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Cardiac Complication Following Subarachnoid Hemorrhage

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ABSTRACT

Besides its severe neurological injuries, spontaneous subarachnoid hemorrhage (SAH) commonly causes cardiac complications. These complications could include three different aspects of cardiac diseases, that is, electrocardiographic abnormalities, myocardial injuries, and left ventricular dysfunction. These complications not only may lead to misdiagnosis of SAH as myocardial infarction, but also it may complicate the management of SAH. In this review, we described all cardiac complications during SAH and explained the appropriate monitoring and management of these problems.

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Introduction

Subarachnoid hemorrhage (SAH) is associated with high mortality and morbidity rates (1). It may lead to cerebral and systemic complications, and cardiac complications are common in SAH. Burch et al. for the first time described electrocardiographic changes in patients with SAH in 1954 (2). Cardiac complications following SAH were reported as electrocardiographic changes, myocardial injury, cardiac biomarkers elevation, and left ventricular dysfunction (LVD) (3). Several studies have shown that cardiac abnormalities are associated with poor outcomes, death, and delayed cerebral ischemia in SAH patients (4, 5). This review focused on cardiac complications, their manifestations, their pathophysiology, and their probable treatments in SAH patients.

Cardiac complications

Electrocardiographic (ECG) abnormalities

Abnormal ECG was reported in 50-100% of SAH patients (5-7). Most ECG abnormalities occurred during 48 to 72 hours of SAH (8-10). They include high-amplitude R waves, a U wave, QTc prolongation, ST changes (ST depression and ST elevation), pathological Q wave, and T wave abnormalities (peaked upright T wave and T wave inversion) (5, 10). Most ECG changes, including ST elevation, resolved within the first or second week of SAH, but T wave changes could persist for three months (11, 12). ECG variation during SAH, especially ST-T changes, could be misdiagnosed as myocardial ischemia or infarction and delay the management of SAH (9). ECG abnormalities within 72 hours of SAH were shown to be associated with poor clinical outcomes (5). The prolongation of QTc was associated with neurologic pulmonary edema and delayed cerebral ischemia (DCI), ST depression with hospital death, nonspecific ST-T changes with neurologic pulmonary edema, delayed

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cerebral ischemia, and death (5).

Cardiac arrhythmia is common in SAH patients and is reported as high as 90% (13). The majority of abnormal cardiac rhythms are insignificant, and they include sinus tachycardia, sinus bradycardia, and premature atrial and ventricular beats (14). However, it could be significant and life-threatening in 5-8% of patients (14, 15). Atrial fibrillation or flutter was reported as the most significant arrhythmia. The others include supraventricular tachycardia, ectopic atrial pacemaker, asystole ventricular tachycardia or flutter, different heart blocks, and torsade de pointes (14, 15). Significant arrhythmia is associated with high mortality in SAH patients and serious cardiac and neurological comorbidities (14, 16).

Cardiac and other biomarkers

CK-MB elevation and peak CK-MB more than 2% were shown in 50% and 20% of SAH patients, respectively, during the first three days of the event (17). In SAH patients, both elevated CK-MB and wall motion abnormalities were associated with LVD, which could increase the risk of decompression illness (DCI) (17).

Cardiac troponin I is more specific than CK-MB for cardiac damage and LVD is more specific in cardiovascular accident (CVA) patients (18). Serum cardiac troponin I increases in 11-21% of SAH patients (3). Several studies showed that the early release of cardiac troponin I and B-type natriuretic peptide (BNP) are related to DCI, poor outcome, and death (19, 20). Recent studies used high-sensitive troponin T (hsTnT) and N-terminal pro B-type natriuretic peptide (NTproBNP), which are more sensitive and specific than previous assays for myocardial injury and heart failure, respectively (21). Early serum elevation in hsTnT and NTproBNP is associated with DCI and early increase in hsTnT with 1-year poor outcome in SAH patients (21).

Left ventricular dysfunction

Left ventricular impairment was shown in 21-53% of SAH patients (4, 7, 22). Regional wall-motion abnormalities were more common than global ventricular dysfunctions (22). LVD rate, regional or global, is higher on days 1 and 2 of SAH and decreases with time during the first week (22). Different studies reported various ventricular segments affected in LVD. Recent studies described an apex-sparing pattern of LVD and proposed that LVD in SAH patients is not due to coronary artery disease and spasm (22, 23). LVD is associated with poor outcomes and DCI in SAH patients (4, 7, 24). As the impairment of cerebral autoregulation during SAH may result in dependence of cerebral perfusion on intravascular volume and cardiac output, LVD could decrease cerebral perfusion, especially in the context of cerebral artery vasospasm, and it could cause DCI (4).

Takotsubo cardiomyopathy, also known as stress-induced cardiomyopathy, broken heart syndrome or apical ballooning syndrome was reported in about 0.8-4.5% of patients with SAH (25). It is defined as reversible LVD, electrocardiographic changes and myocardial enzymes release mimicking myocardial infarction (MI) in the absence of obstructive coronary artery disease (26). Takotsubo cardiomyopathy is caused by high catecholamine release following acute emotional or physical stresses (25). The characteristics of these SAH patients are different from those of other patients with Takotsubo cardiomyopathy in other contexts: they are younger and always present with signs of heart failure and no chest pain (27). Thus, it is defined as neurogenic stress cardiomyopathy (NSC) or stunned myocardium (27). The mortality of these patients is about 25% higher than 1% of patients with Takotsubo cardiomyopathy, and it could be explained by the impact of severe associated neurological comorbidities (25).

In 2003, Bulsara et al. described specific criteria to differentiate neurogenic stress cardiomyopathy (stunned myocardium) from MI that included: no history of cardiac disease, new onset of abnormal cardiac function (<40%), cardiac wall motion abnormalities on echocardiogram that do not correlate with coronary vascular distribution noted on ECG, and cardiac troponin I less than 2.8 ng/ml (28).

Pathophysiology

Cardiac complications following SAH were explained by different mechanisms. SAH could damage hypothalamus, including the paraventricular nucleus, activating sympathetic outflow through the rostral ventrolateral medulla, and induce ECG abnormalities, arrhythmia, and myocardial necrosis (3). Following SAH, activation of hypothalamus-hypophysis axis causes catecholamine surge with massive catecholamine release from myocardial nerve endings and induce cardiomyocyte toxicity (3, 28). Myocardial injury following catecholamine surge could be multifactorial: tachycardia, coronary vasoconstriction, cardiomyocytes toxicity, and an increased intracellular calcium concentration (28) that could lead to contraction band necrosis and myocytolysis (29).

Disruption of the blood-brain barrier associated with neuronal damage can release pro-inflammatory cytokines, microvesicles, and other factors promoting local and systemic inflammation responses (3, 30). Systemic inflammation responses

were reported in 85% of SAH patients during the first four days (3, 30). Coincidence with catecholamine surge, inflammatory cells can flow into the heart and induce cardiac injury, myocarditis, thrombus, and myocardial cell death (31).

Management

Associated cardiac injury during SAH could complicate neurological problems and lead to poor outcomes. Accordingly, all SAH patients were recommended to be evaluated by 12-lead ECG, cardiac enzyme biomarkers (cardiac troponin I), and chest X-ray on admission (7, 32, 33). Serial ECG and serial monitoring of cardiac troponin I and pro-BNP were suggested in patients with high risk of developing LVD (high-grade SAH) (34). For the evaluation of cardiac performance, transthoracic echocardiography is usually recommended when there is evidence of myocardial injury: elevated cardiac troponin I or abnormal EEC, previous history of coronary arterial diseases, or hypotension requiring vasopressors (32-34).

Cardiac output monitoring is suggested when patients have hemodynamic instability or LVD (33). As LVD is usually reversible, it could be managed conservatively with serial ECG monitoring and reassessed 5-7 days later by transthoracic echocardiography (32, 34). If LVD is associated with hypotension (cardiogenic shock) and requires vasoactive drugs, it should be monitored with invasive hemodynamic monitoring and treated aggressively with the involvement of neurointensivists, cardiologists, and neurosurgeons (34, 35). MI rarely occurs in SAH patients as they are younger and usually without any history of coronary artery disease (36). If there is a great suspicion of MI in SAH patients due to abnormal ECG, wall motion abnormalities in a single vascular territory, and high cardiac troponin (> 2.8 µg/l), it is recommended to secure the cerebral aneurysm by endovascular approach in the first step and to assess the patient by cardiac catheterization to rule out MI (36).

Moreover, LVD complicates the treatment of cerebral vasospasm during SAH. While most LVD occurs on admission or during the first three days of SAH and usually resolves at the end of the first week, it is less likely to interfere with vasospasm that presents after three days of the event and mostly on days 7-10. However, nimodipine for vasospasm prophylaxis and 3H therapy for the treatment of vasospasm would easily increase cardiac afterload and oxygen consumption and worsen LVD (32). Besides, LVD could prevent the effective treatment of cerebral vasospasm as cerebral perfusion would be highly dependent on cardiac output after the disruption of cerebral autoregulation following severe SAH.

SAH patients with LVD or NSC should be closely monitored for hemodynamic instability, arrhythmia, cardiogenic shock, pulmonary edema, neurologic pulmonary edema, and sudden cardiac death in NeuroICU (34). NSC could be treated by ß-blockers, inotropes, diuretics, a high concentration of oxygen, Positive end-expiratory pressure (PEEP), and an intra-aortic balloon pump (IABP), especially when it is associated with cardiogenic shock (32, 37). The effect of ß-blockers and their use in SAH patients for the treatment of NSC is controversial. However, ß-blockers are assumed to decrease cardiac injury during SAH by catecholamine surge suppression.

Some studies showed that SAH patients with a history of using ß-blockers experienced NSC less than SAH patients without history of using ß-blockers (38). Nonetheless, other recent studies did not show any effect in this regard (39). In 1978, Dwyer et al. reported that in the course of SAH, propranolol and phentolamine administration had cardioprotective effects and prevented necrotic myocardial lesions (40). Although Landiolol, an ultrashort-acting ß1 antagonist, was revealed to decrease tachycardia during anesthesia for aneurysm clipping without changing blood pressure, it did not have any effects on the incidence of ECG abnormalities, Brain natriuretic peptide (BNP) , and cardiac troponin I level (41).

For the treatment of vasospasm and prevention of DCI, inotropes are usually used to increase blood pressure and maintain the appropriate cerebral perfusion. It is advised to be cautious when it is decided to administer sympathomimetic drugs for NSC patients because of the sympathomimetic origin of NSC (34). Therefore, ß-agonists, like norepinephrine, and α-agonists, like epinephrine, are suggested to be avoided in SAH patients with NSC, especially in the context of cardiogenic shocks (32). However, dobutamine, a ß-agonist (ß1 > ß2), has been found to elevate cardiac output, blood pressure, and cardiac index and mildly decrease systemic vascular resistance in NSC and SAH patients (42-44). Few have reported the occurrence of NSC following dobutamine infusion for the treatment of vasospasm (45).

Non-catecholamine inotrope could be a better choice for the treatment of NSC. Studies showed that milrinone, a phosphodiesterase 3 inhibitor, increases cardiac output and stroke volume more than dobutamine does, but it diminishes systemic vascular resistance and systolic blood pressure more than dobutamine (46). Milrinone was recommended for LVD during SAH when systolic

blood pressure is greater than 90 mmHg or when there is high systemic vascular resistance (46). Levosimendan, a novel calcium sensitizer, was recommended for NSC, and it was used successfully in SAH patients with acute heart failure, improving cardiac ejection fraction and LV filling pressure and normalizing LV wall motion (34, 47). Levosimendan stabilizes troponin C, enhances the calcium sensitivity of cardiac myofilaments, and improves systolic performance and coronary perfusion (34).

When cardiogenic shock is refractory to medical therapy during SAH-associated NSC, intra-aortic balloon pump (IABP) could be life-saving. IABP reduced LV afterload, decreased LV oxygen demand, and increased cardiac output, mean arterial pressure, and cerebral blood flow (32, 48). It also enhanced cerebral and cardiac outcome in high-grade SAH patients (48). Complications included sepsis, leakage, thromboembolic events, and lower limb ischaemia (48). A new IABP device, the NeuroFlo™, was used successfully in SAH patients to increase cerebral blood flow by partial occlusion of the abdominal aorta for the treatment of cerebral vasospasm (49). It also could be deployed in bedside under the guide of two-dimensional, spectral, and color-flow Doppler ultrasound (50, 51).

Conclusion

Cardiac complications are highly common during SAH. SAH can induce ECG abnormalities, myocardial injury, and LVD. Although they are usually benign, they could complicate the treatment of SAH patients and deteriorate their outcomes. Appropriate monitoring considering these complications can help with the early detection of cardiac problems and suitable treatment.

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None.

Conflict of Interest

All the authors declare no conflicts of interest.

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