HIGH CUT-OFF CONTINUOUS VENO-VENOUS HEMODIALYSIS ASSOCIATED WITH HEMOADSORPTION EFFECTIVELY REMOVE BILIRUBIN AND CONTRIBUTE TO PREVENT HYPERBILIRUBINEMIA INDUCED-ACUTE KIDNEY INJURY. A SINGLE CENTER EXPERIENCE

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Introduction

Severe hyperbilirubemia (usually up to 20 mg/dL) is associated with various forms of acute kidney injury (AKI) such as the cholemic nephropathy histologically characterized by the presence of intratubular bile casts and tubular injury. Although the pathogenic effects of bilirubin on renal tubular epithelial cells are incompletely understood, hemodynamic alterations or direct toxic damage have been hypothesized. Recently, it has been demonstrated that bilirubin might induces pro-apoptotic effects on tubular epithelial cells during renal ischemia/reperfusion injury. Indeed, the highest incidence of hyperbilirubinemia induced-AKI has been demonstrated in patients affected by hepatorenal syndrome (HRS) due liver cirrhosis, cardiogenic shock and sepsis.

Aim

The aim of this study was to investigate whether high cut-off continuous veno-venous hemodialysis (HCO-CVVHD) associated with hemoadsorption is able to reduce hyperbilirubinemia preventing hyperbilirubinemia induced-AKI.

Materials and Methods

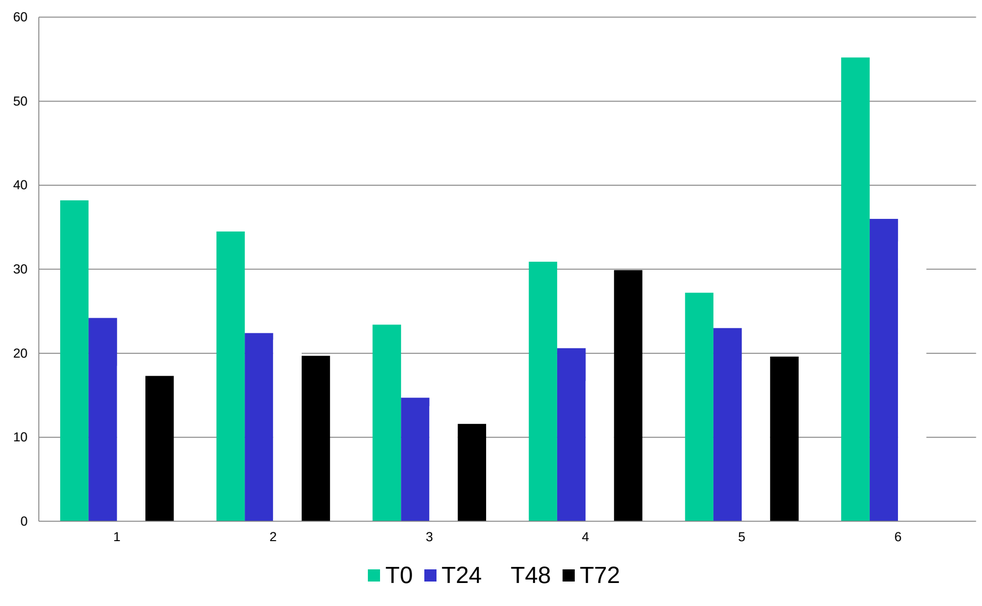
We retrospectively analyzed the clinical data of six patients affected by hyperbilirubinemia due to cholangiocarcinoma (2 pts), sepsis (2 pts) and liver cirrhosis complicated by HRS (2 pts). Patients were treated with citrate anticoagulated HCO-CVVHD associated with CytoSorb, a hemoadsorber characterized by a highly porous, biocompatible polymer capable of binding a broad spectrum of hydrophobic compounds with a molecular weight between 10 and 55 kDa. HCO-CVVHD was performed with EMiC2, a 1.80 m^2 polysulfone hemofilter with a cut-off at ~ 30 kD. Blood flow was maintained at 100 mL/min and dialysate flow rate at 33 mL/min.

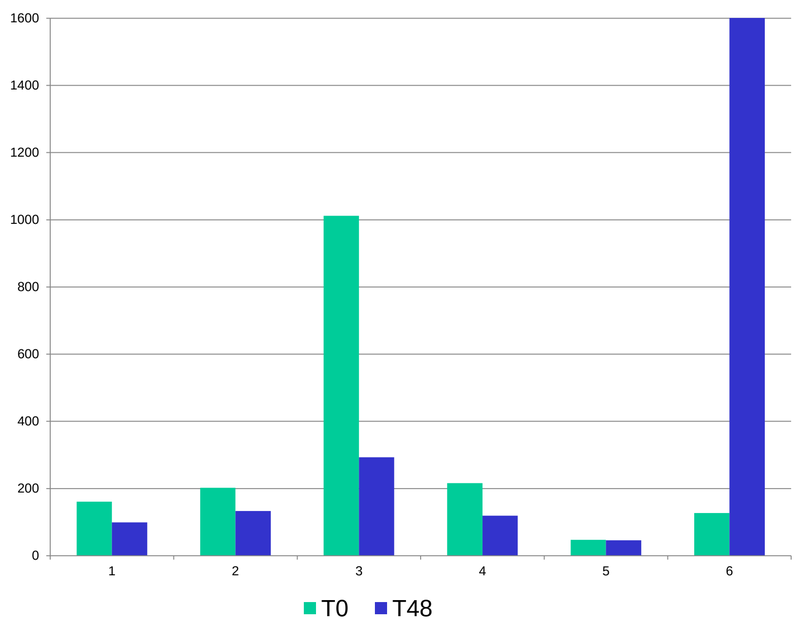
Results

The mean age of the patients was 69.1±17 yrs. Mean serum creatinine and mean urine output at the start of HCO-CVVHD (T0) were 1.2±0.15 mg/dL and 10±5 mL/h , respectively. Mean serum bilirubin at T0 was 34.35±13.05 mg/dL . The planned duration of HCO-CVVHD was 72 hrs. 1/6 patients deceased during the treatment. Mean serum bilirubin significant decrease already after 48 hrs of treatment (T48) (34.35±13.05 vs 20.55±4.85 mg/dL, $p<0.01$) together with a significant increase of the urine output after 72 hrs (T72) (10±5 vs 100±26 mL/h, $p<0.01$)

Conclusions

Our results suggest that high cut-off continuous veno-venous hemodialysis associated with hemoadsorption may contribute to reduce hyperbilirubinemia by a percentage of about 40% allowing a preservation/protection of kidney function.





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