

Therapeutic Hypothermia in Acute Myocardial Infarction: A Systematic Review

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Introduction

- Therapeutic hypothermia preserves neurologic function in post- cardiac arrest patients.
- Several studies suggest that hypothermia may preserve myocardial function in patients with acute myocardial infarction(AMI)

Method & search

- Data: ClinicalTrials.gov, the Cochrane Clinical Trials Register, EMBASE, MEDLINE
- English language & adult population
- Select
- (1)were randomized controlled trials (RCTs), prospective or retrospective cohort studies, or case series
- (2) administered therapeutic hypothermia(core body temperature34°C) to post-AMI patients

(3)reported on infarct size (as a percentage of the left ventricle).



Design

- $\bullet\,$ All case f/u by CT to measure infarct size at 1 month post-AMI.
- All studies reported infarct size
- Only 2 studies reported major adverse cardiac events (MACEs)

Study

- 1 retrospective study: 20p't , catheter-based cooling, in 6hr of symptom, 5MACE , 1died, Median infarct size:4%
- Prospective study, 11p't , TH prior to PCI, protection , Median infarct size: 23%, EF: 45%
- RCT, 42p't, random, in 6 hr of symptom, cooling for 3 hr, no death in TH, 2 death in control, **Median infarct size: 2%/8%,(H/C), p0.8**
- RCT, 392 p't, MACE: 6.2%/3.9%(H/C),p0.45, mean infarct size:14.1%/13.8%(H/C), p0.45
- Although there was no difference in infarct size in the general post-AMI population, there was a reduction in infarct size of patients who sustained an anterior AMI (9.3% /18.2%(H/C), p0.05)
- NOOL-MI, RCT, H.T was safe
- ICE-IT, safe, infarct size:10%/13%(H/C), p0.14

Discussion

- did not find a reduction in infarct size, MACEs, all-cause mortality
- may reduce infarct size exclusively in patients with anterior AMI
- TH does not result in longer door-to-balloon times compared with control, and it does not delay the onset of primary PCI

Conclusion

• More evidence is needed to determine whether TH is associated with improved infarct size, fewer MACEs, or lower all-cause mortality

Thank you for your attention!!

Journal meeting-2 報告者:R2許哲彰 指導者:VS王瑞芳 DATE:2011/7/9

Therapeutic hypothermia after cardiac arrest – cerebral perfusion and metabolism during upper and lower threshold normocapnia

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Introduction

- TH: decrease reperfusion injury
- The ventilation control(alpha-stat strategy)
- Authors: uncorrected pCO2 between 40~45mmHg(5.3–6.0kPa)
- 95%CI lower limit was 32mmHg (4.2kPa) during hypothermia
- corrected lowest pCO2
 3.6kPa

Introduction

- hypocapnia may induce cerebral ischaemia due to vasoconstric-tion
- hypo- or hypercapnia, as high as 55%
- In Finland, corrected:uncorrected=1:1
- All within 4.2 ~ 6.0kPa

Method

- Randomized crooss-over
- 8 victim: >18y/o, witenessd cardiac arrest, shockable, etiology of heart, ROSC in 30 min
- ETT, MV, propofol, fentanyl, cisatracurium, CVP,4~8mmHg ETCO2, SpO2,insulin
- $\bullet\,$ 33C for 24hrs, oesophageal probe & urinary bladder catheter
- Jugular bulb O2, R't IJV
- Microdialysis, 2mm borehole, into lateral ventricle, check lactate glucose pyruvate 1/p ratio 1/g ratio glycerol glutamate

Method

- Tissue oxygenation & ICP were measured (NIRS)
- Transcranial doppler : focus on middle cerebral artery (MCA) mean flow velocity (MFV) and pulsatility index (PI).
- Adjusted primarily according to the continuously monitored EtCO2 levels with intermittent blood gas analysis
- pH-stat approach(temperature corrected pCO2 4.5–5.5)
- Hyper:6.0kpa (corrected) Hypo:4.3kpa (uncorrected)









Discussion

- ↓ jVCO2, No cerebral O2 change
- Microdialysate may not reflect brief hypoperfusion
- appreciate the effects of hypothermia
- even short duration of dyscarbaemia may initiate the bad sequel of events.
- Hypocapnia & lactataemia
- Hypercapnia → no IICP
- Rewarming, O2

Conclusion

- During therapeutic hypothermia, even mild hyperventilation and resulting threshold hypocapnia may predispose to cerebral ischaemia.
- Corrected PCO2 to avoid hyperventilation
- Minor risk $\uparrow\,$ of $\,\uparrow\,$ ICP due to hypercapnia in TH when using corrected PCO2

