

Bridging to transplant a patient with acute-on-chronic hepatic failure: the role of CytoSorb haemoadsorption



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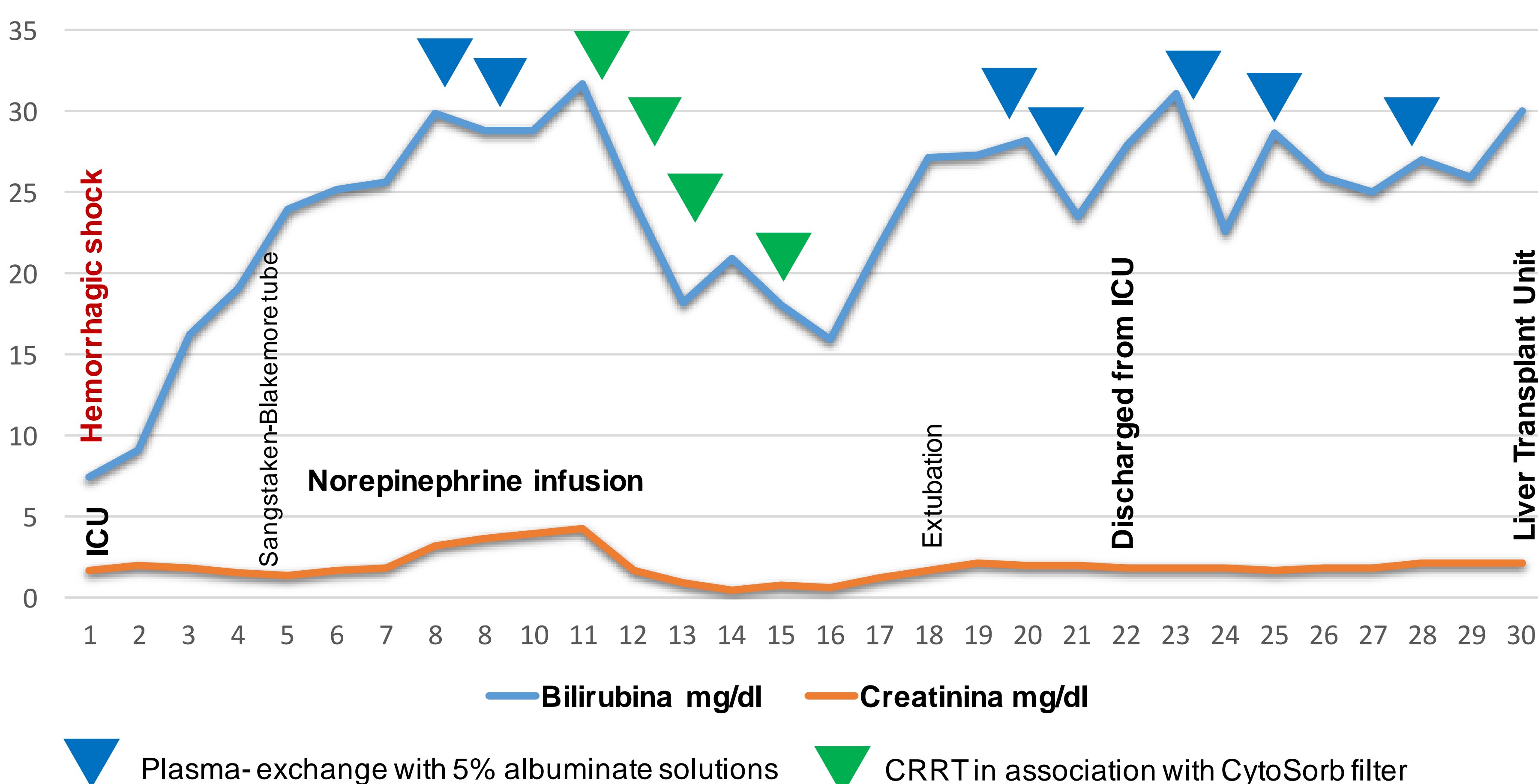
Background: CytoSorb is a synthetic extracorporeal haemoperfusion adsorption column, able to remove inflammatory cytokines and -among others- bilirubin and bile acids, thus supporting impaired liver function as a possible bridge to liver function recovery. We report on a case of acute-on-chronic liver failure, hospitalized in the ICU after gastrointestinal bleeding

Case Report. A 51-year-old male, with liver cirrhosis due to long lasting HCV-related chronic infection, never evaluated for treatment, was brought to the local Emergency Room due to sopor and left hemiparesis after recurrent episodes of hematemesis and melena. Severe anemia (Hb 4.7 mg/dL) as well as hyperglycaemia were evidenced. Massive haemorrhage protocol was started before a CT scan. He underwent emergent endoscopic treatment for cardiac ulcer bleeding; concomitant non-bleeding oesophageal F3 varices were diagnosed. He was admitted to ICU, requiring mechanical ventilation and vasoactive support. His wife reported that she was unaware of his advanced HCV-related liver disease until current episode. After ICU hospitalization, control EGDS for persistent and untreatable bleeding and relapsing anemization led to positioning of a Sangstaken-Blakemore device, held in place for 10 days. Liver function was severely altered, with ensuing coagulopathy (INR 5.4), hyperbilirubinemia (Zenith 31mg/dL) and increased creatinine (4.36mg/dL). The patient was referred to the referent Liver Transplant Center at this stage, but transfer was denied because of neurological impairment, mechanical ventilation and hemodynamic instability.

Plasma-exchange treatments were therefore performed daily, with 5% albuminate solutions; continuous haemodialysis was then started, in association with the CytoSorb filter. A CytoSorb column was plugged into the CRRT circuit, each session lasting approximately 24h. The patient received in total 96h of CytoSorb therapy, over 4 sessions. Bilirubin decreased to 15 mg/dL, creatinine to 0.61 mg/dL. Brain CT scans were persistently negative for ischemic/haemorrhagic lesions, and his consciousness ameliorated, with progressive recovery of left motor deficit. He was extubated after 18 days, with stabilized haemodynamics; he was discharged to the local Infectious Diseases Unit after 22d in ICU. After interruption of CRRT hemofiltration& CytoSorb, hyperbilirubinemia worsened up to 28 mg/dL, with creatinine stabilized at 2.1 mg/dL. His MELD score was 32. At this stage, however, due to persistent haemodynamic stability and full neurological recovery, transfer to the Liver Transplant Unit was granted. Three additional cycles of plasma-exchange were repeated. The patient was successfully and uneventfully transplanted after 14d.



Bilirubin and creatinine plasma levels as modified by sequential plasma-exchange and CRRT/CytoSorb filter



Conclusions. In our patient, with acute on chronic liver failure due to massive digestive haemorrhage, cytokines may well have played a relevant role on persistent, severe impairment of an already hampered organ. The use of CytoSorb may therefore have provided an etiologic, as well as depurative treatment, allowing successful bridging to meet the criteria for eligibility to liver transplantation.