Efficacy of Continuous Multimodality Monitoring on Preventing Cerebral Metabolic Crisis

Michael F Stiefel MD PhD; Corrado Marini MD; Christy Stoller; Nicole R Eiden; Arthur Wang MD; Anu Amin MD; Yin C. Hu MD

Westchester Medical Center, Valhalla, NY

Introduction

Brain hypoxia and metabolic dysfunction are associated with poor outcome in patients with severe brain injury (sBI). We sought to determine the relationship between multimodality monitoring (MMM) parameters and impaired cerebral metabolism in patients with sBI.

Methods

Patients with severe brain injury underwent multi modality monitoring (MMM) with intracranial pressure (ICP), brain oxygen tension (PbtO2), and transcranial oxygen saturation (NIRS). Goal directed therapy was to maintain ICP less than 20 mmHg, cerebral perfusion pressure (CPP) greater than 60 mmHg; PbtO2 greater than 20 mmHg, and NIRS greater than 60%.

In addition, cerebral microdialysis (CMD) samples were collected for the first five days every hour. MMM variables were compared to cerebral microdialysis. Metabolic crisis (MC) events were defined as Lactate:Pyruvate ratio (LPR) > 25. Severe metabolic crisis (sMC) was defined as LPR > 40.

Metabolic crisis was subdivided as (1) Ischemic when glucose and pyruvate were below normal, or (2) Mitochondrial dysfunction when pyruvate was normal or elevated.

Patients were subdivided based on etiology of their brain injury as (1) traumatic brain injury (TBI) or (2) neurovascular (NV).

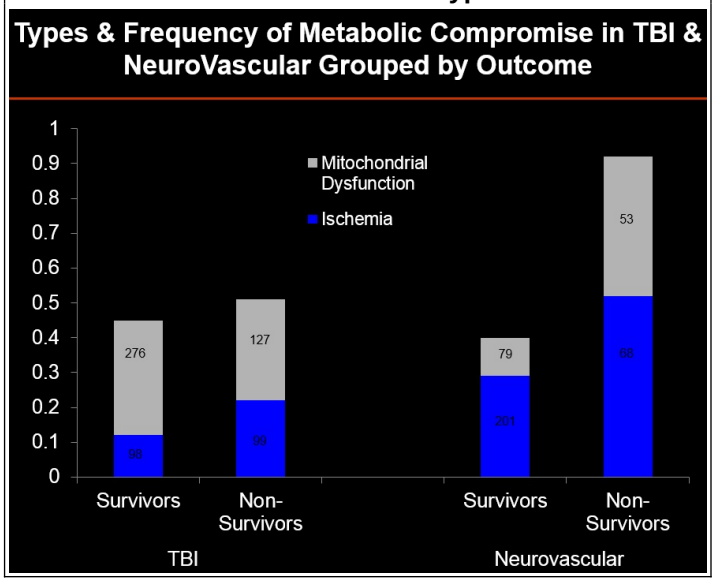
MMM and CMD data for survivors were compared to non survivors.

Results

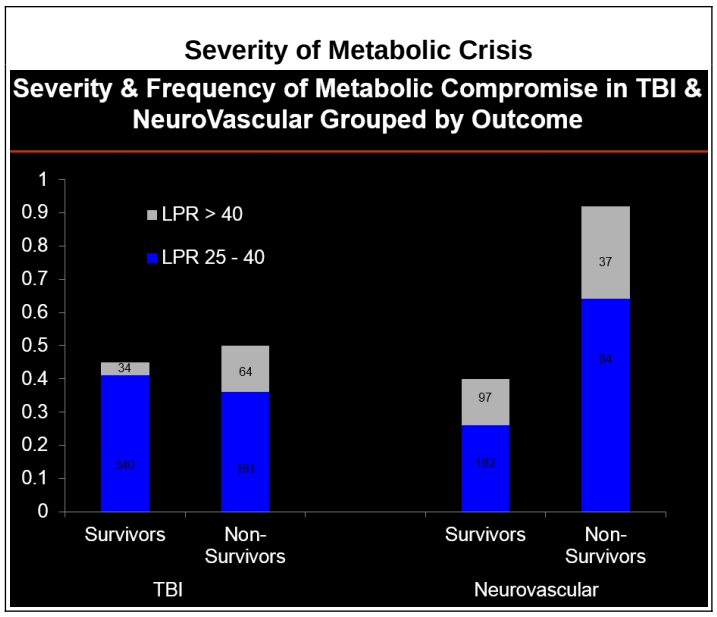
26 consecutive patients were monitored for up to 5 days. 17 patients with Traumatic Brain Injury (TBI) and 9 patients with neurovascular injury (NV). Over 124,652 minutes of continuous multimodality monitoring and 2107 microdialysis periods were analyzed. Following resuscitation, multimodality monitoring goals were obtained 97% of the time.

Metabolic crisis was identified in 1000 (47%) of CMD samples. MC was associated with mitochondrial dysfunction more often in TBI patients, and was associated with ischemic dysfunction more often in NV patients. Nonsurvivors had more episodes of severe metabolic crisis in comparison to survivors.

Metabolic Crisis Type



Severity of Metabolic Crisis



Conclusions

Maintenance of ICP, CPP, PbtO2 and NIRS oxygen saturation does not ensure normal cerebral metabolism.

Non-Survivors have more periods of impaired endpoints of resuscitation

There may be a difference in the cause of the metabolic compromise when comparing TBI to Neurovascular injury.

Studies are needed to identify interventions and therapies effective at reversing and/or protecting against metabolic compromise.

Learning Objectives

Understand the role of Multimodality monitoring in severe brain injury

Understand the relationship between multi modality monitoring and cerebral metabolism

Understand the possible contributing factors to altered metabolism