Medical Literature Summary
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This is a Fresenius Medical Care summary of:

Safety and efficacy of regional citrate anticoagulation in continuous venovenous hemodialysis in the presence of liver failure: the Liver Citrate Anticoagulation Threshold (L-CAT) observational study

Slowinski T et al. Critical Care 2015;19:349

Introduction

Regional citrate anticoagulation (RCA) is frequently used for continuous renal replacement therapy (CRRT). It goes along with a low but constant uptake of citrate. Citrate is metabolised to bicarbonate, which typically is offset by a reduced bicarbonate supply in RCA protocols. Furthermore, citrate binds calcium ions. Thus, citrate metabolism can lead to release of free calcium from calcium-citrate complexes and thereby also interfere with calcium homeostasis. In liver failure, metabolism of citrate may be impaired.

As a consequence, accumulation of citrate may result in disturbances in acid/base balance as well as in calcium homeostasis.

Objective

This study evaluated the safety of the Ci-Ca® RCA protocol in patients with different levels of liver failure. It analysed the incidence of severe acidosis (pH ≤ 7.20), severe alkalosis (pH ≥ 7.55) and impairment of calcium homeostasis (ionised calcium ≤ 0.9 mmol/l or ≥ 1.5 mmol/l).

Design

This was a multicentre, prospective, observational study in 133 adult patients during the first 72 h of CRRT. Patients were stratified according to liver function based on bilirubin levels: ≤2 mg/dl (normal), >2 to ≤7 mg/dl (mild failure), and ≥7 mg/dl (severe failure). Within the Ci-Ca® protocol, citrate was titrated to a post-filter concentration of ionised calcium of 0.25-0.35 mmol/l. Then, calcium was infused via the venous line to adjust its systemic ionised concentration to 1.12-1.20 mmol/l.

Results

Anticoagulation by RCA resulted in sustained filter lifetimes: 69% of patients required only one filter during the 72 h of the study. The overall median filter lifetime for the first filter in each patient was 70.4 h. Filter clotting was rare and occurred independently of the severity of liver failure (2-7%).

Disturbances of the acid-base status (acidosis, alkalosis) and calcium homeostasis (hypo- and hypercalcaemia) developed independently of liver function and mostly resolved subsequently. Clinically relevant citrate accumulation was reported in 3 patients (2%) with 11.6 mg/dl bilirubin or higher. In one patient, citrate accumulation was mild and transient; in another, it was associated with severe lactic acidosis; and in the third, it was presumably related to paracetamol intoxication.

Conclusion

Impairment of acid/base balance or calcium homeostasis did not occur more frequently among patients with liver dysfunction as defined by bilirubin levels. Thus, use of Ci-Ca® for RCA seems to be generally safe in patients with liver dysfunction. Nevertheless, caution is required, e.g., when cellular respiration is disturbed.

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