CASE REPORT

Listening to the head and not the heart: subarachnoid haemorrhage associated with severe acute left ventricular failure

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SUMMARY

Headache is a very common presenting symptom in the emergency department, and distinguishing subarachnoid haemorrhage from more benign causes of headache can be challenging. This particular presentation of subarachnoid haemorrhage was made more difficult by concurrent-related cardiac pathophysiology. This case report describes the evolving differential diagnosis of a 54-year-old woman initially presenting with headache and confusion, with signs and investigations suggestive of ST elevation myocardial infarction and resultant left ventricular failure. The importance of prompt primary percutaneous coronary intervention left clinicians with a difficult decision about which specialty was most appropriate for the patient to be transferred to. Ultimately the symptoms were explained by radiographic confirmation of subarachnoid haemorrhage and subsequent adrenergic storm, causing myocyte injury and myocardial contractile dysfunction. This patient was transferred for coiling of a ruptured cerebral artery berry aneurysm. Her left ventricular failure improved from severe to mild within 48 h of presentation.

BACKGROUND

Subarachnoid haemorrhage carries significant morbidity and mortality. Twenty-five per cent of patients die within the first 24 h, a further 25% die within hospital and up to 50% of the survivors may have residual disability.¹

Historically some neurosurgeons have delayed surgery to overcome hostile operating conditions such as friable cerebral tissue and disordered cerebral autoregulation. However, with the advent of endovascular coiling current best practise has now moved towards urgent intervention within 3–4 days to prevent further cerebral ischaemia.² The intention is to minimise the risk of rebleeding, allow the removal of cisternal blood and permit hypertensive treatment strategies.

The guidelines for myocardial revascularisation in ST elevation myocardial infarction (STEMI) are well established. Patients presenting to hospitals without percutaneous coronary intervention (PCI) should be transferred to appropriate PCI facilities as soon as possible, with the intention of salvaging viable myocardium through reperfusion. Every effort should be made to keep the 'door-to-balloon time' below 90 min.³

Consequently there is considerable expectation to recognise and treat STEMI early to maximise patient outcomes. However as this case illustrates,

ST elevation and raised cardiac enzyme biomarkers should not always automatically warrant a transfer to PCI.

CASE PRESENTATION

A 54-year-old Ethiopian housewife presented to the emergency department with a headache. She had had a gradual onset of pain since the previous evening. This was frontal, constant and dull in nature. She could not localise it to left or right. There was no associated sensory disturbance. She felt it was similar to previous headaches which she got infrequently, approximately every 3–6 months. On further questioning she said that she often got these headaches at the end of the day. She had not been too concerned by this episode, and the pain was at worst 3/10 severity. She fell asleep that evening with the headache.

The following morning she was woken by a very severe headache, the worst she had ever experienced. The headache was exacerbated with coughing but not position. It was generalised across her whole head, and the pain radiated down her neck. She felt nauseous but had not vomited. Her husband found her to be confused, unable to recognise where she was or who she was with. She had been treated for a lower respiratory tract infection 2 weeks before with a course of antibiotics from her general practitioner. She had no chest pain, palpitations, dyspnoea or cough. Her medical history included hypothyroidism only, for which she was prescribed levothyroxine. There was no personal or familial history of migraine. She was a non-smoker, and there was no family history of cardiac disease.

On examination she appeared alert, with a Glasgow Coma Score of 15. Contrary to the husband's earlier evaluation, she was orientated to time place and person, and remained so throughout her time in the emergency department. Speech and language function were entirely normal. However, she was forgetful and easily distractible, often requiring repeated explanations about investigations and treatment plans. Her husband reported this was unusual. Her emotional state was appropriate. She was understandably anxious, and preoccupied by the pain of the headache.

There was no focal neurological deficit. Her headache did not worsen when lying flat. There was no neck stiffness. Funduscopy was normal: there was no papilloedema or evidence of retinal haemorrhage. Oxygen saturations on air were 96%, with a respiratory rate of 20. She talked in full

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sentences. She had fine bibasal crepitations. She was afebrile but tachycardic at 110 bpm. Her blood pressure was 110/70, with no discrepancy between arms. There were no added heart sounds. There was no pedal oedema. Her abdomen was soft and non-tender with no masses.

On the basis of the clinical history, the tachycardia and her relative hypotension given an otherwise hyperadrenergic state, an urgent CT brain was arranged. Prior to the CT scan, an ECG was performed and bloods were taken.

INVESTIGATIONS

The investigations below are listed in the order in which they were obtained and reported on.

- ▶ ECG (see figure 1)—sinus tachycardia. Isolated ST segment elevation in V2, with no reciprocal changes. QTc prolonged at 477 ms.
- ▶ Troponin I 2907 ng/mL 30 min after presentation (3800 ng/mL at 2 h). White cell count 16×10^9 and C reactive protein of 6.2 mg/L. Haemoglobin 12.7 g/dL. Renal function within normal limits.
- ▶ Arterial blood gas—on air—partial pressure of oxygen of 6.7 Kpa (50.2 mm Hg), pH 7.45, partial pressure of carbon dioxide 3.9 Kpa (29.3 mm Hg).
- ▶ Chest X-ray (see figure 2)—extensive diffuse shadowing across both hemithoracies. Upper lobe vascular diversion and fluid within the horizontal fissure.
- ▶ Bedside echocardiogram—severely impaired left ventricular systolic function, with all mid-left ventricular segments akinetic, and reasonable contraction from basal and apical segments. Dilated left atrium and ventricle. Pulmonary artery pressures of approximately 30 mm Hg. No pericardial effusion and no evidence of aortic dissection.
- ▶ CT of the brain with contrast (see figure 3)—acute subarachnoid haemorrhage with blood in the sylvian fissures, quadrigeminal cistern, suprasellar cistern and fourth ventricle due to leakage from a 5 mm aneurysm at the right internal/middle carotid junction. No midline shift.

DIFFERENTIAL DIAGNOSIS

The diagnosis evolved considerably in this case.

With the history of a very severe headache suspicion was instantly raised of a subarachnoid haemorrhage. However, the gradual onset of the headache the evening before had been somewhat (falsely) reassuring. As it became apparent that the patient had been confused, and described a recent infection, other infective processes were considered. These included pneumonia, and central nervous system infection such as encephalitis. Her tachycardia and relative hypotension could be explained by sepsis.

The ECG changes raised suspicion of myocardial infarction, and it was for this reason that a troponin I was requested. However, the patient had neither chest pain nor any previous history of typical angina symptoms, and had no cardiac risk factors. While there was ST segment elevation greater than 2 mm, the ST change was isolated to lead V2 only. The ECG changes were insufficient to meet STEMI diagnostic criteria,⁴ as changes are required in at least two contiguous leads. There was therefore insufficient clinical suspicion to warrant immediate transfer of the patient for PCI. This decision was reinforced by the fact that myocardial infarction and subarachnoid haemorrhage have opposing treatment strategies. The former favours anticoagulation and antiplatelet agents, which are clearly contraindicated in the latter. These factors, together with a presenting

symptom of severe headache, led to a CT of the head being chosen over coronary angiography as the initial investigative imaging.

While the patient was in the CT department for her brain scan, the markedly raised troponin was reported. On returning from CT the patient had become more uncomfortable and breathless, perhaps as a result of having been lain flat. This was interpreted as left ventricular failure as a result of a septal STEMI, a working diagnosis supported by the findings of pulmonary oedema on the chest radiograph. In retrospect this distress may also have been due to raised intracranial pressure as a result of the supine position. Serial ECGs demonstrated no further evolution of the ischaemic change.

Ultimately the definitive diagnosis was made from the CT, demonstrating a subarachnoid haemorrhage following aneurysmal rupture. A bedside echocardiogram confirmed severe left ventricular failure, and it took reassurance from the neurosurgeons to explain that this was most likely neurogenic myocardial stunning, as opposed to a thrombotic cause.

TREATMENT

The patient required high-flow oxygen to maintain her saturations. Her Glasgow Coma Scale did not deteriorate, and there was no requirement for intubation. Her mean arterial pressure remained between 80 and 85 mm Hg. The neurosurgical team advised 60 mg of nimodipine to prevent vasospasm.

The patient was discussed with the cardiologists who advised that management of the intracranial haemorrhage took absolute priority over that of the heart. The patient did not receive any antiplatelet or other anticoagulant medication. Codeine, paracetamol and ibuprofen were used for analgesia. All three analgesics were prescribed prior to the radiographic discovery of her subarachnoid haemorrhage. In retrospect ibuprofen should have been avoided due to its antithrombotic properties.

She was transferred to the nearest neurosurgical facility where she underwent coiling of her intracranial aneurysm, after an angiography confirmed this was an isolated abnormality.

OUTCOME AND FOLLOW-UP

An echocardiogram performed 48 h after admission showed improvement of left ventricular failure from severe to mild. Further diagnostic myocardial imaging such as coronary angiography or stress echocardiography was not performed. It was felt unnecessary due to the absence of cardiac risk factors and pre-morbid symptoms such as angina. Her postoperatively stay on the intensive care unit was complicated by neurogenic pulmonary oedema, and she required a prolonged period of non-invasive ventilation and inotropic support. She is currently rehabilitating well, is autonomous in all her activities of daily living and independently ambulant.

DISCUSSION

It is well recognised that a subarachnoid haemorrhage can cause ECG changes including ST elevation and QT prolongation. Troponin rises have been seen in up to 20% of patients.⁵ Other neurological conditions can have similar effects, including Guillain-Barré syndrome, encephalitis, ischaemic stroke and subdural haemorrhage.⁶ Supportive therapy and correction of the underlying neurological problem typically reverse cardiac ischaemia within a number of days or weeks.⁷ Coronary angiography is invariably normal. The umbrella term ascribed in the literature to this syndrome has been neurogenic stunned myocardium. The exact aetiology is not known, but it is hypothesised that a hypothalamic hypersympathetic state and resultant

Figure 1 ECG—there is a borderline sinus tachycardia. Lead V2 shows 2.5 mm ST elevation, with no ST segment change in adjacent precordial leads, and no reciprocal changes. QTc duration is prolonged at 477 ms.

catecholamine surge may be responsible.⁶ Other theories include coronary artery vasospasm or microvascular dysfunction.

The echocardiographic appearances in this patient of left mid-ventricular segment akinesis and preserved apical contraction are typical of subarachnoid haemorrhage. This is seen in 60–70% of such patients with subarachnoid-induced left ventricular dysfunction.⁶ Stress-induced cardiomyopathy (also known as Takotsubo cardiomyopathy or broken heart syndrome) refers to transient systolic dysfunction of the apical and/or mid-segments, with no evidence of coronary artery disease on angiography. This is in response to an emotional stressor, and the same hyper-sympathetic state is believed to be responsible as for neurogenic stunned myocardium. Apical hypokinesis is more common in stress-induced cardiomyopathy (approximately 60% of cases)

compared with subarachnoid haemorrhage. Attempts exist to unify these two syndromes since despite these differences, they appear to share a similar neurologically driven mechanism.⁶

This patient had a normal neurological examination. This does not exclude subarachnoid haemorrhage as a diagnosis. The history of a ‘worst ever’ headache, so severe as to wake the patient, should be sufficient to raise suspicion to investigate for a subarachnoid bleed.

Figure 2 Chest X-ray—upper lobe vascular diversion (black arrows) and fluid within the horizontal fissure (white arrow).

Figure 3 CT contrast-enhanced axial image—acute subarachnoid haemorrhage with blood visible in the sylvian fissure. There is no midline shift. A 5 mm leaking aneurysm at the junction of the right internal and middle carotid arteries.

The diagnosis was made more difficult by the presence of a milder, insidious headache the evening before the patient's presentation. It might be tempting to label this as a sentinel headache. Debate exists as to the true incidence of 'sentinel' or 'warning' headache: defined as a previous episode of sudden severe headache with recovery before the presentation where a subarachnoid haemorrhage was found. These typically occur within the 3 weeks prior to definitive subarachnoid haemorrhage.⁸ A recent systematic review put the incidence of sentinel headache at between 10% and 43%.⁹ However, this patient's initial headache was neither severe nor sudden, nor was there a clear period of recovery between the evening and morning headaches. It cannot therefore be defined as a sentinel headache.

Learning points

- ▶ A subarachnoid haemorrhage can be associated with ischaemic ECG changes, raised cardiac enzyme biomarkers and impaired left ventricular contractile function.
- ▶ Treatment should be directed at correcting the underlying neurological condition, rather than interventional coronary procedures. Coronary investigations are not typically productive, demonstrating normal coronary arteries and serve only to delay the patient's time to meaningful neurosurgical intervention.
- ▶ Patients may experience a distinct sudden onset severe 'sentinel' headache with subsequent recovery, in the weeks prior to their presentation with subarachnoid haemorrhage.

Contributors CK managed the patient in the emergency department, and wrote this article.

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