Human glia expresses cytoglobin after brain trauma

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A 190-amino acid protein- Cytoglobin(Cygb), is a recently identified member of the vertebrate hemoglobin family. Cygb is an ancient and highly evolutionarily conserved protein. Like the other members of the hemoglobin family, functions of Cygb are mainly based on its oxygen binding ability. Cygb was previously demonstrated to be exclusively expressed by neurons of brain. However, a recent research reported the expression of Cygb in human GBM cells, hinting that glia cells may also express Cygb under certain pathological states. Therefore, to assess the cellular localization of human Cygb under physiological and pathological state, we performed immuno-staining to post-mortem brain specimens from people who died of traumatic brain injury and the deceased without neuropathy, respectively. In uninjured human brains, the immuno-signal of Cygb was restricted in neurons but not glia cells. Whereas in the chronic brain trauma group, expression of Cygb was also detected in astrocytes and microglia cells in the injury repairing area. Results of this present study offered the neuro-anatomical basis for further exploration of the neuro-protective role of Cygb and suggested Cygb to be a novel therapeutic target in various neurological disorders.

Gut dysfunction in patients with abdominal compartment syndrome during acute pancreatitis

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The incidence of abdominal compartment syndrome (ACS) in patients with severe acute pancreatitis (SAP) is around 20%. Enteral nutrition (EN) is a gold standard for the patients with SAP, but in those with ACS can aggravate bowel ischaemia. Total parenteral nutrition (TPN) may increase bacterial translocation and deteriorate gut functions. Study included 36 patients with SAP (21 with ACS and 15 without). Intra-abdominal pressure (IAP) was measured daily and maximal noted IAP (maxIAP) during hospitalization was included in the study. In each patient a serum level of procalcitonin (PCT) was measured daily. A value of PCT at 48h on hospital admission and maximal noted PCT (maxPCT) during hospitalization were included. Nutritional support during organ failure was EN, TPN or EN+TPN in the same time. MaxIAP in patients with ACS was significant higher than in those without (p<0.001). There were no difference in PCT values between patients with and without ACS (p=0.64). MaxPCT in patients with ACS was 3.1 ng/mL (1.3-34.6) and in those without ACS was 2.3 ng/mL (0.4-12), (p=0.03). In patients who received TPN 20 (95%) suffered from ACS and 8 (53%) did not (p=0.005). In patients who received EN 12 (57%) suffered from ACS and 15 (100%) did not (p=0.005). In patients who received EN+TPN in the same time 11 (52%) suffered from ACS and 8 (53%) did not (p=1). In patients who received TPN a significant higher PCT (p=0.01) and maxPCT values (p<0.001) were noted than in those who did not received. In patients who received EN there was no difference in value of PCT (p=0.06), but significant higher maxPCT value was found in patients who received EN than in those who did not (p=0.04). There was no difference in PCT and maxPCT values in patients who received EN+TPN in the same time than in those who did not (p=0.68, p=0.29). In ACS during SAP serum values of PCT are higher in patients who receive TPN. Balanced usage of EN+TPN has a less influence on gut functioning than EN or TPN alone in patients with ACS and SAP.