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Dear ladies and gentlemen, dear ADVOS users and interested parties,
we are pleased to present you another issue of our ADVOS Literature Service. We regularly select one or more papers from international journals which might be of interest to you in connection with our ADVOS procedure. This month we have selected the following:

CHOLEMIC NEPHROPATHY CAUSES ACUTE KIDNEY INJURY AND IS ACCOMPANIED BY LOSS OF AQUAPORIN 2 IN COLLECTING DUCTS.

Bräsen et al.

Background

Acute Kidney Injury (AKI) is a common complication in patients with liver cirrhosis. 20% of hospitalized patients with decompensated cirrhosis develop AKI due to hepatorenal syndrome. However, AKI can be also caused by cholemic nephropathy. These patients show characteristic histomorphological kidney alterations including intratubular bile casts and tubular injury.
In this new issue of the literature-service, we review the study from Bräsen et al. regarding the importance of bilirubin levels in the development of kidney injury.

Methods

149 patients who underwent kidney biopsy between 2000 and 2016 at the Department of Gastroenterology, Hepatology, and Endocrinology of the Hannover Medical School (MHH) were identified. 79 patients with a history of liver disease and renal deterioration were finally included in the retrospective study.

Patients were classified to the type of kidney disease based on the 2018 EASL clinical practice guideline on the management of patients with decompensated cirrhosis. Among others, bilirubin, transaminases, creatinine, and complete blood count were analyzed. Survival rate was also assessed.

Results

When applying EASL criteria 57% (n=45) of the patients presented with acute kidney injury, while 43% (n=34) had chronic kidney disease (CKD). Renal biopsy revealed the diagnosis of cholemic nephropathy in 17.8% of the acute patients (n=8), whereas none of the chronic patients was diagnosed with cholemic nephropathy.

Moreover, 44.4% (8 of 18) of the patients with a bilirubin > 100 µmol/l (5.8 mg/dl) in the cohort of acute patients were diagnosed with cholemic nephropathy. Three patients recovered, while the remaining five patients had advanced stages of various diseases and needed renal replacement therapy. Four out of five died within two years, the remaining patient underwent combined liver/kidney transplantation.

Serum bilirubin, alkaline phosphatase and urinary bilirubin were identified as predictive factors for the diagnosis of cholemic nephropathy. Finally, patients with acute kidney injury had a significantly higher mortality after one year.

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The authors conclude:

In this cohort, two types of cholemic nephropathy were noted. First, a reversible form was observed in patients with acute liver disease where a specific treatment was available. All these patients developed acute kidney injury which resolved once bilirubin declined. In contrast, the five other patients suffered from disease conditions that are per se considered major risk factors for the development of severe acute kidney injury (stage 3) including liver cirrhosis or sepsis. Cholemic nephropathy in these patients did not resolve as hyperbilirubinemia could not be improved by medical intervention.

The authors speculate that the extracorporeal elimination of hepatic metabolites including bilirubin has a beneficial effect on the renal deposition of bile cast, and that this prophylactic approach would be more efficient as opposed to a belated treatment once casts and cellular pigments are already deposited.

We think that:

This paper of the Department of Gastroenterology, Hepatology, and Endocrinology of the Hannover Medical School shows a case-series of patients with underlying liver failure who developed kidney failure due to cholemic nephropathy.

They identified that a bilirubin level 5 times over the normal upper limit with a cutoff of 100 µmol/l (5.8 mg/dl) represents a risk factor for intratubular bile cast formation, and consequently for kidney injury development. In fact, they speculate with the possibility of a prompt attempt for bilirubin elimination through extracorporeal support devices in order to prevent renal damage.

This is in line with other studies that already demonstrated the risks of high bilirubin levels, not only for kidney injury progress, but also for mortality increase, even with levels > 1.2 mg/dl (20 µmol/l). Therefore, we agree with the idea of the authors regarding the prompt initiation of bilirubin removal.

In this regard, ADVOS should be understood as an advanced renal support device as it helps the kidney to
i) eliminate water soluble substances and equilibrate electrolyte levels
ii) remove protein-bound toxins (e.g. bilirubin) otherwise excreted through kidney's anionic or cationic transporters
iii) directly eliminates protons and generates bicarbonate imitating the renal compensatory mechanism for acidosis.

If you have further questions or suggestions - please contact us at marketing@advitos.com.

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