Time Course and Physiological Determinants of Cerebral Lactate/pyruvate Ratio Following Traumatic Brain Injury

Mathew Guilfoyle; Adel Helmy MD, PhD; Ivan Timofeev; Keri Carpenter PhD; David K. Menon MBBS, MD, PhD, FRCP; Peter Smielewski; Marek Czosnyka; Peter J. Hutchinson PhD, FRCS (SN)

Introduction

Brain lactate/pyruvate ratio (LPR) is an important indicator of cerebral metabolism and an independent predictor of neurological outcome following traumatic brain injury (TBI)[1]. This study examined the temporal trends and physiological factors driving fluctuations in LPR following TBI.

Methods

This was a retrospective analysis of prospectively collected data on patients with TBI requiring neurocritical care and invasive multi-modality monitoring. Microdialysis parameters (glucose, pyruvate, lactate) were co-registered with continuous recordings of intracranial pressure (ICP), mean arterial pressure (MAP), PRx (a measure of cerebrovascular autoregulation), and brain tissue oxygen tension (PbtO2). Analysis using linear mixed-effects and generalised additive models was conducted in R (v.3.2.5)

Conclusions

Higher LPR is associated with worse outcome following TBI. Brain glucose is a major determinant of LPR, both directly and via interaction with other physiological parameters. These findings suggest potentially modifiable factors that could be employed in therapeutic approaches to correct deranged LPR following TBI.

Results

566 patients were included; median age was 36 years and 76.3% were male. Mortality at 6 months was 21.2% and 32.0% of patient had a poor neurological outcome (extended Glasgow Outcome Scale < 4). Mean LPR was higher amongst patients with poor outcome in the first several days following injury. Cerebral glucose was the strongest independent predictor of LPR amongst the monitoring parameters ($p<0.00001$), and demonstrated a highly nonlinear relationship with LPR rising rapidly at very low glucose concentrations. Cerebral perfusion pressure (CPP=MAP-ICP; $p<0.0001$), ICP ($p<0.01$), and PRx ($p<0.0001$). Further, there were significant interactive effects on LPR between CPP, glucose, and PbtO2.

Learning Objectives

Metabolic dysfunction following TBI is associated with poor outcome and low brain glucose is a key factor.

References

[1] Timofeev I et al. (2011) Cerebral extracellular chemistry and outcome following traumatic brain injury: a microdialysis study of 223 patients. Brain 134(2):484-94

[Default Poster]