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REVIEW ARTICLE

ECG Changes in Subarachnoid Haemorrhage: A Synopsis

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Abstract Subarachnoid haemorrhage (SAH) is a neurological emergency with high mortality rates. It is mainly caused by rupture of an aneurysm (congenital/infectious/traumatic) or rupture of an arteriovenous malformation. Electrocardiograms (ECGs) done in patients with SAH have shown morphological changes as well as arrhythmias. Subarachnoid haemorrhage (SAH) patients have often been misdiagnosed to have cardiac abnormalities based on their ECGs when in many of those instances the ECG change had been the result of the SAH itself. They have led to unnecessary and wasteful investigations and therapies in many occasions. Hence the current article is an effort at consolidating the information available in an attempt to avoid possible errors in diagnosis by house staff and internists. There are two mechanisms that might mediate ECG changes in patients with SAH, i.e. autonomic neural stimulation from the hypothalamus or elevated levels of circulating catecholamine. Hypothalamic stimulation may cause ECG changes without associated myocardial damage whereas elevated catecholamine levels have been correlated with QT-interval prolongation and myocardial damage.

Keywords Subarachnoid haemorrhage · Electrocardiogram · Arrhythmias

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Significance of ECG Changes Seen in SAH

Subarachnoid haemorrhage (SAH) has long been known to be associated with electrocardiogram (ECG) changes [1]. However, the aetiology, pathophysiology and prognosis of underlying cardiac abnormalities remain unexplained to a large extent. Hypothalamic stimulations [2] and autonomic dysfunctions have been linked but not conclusively proven to be causative. These considerations may influence therapeutic interventions as infusions of large volumes of fluids or administrations of vasopressors may prove detrimental in patients with a compromised heart [3].

ECG changes in SAH commonly reflect ischaemic heart disease and have been known to present with ST-segment elevation and T-wave inversion [4]. Therapeutic thrombolytic therapy and anticoagulation as well as withholding of life-saving neurosurgery in such cases may well endanger the life of the patient concerned [4, 5].

SAH has been associated with malignant ventricular arrhythmias, including ventricular tachycardia, torsades de pointes, and ventricular fibrillation, particularly if the corrected QT (QTc) interval is prolonged, often leading to compromise in a haemodynamically unstable patient [6, 7].

Implications

Documented ECG abnormalities in a patient with SAH who has brain death mean the heart is not accepted as a donor organ because of the possibility of cardiac abnormalities. Greater knowledge about the pathophysiology and management of ECG changes in SAH may make heart donations possible in these cases [8]. Also in

Table 1 Morphological changes in ECG in SAH

ECG changes	Percentages (%)
High R waves	19
ST depression	15
T-wave abnormalities	32
Large U waves	47
Prolonged QTc	23

such a situation it is essential that a proper history is elicited and a thorough physical examination is conducted to rule out SAH and the threshold for ordering diagnostic cerebral imaging should be very low in these instances for thombolysis and anticoagulation may have catastrophic consequences in such patients.

Prevalence of ECG Abnormalities in SAH

The reported prevalence of ECG changes in patients with SAH ranges from 27% to 100% [3, 8–12]. Such wide variation may be due to differences in study design, investigators' definitions of ECG abnormalities, or the methods used to evaluate ECG changes. Admissions with neurological diagnoses often do not have routine ECGs. Thus, there always remains a possibility of selection bias leading to greater inclusion of subjects who have had cardiac events during their hospitalisation, if ECG tracings are used as inclusion criteria. Also, baseline ECGs were unavailable in many cases making it impossible to differentiate between preexisting changes and de novo events associated with an episode of SAH.

Promptness of ECG data collection may influence conclusions. Brouwers et al. [9] found that during the first 72 h after SAH, ECG changes were the most pronounced. Di Pasquale et al. [10] found that 90% of patients had ECG abnormalities within the first 48 h following SAH, suggesting that studies in which surveillance is started later in the course of illness may miss significant data [8].

Due to paucity of follow-up data, the chronological progression of the changes remains unclear. ST elevations seem to resolve while 'T' wave inversions appear to persist—even for months/years [13, 14].

Possible Mechanisms of the ECG Changes in SAH

Hypothalamic stimulation as well as autonomic dysregulations have been implicated as causative for the ECG changes in SAH without corroborative conclusive evidence.

Morphological ECG Changes

In SAH, ST-T wave changes such as associated with myocardial infarction seem to be predominant (Table 1). Other changes seen commonly include presence of a U wave, a Q wave, prolonged QTc intervals and large, flattened or notched T waves [15].

In the largest study [16] to date, a single preoperative 12-lead ECG from each of the 406 patients with SAH was examined. ECG findings included high-amplitude R waves in 19% of subjects, ST depression in 15%, T-wave abnormalities in 32%, U waves greater than 1 mm in amplitude in 47%, and prolonged QTc interval (>440 ms) in 23%. Most of these seem to point to a repolarisation abnormality in SAH patients. Abnormalities involving atrial depolarisation, particularly peaked P waves (>2.5 mm in amplitude) and short PR intervals (<100 ms), have also been reported (Fig. 1) [17, 18].

Rhythm Abnormalities

Commonly detected rhythm disturbances in SAH include sinus bradycardia and sinus tachycardia, wandering atrial pacemaker and atrial fibrillation and have been detected in two studies [19, 20]. Premature atrial, junctional and ventricular complexes, ventricular tachycardia and atrioventricular block have also been detected occasionally [21]. Holter monitoring of patients in recent times has led to greater detection and reporting rates of arrhythmias in SAH patients. In the largest study to date in which Holter technology was used, Di Pasquale et al. [10] first obtained a 12-lead ECG at the time of admission from a sample of 120 patients with SAH. Holter monitoring was started on the same day, and a total of 107 adequate Holter recordings were obtained. Cardiac arrhythmias were detected in

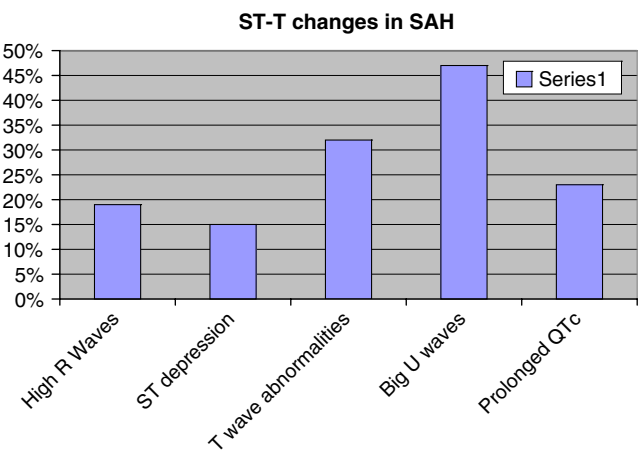

Fig. 1 ST-T changes in subarachnoid haemorrhage

96 (90%) of the 107 patients. Premature ventricular complexes, including multiform premature ventricular complexes, couplets or triplets, and R-on-T phenomenon, were detected in 49 patients (46%). Five of the patients with frequent premature ventricular complexes also had nonsustained ventricular tachycardia (defined as three or more consecutive premature ventricular complexes). Torsades de pointes occurred in four patients and progressed to ventricular fibrillation and asystole in one of the four. Holter monitoring was repeated in 48 h for all patients with malignant ventricular arrhythmias, but no further similar arrhythmias were recorded.

Of the 107 patients, 39 (36%) had supraventricular arrhythmias, including premature supraventricular complexes, nonsustained supraventricular tachycardia, and atrial fibrillation. Thirty-two patients (30%) had sinus tachycardia (heart rate >120/min), 32 (30%) had sinus arrhythmia, 42 (39%) had sinus bradycardia (heart rate <50/min) and 23 (21%) had sinoatrial blocks. Ventricular premature complexes in 49, nonsustained ventricular tachycardia in five, supraventricular premature complexes in 29, paroxysmal supraventricular tachycardia or atrial fibrillation in nine, sinoatrial block and arrest in 29, second-degree atrioventricular block in one, atrioventricular dissociation in four and idioventricular rhythm in two. Life-threatening ventricular arrhythmias (torsades de pointes type ventricular tachycardia) occurred in four patients, degenerating into either ventricular flutter or fibrillation in two. ST-segment changes suggestive of acute transitory myocardial ischaemia were found in eight patients (1.5 mm or more of ST depression in seven patients and 1.5 mm or more of ST elevation in one patient).

In their attempt at correlating arrhythmias with time elapsed since SAH, Di Pasquale et al. [10] found that both frequency and severity of arrhythmias were higher in the 62 subjects studied within 48 h of SAH.

However, all patients with malignant ventricular arrhythmias had a much longer QTc interval (590±52 ms) and serum levels of potassium less than 3.5 mmol/l.

The study by Di Pasquale et al. found no correlation between clinical condition, site of aneurysm, extent of intracranial haemorrhage, age or preexisting heart disease and the frequency and severity of arrhythmias [8, 10].

Summary

The morphological ECG changes most frequently encountered in a subject with SAH are U waves, T-wave abnormalities, R-wave abnormalities and non-specific ST-T changes. The predominant rhythm abnormalities seem to be relatively benign sinus tachycardias and bradycardias. However, the possibility of development of supraventricular arrhythmias, sinoatrial blocks and arrests, and life-threatening ventricular arrhythmias warrants a close monitoring of the ECG in these patients.

A recent study has found that arrhythmias are associated with an increased risk of cardiovascular comorbidity, prolonged hospital stay and poor outcome or death after SAH, after adjusting for other predictors of poor outcome [22]. Although no correlation was found between the frequency and severity of cardiac arrhythmias and the neurological condition, the site and extent of intracranial blood on computed tomography scan, or the location of ruptured malformation, the extremely high incidence of cardiac arrhythmias, sometimes serious, in the acute period after subarachnoid haemorrhage and the absence of clinical and radiological predictors make systematic continuous ECG monitoring compulsory to improve the overall results of subarachnoid haemorrhage, irrespective of early or delayed surgical treatment.

If kept in mind, the ECG changes as enumerated might be beneficial when an SAH patient is evaluated for an abnormal ECG.

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