Predictors of LV dysfunction in patients with subarachnoid haemorrhage

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Background and Objectives

Subarachnoid haemorrhage (SAH) is known to be associated with biochemical and echocardiographic evidence of cardiac injury. Studies have shown that regional wall motion abnormalities can be seen in 13 - 31% of patients with SAH, and elevated troponin can be seen in 21 - 50%1. The presence of wall motion abnormalities caused by SAH has also been shown to correlate with poor outcome and death, possibly by contributing to the occurrence of delayed cerebral ischaemia2.

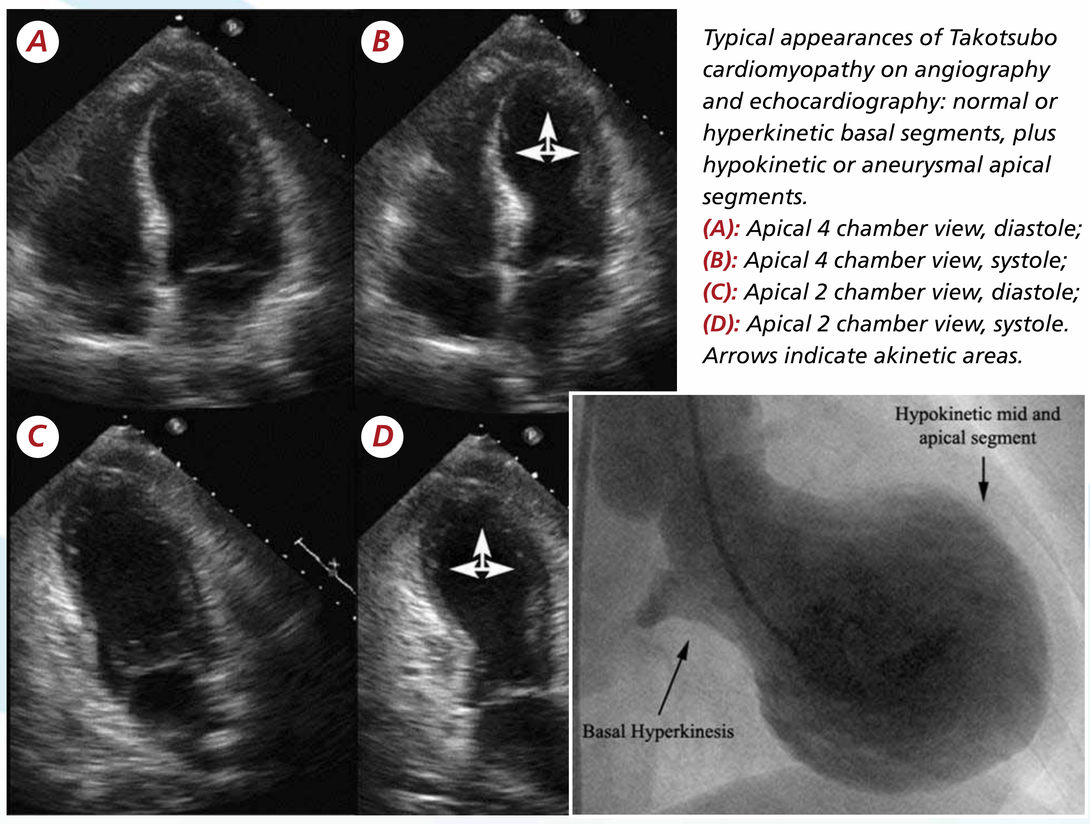
The presence of a simple marker of increased risk of poor outcome may help in informing whether patients with SAH should be cared for in a critical care setting. As a result, we set out to investigate the presence of left ventricular dysfunction in our local SAH population, and assess the degree of correlation between this abnormality and a number of other physiological and biochemical variables.

Methods

Two tertiary neurosciences centres in North West England participated in this retrospective observational study. We identified all patients presenting with SAH between April 2012 and April 2013 who had been investigated with echocardiography as part of their routine care. Microsoft Excel was used to collect data on the following variables for each patient:

- Presence of echocardiographic abnormalities: Takutsubo cardiomyopathy (diagnosed using the Modified Mayo Clinic criteria), regional wall motion abnormalities or reduced left ventricular ejection fraction
- Age
- Aex
- Ethnicity
- Medications
- Sodium
- Comorbidities
- Creatinine
- Troponin I
- Haemoglobin
- Type of aneurysm
- Fisher grade
- Admission ICP and GCS,
- Presence of hydrocephalus
- Need for vasopressors
- Need for ventilation
- Mortality

Multiple regression analysis was carried out using StatsDirect in order to assess for correlation between each variable.



Typical appearances of Takotsubo cardiomyopathy on angiography and echocardiography: normal or hyperkinetic basal segments, plus hypokinetic or aneurysmal apical segments.
(A): Apical 4 chamber view, diastole;
(B): Apical 4 chamber view, systole;
(C): Apical 2 chamber view, diastole;
(D): Apical 2 chamber view, systole.
Arrows indicate akinetic areas.

Results

31 patients were identified as having had echocardiography following their SAH, with a median age of 55 years. Of these, 24 had abnormal echocardiograms: seven had evidence of Takutsubo cardiomyopathy and 17 had left ventricular dysfunction characterised by either reduced ejection fraction or regional wall motion abnormalities. 19 patients had an elevated troponin; of these only two had a normal echocardiogram. Multiple regression analysis identified presenting Glasgow Coma Score (P=0.004) and elevated troponin (P=0.028) as being the only variables that were associated with either LV dysfunction or Takutsubo cardiomyopathy in this cohort of patients.

Discussion

This small study confirms the results of previous investigations that have shown a correlation between the presence of elevated troponin or low presenting GCS and either biochemical or echocardiographic evidence of cardiac injury3,4. The proposed mechanism of cardiac injury following SAH is one of neurologically mediated massive catecholamine release occurring at the time of ictus. It has been proposed that the correlation between poor neurological grade and increasing severity of cardiac injury may be due to the fact that SAH tends to cause direct release of catecholamines from cardiac sympathetic terminals, rather than a systemic catecholamine release3.

The study is limited by the fact a relatively small number of patients were identified as having had echocardiography following their SAH. This may represent a shortcoming in the investigation of our population of patients suffering SAH. Furthermore, not all patients presenting with SAH had their serum troponin measured, leading to the possibility that some patients who had cardiac injury were not identified.

Given recent evidence relating presence of regional wall motion abnormalities to delayed cerebral ischaemia and poor outcome in SAH2, it may be prudent to screen for elevated troponin in all patients presenting with SAH. Whilst patients with a poor neurological grade at presentation are automatically cared for on a neurocritical care unit, those with a good grade but elevated troponin may benefit from close observation and invasive monitoring so that any deterioration may be aggressively treated at the earliest opportunity.

References

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