tests for detecting and quantifying neurological damage and POCD, and also, there is no agreement on the optimal timing for postoperative testing for research, as well as, day-to-day clinical use. Future studies need to include a broader range of relevant clinical scenarios using a wider consensus on the methodological approaches, including timing and dosing of drug administration, patient selection and perioperative neurological and cognitive testing. The need for shared methodological approach, when clinical studies on pharmacological neuroprotection are designed, is recommended.

REFERENCE

1. Bilotta F, Gelb AW, Stazi E, Titi L, Paoloni FP, Rosa G. Pharmacological perioperative brain neuroprotection: A qualitative review of randomized clinical trials. Br J Anaesth 2013;110 (suppl 1):i113-20.

Rogachev B, Tsesis S, Gruenbaum BF, Gruenbaum SE, Boyko M, Klein M, *et al*. The effects of peritoneal dialysis on blood glutamate levels: Implementation for Neuroprotection. J Neurosurg Anesthesiol 2013;25:262-6.

Glutamate, an excitatory amino acid, is released in high concentrations following brain insults and is an important causative factor for secondary neuronal damage after brain injury; elevations in extracellular fluid (ECF) glutamate levels have been seen in patients with severe head injury using cerebral microdialysis. In stroke patients too, glutamate levels are shown to correlate closely with the volume of ischaemic lesion and neurological outcome. Animal experiments have further demonstrated that infusions of glutamate-lowering drugs can improve neurological outcome after severe brain insults. Hence, human clinical trials were conducted to examine the efficacy of glutamate release inhibitors and glutamate receptors antagonists; however, these experiments were aborted due to several drug-induced adverse effects. Extracorporeal methods are suggested as other means for lowering blood and brain glutamate levels, thus serving as effective neuroprotective tools in acute and chronic brain ailments. The effectiveness of haemodialysis (HD) in lowering blood glutamate has already been demonstrated by these authors in a previous study. However, the use of HD in acute brain injury settings is limited by the requirement for heparinisation and presence of haemodynamically unstable patients. In such situations, peritoneal dialysis (PD) may be a viable option as it is a short and minimally invasive procedure that does not adversely impact patient haemodynamics. This study aims to investigate the efficacy of PD as a blood glutamate lowering method and describe the pattern of glutamate concentration changes in blood, as well as, the dialysis solution.[1]

Eighteen patients (aged 26-87 years) with stage V chronic kidney disease on PD were studied. The study protocol was based on the routine peritoneal equilibrium test (PET), which assesses the efficacy of PD to remove water and waste from the body in patients undergoing PD. Following drainage of PD fluid from an overnight exchange, two litres of dialysis solution were infused in these patients over 10 minutes. After completing the infusion ($t = 0$), 200 mL of dialysis solution was drained every hour till completion of the test (t = 60, 120, 180 and 240 min). A 10 mL sample was extracted from each collected volume for analysis. Blood samples were also collected before initiation of PD and hourly thereafter in accordance with the PD sample collections. The blood samples were analysed for glutamate, creatinine, urea, glucose, glutamate oxaloacetate transaminase (GOT) and glutamate pyruvate transaminase (GPT) and PD samples were analysed for glutamate, creatinine, urea, and glucose at the same time points as the blood samples. A comparison between baseline levels of glutamate and other parameters to levels over time was made.

Blood glutamate concentrations were significantly reduced by 60 minutes after the infusion of dialysis solution ($P < 0.0001$) but slowly returned towards baseline values as time progressed. The levels of glutamate in the dialysis solution were increased significantly by 60 minutes ($P < 0.0001$), maximised at 180 minutes and started to decrease thereafter. The levels of GOT, GPT, urea, creatinine, glucose, bicarbonate and pH in plasma did not change significantly throughout the experiment.

PD was capable of significantly reducing glutamate concentrations in the blood (by almost 20% when compared to baseline values) for at least an hour after dialysis solution infusion. However, the observed effect was short-lasting, which was attributed to the very small capacity of the dialysis solution and continuous shift of glutamate from peripheral ECF and intracellular compartments into the blood; it is recommended to replace the dialysis solution in the peritoneal cavity often (every 60 minutes) to avoid saturation and the deceleration of a blood glutamate-reducing effect. Thus, this study demonstrates that PD is an effective modality in reducing blood glutamate concentrations and may be potentially utilised for the treatment of acute and chronic brain disorders that are accompanied by elevated glutamate in the brain's ECF.

REFERENCE

1. Rogachev B, Tsesis S, Gruenbaum BF, Gruenbaum SE, Boyko M, Klein M, *et al*. The effects of peritoneal dialysis on blood glutamate levels: Implementation for Neuroprotection. J Neurosurg Anesthesiol 2013;25:262-6.

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