Evaluation of Lipid Emulsion on the Hemodynamic Effects of Ropivacaine: Study in an Experimental Porcine Model of Ropivacaine Intoxication

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Background and Goal of Study

Recently, experimental studies have shown that lipid emulsions are effective in reversing local anesthetic cardiac toxicity. The so-called â€œlipid sinkâ€ effect is suggested to be the basic mechanism of this treatment and its efficacy varies depending on the physico-chemical properties of the local anesthetics. Ropivacaine is considered a less cardiotoxic agent in comparison with other long duration amide local anesthetics. There are, however, several case reports in which significant cardiac side effects have occurred after ropivacaine administration. Only a limited amount of studies have evaluated the efficacy of intralipid in the reversion of the hemodynamic effects of ropivacaine.

Material and methods:

Eleven Large-White pigs were premedicated with ketamine and anesthetized with intravenous sodium thiopental 5 mg/kg. The anesthetic maintenance was performed with sevoflurane (2.6%). A 5-French catheter (PICCO, Pulsion Medical Systems AG) was inserted through the femoral artery to record mean aortic pressure, cardiac output (measured by transpulmonary thermodilution), LVdP/dtmax, and systemic vascular resistance index. Contralateral femoral artery was canalized for analytical blood gas samples and ropivacaine levels determination, in addition, contralateral femoral vein was canalized for measurement of central venous pressure and for intermittent thermodilution cardiac output measurement. After instrumentation and monitorization, a ropivacaine bolus of 5 mg.kg-1 was administered. The animals were randomly assigned in two groups: control group (C-group, n=5) and intralipid group (IL-Group, n=6). Hemodynamic data: Heart rate (HR); mean arterial pressure (MAP); cardiac index (CI); maximal first derivative of left ventricular pressure (LVdP/dtmax) and systemic vascular resistance index (SVRI) were measured at baseline, after ropivacaine administration and at 1, 5, 15 and 30 minutes after Intralipid infusion (1.5 mL/kg over 1 minute followed by an infusion of 0.25 mL/kg/min). In C-group a saline infusion was administered instead of Intralipid. Statistical analysis: Mann-Whitney test and Wilcoxon test as appropriate.

Results: Plasma ropivacaine levels ranged between 7,940 to 5,450 ng/dl from 5 to 30 min respectively. Representative hemodynamic data is provided in Table 1. Ropivacaine induced a significant decrease in MAP (Î” 13%), CI (Î” 28%), LVdP/dtmax (Î” 41%) and in SVRI (Î” 16%) without significant differences between groups. There were not significant differences between lipid
infusion and saline in the recovery of the hemodynamic parameters previously altered by ropivacaine through any timepoint of the experiment.

Conclusion: In the present study the lipid emulsion did not enhance hemodynamic recovery in pigs intoxicated with ropivacaine. More research is warranted to define the role of intralipid on ropivacaine cardiac toxicity.