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Therapeutic hypothermia after cardiac arrest: a literature review

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Abstract: Cardiac arrest is an event that provides an elevated mortality rate. When not fatal, it can lead to neurological and other systems impairment, which may affect the prognosis of the patient. To avoid cerebral damage related to the event, some methods are indicated including therapeutic hypothermia. However, it remains unknown yet how effective is this procedure to mitigate such risk. The purpose of this literature review consisted in the systematic review of current data in order to collect what is most relevant about hypothermia as a therapeutic modality in patients after cardiac arrest. Online databases like SCIELO, MEDLINE, LILACS and PUBMED were used for the development of this review. The choice of hypothermia as a therapeutic measure in the moment post-cardiac arrest has appeared as an effective choice, especially after the analysis of biological and biochemical markers and imaging exams, which are important neurologic outcomes predictors. The achievement of a lower temperature at a specific time has showed itself as an important aspect in the neurologic outcome improvement. Therefore, it contributes to the consolidation of the hypothermia as an effective approach post-cardiac arrest. Therefore, it is possible to determine that its use has been proven as beneficial.

Keywords: Therapeutic Hypothermia; Induced Hypothermia; Cardiac Arrest.

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1. Introduction

The occurrence of cardiac arrest is an event that correlates directly to a high mortality rate. When not fatal, it can lead to neurologic impairment that will affect directly patient prognosis and quality of life. In order to avoid additional damage some strategies are implemented, including therapeutic hypothermia. The efficacy of this method to minimize aggression to body systems, although it is believed to be a successful strategy. The hypothermia effects were described initially in the 18th century, but only in 1942 during a medical conference in Berlin/Germany research presented by Dr. Sigmund Rasher on the effects of induced hypothermia in the achievement of improved prognosis in German pilots during World War II [1].

In 1950, the first experience in patients after cardiac arrest occurred. The purpose was to reduce ischemic lesion in several body tissues [2]. Still in de 1950s, Peter Safar studied the effects of hypothermia after cardiac arrest in animals. No beneficial data was observed at the time. Negative aspects of the strategy include tremors, arrhythmias, coagulopathy, and pulmonary infections [3]. Studies is four Australian Hospitals conducted in 2002 consolidated the therapeutic use of hypothermia in patients that survived cardiopulmonary arrest. A temperature target between 32ºC and 34ºC for 12-24 hours was used in the study. When compared to patients maintained in normal temperatures, those on the lower temperature group had reduced mortality rate and greater improvement in neurologic benefit. These results have constantly been reproduced in other studies [1,2].

In order to achieve a better understanding of the mechanisms associated with therapeutic hypothermia, a throughout understanding on the pathophysiology of anoxic encephalopathy is needed. It is characterized by a systemic inflammatory response mediated by oxygen-reactive species that occurs mainly during the reperfusion period, which can extend for 48-72 hours. It is well known that encephalic metabolism reduces its activity up to 6-10% for each degree Celsius decrease in temperature [4]. During the cerebral hypoperfusion period there is a shift from aerobic to anaerobic metabolism with subsequent increase in lactic acid from anerobic glycolysis that results in increased lactate and hydrogen levels. The result of this process is intra and extracellular acidosis, which leads to calcium influx and cell apoptosis [4].

Hypoxia is also responsible for neuronal membrane permeability changes and cytotoxic edema that generates increased cranial pressure and reduced cerebral blood flow. Hypothermia targets all these mechanisms described in cellular lesion: inhibition of harmful excitatory processes, reduction of neuronal permeability, reduction of cerebral edema and oxidative damage. In temperatures below 35ºC, there is also an anticoagulant effect and decreased release of thromboxane A2 and endothelin with a resultant effect of local vasodilation [4]. Non-invasive cooling methods include ice packs, thermic blankets, cooling devices and refrigerated solution infusion. These approaches are highly effective, but caution is needed to avoid hyper-cooling [5].

The objective of this literature review consists in collection of the most recent available data regarding the applicability of therapeutic hypothermia as a treatment approach to patients after successful return to spontaneous circulation and its prognosis. A possible association between these factors was analyzed, a hypothesis was made, and we were able to evaluate cases of both success and failure of this strategy. After careful review of the available data, it is possible to determine that therapeutic hypothermia has proven to be beneficial in selected patients.

2. Material and methods

To the elaboration of this literature review were mainly used scientific articles published at the online version of the Brazilian Journal of Critical Care Medicine. Research in electronic databases such as SCIELO, MEDLINE, LILACS, and PUBMED were also conducted using the following keywords in English and Portuguese: therapeutic hypothermia and induced hypothermia. The combination of these keyword was also utilized during manual search in the references of the consulted articles. The selection of papers was based in the following inclusion criteria: articles published in the last 15 years.

3. Results and discussion

The choice of hypothermia as a therapeutic approach in patients after cardiac arrest has revealed to be a viable strategy with potential prognostic benefit. In the intensive care setting, a prospective study performed by [6] evaluated 52 patients with a mean age of 62 years-old that were submitted to hypothermia after return to spontaneous circulation. The data was compared to a historic cohort (n=74) treated before the formal recommendation of the use of hypothermia. The intervention group showed significantly reduced intensive care unit stay and time on mechanical ventilatory support. There were also better neurologic outcomes up to 1 year in follow-up.

The efficacy of the method was also observed in the analysis of biochemical, biological, and imaging markers. In this context, neuroenolase, hypoxia-induced cerebral lesion and rapid achievement of target temperature were factors associated to a negative prognosis. An improvement in neurologic outcomes was associated to an ideal cooling time, which contributed to the consolidation of induced hypothermia as a therapeutic approach after successful cardiopulmonary resuscitation [5,6]. Thus, the subject is question remains controversial and lacks relevant studies to support its extended use. During this literature review, we have noticed a reduced number of reports related to the method

and a series of biased articles, which significantly compromises the data analysis regarding efficacy and benefits of this treatment modality.

4. Conclusion

In view of the searched data, we can conclude that therapeutic hypothermia in patients after cardiac arrest may have a potential benefit. Although, the application of this strategy is not risk-free, more studies and technical improvement are required, as well as better definition of the inclusion criteria and selection of patients.

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