiac rhythm and the focal myocardial lesions that sometimes occur after SAH. Currently, much research focuses on the release of catecholamines, either systemically or within the myocardium, as a cause of ECG abnormalities. The characteristic pattern of myocardial lesions suggested to some researchers that the damaging catecholamines are released from intramyocardial nerve endings rather than from the general circulation. Factors that may influence the development of arrhythmias in patients with SAH include cerebral vasospasm, hypoxia, electrolyte imbalance, and sudden increase in intracranial pressure triggering a sympathetic or vagal discharge due to compression of brain structures. The basic hemorrhagic nature of SAH may play a role in producing arrhythmias in the period immediately after hemorrhage.17-21

Some SAH patients do show evidence of structural cardiac damage. Plasma levels of creatine kinase myocardial isoenzyme (CK-MB) are mildly elevated in 20% to 50% of patients. A characteristic form of myocardial pathology, contraction band necrosis, is commonly found at autopsy and has been produced in experimental SAH models. Troponin I (cTnI) is a reliable marker of myocardial injury and poor prognosis in patients with unstable cardiac ischemia and also in patients with septic shock. An abnormal cTnI is a powerful predictor for the occurrence of pulmonary and cardiac complications in patients with aneurysmal SAH. The additional prognostic information of troponin I for poor outcome is limited. The severity of brain injury is the leading cause of a poor outcome and pulmonary and cardiac complications are less important factors for the eventual outcome.22,24

Excessive catecholamine release after SAH is the driving force for left ventricular dysfunction of the heart and pulmonary edema. The severity of the brain injury is strongly related to the occurrence of these cardiac and pulmonary complications. Pathogenesis of neurogenic pulmonary edema is noncardiogenic, resulting primarily from injury to the pulmonary circulation. Neurogenic left ventricular dysfunction, in combination with noncardiogenic mechanisms, may contribute to the formation of pulmonary edema.25-27

**Conclusion**
1. Neurologically mediated ECG changes are often misdiagnosed as myocardial ischemia or infarction, resulting in inadvertent or delayed treatment of the primary problem.

2. Routine measurement of the length of the QTc interval in patients with SAH may help detect predisposition to potentially lethal tachyarrhythmias, particularly if the patient also has low serum levels of potassium. All these cardiac manifestations in aneurysmal SAH are transient and need supportive care.

References


