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## Troponin elevation in subarachnoid hemorrhage

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### ABSTRACT

Troponin (tr) elevation in aneurysmal subarachnoid hemorrhage (SAH) patients is often difficult to be appropriately assessed by clinicians, causing even disagreements regarding its management between neurosurgeons and cardiologists. The purpose of this article was to review the literature regarding the clinical interpretation of tr elevation in SAH. We searched for articles in PubMed using the key words: "troponin elevation" and "subarachnoid hemorrhage". All of them, as well as relative neurosurgical books, were used for this review. Some type of cardiovascular abnormality develops in most SAH patients. Neurogenic stunned myocardium is a frequent SAH complication, due to catecholamine surge which induces cardiac injury, as evidenced by increased serum tr levels, electrocardiographic (ECG) changes and cardiac wall motion abnormalities. Tr elevation, usually modest, is an early and specific marker for cardiac involvement after SAH and its levels peak about two days after SAH. Cardiac tr elevation predictors include poor clinical grade, intraventricular hemorrhage, loss of consciousness at ictus, global cerebral edema, female sex, large body surface area, lower systolic blood pressure, higher heart rate and prolonged Q–Tc interval. Elevated tr levels are associated with disability and death (especially tr >1 µg/L), worse neurological grade, systolic and diastolic cardiac dysfunction, pulmonary congestion, longer intensive care unit stay and incidence of vasospasm. Tr elevation is a common finding in SAH patients and constitutes a rightful cause of worry about the patients' cardiac function and prognosis. It should be therefore early detected, carefully monitored and appropriately managed by clinicians.

## 1. Introduction

A wide variety of cerebral pathology, from acute ischaemic stroke[1], to the commonest and most important of the group, subarachnoid hemorrhage (SAH), or even rare entities such as transient global amnesia[2], has been associated with transient cardiac dysfunction with electrocardiographic (ECG) changes and simultaneous release of cardiac biomarkers[3]. These two findings, as well as left ventricular systolic dysfunction, constitute cardiac abnormalities that have been reported after SAH[4]. Cardiopulmonary

complications after aneurysmal SAH negatively affect overall morbidity and mortality and have been linked to worsened clinical outcome, suggesting a role for cardiac monitoring and interventions[5].

Although Cardiac tr elevation, in addition to ECG changes or alone, considered as an indicator of cardiac complications in SAH, it proves usually to be a misleading finding in the emergency department in the process of diagnosing the underlying disease. Thus tr elevation in SAH patients is often difficult to be appropriately assessed by clinicians, causing even disagreements regarding its management between neurosurgeons and cardiologists. But is this laboratory finding a rightful cause of worry about the patients' cardiac function? Does it affect their prognosis and how? Approaching answers to these questions, purpose of this article was to review the literature regarding the

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clinical interpretation of tr elevation in SAH.

We systematically reviewed the literature regarding tr elevation in patients with SAH. We searched for articles in PubMed using the key words: “troponin elevation” and “subarachnoid hemorrhage”, which revealed 30 articles. All of them, as well as relative neurosurgical books, were used for this review. The available literature was mostly about cardiac complications following SAH. We classified the data we collected into two main categories; the tr elevation is caused by cardiac complications of SAH and the prognostic value of this elevation.

## 2. Cardiac complications of SAH

Cardiac alterations associated with SAH have been recognized and frequently reported[6]. Some type of cardiovascular abnormality develops in most patients with SAH. Hypertension and hypotension are common (27% and 18% respectively). Other cardiovascular events include life-threatening arrhythmias (8%), myocardial ischemia (6%) and successful resuscitation from cardiac arrest (4%)[7].

SAH may be associated with ECG changes in over 50% of cases, occasionally producing ECG abnormalities indistinguishable from an acute myocardial infarct[6]. Arrhythmias also occur in about one-third of patients after SAH[5].

More specifically, SAH may cause ECG changes that include ST segment elevation in lateral leads, ST segment depression in inferior leads and T wave inversion[8,9]. In the study of Sommargren *et al.*[10], repolarization abnormalities occurred in 41% of SAH patients. These included prolonged QTc interval (>460 ms) in 16%, ST segment elevation in 9%, ST depression in 3%, T wave inversion in 7% and U wave ( $\geq 100 \mu\text{V}$ ) in 15%. ECG criteria for left ventricular hypertrophy were met in 14% and 43% of those patients who had no history of hypertension. They interestingly reported that prolonged QTc interval after SAH is significantly related to myocardial injury[10].

SAH can cause transient abnormal left ventricular wall motion with echocardiographic findings of diffuse or localized left ventricular hypokinesis around the apical area (Takotsubo-like cardiomyopathy), but with negative coronary angiography[8]. Certain echocardiographic criteria for the diagnosis of apical ballooning syndrome as a result of SAH (or other cerebral pathology) have been established (such as transient akinesis or dyskinesis of the central left ventricle with or without apex involvement extending over the region of vascularisation of more than one coronary artery). However, its diagnosis in the emergency department remains a challenge largely due to the lack of significantly

constricted coronary vessels or angiographic features of atheromatous plaque rupture, together with ECG changes (ST segment elevation or negative T waves) and slightly increased concentrations of cardiac tr[11–13].

## 3. Underlying pathophysiological mechanisms

The mechanisms of cardiac dysfunction after SAH used to be controversial until recently[4]. Historically, cardiac necrosis after SAH had been attributed to coronary artery disease, coronary vasospasm or oxygen supply–demand mismatch. Experimental evidence, however, indicated that excessive release of norepinephrine from the myocardial sympathetic nerves was the most likely cause[14].

Nowadays we know that acute left ventricular dysfunction associated with SAH can be the expression of a stress-related cardiomyopathy[15], (“catecholamine hypothesis” of neurogenic cardiac stunning causing non-ischemic myocardial injury[16]). Neurogenic stunned myocardium (also known as apical ballooning syndrome or Takotsubo syndrome) is a frequent complication of aneurysmal SAH, with a significant impact on disease course. The presumed cause is catecholamine surge at the time of aneurysm rupture[17]. A possible mechanism is that hypothalamic ischemia causes increased sympathetic tone and resultant catecholamine surge producing subendocardial ischemia or coronary artery vasospasm[18].

So SAH-related cardiac complications have been attributed to catecholamin toxicity that could be triggered by physical or psychological stress. This mechanism is similar to acute myocardial infarction and this is probably the reason of both ECG abnormalities and tr release but with a different pathological background[3,19]. Soon after the event of SAH, a generalized sympathetic response is often encountered, which has profound effects on the cardiopulmonary system as well as the systemic vasculature. The massive surge of catecholamines after SAH likely induces various degrees of cardiac injury, as evidenced by increased serum tr T levels, ECG changes and sometimes severe cardiac wall motion abnormalities[20].

## 4. Tr elevation due to cardiac complications of SAH

As previously mentioned, SAH frequently results in myocardial necrosis with release of cardiac enzymes[14]. Cardiac tr I release occurs frequently after SAH and has been associated with a neurogenic form of myocardial injury[21]. SAH can also cause tr T elevation accompanied by transient abnormal left ventricular wall motion[8,20]. Cardiac injury,

evidenced by an elevation in tr levels, is reported in about one-third of patients after aneurysmal SAH[5]. Elevated tr I has been reported in up to 68% of SAH patients[7]. Tr elevation, usually modest, is an early and specific marker for cardiac involvement after SAH[22,23], and its levels peak about two days after SAH[7].

In the study of Kothavale *et al.*[4], among SAH patients with a peak cardiac tr I level  $>1.0 \mu\text{g/L}$ , 65% had regional wall motion abnormalities[4]. Concentration of cardiac tr  $<2.8 \mu\text{g/L}$  in patients with ejection fraction  $<40\%$  is characteristic for stunned myocardium[11].

Lee *et al.*[16], reported a case that a SAH patient with normal myocardium perfusion (in the setting of normal coronary arteries on coronary angiogram) and midventricular akinetic segments, which is compatible with non-ischemic injury. The patient developed transient ST segment elevation on lateral ECG leads and elevated cardiac enzymes with creatine-kinase MB isoenzyme of  $44.3 \text{ ng/mL}$  and tr of  $0.62 \text{ ng/mL}$ [16].

Admission predictors of cardiac tr I elevation include poor clinical grade, intraventricular hemorrhage, loss of consciousness at ictus, global cerebral edema and a composite score of physiological derangement[21]. Tung *et al.*[14], found that a Hunt-Hess score  $>2$ , female sex, larger body surface area, left ventricular mass, lower systolic blood pressure, higher heart rate and phenylephrine dose are independent predictors of tr I elevation ( $>1.0 \mu\text{g/L}$ ). They also found that the degree of neurological injury as measured by the Hunt-Hess grade is a strong, independent predictor of myocardial necrosis after SAH, supporting the hypothesis that cardiac injury after SAH is a neurally mediated process[14]. Moreover, Sommargren *et al.*[10], found that serum cardiac tr I was elevated in 21% SAH patients with abnormalities on the initial ECG. Controlling for gender, those with QTc interval  $>460 \text{ ms}$  were 5.5 times more likely to have elevated serum cardiac tr I[10]. Tr elevation predictors in aneurysmal SAH patients are poor clinical grade, intraventricular hemorrhage, global cerebral edema, female sex, large body surface area, left ventricular mass, lower systolic blood pressure, higher heart rate, prolonged qtc interval, composite score of physiological derangement and phenylephrine dose.

Furthermore, levels of troponin Ic (and Serum B-type natriuretic peptide) are correlated and seem to be more sensitive to cardiac stress occurring in SAH as compared with Doppler variables of diastolic function[24].

Interestingly, cardiac tr I (and creatine kinase MB) levels in blood (and pericardial fluid) show also a mild and gradual postmortem time-dependent elevation, larger for specific causes of death including cerebrovascular diseases[25].

Of course, regarding the clinical management of SAH

patients, early detection of cardiac involvement, through ECG abnormalities and tr elevation, and monitoring of these changes, as well as direct approach and medical intervention, holds an important role in amelioration of patient outcome[26].

## 5. Tr elevation as a prognostic factor in SAH

The prognostic significance and clinical impact of cardiac tr I elevation used to be poorly defined until recently[21]. Several studies have been published concerning the use of tr levels in SAH as a predictor of outcome. Most of these studies have associated higher levels of tr with poor outcome[22]. Nowadays we know that cardiac tr I is a good indicator not only for cardiopulmonary complications but also for neurological complications[27], and outcome following SAH[22,27]. More specifically, elevated tr levels are associated with disability and death after SAH, as well as worse neurological grade, systolic and diastolic cardiac dysfunction and pulmonary congestion[7].

A cardiac tr I level  $>1.0 \mu\text{g/L}$  is independent predictor for the development of regional wall motion abnormalities, which are, as already mentioned, frequent after SAH[4]. Thus left ventricular wall motion abnormalities seen on echocardiography are more common in patients with elevated tr[7].

According to some studies, cardiac tr I elevation is also related with variable clinical factors such as poor Glasgow Outcome Scale score, profound pulmonary complication, higher heart rate during initial three days following SAH and even incidence of vasospasm and duration of hyperdynamic therapy for vasospasm. So this mark could contribute to the management of SAH and treatment of concomitant complications[27].

Tanabe *et al.*[28] studied the levels of tr and associated outcomes in SAH patients. Highly positive cardiac tr I ( $>1.0 \text{ ng/mL}$ ) was associated with clinical neurological severity, systolic and diastolic cardiac dysfunction, pulmonary congestion and longer intensive care unit stay. Even mild increases in cardiac tr I ( $0.1\text{--}1.0 \text{ ng/mL}$ ) were associated with diastolic dysfunction and pulmonary congestion[28].

In the study of Naidech *et al.*[21] peak cardiac tr I level was associated with an increased risk of echocardiographic left ventricular dysfunction, pulmonary edema, hypotension requiring pressors and delayed cerebral ischemia from vasospasm. Peak cardiac tr I levels were predictive of death or severe disability at discharge after controlling for age, clinical grade, and aneurysm size but this association was no longer significant at 3 months. Cardiac tr I elevation after SAH was also associated with an increased risk of death or

poor functional outcome at discharge<sup>[21]</sup>.

In the same direction, Kumar *et al.*<sup>[23]</sup> showed that neurological outcome was adversely related to increase in tr levels and Ahmadian *et al.*<sup>[22]</sup> reported that associated levels of tr >1 µg/L are ten times higher risk of death. Interestingly, Ramappa *et al.*<sup>[29]</sup> found out that patients with SAH and elevated cardiac tr I usually have worse neurological status at admission, as well as a worse neurological outcome and in-hospital mortality<sup>[29]</sup>. As prognostic factors, clinical parameters are disability, mortality, worse neurological grade, systolic cardiac dysfunction, diastolic cardiac dysfunction, higher heart rate during initial three days following sah, pulmonary congestion, longer intensive care unit stay, incidence of vasospasm, duration of hyperdynamic therapy for vasospasm, and poor functional outcome at discharge from some published reports.

Additionally, Martini *et al.*<sup>[30]</sup> found that tr elevation >0.4 ng/mL was one of some important factors (such as age, Hunt–Hess and Fisher scores and mechanical ventilation at intensive care unit admission), which were adjusted with the usage of multivariable logistic regression to come to the conclusion that patients with SAH and early positive fluid balance had worse clinical presentation, prolonged length of hospital stay and greater resource use during the hospital course<sup>[30]</sup>.

On the other hand, other analyses failed to prove a predictive value of elevated tr in SAH patients. Gupte *et al.*<sup>[31]</sup>, for example, although associated high tr levels with higher in-hospital mortality, statistically significant difference was not proven when results were adjusted for age and Hunt–Hess and Fisher grades<sup>[31]</sup>. Furthermore, though Sanchez–Peña *et al.*<sup>[32]</sup>, initially observed that tr initial value was associated, among other factors, with poor 12-month outcome (namely Glasgow Outcome Scale 1–3), they concluded, after multivariate analysis, that only mean 15-day S100–B value significantly predicted outcome<sup>[32]</sup>. Finally, according to Pereira *et al.*<sup>[33]</sup>, only persistent intracranial pressure elevation and higher mean 8-day S100–B value significantly and independently predicted a poor outcome after one year<sup>[33]</sup>.

## 6. Conclusion

Neurogenic stunned myocardium is a frequent complication of aneurysmal SAH due to catecholamine surge and tr elevation which is a common finding in aneurysmal SAH patients. Answering our purpose's questions, we could say that this laboratory finding is a definitely rightful cause of worry about the patients' cardiac function and prognosis. More specifically, tr elevation,

usually modest, is an early and specific marker for cardiac involvement after SAH and its levels peak about two days after SAH. Predictors of cardiac tr I elevation include poor clinical grade, intraventricular hemorrhage, loss of consciousness at ictus, global cerebral edema, female sex, large body surface area, lower systolic blood pressure, higher heart rate and prolonged QTc interval. Elevated tr levels are associated with disability and death (especially tr >1 µg/L), worse neurological grade, systolic and diastolic cardiac dysfunction, pulmonary congestion, longer intensive care unit stay and even the incidence of vasospasm (an often devastating neurological consequence of aneurysmal SAH). Tr elevation should be therefore early detected, carefully monitored and appropriately managed by a multidisciplinary team of clinicians.

## Conflict of interest statement

The authors report no conflict of interest.

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