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Out-of-Hospital Cardiac Arrest and Acute Coronary Syndromes: Reviewing Post-Resuscitation Care Strategies

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ABSTRACT

Out-of-hospital cardiac arrest (OHCA) carries an enormous global burden of mortality and morbidity. The post-cardiac arrest syndrome consists of complex pathophysiological changes that result in hypoxic brain injury, myocardial and peripheral organ dysfunction, and the systemic ischemia-reperfusion response. We review common cardiac arrest cases to highlight key management issues and recommendations in post-resuscitation care, including therapeutic hypothermia, coronary angiography and revascularization, and circulatory support. Guidelines still suggest mild therapeutic hypothermia be administered for OHCA over targeted temperature management preventing pyrexia. Similarly, early invasive coronary angiography is particularly beneficial when there is ST-elevation on the post-resuscitation electrocardiogram, but might be considered in the absence of ST-elevation if there is no noncardiac cause to explain the OHCA. However, there remain a large number of unanswered questions that require ongoing research.

RÉSUMÉ

L'arrêt cardiaque hors de l'hôpital (ACHH) entraîne un fardeau énorme de mortalité et de morbidité. Le syndrome post-arrêt cardiaque consiste en des changements physiopathologiques complexes qui entraînent des lésions cérébrales hypoxiques, une dysfonction du myocarde et des organes périphériques, et une réponse systémique d'ischémie-reperfusion. Nous avons passé en revue des cas communs d'arrêt cardiaque pour souligner les principaux problèmes et les recommandations de prise en charge des soins post-réanimation, dont l'hypothermie thérapeutique, la coronarographie et la revascularisation, et l'assistance circulatoire. Les lignes directrices suggèrent encore que l'hypothermie thérapeutique légère soit administrée lors d'ACHH par rapport à la prise en charge ciblée de la température pour prévenir la pyrexie. De la même façon, la coronarographie effractive précoce est particulièrement bénéfique lorsqu'il y a sus-décalage du segment ST à l'électrocardiogramme post-réanimation, mais serait considérée en l'absence de sus-décalage du segment ST s'il n'y a pas de cause non cardiaque pour expliquer l'ACHH. Cependant, nombreuses sont les questions non résolues qui obligent à poursuivre la recherche.

Out-of-hospital cardiac arrest (OHCA) carries an enormous global burden with in-hospital mortality of 50%-90%.[1] Among survivors, a third suffer poor quality of life and neurological sequelae from cerebral ischemia.[1] Few recover to "good" neurological status, with a Cerebral Performance Category score of 1-2 for minimal or moderate neurologic disability.[1] Cardiac arrest leads to complex but modifiable pathophysiological changes that constitute the post-cardiac arrest syndrome, including hypoxic brain injury, myocardial and peripheral organ dysfunction, and the systemic ischemia-reperfusion response. Herein, we review components of post-resuscitation care that might affect survival and neurological status in OHCA with common scenarios.

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See page 1480 for disclosure information.

Clinical Case 1

A 65-year-old woman collapses suddenly with pulseless electrical activity. After return of spontaneous circulation, she is transferred to a tertiary care centre intubated. The initial post-resuscitation electrocardiogram (ECG) reveals inferolateral ST-depression, accompanied by slightly increased levels of cardiac biomarkers. Therapeutic hypothermia is started as is acute coronary syndrome (ACS) management with aspirin, clopidogrel, and intravenous heparin. The ECG evolves to anterior ST-elevation, which prompts coronary revascularization of a proximal left anterior descending lesion.

Question 1: Is there a role for cooling in OHCA, especially with an ACS? What cooling modalities can be used?

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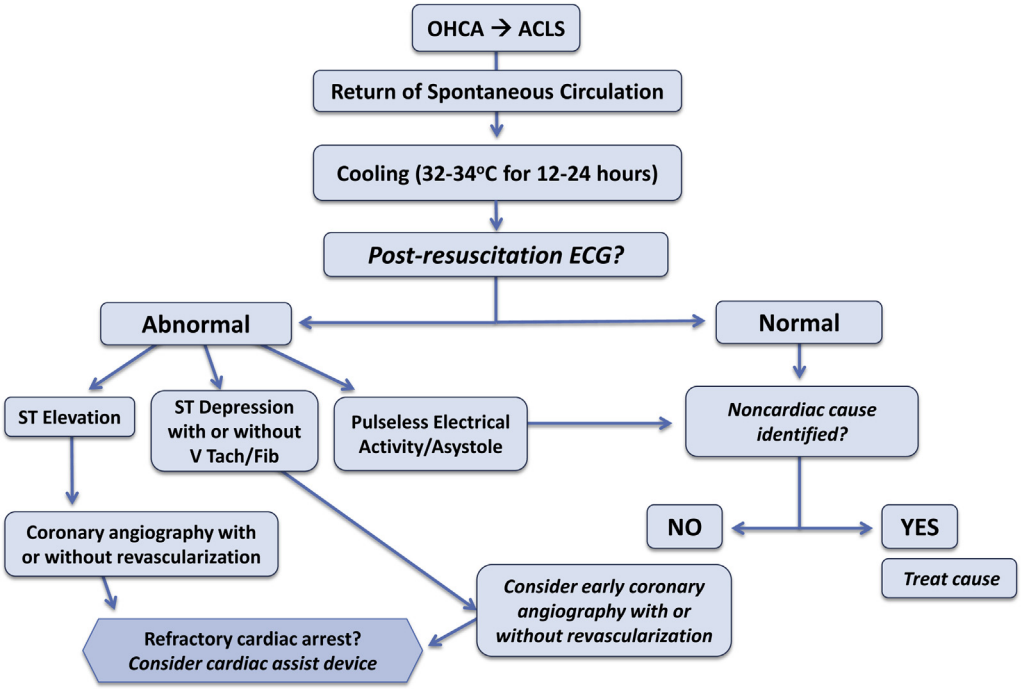

Figure 1. Proposed algorithm for post-resuscitation care in out-of-hospital cardiac arrest (OHCA) and acute coronary syndromes (ACS). In OHCA, comatose survivors resuscitated using advance cardiac life support (ACLS) should undergo mild therapeutic hypothermia (class I and IIb indications for cooling for ventricular and other rhythms, respectively). The post-resuscitation electrocardiogram (ECG) can guide subsequent therapy; early invasive coronary angiography and revascularization should be considered if ECG changes are suggestive of an acute coronary syndrome (ie, ST elevation or depression with or without ventricular tachycardia or fibrillation [V Tach or Fib]). The underlying cause should be sought and treated accordingly if the ECG reveals pulseless electrical activity or asystole, or is normal. Refractory cardiac arrest might require circulatory support with ventricular assist devices or extracorporeal membrane oxygenation.

What is the optimal target temperature? How does cooling influence efficacy of ACS management?

Therapeutic Hypothermia and ACS in OHCA

Early implementation of therapeutic hypothermia in OHCA improves neurological outcomes by reducing cerebral glucose and oxygen metabolism and by providing neuroprotection against reperfusion injury.[1,2] Two landmark studies in 2002 demonstrated improved survival and Cerebral Performance Category score of 1-2 neurological outcomes with cooling between 32°C and 34°C for 12-24 hours vs normothermia after OHCA from ventricular fibrillation.[1,2] The International Liaison Committee on Resuscitation recommends mild hypothermia for comatose survivors of ventricular fibrillation-induced OHCA.[1] Although the evidence is strongest for shockable ventricular arrhythmias (with a number needed to treat of 6), cooling conferred a pooled relative risk of 0.85 for 6-month mortality for nonshockable rhythms.[1] Thus, therapeutic hypothermia might be beneficial for nonshockable rhythms of asystole and pulseless electrical activity in OHCA.[1]

Observational data suggest survival and good neurological benefits from adjunctive cooling for ST-elevation myocardial infarction (STEMI) and non-STEMI in the presence of ventricular arrhythmias after OHCA.[1,3,4] Interestingly, OHCA patients who received thrombolysis were cooled in the 2002 studies, inferring feasibility and benefit in this setting.[1] Because hypothermia can contribute to hypercoagulability, dysrhythmias, infections, hemodynamic instability, and electrolyte abnormalities,[1] research is required to explore the potential effect on ACS therapies such as percutaneous coronary intervention (PCI). Increased risk of stent thrombosis has been noted after angioplasty for an acute myocardial infarction after resuscitated cardiac arrest treated with hypothermia, perhaps because of infrequent administration of standard therapy with aspirin, dual antiplatelet agents, or heparin.[1,3] Pharmacokinetics also differ with temperature[1,3]: ticagrelor provides more rapid and sustained reduction in platelet reactivity over clopidogrel during hypothermia, and yet is less efficacious during normothermia because of gastric hypofunction and platelet hyperfunction. In contrast, glycoprotein IIb IIIa inhibitors such as eptifibatide maintain activity at all temperatures, highlighting that these agents and ticagrelor might be more effective than clopidogrel in OHCA patients who require coronary revascularization.

Hypothermia can be achieved most feasibly through external cooling blankets, cold ice packs (applied to the groin, axillae, torso, and neck), and intravenous cold crystalloid infusions.[1] Cooling helmets, fans, and internal cooling devices (eg, intranasal, esophageal, or endovascular) have been used with varying success.[1] Recent studies suggest no added benefit of prehospital- over in-hospital-initiated cooling in terms of mortality outcomes, with adverse consequences such as an increased risk of pulmonary edema. A direct comparison of cooling to 33°C vs 36°C revealed similar outcomes,