Interventional Cardiology Journal
ISSN: 2471-8157

Open access Commentary

Causes and Mechanisms of Post-Cardiac Arrest Syndrome

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DESCRIPTION

After a cardiac arrest, a patient may have Post-Cardiac Arrest Syndrome (PCAS), an inflammatory pathophysiological disorder. During cardiac arrest, the body goes into a distinct state of global ischemia. This ischemia causes a build-up of metabolic waste products that trigger the production of inflammatory mediators. If Recovery of Spontaneous Circulation (ROSC) can be achieved after CPR, circulation is restored, leading to global reperfusion and subsequent distribution of the products of ischemia throughout the body. Although PCAS has different causes and consequences, it can be considered a form of global ischemia-reperfusion injury. The duration of the patient's ischemic phase often affects the damage caused by PCAS and, consequently, the prognosis; as a result, not all patients experience PCAS of equal severity. Just before the heart stops, the body is in a state of equilibrium. Blood in the body circulates normally, venous blood collects waste products of metabolism that are used elsewhere or thrown out of the body. Venous blood also transports oxygen to the tissues during this process. In the body, however, blood circulation and lungs stop during cardiac arrest. Blood no longer circulates around the body, and the lungs no longer ventilate for oxygen. As a result, all tissues in the body begin to enter a state of ischemia. In this condition, metabolic waste products such as lactic acid and carbon dioxide begin to accumulate as there is no circulation to move these products to the appropriate organs. This state of ischemia will continue until ROSC is achieved with cardiopulmonary resuscitation, at which time reperfusion of blood throughout the body will begin. This reperfusion induces an inflammatory injury using three encompassing instruments. The severity of PCAS depends solely on many factors, including: The underlying cause of the seizure, the duration of the ischemic period, the nature of the CPR received, and the patient's physiological condition. However, organs for the most part respond to the ischemic period in an unremarkable manner, and thus PCAS has a typical show. Adverse effects of PCAS are related to the effects of ischemia-reperfusion injury on individual structures, but there is a huge discrepancy between the totality of organ responses. Being profoundly metabolic with a low blood supply, the mind is the most vulnerable organ to ischemia. Accordingly, any measure of ischemia of consciousness, especially if it is delayed in cases of heart failure, usually results in brain damage. Progressively severe damage can lead to long-term effects such as mental disorder, a certain vegetative state, and finally loss of mind. The brain sustains permanent damage after approximately 20 minutes of ischemia. Indeed, even after blood flow is restored to consciousness, patients may experience hours-long periods of hypotension, hypoxemia, impaired cerebrovascular autoregulation, cerebral edema, fever, hyperglycemia, or potential seizures that further damage brain tissue. Detection of psychiatric injury includes neurological assessment, EEG, brain imaging, or potential assessment of biomarkers (e.g., S100B and NSE). In outpatient heart failure, mental trauma is the cause of death in many patients undergoing ROSC at the end of the day.

CONCLUSION

After the mind, the heart is the second most vulnerable organ to ischemia. In the event that the cause of heart failure was coronary pathology, the results of heart tests may include complications of dead myocardial tissue. While the lungs are largely oxygenated during ischemic arrest, they are still vulnerable to ischemic injury. Although ischemia is not an instrument of injury, evidence suggests that the lack of perfusion through the pulmonary vasculature during entrapment reduces the tilt of the alveolar blood vessels that creates dead space. Aggregation of oxygen in the alveoli contributes to the generation of ROS, which causes lung damage.

ACKNOWLEDGMENT

The author is grateful to the journal editor and the anonymous reviewers for their helpful comments and suggestions.

CONFLICT OF INTEREST

The author declared no potential conflicts of interest for the research, authorship, and/or publication of this article.

Received:	29-June-2022	**Manuscript No:**	IPIC-22-14226
Editor assigned:	01-July-2022	**PreQC No:**	IPIC-22-14226 (QC)
Reviewed:	15-July-2022	**QC No:**	IPIC-22-14226
Revised:	20-July-2022	**Manuscript No:**	IPIC-22-14226 (R)
Published:	27-July-2022	**DOI:**	10.21767/2471-8157.8.7.33

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Citation Yacine K (2022) Causes and Mechanisms of Post-Cardiac Arrest Syndrome. Interv Cardiol J. 8:33.