Journal Of Nephrology & Kidney Diseases
ISSN# 2833-308X

Research Article

Successful Treatment of Cholemic Nephropathy by Extracorporeal Albumin Dialysis in Liver Failure

Katharina Kornhuber[1*], Lutz Renders[1], Florian Voit[1], Lena Fürst[1], Veronica Encalada Guerrero[1], Joachim Velden[2], Uwe Heemann[1] and Claudius Küchle[1]

[1]Department of Nephrology, Klinikum Rechts der Isar, Germany

[2]Department of Nephropathology, Uniklinikum Erlangen, Germany

ARTICLE INFO

Received Date: November 19, 2024
Accepted Date: December 10, 2024
Published Date: December 12, 2024

KEYWORDS

Bile cast nephropathy; Molecular adsorbent recirculating system; Albumin binding capacity; Extracorporeal albumin dialysis; Plasmapheresis; acute kidney injury; Hepatorenal syndrome

Citation for this article: Katharina Kornhuber, Lutz Renders, Florian Voit, Lena Fürst, Veronica Encalada Guerrero, Joachim Velden, Uwe Heemann and Claudius Küchle. Successful Treatment of Cholemic Nephropathy by Extracorporeal Albumin Dialysis in Liver Failure. Journal of Nephrology & Kidney Diseases. 2024; 6(1):139

Corresponding author:
Katharina Kornhuber,
Abteilung für Nephrologie, Klinikum Rechts der Isar, Ismaningerstraße 22, 81675 München,
Tel: +4915757796575,
Email: katharina.kornhuber@mri.tum.de

ABSTRACT

Bile cast nephropathy is an often-overlooked diagnosis, currently lacking treatment guidelines and definitive diagnostic criteria for early recognition. However, several studies suggest that its incidence is higher than previously believed, especially when assumed cases of hepatorenal syndromes are investigated via renal biopsy.

Nonetheless, bile cast nephropathy can be effectively managed with early extracorporeal interventions. We report the case of a 32-year-old man with bile cast nephropathy and liver failure most likely steroid induced, successfully treated using extracorporeal albumin dialysis. We will give a brief overview on bile cast nephropathy focused on diagnostic and treatment options. The primary aim of this paper is to underscore the importance of early diagnosis and treatment initiation and to highlight available therapeutic modalities.

INTRODUCTION

Cholemic nephropathy describes an acute kidney injury characterized by typical histomorphological changes in the kidney observed in patients with liver failure. The gold standard for diagnosis is kidney biopsy, which is though associated with risks in patients with liver failure due to coagulation issues. Early therapy is crucial to prevent long-term kidney injury, as perpetuated tubular necrosis can lead to scarring of kidney parenchyma.

Nevertheless, standardized therapeutic procedures are currently lacking. The primary aim of all therapies is to address the underlying causes and eliminate bilirubin and bile acids. Possible treatment options include albumin dialysis and plasmapheresis. However, these procedures have thus far been utilized only in rare individual cases.

CASE REPORT

A 32-year-old man presented to our emergency department with skin and scleral jaundice, dark urine and diffuse abdominal pain. He had a history of haemophilia A with factor VIII substitution. The patient had been indiscriminately taking steroids (trenbolone) and human growth hormone, combined up to 30 tablets a day, intermittently over several years to support his weight training.

Upon admission laboratory test results revealed elevated transaminases and cholestasis parameters with leading hyperbilirubinemia of 26.5 mg/dl. Coagulation parameters were within normal limits. Laboratory test results are summarized in Table 1. Clinical examination revealed no evidence of hepatic encephalopathy, and

physical examination showed diffuse abdominal tenderness and jaundice as the only notable abnormalities. Ultrasound and CT abdomen examinations ruled out intra- or extrahepatic cholestasis, as well as liver tumor or vascular diseases. Microbiology, virology and autoimmune serology results were unremarkable. Additionally, there was no evidence of Wilson's disease, alpha-1-antitrypsin deficiency, or haemochromatosis. Liver biopsy revealed a regular lobular structure of the parenchyma without evidence of cirrhotic remodeling but with signs of cholestasis and the presence of intracellular eosinophilic pigmented material, consistent with steroid-induced cholestatic liver disease.

Table 1: Laboratory values.

Laboratory tests	Normal range	On admission	Two weeks later	Pre-dialysis	At last follow-up
White blood cells	4-9 G/l	9.58	11.62	9.98	8.45
Hemoglobin	14-18 g/dl	14.9	12.5	10.6	10.4
Platelets	150-450 G/l	422	382	449	267
Sodium	135-145 mmol/l	135	140	141	140
Potassium	3.5-5.0 mmol/l	4.4	4.5	4.4	5.3
Alanine aminotransferase (ALT)	10-50 U/l	100	89	68	182
Aspartate aminotransferase (APT)	10-50 U/l	74	58	57	112
Alkaline phosphatase	40-129 U/l	427	435	369	263
Creatinine	0.7-1.3 mg/dl	0.8	1.5	2.4	1.4
Urea	7-18 mg/dl	10	18	24	27
Bilirubin, total	<1.2 mg/dl	29.7	45.6	44.1	5.4
Bile acids	<10 µmol/l		136	99	16
INR		0.9	0.9	1.0	1.0

Subsequently, kidney function parameters deteriorated. Ultrasound examination of the kidney revealed acute renal failure without evidence of obstructive uropathy. Urine sediment analysis showed tubular damage with proteinuria of 403 mg/g creatinine without dysmorphic red blood cells. The urine test strip was strongly positive for bilirubin. Transjugular renal biopsy showed bile cast nephropathy with moderate acute tubular injury, interstitial inflammation, and the presence of intraluminal and intracytoplasmic bile casts (Figure 1, 2).

Initially, extracorporeal albumin dialysis was performed twice by Advanced Organ Support (ADVOS) system at three-day intervals (first treatment over 8 h, second treatment over 18 h). However, since the bilirubin (max. 50.2 mg/dl), bile acids (max. 161 µmol/l), transaminases and kidney function parameters (creatinine max. 8.3 mg/dl) subsequently increased again, treatment continued with one session of hemodialysis followed by plasmapheresis. On the following day, extracorporeal albumin dialysis using the ALBUNIQUE system (Fresenius Multifiltrate, Albutec GmBH) was initiated for the next 13 days with a total of 7 treatments (on average over 6.85 hours). This resulted in a significant improvement in laboratory values (bilirubin 10.9 mg/dl, bile acids 60 µmol/l, creatinine 1.9 mg/dl), and the patient was discharged home. Figures 3 and 4 depict the trends of creatinine, bilirubin, and bile acids.

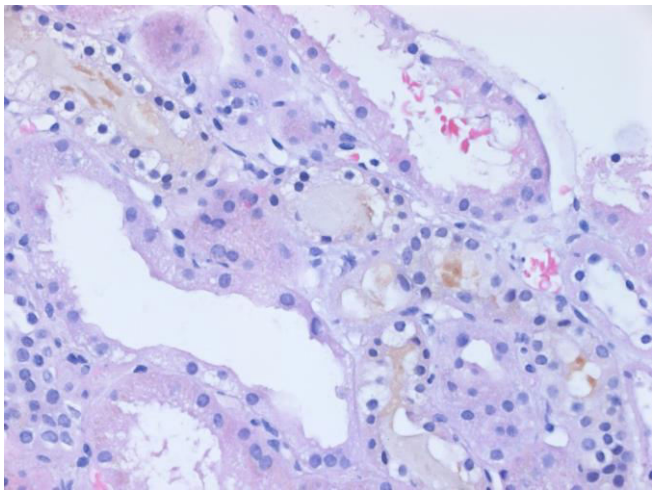

Figure 1: H&E shows tubules with intraluminal and intracytoplasmic brown pigment consistent with bile casts.

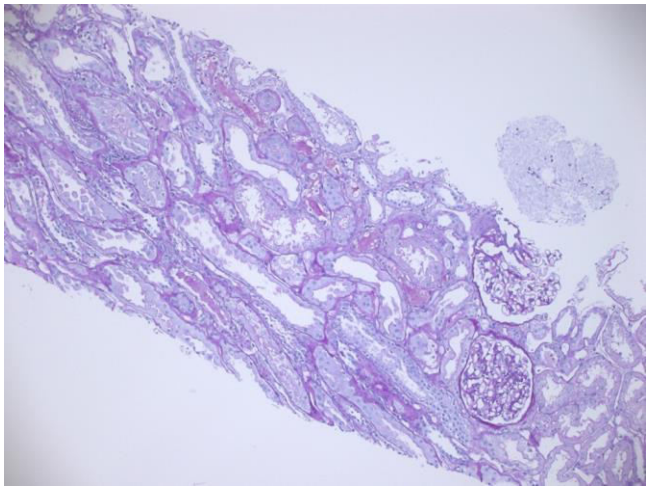

Figure 2: H&E shows two glomeruli and several tubules with moderate acute tubular injury and interstitial cell proliferation.

Successful Treatment of Cholemic Nephropathy by Extracorporeal Albumin Dialysis in Liver Failure. Journal of Nephrology & Kidney Diseases. 2024; 6(1):139.

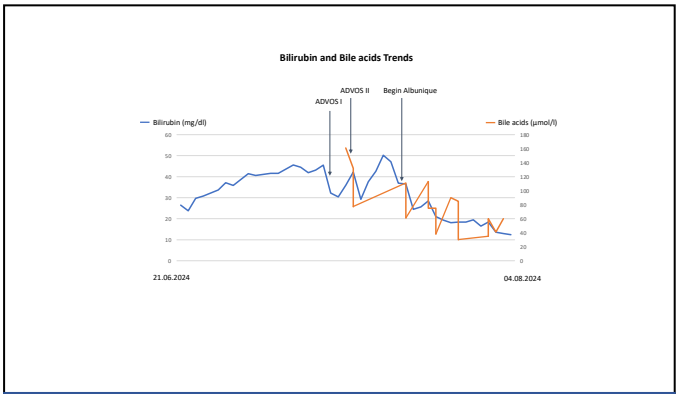

Figure 3: Bilirubin and Bile acids Trends.

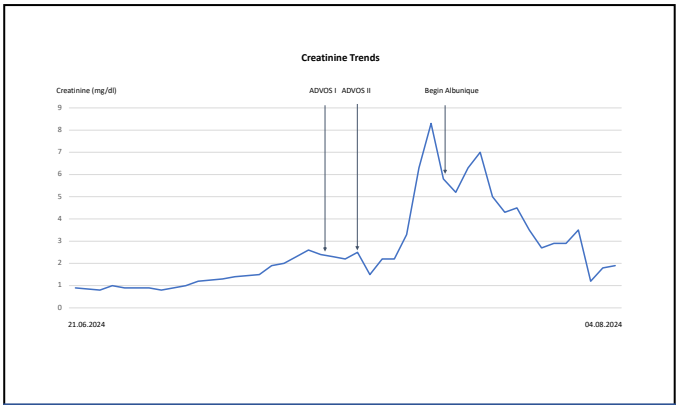

Figure 4: Creatinine Trends

During outpatient follow-up after two weeks, improved laboratory parameters (bilirubin 3.8 mg/dl, bile acids 16 µmol/l, creatinine 1.2 mg/dl) were observed, along with decreasing proteinuria (338 mg/g creatinine) in the urine sediment and stable clinical findings.

The patient was fully informed about his diagnosis and treatment options throughout the treatment and subsequently about the publication of his case and gave his written consent.

DISCUSSION

Prevalence, etiology, and pathophysiology

Acute renal failure is a common complication in patients with liver disease. The incidence of acute renal failure in hospitalized patients with liver cirrhosis tripled in the last 10 years [1]; in ICU patients, an incidence of 30-60% has been reported [2]. It´s associated with poor clinical outcomes and high mortality rates, as the mortality rate in acute renal failure is approximately three times higher than in chronic renal failure alone or in the absence of renal dysfunction [1]. Therefore, early diagnosis and prompt treatment plays a crucial role. In most cases, the cause of acute renal failure is prerenal. In intensive care units, sepsis is described as the most common cause of acute renal failure, accounting for 38.6% [2]. Hepatorenal syndrome also plays a major role with a reported incidence of 20% in hospitalized patients with liver cirrhosis [3]. Cholemic nephropathy, as a differential diagnosis, is often overlooked, despite various studies indicating that its incidence is higher than previously believed. For instance, van Slambrouck, et al. [4] demonstrated the presence of bile casts in 55% of kidney autopsies or biopsies of icteric patients, 85% of whom had a the pre-mortem diagnosis of hepatorenal syndrome. Similary, Nayak, et al. [5] conducted a clinicopathologic study, finding bile casts in 44.8% of kidney autopsies of patients with a pre-mortem diagnosis of hepatorenal syndrome. These studies highlight the importance of considering cholemic nephropathy as a significant differential diagnosis, particularly in distinguishing it from hepatorenal syndrome.

Hepatorenal syndrome is a diagnosis of exclusion and and requires the elimination of other potential etiologies. Distinguishing features from cholemic nephropathy include the lack of response to terlipressin, noradrenaline and albumin, which are standard treatments for hepatorenal syndrome. Additionaly, cholemic nephropathy is characterised by typical structural changes, such as histomorphological changes and abnormal urine findings.

Cholemic nephropathy was first described by Quincke in 1899, who observed these changes in kidney autopsies of patients with icterus and acute kidney injury. The pathomechanism is not fully understood. Normally, bile acids undergo the enterohepatic circulation, whereas the enterorenal circulation plays a secondary role. However, in the context of cholestasis, compensatory mechanisms lead to increased elimination of bile acids via urine. This is facilitated by upregulated transport mechanisms in hepatocytes, inhibition of reabsorption in proximal tubular cells, and increased tubular secretion. Renal clearance of bilirubin thus increases from less than 5% to almost 90% [6] . Ghallab, et al. [7] were able to show that the transport protein SLC10A2 (also known as ASBT), produced also by human kidney cells, is responsible for the accumulation of bile acid and that the progression of cholemic nephropathy could be prevented by inhibiting this protein in the mouse

model. The exact pathogenic effect of bile acids and bilirubin on the kidney remains controversial. It is hypothesized that direct toxicity of bilirubin and bile acids may contribute to tubulointerstitial nephritis. Additionally, the formation of "bile casts" may lead to tubular obstruction [8-10].

Approach and diagnosis

Achieving an accurate diagnosis of acute kidney injury in patients with liver failure is often challenging. Creatinine, typically used as a routine parameter, has limitations in this context. Furthermore, the possibility of performing a biopsy, currently considered the gold standard for diagnosis, is restricted in cases where coagulation problems are present. Nonetheless, early diagnosis and prompt treatment are crucial, underscoring the need for simple and early diagnostic options to be established. Indeed, there are certain indicators that can aid in the diagnosis of cholemic nephropathy.

Firstly, serum bilirubin levels should be considered as a non-invasive diagnostic parameter. While a serum bilirubin level of 20 mg/dl is commonly regarded as the cut-off value, it's worth noting that case reports and autopsy studies have documented the presence of cholemic nephropathy at serum bilirubin levels below this threshold. However, it has been suggested that this may occur in cases of prolonged elevated bilirubin levels, such as in chronic obstructive jaundice [2,11]. In the context of acute liver failure, histomorphological changes indicative of cholemic nephropathy are typically observed at serum bilirubin levels above 20 mg/dl [11] and a concomitant increase in creatinine and bilirubin has been documented [12]. Secondly, urine examination plays a crucial role in the diagnosis, particularly with the detection of bile casts in the urine sediment, which serves as an important diagnostic criterion [3,6,13]. Maiwall, et al. [14] have demonstrated that in cases where histological findings of cholemic nephropathy are present, bilirubin and its degradation product urobilinogen can be detected in the urine. In summary, serum bilirubin levels exceeding 20 mg/dl should prompt attention, followed by a detailed urine examination. Urinalysis stands out as a cost-effective, non-invasive, readily available, and straightforward diagnostic option.

Currently, a definitive diagnosis of cholemic nephropathy is typically achieved through biopsy and subsequent histological examination. Transjugular biopsy is a good alternative in settings with coagulation problems. Studies point it out as a safe and effective method [15,16]. Additionally, compared to classic transcutaneous renal biopsy, transjugular biopsy offers superior capability for capturing material in the area of the distal nephron, where bile casts predominantly form [6].

Bile casts promoting factors (BCPFs), such as metabolic acidosis and hypalbuminemia, are commonly observed in patients with concurrent kidney and liver failure and warrant attention. Hypalbuminemia contributes to reduced renal blood flow due to intravascular volume deficiency, as well as to elevated levels of free serum bilirubin, subsequently leading to increased bilirubin excretion in the urine. Metabolic acidosis exacerbates bile acid precipitation and thus increases the formation of bile casts [3].

In our patient, serum bilirubin level upon admission was 26.5 mg/dl. Subsequently, both serum bilirubin and creatinine increased in parallel, reaching maximum values of 50.2 mg/dl and 8,3 mg/dl, respectively. Additionally, the urine test strip showed a strong positive result for bilirubin. A transjugular biopsy was performed to obtain an exact diagnosis, confirming the presence of bile cast nephropathy. Taking together the kidney function parameters, the serum bilirubin, the urine findings and the clinical presentation, it can be discussed whether the biopsy was really mandatory, or whether it will be needed in future cases.

Treatment and outcome

As mentioned earlier, early treatment is imperative to prevent long-term kidney damage in cholemic nephropathy [17]. However, established treatment guidelines are currently lacking. Generally, therapy focuses on two main objectives: addressing underlying causes and reducing levels of bilirubin and bile acids. Additionally, it is crucial to recognize and manage bile casts promoting factors (BCPFs), and in cases of severe renal dysfunction, hemodialysis may be necessary. Extracorporeal procedures can be employed to eliminate bilirubin and bile acids. These procedures involve plasmapheresis or extracorporeal albumin dialysis. Extracorporeal Albumin dialysis in general removes water-soluble substances bound to albumin, such as bilirubin, bile acids and other albumin-bound toxins that can only be detected by the albumin binding capacity. Various options exist for extracorporeal albumin dialysis, such as MARS (Molecular Adsorbent Recirculating System), ALBUNIQUE,

ADVOS (Advanced Organ Support), and SPAD (Single Pass Albumin Dialysis). MARS dialysis is effective for eliminating bile acids, but hemodialysis can only be carried out to a limited extent. In contrast, ADVOS dialysis offers effective hemodialysis, CO2 elimination, and adjustment of acid-base balance and electrolytes. However, ADVOS dialysis is a complex procedure primarily utilized in intensive care units, and its additional features may not be necessary in most cases. In our case, we opted for ALBUNIQUE (Albutec GmbH, Rostock), which utilizes a new adsorber with enhanced albumin binding capacity, resulting in improved elimination of albumin-bound toxins. It's important to consider the different consumption rates of human albumin with each dialysis method: ADVOS dialysis requires low consumption (40 g per session), ALBUNIQUE requires moderate consumption (80 g per session), while MARS dialysis demands higher consumption (120 g per session) and SPAD dialysis entails the highest consumption (260 g per session) as the albumin dialysate is not regenerated.

Several studies have compared SPAD and MARS dialysis. One study involving patients with liver failure demonstrated that both methods could reduce serum bilirubin levels, while only MARS dialysis positively impacted bile acids, kidney function parameters, and albumin binding capacity [18]. A more recent study found equivalence between SPAD and MARS dialysis in terms of reducing total and conjugated bilirubin and bile acid levels, with no significant differences observed in patients with liver failure [19]. However, it's worth noting that the trend of bile acid reduction there was higher with MARS, and the duration of the two methods differed (8 hours with MARS compared to 10 hours with SPAD).

In comparison to albumin dialysis, plasmapheresis is a well-established procedure with known benefits. Plasmapheresis has a positive influence on blood coagulation and can aid in replenishing albumin levels, as well as hepatic regenerative stimulating factors [6], especially when FFP is used as a substitute. Case reports involving patients with bile cast nephropathy have demonstrated promising results with resolution of kidney injury with plasmapheresis [20,21]. However, it's important to acknowledge that the requirement for FFP substitution is not without concerns.

In this case report, the treatment protocol consisted of two initial sessions of ADVOS dialysis followed by seven sessions of ALBUNIQUE dialysis. After performing the ADVOS dialysis, a recurred increase of laboratory values (bilirubin, bile acids, kidney function parameters) was recorded, which we attribute to the three-day interval between treatments and the insufficiency of only two sessions. Subsequent to the seven sessions of ALBUNIQUE dialysis, there was a notable decrease in the aforementioned laboratory values and an improvement in the patient´s clinical presentation. Notably, in this case, ALBUNIQUE dialysis emerged as the optimal treatment option, as there was no requirement for hemodialysis or adjustments to the acid-base balance and electrolytes. Furthermore, the patient did not necessitate intensive care unit treatment with ALBUNIQUE dialysis. To the best of our knowledge, this is the first case report on bile cast nephropathy successfully treated with ALBUNIQUE dialysis.

A review [6] summarized case reports from 2000 to 2020 on histologically confirmed bile cast nephropathy and their treatment modalities and outcomes. In total, 22 case reports involving 87 patients were described. Among the various treatment modalities, hemodialysis was the most frequently performed treatment, documented in 11 cases. Plasmasepheresis was utilized in only one case [20], as well as albumin dialysis [22].

Overall, there are few case reports documenting the treatment of bile cast nephropathy with extracorporeal albumin dialysis. In the literature we identified three such reports: First, Issac, et al. [23] reported a case of bile cast nephropathy and obstructive jaundice successfully treated with five sessions of MARS dialysis. Saich, et al. [24] documented about a case of secondary renal impairment due to BRIC (Benign recurrent intrahepatic cholestasis) effectively treated with three sessions of MARS dialysis. Third, a patient with TCF 2 Mutation induced biliary duct dystrophy and bile cast nephropathy, was initially treated with hemodialysis, followed by one session of MARS and eight sessions of SPAD. This treatment regimen resulted in a significant improvement in kidney function, although simultaneous liver and kidney transplantation became necessary later on due to other complications in the patient´s clinical course [22].

Overall, good outcomes have been documented with extracorporal procedures in the case reports described above, as well as in our case However, it's important to recognize that

these procedures are associated with high costs, effort, and expertise, and therefore should be utilized only as a temporary solution after careful verification of the indication. Further studies are warranted to compare different treatment methods and establish standards for the management of cholemic nephropathy in the future. The particular focus should be placed on investigating ALBUNIQUE dialysis, as a new innovation with higher albumin binding capacity. To the best of our knowledge, this is the first case report using ALBUNIQUE dialysis to treat bile cast nephropathy.

CONCLUSION

In patients with liver disease and acute kidney injury, it is crucial to determine serum bilirubin and bile acids, as well as urinalysis at an early stage. These simple measures can differentiate between bile cast nephropathy and hepatorenal syndrome, thereby avoiding unnecessary treatments. These tests provide a straightforward, cost-effective, and non-invasive means of suspecting cholemic nephropathy. Early treatment can prevent long-term kidney injury and alleviate other symptoms resulting from elevated bile acids, hyperbilirubinemia and other albumin-bound toxins. Extracorporeal albumin dialysis offers promising treatment options, although it has so far only been documented in individual case reports. This is the first case report about bile cast nephropathy treated with ALBUNIQUE dialysis. While further studies are necessary to establish therapeutic standards, treatment is already feasible and indeed essential.

REFERENCES

1. Cullaro G, Kanduri SR, Velez JCQ. (2022). Acute Kidney Injury in Patients with Liver Disease. Clin J Am Soc Nephrol. 17: 1674-1684.
2. Mohamed A, Peniston M, Mahmood R. (2023). Acute Kidney Injury in Patients Admitted to the Intensive Care Unit: A Case Report. Cureus. 15: e40380.
3. Tinti F, Umbro I, D'Alessandro M, Lai S, Merli M, et al. (2021). Cholemic Nephropathy as Cause of Acute and Chronic Kidney Disease. Update on an Under-Diagnosed Disease. Life (Basel). 11.
4. van Slambrouck CM, Salem F, Meehan SM, Chang A. (2013). Bile cast nephropathy is a common pathologic finding for kidney injury associated with severe liver dysfunction. Kidney Int. 84: 192-197.
5. Nayak SL, Kumar M, Bihari C, Rastogi A. (2017). Bile Cast Nephropathy in Patients with Acute Kidney Injury Due to Hepatorenal Syndrome: A Postmortem Kidney Biopsy Study. J Clin Transl Hepatol. 5: 92-100.
6. El Chediak A, Janom K, Koubar SH. (2020). Bile cast nephropathy: when the kidneys turn yellow. Renal Replacement Therapy. 6: 15.
7. Ghallab A, González D, Strängberg E, Hofmann U, Myllys M, et al. (2024). Inhibition of the renal apical sodium dependent bile acid transporter prevents cholemic nephropathy in mice with obstructive cholestasis. J Hepatol. 80: 268-281.
8. Sequeira A, Gu X. (2015). Bile cast nephropathy: an often forgotten diagnosis. Hemodial Int. 19: 132-135.
9. Al Awadhi H, Al Qassimi S, Akhras A, Herlitz L, Ghosn M. (2021). Bile acid nephropathy induced by anabolic steroids: A case report and review of the literature. Clin Nephrol Case Stud. 9: 123-129.
10. Alkhunaizi AM, ElTigani MA, Rabah RS, Nasr SH. (2016). Acute bile nephropathy secondary to anabolic steroids. Clin Nephrol. 85: 121-126.
11. Foshat M, Ruff HM, Fischer WG, Beach RE, Fowler MR, et al. (2017). Bile Cast Nephropathy in Cirrhotic Patients: Effects of Chronic Hyperbilirubinemia. Am J Clin Pathol. 147: 525-535.
12. Somagutta MR, Jain MS, Pormento MKL, Pendyala SK, Bathula NR, et al. (2022). Bile Cast Nephropathy: A Comprehensive Review. Cureus. 14: e23606.
13. Bräsen JH, Mederacke YS, Schmitz J, Diahovets K, Khalifa A, et al. (2019). Cholemic Nephropathy Causes Acute Kidney Injury and Is Accompanied by Loss of Aquaporin 2 in Collecting Ducts. Hepatology. 69: 2107-2119.
14. Maiwall R, Pasupuleti SSR, Bihari C, Rastogi A, Kumar Singh P, et al. (2020). Incidence, Risk Factors, and Outcomes of Transition of Acute Kidney Injury to Chronic Kidney Disease in Cirrhosis: A Prospective Cohort Study. Hepatology. 71: 1009-1022.
15. P Cluzel 1, F Martinez, M F Bellin, Y Michalik, H Beaufils, et al. (2000). Transjugular versus percutaneous renal biopsy for the diagnosis of parenchymal disease: comparison of sampling effectiveness and complications. Radiology. 215: 689-693.

16. R Sam, DJ Leehey, MM Picken, MA Borge, EM Yetter, et al. (2001). Transjugular renal biopsy in patients with liver disease. Am J Kidney Dis. 37: 1144-1151.
17. Tanner GA, Evan AP. (1989). Glomerular and proximal tubular morphology after single nephron obstruction. Kidney Int. 36: 1050-1060.
18. Christoph Sponholz, Katja Matthes, Dina Rupp, Wolf Backaus, Sebastian Klammt, et al. (2016). Molecular adsorbent recirculating system and single-pass albumin dialysis in liver failure--a prospective, randomised crossover study. Crit Care. 20: 2.
19. Grégoire Wallon, Cécile Guth, Céline Guichon, Sylvie Thevenon, Mathieu Gazon, et al. (2022). Extracorporeal Albumin Dialysis in Liver Failure with MARS and SPAD: A Randomized Crossover Trial. Blood Purif. 51: 243-250.
20. Flores A, Nustas R, Nguyen HL, Rahimi RS. (2016). Severe Cholestasis and Bile Acid Nephropathy From Anabolic Steroids Successfully Treated With Plasmapheresis. ACG Case Rep J. 3: 133-135.
21. El Khoury C, Sabbouh T, Farhat H, Ferzli A. (2017). Severe Cholestasis and Bile Cast Nephropathy Induced by Anabolic Steroids Successfully Treated with Plasma Exchange. Case Rep Med. 2017: 4296474.
22. Sens F, Bacchetta J, Rabeyrin M, Juillard L. (2016). Efficacy of extracorporeal albumin dialysis for acute kidney injury due to cholestatic jaundice nephrotoxicity. BMJ Case Rep. 2016.
23. Issac AG, Yu MA, Rogers DM, Subramanian RM. (2023). Case Report: Efficacy of albumin dialysis for the reversal of bile cast nephropathy-induced acute kidney injury. Front Nephrol. 3: 1256672.
24. Saich R, Collins P, Ala A, Standish R, Hodgson H. (2005). Benign recurrent intrahepatic cholestasis with secondary renal impairment treated with extracorporeal albumin dialysis. Eur J Gastroenterol Hepatol. 17: 585-588.