Acute subarachnoid haemorrhage and the mysterious electrocardiogram

Nevin D, MBBCh, DA
Lines D, MBBCh, FFA
Department of Anaesthesia, University of the Witwatersrand, Johannesburg, South Africa
Correspondence to: Dr Daniel Nevin, e-mail: dnevin@doctors.org.uk
Keywords: subarachnoid haemorrhage; electrocardiogram; cardiovascular dysfunction; myocardial injury

Abstract

Subarachnoid haemorrhage (SAH) is a devastating neurological insult, and is increasingly understood as a multi-system condition initiated in the central nervous system. Perioperative investigation of patients presenting for aneurysm surgery often includes a routine electrocardiogram (ECG) which frequently reveals an abnormality. We describe a patient who presented with SAH and who was found to have significant Q waves on the ECG suggestive of a trans-mural myocardial infarction, despite a negative medical history for such an event. We briefly highlight the issues faced by the anaesthesiologist when dealing with the patient with SAH and ECG abnormality, and the implications for cardiovascular dysfunction.

Introduction

Subarachnoid haemorrhage (SAH), a devastating neurological insult, is increasingly being understood as a multi-system condition initiated in the central nervous system.[1,2] Occurring in approximately 5–20 per 100 000 people (with wide geographical variability) and with a 30 day mortality rate of 25–50% (and up to 70% in patients who re-bleed), it is frequently encountered by neurosurgical teams and consequently by anaesthesiologists.[1,3,4]

Perioperative investigation of such patients often includes a routine electrocardiogram (ECG) which not infrequently reveals one or more repolarisation abnormalities.[1-3,5-7] Other atypical ECG abnormalities have been described, but their presence is infrequent and their clinical significance poorly understood.

We describe a patient who presented with SAH and who was found to have significant Q waves on the ECG suggestive of a previous trans-mural myocardial infarction, despite a negative medical history for such an event. We highlight this case and the issues faced by the anaesthesiologist encountering patients with SAH and ECG abnormalities, and the implications for potential cardiovascular dysfunction.

Case report

Mrs AB, a 46 year old black woman, was booked for cerebral aneurysm clipping after suffering SAH five days earlier. The medical history was significant only for essential hypertension, for which the patient was being treated with hydrochlorothiazide and enalapril, and which was well controlled. There were no risk factors for a cerebrovascular accident or cardiovascular disease and no previous history of congestive cardiac failure, chest pain, shortness of breath or palpitations. Mrs AB readily admitted to excellent prior health and a good effort tolerance. The patient looked clinically well and had an unremarkable physical examination, which was noticeably lacking in any signs of cardio-respiratory disease or residual neurological deficit from the SAH. Her ECG (Figure 1) revealed her to be in sinus rhythm at a rate of 95 beats per minute, with an axis of approximately zero degrees. Significant Q waves were noted in leads II, III, aVF as well as V5 and V6, each with associated T wave inversion. R wave progression was normal. Initial laboratory results recorded the following: white cell count 9.68 x 10^9/L, haemoglobin 12.8 g/dl, platelets 204 x 10^9/L, sodium 139 mmol/L, potassium 3.3 mmol/L, chloride 100 mmol/L, urea 4.3 mmol/L, creatinine 62 µmol/L. Calcium, magnesium, phosphate and C-reactive protein values were

within normal limits. Computed tomography (CT) of the brain revealed a small SAH extending into the Sylvian fissure. Cerebral angiography further revealed an aneurysm in the right anterior cerebral artery. Cardiology assessment was requested in light of the ECG changes consistent with prior infero-lateral myocardial infarction. Echocardiography revealed good left ventricular systolic function with an ejection fraction of 60%. There were no regional wall motion abnormalities (RWMA). Neither of the ventricles was dilated and the valves were considered normal. Within 48 hours a repeat ECG showed normalising T waves and a reduction in the size of the Q waves in leads V5 and V6. Surgery proceeded as planned, with unremarkable intra- and postoperative courses. An ECG five days after surgery (Figure 2) revealed no Q waves and normal T waves in leads V5 and V6. T waves in lead II were noted to be normalising, however Q waves and inverted T waves persisted in standard leads III and aVF. The patient was discharged with no residual neurological or cardio-respiratory sequelae.

Discussion

Systemic organ dysfunction occurring in SAH is not infrequent and has significant impact on morbidity and mortality accounting for 23–42% of all deaths.[1] The mechanisms by which this dysfunction is manifested remain poorly understood. Current hypotheses suggest that the neurological insult precipitates a neuroendocrine and autonomic dis-equilibrium with resultant widespread inflammatory and immunological signalling and sequelae.[1,3]

In 1947 the first documented relationship between SAH and the ECG began to appear in the literature.[1-3,5,7] Thus began the search for a correlation between ECG abnormalities and outcomes in an attempt to prognosticate and risk stratify such patients. The clinical importance of this is demonstrated in two separate case reports where patient management was directly affected by the over-consideration of significant ECG findings to the detriment of both patients.[3] In one case a patient presented
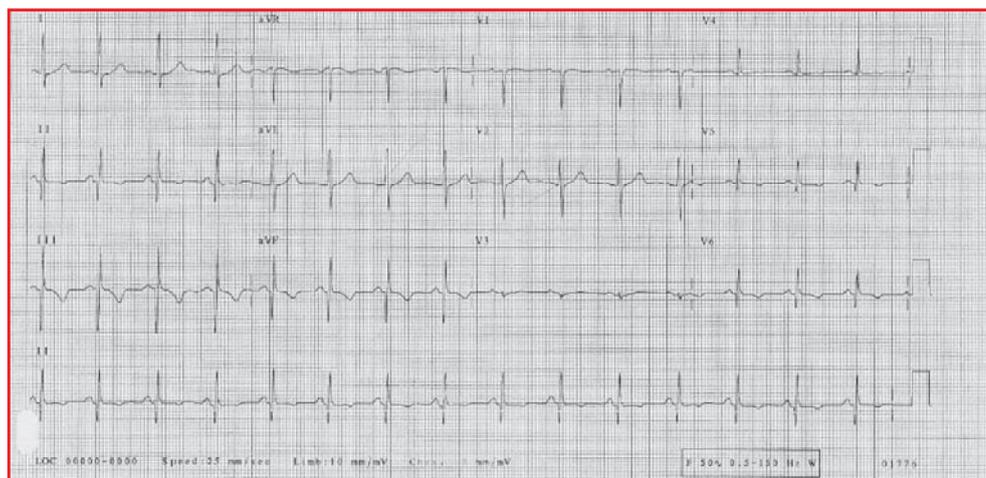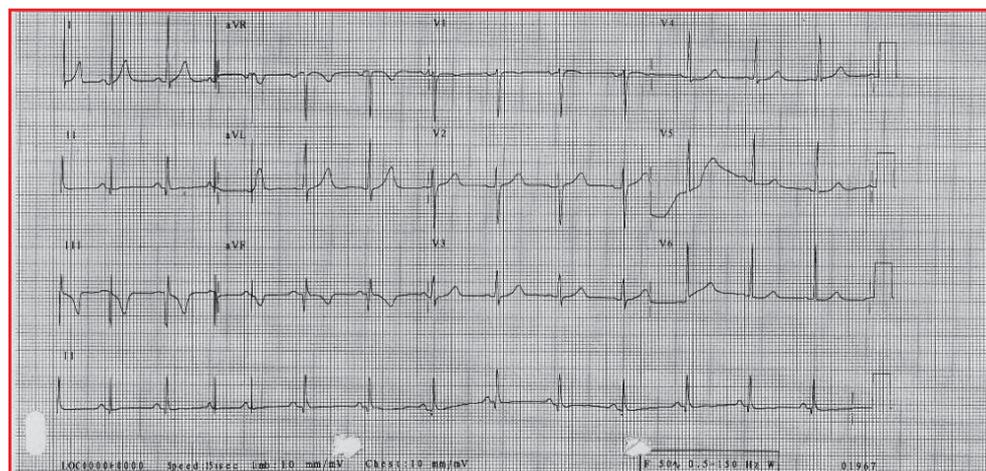
Figure 1: ECG on admission

Figure 2: ECG five days post surgery

and was treated for myocardial 'ischemia' on the basis of the ECG after presenting to hospital following a brief episode of collapse but no other neurological signs or symptoms. A neurological diagnosis was not even entertained. In another case a patient was diagnosed with SAH, but surgery was delayed pending treatment for a 'myocardial infarction'. Both cases had fatal outcomes and in both autopsy revealed cerebral aneurysms, SAH and normal hearts.[3] The 2009 American Heart Association guidelines on SAH cited the commonest reason for (the frequent) misdiagnosis (and consequently poorer outcome) to be the failure to obtain a non-contrast CT of the brain at presentation.[4] These reports, and other 'near misses' demonstrate that significant ECG changes have previously been instrumental in misdirecting the unsuspecting clinician and further confounding management decisions even after the diagnosis of SAH has been made.

ECG changes in SAH commonly reflect abnormalities of repola-risation and include ST segment changes, abnormal T wave morphology, QT interval prolongation (with antecedent risk of arrhythmia) and less frequently U waves.[1,3] There exists little documentation of Q wave abnormalities. A single case report from 1985 was found, describing the cancellation of surgery due to Q waves and T wave inversion consistent with previous inferior myocardial infarction. In this particular case no cardiac enzyme abnormalities were detected and nuclear heart scans were found to be normal. Surgery was performed with an uneventful intraoperative course. ECG changes were subsequently noted to have resolved five months after admission.[3]

Though extensively investigated, research has yet to produce definitive conclusions as to the significance or expected duration of ECG abnormalities in SAH. It has been noted that such changes occur most frequently within the first 72 hours after the insult and steadily resolve with the passage of time. In addition, the incidence of cardiovascular complications is noted to be related to the severity of the neurological insult.[2,3,5,6,8,9] Furthermore, there is little correlation between ECG abnormalities and the presence of underlying coronary artery disease (CAD) or myocardial injury.[1,3,9-12] Consequently, the interpretation of the ECG in light of SAH remains uncertain, and prognosticating on the basis of ECG abnormalities remains highly inaccurate.[1-3,5,6] However, cardiovascular complications in SAH are readily acknowledged and have been reported in 39–63% of patients with SAH.[1,2,9] A recent meta-analysis evaluating data from 1960 onwards and including over 2 600 patients has clearly demonstrated an association between increased mortality, poor outcome and delayed cerebral ischaemia in patients with ECG changes, cardiac enzyme abnormalities and echocardiographic evidence of myocardial dysfunction. Most notably, Q waves were associated with an increased mortality (relative risk 2.9).[8]

Other modalities of cardiovascular investigation have been found to demonstrate myocardial injury. Elevated troponins have been found to be associated with increased risk of cardiovascular complications and increased morbidity and mortality.[7-9,11,12] Occurring in as many as 20–40% of patients suffering SAH, troponin I has been found to be not only sensitive and specific but it is also an independent predictor for poor functional outcome.[8,9,11] Correlation has also been demonstrated between severity of neurological insult and degree of enzyme leak from the myocardium.[9,12] Most importantly though, cardiac enzyme assays are elevated despite exclusion of coronary artery disease or spasm.[1,9,11] Jeon et al go as far as to recommend that consideration be given to regular monitoring of troponin I for the first 72 hours following SAH in order to quantify and risk stratify potential cardio-respiratory sequlae.[9] Findings of elevated biomarkers in the face of an abnormal ECG may thus herald the need for further cardiac investigation.

Echocardiography has also been shown to reveal abnormalities (both systolic and diastolic dysfunction) in 10–31% of patients with SAH.[1] These are typically areas of regional wall motion abnormality and dilation of the left ventricle, which have been found to be reversible (usually within 5–10 days of insult), and have also been documented in patients sustaining other forms of neurological injury.[1,9,12] In this setting, inadequate ventricular function is termed a 'stunned myocardium' and significant CAD in such patients is unusual.[1,7] In fact, prospective evaluation of microvascular myocardial blood flow in patients with SAH utilising real-time perfusion contrast echocardiography has demonstrated normal blood flow even within the areas of RWMA.[10] No universally accepted explanation of the link between or significance of SAH and cardiovascular dysfunction currently exists.[2,7] Theories include genetic predisposition, catecholamine surge at the time of aneurysm rupture with resultant sympathetic 'storm', ischaemic injury secondary to coronary artery spasm, free radical related injury and significant hypercalcaemia.[7,9,10,12]Aetiologically, there are thought to be corresponding neural (autonomic mismatch) and humoral mechanisms responsible, with most studies now implicating catecholamine excess. Indeed, some literature report diminished

myocardial injury and reduced mortality with the use of beta-blockade during patient treatment. This was supported by rapid resolution of ECG changes and diminished myocyte necrosis at autopsy.[11] Typically, there is a conspicuous absence of significant CAD on autopsy specimens, though myocardial injuries have been noted, which include sub-endocardial haemorrhage, fibrosis and inflammatory cell in-filtrates.[1,9,11] This so-called 'neurogenic stress cardiomyopathy' can be compared to the 'transient left ventricle apical ballooning syndrome' (also known as Takotsubo Syndrome/Cardiomyopathy), which has been described in women exposed to a sudden emotional stress.[1,9,12] In a recent case report of an out-of-hospital cardiac arrest secondary to SAH induced Takotsubo cardiomyopathy, the diagnosis was once again nearly overlooked as the post-resuscitation ECG, cardiac enzymes and echocardiography were supportive of a diagnosis of an acute coronary syndrome. However, cerebral CT scanning revealed aneurysmal subarachnoid haemorrhage which responded to interventional radiological therapy with excellent clinical outcome.[13] Despite similarities in these conditions, and despite similarities found in the ECGs of patients with SAH and patients with Takotsubo cardiomyopathy, recent work done in Korea has demonstrated a poor correlation between ECG abnormalities in SAH and the presence of Takotsubo cardiomyopathy, further clouding the diagnostic significance of the ECG in SAH.[14]

Of importance to the anaesthesiologist is the understanding and awareness of the propensity for significant myocardial dysfunction in patients with SAH despite the apparent or even absolute lack of previous cardiovascular history. Furthermore, the presence of significant markers of cardiovascular injury has bearing on clinical course and has an association with a worsened clinical course and overall outcome. This needs to be counterbalanced against abnormal ECG findings suggesting myocardial dysfunction, but which are difficult to interpret, and ultimately may not have further bearing on the conduct of anaesthesia. This case not only highlights just such a scenario but further confuses the clinical picture by presenting ECG changes that could be considered uncommon, even for SAH. In the light of this, sufficient clinical suspicion or the presence of an abnormal ECG should prompt the consideration for further investigation including cardiac enzymes (specifically troponin I) and echocardiography to further define the presence and degree of myocardial dysfunction, which may have bearing on anaesthetic management, technique and overall morbidity, mortality and functional outcome.

References

1. Stevens RD, Nyquist PA. The systemic implications of aneurismal subarachnoid haemorrhage. J Neurol Sci 2007 Oct;261(1–2):143–56.
2. Macrea LM, Tramer MR, Walder B. Spontaneous subarachnoid haemorrhage and serious cardiopulmonary dysfunction – a systematic review. Resuscitation 2005 May;65(2):139–48.
3. Sommargren CE. Electrocardiographic abnormalities in patients with subarachnoid haemorrhage. Am J Crit Care 2002 Jan;11(1):48–56.
4. Bederson JB, Connolly ES Jr., Batjer HH, et al. Guidelines for the management of aneurismal subarachnoid haemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. Stroke 2009 Mar;40(3):994–1025.
5. Sakr YL, Ghosn I, Vincent JL. Cardiac manifestations after subarachnoid haemorrhage: a systematic review of the literature. Prog Cardiovasc Dis 2002 Jul–Aug;45(1):67–80.
6. Sakr YL, Lim N, Amaral ACKB, Ghosn I, et al. Relation of ECG changes to neurological outcome in patients with aneurismal subarachnoid haemorrhage. Int J Cardiol 2004 Sep;96(3):369–73.
7. Hamilton JC, Korn-Naveh L, Crago EA. Case studies in cardiac dysfunction after acute aneurismal subarachnoid haemorrhage. J Neurosci Nurs 2008 Oct;40(5):269–74, 317.
8. Van der Bilt IA, Hasan D, Vandertop WP, et al. Impact of cardiac complications on outcome after aneurismal subarachnoid haemorrhage: a meta-analysis. Neurology 2009 Feb;72(7):635–42.
9. Tung P, Kopelnik A, Banki N, et al. Predictors of neurocardiogenic injury after subarachnoid haemorrhage. Stroke 2004 Feb;35(2):548–51.
10. Abdelmoneim SS, Wijdicks EFM, Lee VH, et al. Real-time myocardial perfusion echocardiography and regional wall motion abnormalities after aneurismal subarachnoid haemorrhage. J Neurosurg 2009 Nov;111(5):1023–8.
11. Jeon IC, Chang CH, Choy BY, Kim, MS, Kim SW, Kim SH. Cardiac troponin I elevation in patients with aneurismal subarachnoid haemorrhage. J Korean Neurosurg Soc 2009 Aug;46(2):99–102.
12. Tanabe M, Crago EA, Suffoletto ES, et al. Relation of elevation in cardiac troponin I to clinical severity, cardiac dysfunction, and pulmonary congestion in patients with subarachnoid haemorrhage. Am J Cardiol 2008 Dec;102(11):1545–50.
13. Suzuki H, Sakuai M, Fujimoto M, Tsuchiya T, Sakaida H, Taki W. Complete recovery from aneurismal subarachnoid haemorrhage associated with out-of-hospital cardiopulmonary arrest. Eur J Emerg Med 2010 Feb;17(1):42–4.
14. Jung JH, Min PK, Rim SJ, Ha JW, Chung N, Lee KC. Are electrocardiographic changes in patients with acute subarachnoid haemorrhage associated with Takotsubo cardiomyopathy? Cardiology 2010 Jan;115(2):98–106.