A Review of Neurogenic Cardiomyopathy in Subarachnoid Hemorrhage and the Prognostic Implications of Cardiac Biomarkers

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INTRODUCTION

The incidence of subarachnoid hemorrhage (SAH) is approximately 2 to 22 per 100,000, with incidence being about twice as high in women than men [1]. Neurogenic cardiomyopathy has become a well known sequelae of SAH, occurring in approximately 20-40% of patients [2,4,6,7]. It is generally believed that sympathetic stimulation of the myocardium causes contraction band necrosis leading to the cardiomyopathy. ECG changes observable in 50-100% of patients, and detectable cardiac troponins observed in 20-40% of patients [2,4,6,7]. Left ventricle wall motion abnormalities (WMA) are detectable on echocardiograms in 20% of patients [2,3,5,7]. These cardiac changes have been associated pulmonary edema, hypotension requiring vasopressor support as well as delayed cerebral ischemia (DCI), a known cause of death in SAH, and longer ICU stay [2,3,5,8,9,10]. It is not well understood how the severity of cardiac biomarkers elevation correlates with clinical outcomes, however. The purpose of this review is to identify and summarize the incidence and prognostic implications of elevated cardiac biomarkers on SAH outcomes.

METHODS

A search was performed on Pubmed database for the keywords ‘aneurysmal subarachnoid hemorrhage’ AND ‘cardiomyopathy’ OR ‘troponin’ OR ‘BNP’ OR ‘ECG’ OR ‘echocardiography’ for the time period of 2001 to 2021. Studies among references in articles identified by this search were also screened for inclusion in the systematic review. A study was considered eligible if it was an English language article and reported quantitative data cardiac biomarkers (troponin, BNP, pro-BNP), ECG, or echocardiogram with ejection fraction and wall motion abnormalities. Data were extracted about the incidence of cardiac complications following subarachnoid hemorrhage, qualitative measurement of severity of the cardiac complications (cardiac biomarkers, ECG, echocardiogram), clinical interventions, inpatient complications, and clinical outcomes.

RESULTS

This search yielded thirty-eight articles for review, including case reports, prospective and retrospective studies, and prior literature reviews. The parameters reported included EKG changes, BNP and troponin elevations over time, pulmonary artery catheter pressures, echocardiographic findings, and their relation to morbidity and mortality. These publications and their key findings were summarized in a collected table. Their results and findings are compared within related measured outcomes. Of the articles, 12 of the 38 focused on the relation between cardiac biomarkers, morbidity, and mortality. Of the 12, 10 articles showed elevated cardiac biomarkers to correlate with increased morbidity and mortality

Summary of clinical studies and reviews evaluating the prevalence of cardiac biomarkers and prognostic impact in aneurysmal SAH	

Naidech, A. M., et. al. (2005). Cardiac troponin elevation, cardiovascular morbidity, and outcome after subarachnoid hemorrhage. Circulation, 112(18), 2851–2856.	• cTnI reached peak around day 3 from SAH onset
	• Peak cTnI was significantly associated w/ a significantly increased risk of abnormal LV wall motion on echo, pulmonary edema, hypotension treated with pressors, Delayed cerebral ischemia (DCI) from vasospasm, and cerebral infarction from any cause
	• Peak cTnI lvl:
	  ○ >0.5 mcg/L → 50% risk for DCI
	  ○ >2.0 mcg/L → 30% risk for Pulm Edema
	  ○ >10.0 mcg/L → 40% risk for developing hypotension
	• cTnI elevation was significantly associated w/ an increased likelihood of death or severe disability at discharge
	• cTnI elevation was associated w/ increased hospital stay (mean: 22.9d)

Schuiling, W. J. et. al. (2006). ECG abnormalities in predicting secondary cerebral ischemia after subarachnoid haemorrhage. Acta neurochirurgica, 148(8), 853–858.	• Only ST segment depression predicted the occurrence of DCI • ST depression and ischemic ECG abnormalities (presence of ST depression or TWI, or both in at least 2 leads) appeared independent predictors of poor outcome
van der Bilt, I. A. et. al. (2009). Impact of cardiac complications on outcome after aneurysmal subarachnoid hemorrhage: a meta-analysis. Neurology, 72(7), 635–642.	• Wall motion abnormalities (WMAs), elevated troponin, and proBNP lvls, tachycardia, and Q aves, ST depression, and T wave abnormalities were significantly associated w/ an increased risk of death • WMAs, elevated troponin/CK-MB/proBNP lvls, ST-segment depression were significantly associated w/ an increase risk of the development of DCI • Elevated troponin and CK-MB lvls, ST-depression were significantly associated w/ poor outcome
Murthy, S. B. et. al.. (2015). Neurogenic Stunned Myocardium Following Acute Subarachnoid Hemorrhage: Pathophysiology and Practical Considerations. Journal of intensive care medicine, 30(6), 318–325.	• Recent studies have shown that ST-T changes and QTc prolongation have no correlation with the development of delayed cerebral ischemia, raised intracranial pressure, or mortality • A small retrospective study observed that cTI elevation was significant associated with vasospasm and mortality, after adjusting for admission Hunt-Hess grade, age, and aneurysm size • Patients having elevated cTI are more likely to have severe vasospasm
Akkermans, A. et. al. (2019). Cardiac events within one year after a subarachnoid haemorrhage: The predictive value of troponin elevation after aneurysm occlusion. European journal of preventive cardiology, 26(4), 420–428.	• Troponin I elevation after occlusion of a ruptured intracranial aneurysm predicts the occurrence of a major adverse cardiac event within one year after ASAH, as measured by MACE.
Zahid, T. et. al. (2020). Cardiac Troponin Elevation and Outcome in Subarachnoid Hemorrhage. Cureus, 12(8), e9792.	• Cardiac troponin elevation post-SAH is associated w/ many systemic complications, poor outcomes and increase mortality • Elevated troponin and BNP predict worse outcomes after SAH • Cardiac dysfunction defined by myocardial WMAs or positive troponin after SAH is also linked to decreased focal and global cerebral perfusion • WMAs are risk factor for poor clinical outcome after SAH

CONCLUSION

The relevance of elevated cardiac biomarkers following an aneurysmal SAH impact on morbidity and mortality is an under-researched topic, and warrants further investigation and development of standardized guidelines. A trend exists between the amount of cardiac biomarker elevation and poor outcomes, however there exist no guidelines aimed at monitoring and managing cardiac complications.

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