Sevoflurane sedation in a rat model of sepsis: systemic and neuro-inflammation

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Background

Volatile anesthetics such as sevoflurane have shown anti-inflammatory properties in various organs and settings [1]. We were interested in evaluating the safety profile and the potential of sevoflurane to reduce neuro-inflammation in sepsis.

Methods

Adult male Wistar rats (329±21g) were subjected to intravenous injection of 1mg/kg lipopolysaccharides (LPS) or sham procedure (phosphate-buffered saline, PBS) and randomly assigned to sedation with propofol or sevoflurane for 12h. Blood samples were taken every 3 hours. The inflammatory mediators cytokine-induced neutrophil chemoattractant protein-1 (CINC-1) and monocyte chemoattractant protein-1 (MCP-1) were evaluated in serum and brain tissue. Brain damage marker mRNA s100B, transforming growth factor β (TGF- β) and glial fibrillary acidic protein (GFAP) as well as wet-to-dry ratio were assessed. Statistical analysis was performed using one way analysis of variance, $p < 0.05$ was considered significant.

Results

There was no difference with regard to ventilation or mean arterial blood pressure in the two sedation groups. Pro-inflammatory cytokines in serum were upregulated with two peaks at 3h and at 12h in the propofol and sevoflurane sepsis group (12h CINC-1: 401±305 vs 96±44ng/ml; 12h MCP-1 4.82±1.92 vs. 2.51±0.54 μ g/ml; both $p < 0.001$), values were higher in the propofol compared to the sevoflurane group. (**Fig. 1A and Fig. 1D**)

Also in brain tissue CINC-1 and MCP-1 protein was increased upon LPS injection, however, with similar values in both sedation groups. (**Fig. 1C and Fig. 1F**)

Brain damage marker mRNA s100B, TGF- β and GFAP were decreased by 2%, by 25% and increased by 43% by sevoflurane, but without reaching statistical significance ($p = 0.99, 0.35$ and 0.42 , **Fig. 2B-D**).

Wet to dry ratio was not affected by the sedative regimen in septic animals (4.89±0.07 vs. 4.80±0.07, $p = 0.23$, **Fig. 2A**).

Conclusion

Sevoflurane seems to be safe in a rat sepsis model with regard to neuro-inflammation and offering the benefit of attenuating the systemic inflammatory reaction.

References

[1] Lee YM et al: Biomed Res Int. 2015

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