Journal of Diabetes Science and Technology
Volume 5, Issue 3, May 2011

SYMPOSIUM

Monitoring of Glucose in Brain, Adipose Tissue, and Peripheral Blood in Patients with Traumatic Brain Injury: A Microdialysis Study

Elham Rostami, M.D.[1] and Bo-Michael Bellander, M.D., Ph.D.[2]

Abstract

Background:

Episodes of hyperglycemia are considered to be a secondary insult in traumatically brain-injured patients and have been shown to be associated with impaired outcome. Intensive insulin therapy to maintain a strict glucose level has been suggested to decrease morbidity and mortality in critically ill patients but this aggressive insulin treatment has been challenged. One aspect of strict glucose control is the risk of developing hypoglycemia. Extracellular intracerebral hypoglycemia monitored by intracerebral microdialysis has been shown to correlate with poor outcome. Monitoring of blood glucose during neurointensive care is important because adequate glucose supply from the systemic circulation is crucial to maintain the brain's glucose demand after brain injury. This study investigates the correlation of glucose levels in peripheral blood, subcutaneous (SC) fat, and extracellular intracerebral tissue in patients with severe traumatic brain injury during neurointensive care.

Methods:

In this study, we included 12 patients with severe traumatic brain injury. All patients received one microdialysis catheter each, with a membrane length of 10 mm (CMA 70, CMA Microdialysis AB) in the injured hemisphere of the brain and in the noninjured hemisphere of the brain. An additional microdialysis catheter with a membrane length of 30 mm (CMA 60, CMA Microdialysis AB) was placed in the periumbilical subcutaneous adipose tissue. We studied the correlation among levels of glucose measured in peripheral blood, adipose tissue, and the noninjured hemisphere of the brain during the first 12 hours and during 3 consecutive days in neurointensive care.

Results:

We found a significant positive correlation between levels of glucose in peripheral blood, SC fat, and the noninjured brain during the initial 12 hours but not in injured brain. However, the result varied between the patients during the 3-day measurements. In 7 patients, there was a significant positive correlation between glucose in blood and noninjured brain, while in 4 patients this correlation was poor. In 4 patients, there was a significant positive correlation in injured brain and blood. Furthermore, there was a significant correlation between brain and adipose tissue glucose during the 3-day measurements in 11 out of 12 patients.

continued →

Author Affiliations: [1]Department of Neuroscience, Karolinska University Hospital Solna, Karolinska Institute, Stockholm, Sweden; and [2]Department of Clinical Neuroscience, Section for Neurosurgery, Karolinska University Hospital Solna, Stockholm, Sweden

Abbreviations: (ANLSH) astrocyte-neuron lactate shuttle hypothesis, (CT) computed tomography, (GCS) Glasgow Coma Scale, (GLUT) glucose transporter, (IB) injured brain, (ICP) intracranial pressure, (L/P) lactate/pyruvate, (MAP) mean arterial blood pressure, (NIB) noninjured brain, (NICU) neurointensive care unit, (SC) subcutaneous, (SD) standard deviation, (TBI) traumatic brain injury

Keywords: human, hypoglycemia, microdialysis, traumatic brain injury

Corresponding Author: Elham Rostami, M.D., Department of Neuroscience, Karolinska University Hospital Solna, Karolinska Institute, Retzius väg 8, S-17177 Stockholm, Sweden; email address elham.rostami@ki.se

Abstract cont.

Conclusion:

This study indicates that there is a good correlation between blood glucose and adipose tissue during initial and later time points in the neurointensive care unit whereas the correlation between blood and brain seems to be more individualized among patients. This emphasizes the importance of using intracerebral microdialysis to ensure adequate intracerebral levels of glucose in patients suffering from severe traumatic brain injury and to detect hypoglycemia in the brain despite normal levels of blood glucose.

J Diabetes Sci Technol 2011;5(3):596-604