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Intracerebral Hemorrhage Electrocardiogram Changes in Patients with Myocardial Ischemia

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Editorial

Cardiac involvement is prevalent following acute CNS involvement, regardless of the severity of cerebral involvement. Up to one-third of acute cerebrovascular events had a longer QT interval, ST-segment deviations, T wave abnormalities, increased NT-proBNP, elevated cardiac biomarkers, and/or aberrant LV function. The changes may resemble those seen in Myocardial Infarction (MI), although they are not always the same as those seen in coronary thrombosis. The current study shows that the right insular cortex (posterior, medial, and superior portions) and the right inferior parietal lobe are both associated with stroke related myocardial damage. In stroke patients, the insular cortex is involved in the modulation of the cardiac autonomic nervous system, which might result in myocardial damage. The relationship between the right inferior parietal lobe and cardiac damage is currently unknown, in contrast to the insula. It's critical to make a quick and correct differential diagnosis, especially in unconscious patients who have symptoms that are similar to those of myocardial ischemia. In individuals with symptoms of cardiac involvement during acute cerebrovascular episodes, it may be beneficial to continue cardiac evaluation to rule out simultaneous ischemic heart disease. The etiopathological basis of myocardial damage caused by a stroke is unknown. Neurogenic Stunned Myocardium (NSM) was formerly defined as myocardial injury and dysfunction with a quick onset that occurs after acute brain disease as a result of autonomic abnormalities, which may be responsible for cardiac injury following stroke. Arrhythmia, myocardial necrosis, ECG abnormalities, pulmonary edoema, and acute left HF with cardiogenic shock are all clinical characteristics that have been seen.

Pathophysiology

Induced ECG alterations in rats that were identical to those seen after an ischemic stroke, indicating that neurogenic heart damage is a possibility. NSM is a poorly known condition that can occur in any acute CNS disorder. The majority of etiopathology models for NSM include a Subarachnoid Haemorrhage (SAH). After ICH, ECG alterations and increases in cardiac enzyme levels, indicating autonomic nerve dysfunction, are also seen, however there is little data on how common this syndrome is. The peripheral causes of CCS are thought to include brain-induced autonomic dysfunction, neuroendocrine dysregulation, and systemic inflammatory processes, whereas the central mechanisms are unclear.

Neuroendocrine dysregulation and autonomic dysfunction: Put forth the sympathetic overload hypothesis, which states that central neuropathy causes autonomic dysfunction and increases catecholamine release from neural terminals. This theory explains the damage to the heart stated in NSM. Stroke causes autonomic nervous system dysfunction, which stimulates the hypothalamus-pituitary-target gland axis, resulting in sympathetic-adrenal system hyperfunction, which is exacerbated by the stress response, and increased levels of catecholamine in the body, which can be used as a proxy for sympathetic activity in neurological studies. While catecholamines have a beneficial inotropic impact on the heart under normal circumstances, excessive production can cause hypermetabolism and hyperdynamic circulation, which can cause cardiac problems. In NSM patients, plasma catecholamine levels might be up to 20 times greater than in healthy people. Excess catecholamine levels can induce heart injury by increasing myocardial oxygen demand or producing coronary vasospasms, resulting in ischemia. Venous plasma catecholamines, on the other hand, may not be a reliable indicator of sympathetic activity. Localized and excessively sharp rises in catecholamine levels cause coronary vasoconstriction and microvascular dysfunction, resulting in myocardial myocyte destruction and alterations in contractile performance.

Furthermore, due to the local increase in catecholamines, heart damage might occur in the form of direct cardiotoxic consequences. Increased catecholamine levels stimulate adrenergic receptors, leading in (over) activation of calcium channels, the generation of free radicals and peroxide in cells, and reduced adenosine triphosphate levels, all of which accelerate mitochondrial dysfunction and ultimately to cardiomyocyte mortality. Myocardial damage caused by inflammation: The second primary NSM induction mechanism is an increase in stroke induced inflammation. Some studies have shown that immunologically active components (such as cytokines, tumour necrosis factor, adhesion molecules, and bioactive peptides) are produced in the brain and released into the circulation, where they can cause systemic inflammatory response syndrome, which can lead to organ dysfunction, including heart dysfunction. Patients receiving heart transplants from donors showed conclusive evidence of these phenomena, with greater mortality rates in patients receiving heart transplants from non-traumatic ICH donors.

Further research has revealed that cardiac myocytes from ICH donors express greater levels of matrix metalloproteinases, which are hallmarks of early inflammatory conditions. Inflammatory reactions can make it easier for enzymes from brain cells to enter the bloodstream and breach the blood-brain barrier. Recent research has also suggested that the parasympathetic nervous system may play a role in cardiomyocyte injury in stroke by influencing the cardiac inflammatory response via acetylcholine receptors. Although the exact mechanism by which changes in the autonomic nervous system are influenced is unknown, persistent sympathetic dysfunction with excessive catecholamine release and inflammation has been shown to aggravate myocardial injury, increase serum myocardial enzyme levels, and lead to cardiac insufficiency. Furthermore, the majority of stroke patients are given diuretics, and eating difficulties can result in insufficient blood volume, hemorheological and hemodynamic changes, and abnormal antidiuretic hormone secretion due to cerebral haemorrhage involving the hypothalamus can result in electrolyte disturbances (especially hypokalemia and hypomagnesemia), all of

which can affect and aggravate CCS. The aberrant cardiac contraction and metabolism are a result of autonomic imbalance with a concurrent increase in the release of catecholamines and inflammatory substances. Histopathological observations in animals after blockage of the middle cerebral artery have added to the evidence for neurogenic myocardial damage. Micro-islands of necrosis with monocyte infiltration and subendocardial bleeding, sometimes known as myocytolysis, are a typical pattern of stroke related widespread myocardial injury. Unlike the delayed coagulative necrosis of the myocardium seen in CAD myocardial ischemia along the vascular distribution area, NSM myolysis is focused at the nerve ends.