CASE REPORT

Complete heart block as the presenting feature in subarachnoid haemorrhage

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Accepted 20 November 2017

SUMMARY

Cardiac manifestations of subarachnoid haemorrhage (SAH) are well-documented phenomena that can complicate the treatment of this devastating condition. Here, we present a case of SAH presenting as complete heart block on initial assessment, an extremely rare event.

A 53-year-old woman presented with a witnessed fall, sustaining a mild head injury. She denied any symptoms of SAH. Initial ECG revealed complete heart block, for which the patient was accepted under the cardiology team. For completion, a CT head scan was requested, this demonstrated significant SAH blood load in an aneurysmal rather than traumatic pattern. CT angiogram and subsequent digital subtraction angiography confirmed a posterior communicating artery aneurysm as the cause of the SAH. This case highlights the importance of considering neurological diagnoses in patients with collapse even with concomitant cardiac abnormalities, as the two are often inextricably linked.

BACKGROUND

Subarachnoid haemorrhage (SAH) is a severe and debilitating form of haemorrhagic stroke. It has a global incidence of roughly 9/100 000.¹ With a mortality rate of up to 10%–15% prior to reaching hospital and up to 40% within the first week of diagnosis,² early recognition is crucial.

Commonly, SAH produces prodromal symptoms, typically sudden onset severe ‘thunderclap’ headache alongside nausea, vomiting, focal neurological deficit, visual disturbances and meningism. However, it may also present as seizures or collapse. It is therefore vital to include SAH in the differential diagnosis of a collapse admission. Cardiogenic manifestations of SAH are very common with between 27% and 100% of patients presenting with ECG abnormalities on primary hospital presentation.³ In addition, up to 50% of patients with SAH may have echocardiographic abnormalities such as regional wall motion abnormalities and reduced contractility which may contribute to outcome.^{4 5} Clinically, these changes may manifest as myocardial infarction (MI), cardiac arrhythmia and congestive failure.⁶ However, these features often develop after a diagnosis of SAH is made.

With our patient, the primary sign of SAH being an isolated heart block without cardinal SAH symptoms is extremely rare and, to the best of our knowledge, this is the only documented case. With this being the situation, SAH should be considered

in the list of differentials in the initial presentation of isolated cardiac arrhythmias.

With a reported 46% of patients suffering from cognitive/functional impairment as a direct result of their SAH,⁷ rapid recognition is essential not only to survival chances but also to quality of life.

CASE PRESENTATION

A 53-year-old woman, who is otherwise fit and well, suffered a fall resulting in a mild traumatic head injury while out with her husband. According to witnesses, the patient tripped over and hit her head on a metal railing before falling to the floor. She sustained a brief episode of loss of consciousness from which she was easily roused. An ambulance was called as the patient was talking ‘nonsense’ immediately after the fall for a number of minutes. This had recovered completely by the time the ambulance had arrived about 20 min later. In the ambulance, the patient was noted to have a Glasgow Coma Scale (GCS) of 15, with no headache, neurological deficit, nausea, meningism or other significant symptoms. The patient was unable to recall the exact events causing the fall but was otherwise orientated to person, place and time. ECG at this point revealed complete heart block at a rate of 40 bpm. The patient was given 500 µg intravenous atropine in the ambulance just prior to reaching the emergency department.

On arrival in accident and emergency, the patient’s ECG revealed sinus rhythm at a rate of 80 bpm. At this point, the patient denied any symptoms associated with SAH and on examination was GCS 15 and neurologically intact. The patient was referred and accepted by the cardiology team for admission and observation. However, due to the mechanism of injury, the cardiology registrar organised a CT head before transfer to the ward to rule out a concomitant head injury that might require neuro-observations.

INVESTIGATIONS

Blood investigation revealed normal full blood count, renal function, clotting and glucose. CT head revealed a significant left-sided SAH in a typical aneurysmal rather than traumatic pattern. An emergent referral to neurosurgery was made at this point. This patient was clinically graded as a World Federation of Neurosurgery grade 1 and radiologically graded as Fisher grade 3 (see [figure 1](#)). The neurosurgical team organised a CT angiography which demonstrated a 2.7×2.9 mm left-sided posterior

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To cite: Pepper J, Fenton C, Brydon H. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-222423

Figure 1 Plain CT scan showing subarachnoid haemorrhage.

communicating (PComm) artery aneurysm (see [figure 2](#)), which was the source of the SAH. Digital subtraction angiography of the cerebral vessels confirmed the presence of a PComm aneurysm as the likely source of the SAH.

TREATMENT

Within 24 hours, the patient, under general anaesthetic, underwent endovascular coiling of the aneurysm with satisfactory occlusion of the vascular lesion to prevent the risk of aneurysmal rehaemorrhage. After coiling, the patient was admitted to the intensive care unit (ICU) for close monitoring of mean arterial

Figure 2 CT angiogram three-dimensional rendering showing a left-sided 2.7x2.9 mm posterior communicating aneurysm.

Figure 3 Plain CT head at day 8 showing resolution of the subarachnoid blood.

blood pressure, neurological status and GCS. In the ICU, the patient was started on intravenous metaraminol infusion (10 mg/mL) at a rate of 3 mL/hour with a target systolic blood pressure of 150 mm Hg. Intravenous fluids were started with a target fluid input of 3 L to ensure euvolaemia. This was done to prevent the risk of arterial vasospasm. During the intensive therapy unit admission, the patient did not have any focal neurological deficits. The patient was weaned off metaraminol over 5 days, and when maintaining normotension, she was transferred to a neurosurgery ward. In addition, on admission to hospital, she was started on nimodipine 60 mg four times a day. This was done as per standard postaneurysmal SAH management.⁸ On day 8, the patient was noted to be confused, and a CT head scan was organised to rule out a cerebral infarct, hydrocephalus and aneurysmal rebleed. This CT scan was unremarkable and demonstrated reducing and redistributed subarachnoid blood load (see [figure 3](#)). Laboratory blood investigations and urine dipstick were normal, and this clinical state improved spontaneously over 24 hours. The patient was observed for a period of 11 days in total. This period of in-hospital observation is normally conducted as it corresponds to the clinical window during which arterial vasospasm may occur.⁸ It also serves as a useful period to observe for worsening headaches which may be a symptom of a developing communicating hydrocephalus.

OUTCOME AND FOLLOW-UP

On discharge, the patient was GCS 15, neurologically intact with minimal and improving headache. There were no further cardiac concerns; specifically, there were no episodes of heart block or cardiac anomalies identified during general anaesthesia or while in the ICU, and no cardiac concerns were noted while the patient was on the neurosurgery ward. The patient was discharged on nimodipine 60 mg four times a day for a total course of 21 days. When seen in clinic 3 months after the event, the patient remains independent and complained of occasional but improving

Figure 4 MR angiogram at 6 months showing occlusion of the left posterior communicating aneurysm.

headaches. This would equate to a Modified Rankin Scale score of 1 (no significant disability despite symptoms) and a Glasgow outcome scale of 5 (good recovery). Follow-up MR angiogram 6 months from ictus demonstrated complete occlusion of the aneurysmal defect (figure 4).

DISCUSSION

Cardiac manifestations of SAH without obvious neurological features could lead to misdiagnosis. Moreover, the presence of cardiac abnormalities in a patient with a primary cerebral diagnosis may represent a prediction of poor outcome.⁹ Therefore, awareness of the cardiac changes as a result of SAH is crucial to reduce patient morbidity and mortality. Although patients may not present initially with a life-threatening arrhythmia, these can progress to life-threatening changes such as sinoatrial heart blocks and atrioventricular arrhythmias.³ A variety of cardiac pathology, including MI, arrhythmia and congestive heart failure, is seen in patients with SAH and contributes to an overall mortality of 23%.⁶

Additionally, SAH with cardiac involvement increases the risk of cardiovascular comorbidities, undesirable post-SAH outcomes and lengthened ICU stays.¹⁰ This may be because cardiac dysfunction results in a reduction in cerebral blood flow and increase in time-to-peak, causing cerebral perfusion deficits.¹¹

Takotsubo cardiomyopathy (TCM) is a phenomenon of transient dyskinesia of the left mid-ventricular segment and may occur in up to 10% of patients presenting with an SAH.^{12 13} It is more common in patients with poor clinical grade (World Federation of Neurological Societies (WFNS) grade 4–5) SAH and when present may increase the mortality rate by up to 7%.¹⁴ The pathology of this entity is not well understood but is believed to be related to catecholamine surges. Therefore, it is important that further surges caused by haemorrhage are avoided, and aneurysm treatment strategies should not be delayed because of the presence of TCM.¹³ Importantly, TCM secondary to SAH has been reported to present as an acute ST elevation MI in a collapsed patient resulting in mistreatment and diagnostic delay.¹⁵

Indeed, TCM is believed to be related to another cardiac phenomenon, neurogenic stunned myocardium (NSM). NSM is characterised by reversible left ventricular dysfunction and is hypothesised to result from a combination of hypothalamic

dysfunction, catecholamine surges and myocardial perivascular lesions.¹⁶ There is no clear definition of diagnosis, but it resolves around severe cardiac dysfunction which may include elevation of troponin, ejection fraction <55% or significant ECG abnormalities¹⁷ and results in higher mortality and morbidity rates¹⁸ in patients after SAH. There is no specific therapy, and these patients undergo cardiorespiratory support measures until the cardiac dysfunction improves. They should also undergo a coronary angiogram to rule out occlusive disease. These patients will often have troponin I elevation (0.1–1.0 ng/mL and >1.0 ng/mL, respectively) which is also related to acute diastolic dysfunction and pulmonary congestion.¹⁹ Additionally, patients presenting with MI on ECG with troponin >1.0 µg/L had a 10-fold increase in mortality than those without.⁶ This is indicative of poorer neurological function and more severe cardiac dysfunction.

With troponin elevation directly correlated to MI in conjunction with the previously discussed surge in adrenergic compounds postaneurysmal rupture, it is unsurprising that cardiac abnormalities may delay a diagnosis of SAH.^{20–23}

Pathophysiology

The existence of cerebral–cardiac interactions in cerebral injury is well acknowledged; however, the intricacies of this are not well understood. The widely accepted hypothesis is that hypothalamic dysregulation combined with adrenergic releases (attributable to sympathovagal changes) disrupts cardiac control. With the presumption that first degree and type 1 second degree heart block are the manifestations of high vagal tone, it is unsurprising that these are commonly associated with SAH. However, complete heart block is believed to be attributable to a structural abnormality of the His–Purkinje system, and so the interaction of this with cerebral injury is poorly understood.

Cropp and Manning postulate that there are vagally controlled cardiac representations in the frontal lobe²⁴; however, this theory has been overshadowed by the consensus that hypothalamic dysregulation³ (potentially of sympathetic/parasympathetic tone)²⁵ is responsible for cerebral-attributable ECG changes. Other publications suggest that hypothalamic lesions (resulting from haemorrhagic vasospasm) cause cell-damaging and arrhythmia-producing catecholamine surges.^{3 26} Additionally, myocardial injury resulting from intracellular potassium changes²⁷ and autonomic dysfunction due to SAH (with subsequent subendocardial necrosis)²⁸ could also contribute to ECG abnormalities. This pathophysiology explains the common cardiac manifestations seen in SAH, such as P-wave peaking, P–R interval changes, ST elevation/depression, QT interval length changes and pathological Q-waves,²⁹ but does not adequately explain complete heart block.

CONCLUSION

One of the few ways in which patient mortality can be improved is by earlier recognition and treatment of SAH. As well as this, awareness of a patient's predisposing risk factors such as

Learning points

- ▶ Subarachnoid haemorrhage (SAH) should always be considered in the differential diagnosis of patients with collapse.
- ▶ Cardiac abnormalities occur commonly in patients with SAH.
- ▶ Prompt diagnosis of SAH is vital to prevent adverse outcomes.

genetics, hyperlipidaemia, hypertension and diabetes could aid in faster recognition of a patient's likelihood of having an SAH versus a primary cardiac disease. Most importantly, clinicians should always consider a primary neurological diagnosis in cases of collapse attributed to cardiac abnormalities as the two are intimately linked.

Contributors JP selected the case, wrote the case report with CF, corrected the drafts and additional write-up, revised the manuscript as per reviewer's comments and provided a point-by-point response to reviewers. CF wrote the manuscript draft under guidance and researched the paper. HB corrected the drafts, gave final approval and provided support with background research.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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