Usporedba reaktivnosti trombocita uz neinvazivni i invazivni postupak održavanja ciljne tjelesne temperature nakon kardiorespiratornog aresta: kohortna studija

Comparison of platelet reactivity levels with noninvasive and invasive targeted temperature treatment after cardiorespiratory arrest: a cohort study

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Uvod: Rano održavanje tjelesne temperature ispod 36°C kod bolesnika koji nisu oporavili svijest nakon izvanbolničkog aresta je vrlo važno kako bi se smanjila mogućnost većeg oštećenja centralnog živčanog sustava. Česti uzrok aresta srca je infarkt miokarda u kojemu je nužno primjenjivati dvojnu antiagregacijsku terapiju. Snižena tjelesna temperatura utječe na funkciju trombocita, a ne postoje posebne smjernice kako navoditi antitrombocitnu terapiju kod ovih bolesnika. Nejasno je kako različite metode kontrole tjelesne temperature utječu na reaktivnost trombocita.^{1,2} U ovom prikazu uspoređujemo reaktivnost trombocita u dvije manje kohortne skupine bolesnika koji su podvrgnuti neinvazivnom i invazivnom načinu kontrole tjelesne temperature nakon kardiorespiratornog aresta u slici akutnog infarkta miokarda s ST-elevacijom.

Metode: Analizirali smo promjene reaktivnosti trombocita tijekom prva tri dana koristeći Multiplate uređaj s arahidonskom kiselinom kao agonistom agregacije.

Rezultati: U skupini bolesnika koji su podvrgnuti neinvazivnoj kontroli temperature je bilo tri bolesnika, a u skupini s invazivnom kontrolom temperature je bilo dva bolesnika. Svi bolesnici osim jednoga su imali mjerenja reaktivnosti trombocita tijekom sva tri dana. Navedeni bolesnik koji je podvrgnut invazivnoj metodi kontrole tjelesne temperature je preminuo drugi dan te nema mjerenje učinjeno treći dan. Bolesnici podvrgnuti neinvazivnoj kontroli temperature su imali višu reaktivnost trombocita (slika 1).

Zaključak: Podaci ukazuju kako postoji razlika u reaktivnosti trombocita između različitih metoda održavanja tjelesne temperature nakon aresta i infarkta miokarda. Mogući mehanizam bi mogao biti izravan učinak invazivne primjene snižene temperature na trombocite čime se smanjuje osjetljivost na acetilsalicilatnu kiselinu. Daljnja istraživanja su nužna kako bi se ovi rezultati potvrdili, uključujući svakako i mjerenje aktivacije trombocita preko ADP-ovisnog puta preko kojega djeluju P2Y12 inhibitori.

Introduction: Early targeted temperature management (TTM) below 36°C in patients who did not recover conscience after out of hospital arrest is crucial to reduce central nervous system damage. Myocardial infarction is often a cause of cardiac arrest and warrants dual antiplatelet therapy. Decrease in body temperature affects platelet reactivity and there are no special guidelines for antiplatelet management in this clinical setting. It is unclear how different TTM methods affects platelet reactivity.^{1,2} Herein, we compared platelet reactivity (PR) in two smaller cohort groups of patients with acute ST segment elevation myocardial infarction who underwent non-invasive and invasive TTM after cardiorespiratory arrest.

Methods: We analyzed PR changes in patients during first three days using Multiplate function analyzer and arachidonic acid as platelet aggregation agonist.

Results: Two and three patients underwent invasive and noninvasive TTM, respectively. All but one had PR measurement on all three days. One patient in the invasive TTM group died on the second day and had no PR measure on day three. Patients in the noninvasive TTM group had higher PR levels (Figure 1).

Conclusion: Data shows that there is a difference in PR in patients undergoing different methods of TTM after cardiac arrest and myocardial infarction. We hypothesize that this could be due to direct effect of invasive body temperature lowering on platelets which decreases their sensitivity on aspirin. Further investigations are needed to confirm our results, including measurements of ADP-dependant PR which is inhibited by P2Y12 antagonists.

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