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DISTAL RENAL TUBULAR ACIDOSIS SECONDARY TO HYPERBILIRUBINEMIA

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ABSTRACT

Introduction: Bile cast nephropathy (BCN) is a rare and often overlooked cause of acute kidney injury (AKI), usually seen in the context of severe hyperbilirubinemia. It results from the deposition of bile pigments and bile casts in renal tubules, specifically distal and collector tubules, leading to obstruction and direct tubular toxicity.

Case description: We describe the case of 49-year-old male who presented with severe jaundice and oliguric AKI. With the gradual resolution of liver dysfunction, the patient's kidney function also improved. However, during the recovery phase, the patient developed persistent hypokalaemia and normal anion gap metabolic acidosis. Further work-up was consistent with distal renal tubular acidosis (dRTA).

Discussion: While BCN is being increasingly recognized in patients with cholestatic liver diseases, complications arising during the recovery phase – particularly acid-base disturbances like dRTA – are rarely reported. In this case, the onset of dRTA during renal recovery may reflect delayed or selective tubular healing after bile-induced injury. This case highlights the importance of continued monitoring for renal tubular defects even after apparent improvement in glomerular function.

Conclusion: This case emphasizes the need to consider dRTA as a potential complication in patients recovering from BCN. Close follow-up of electrolyte and acid-base parameters is advised during renal recovery, especially in the presence of persistent hypokalaemia.

KEYWORDS

Bile cast nephropathy, hyperbilirubinemia, acute kidney injury, electrolyte imbalance, distal renal tubular acidosis

LEARNING POINTS

- Bile cast nephropathy (BCN) is an under-recognized cause of acute kidney injury (AKI) in patients with severe hyperbilirubinemia and should be considered in the differential diagnosis of AKI in jaundiced patients.
- Distal renal tubular acidosis (dRTA) can emerge during the recovery phase of BCN-related AKI, possibly due to tubular dysfunction from bile-induced injury, highlighting the need for close metabolic monitoring after renal recovery.
- The co-occurrence of BCN and dRTA emphasizes the importance of serial electrolyte assessments and acid-base evaluation in jaundiced patients with AKI, even after apparent improvement in renal function.

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INTRODUCTION/BACKGROUND

Acute kidney injury (AKI) is a common complication in patients with decompensated cirrhosis, affecting up to 50% of hospitalized cases[1]. The majority of cirrhosis-associated AKI cases are pre-renal or functional in nature, often driven by hemodynamic alterations and splanchnic vasodilation. Among the primary causes are pre-renal AKI, hepatorenal syndrome, and acute tubular necrosis (ATN), each with distinct pathophysiologic mechanisms and management strategies[1,2].

Bile cast nephropathy (BCN), also referred to as cholaemic nephrosis, is a lesser-known cause of intrinsic AKI in cirrhosis in the context of severe hyperbilirubinemia and cholestasis[3]. It is characterized by the intratubular deposition of bile casts, resulting in direct toxicity and obstruction. Although under-recognized, BCN can lead to significant renal impairment[3]. Fortunately, with appropriate treatment of the underlying liver dysfunction, renal function often recovers without lasting damage.

Here, we present the case of BCN complicated by dRTA during the recovery phase of AKI. To our knowledge, this is among the first descriptions of such an association in the literature.

CASE DESCRIPTION

A 49-year-old Caucasian male with a history of alcohol-related cirrhosis (Child-Pugh B, diagnosed in 2018) presented to the emergency department with a 2-week history of progressive jaundice, pruritus, and abdominal discomfort. He had not engaged in medical follow-up since 2023. His history was notable for heavy alcohol use (1-2 bottles of whiskey daily for 30 years, ceased 6 weeks prior) and chronic tobacco use. There was no family history of liver or kidney disease.

On examination, the patient was alert and oriented but appeared jaundiced, with a distended abdomen and bilateral lower extremity oedema. No asterixis or signs of hepatic encephalopathy were observed. Initial laboratory results showed a potassium of 2.6 mmol/l, bicarbonates of 23 mmol/l, creatinine of 2.3 mg/dl, total bilirubin of 31.4 mg/dl (predominantly conjugated), aspartate aminotransferase of 184 U/l, alanine aminotransferase of 83 U/l, alkaline phosphatase of 271 U/l, gamma-glutamyl transferase of 217 U/l, C-reactive protein of 48.8 mg/dl, albumin of 29 g/l. His international normalized ratio (INR) was 1.89. Compared to baseline labs from prior outpatient visits, he had a potassium of 4.2 mmol/l, bicarbonates of 25 mmol/l, creatinine of 0.7 mg/dl, total bilirubin of 1.9 mg/dl, and his INR was 1.04.

Urinalysis revealed the presence of bile, a urinary sodium < 20 mmol/l, a urinary potassium of 51 mmol/l, a microalbuminuria of 8.7 mg/l, and a β2-microglobulinemia of 7.7 mg/l. Abdominal computed tomography (CT) scan showed a dysmorphic liver without biliary obstruction or focal lesions.

The patient was admitted with presumed alcoholic hepatitis and decompensated cirrhosis. His Maddrey score was 82. A transjugular liver biopsy confirmed severe acute alcoholic hepatitis. Workup for alternative aetiologies of acute liver failure – including viral serologies, autoimmune markers, and vascular imaging - was negative.

Supportive care included fluid administration, electrolytes correction, cessation of nephrotoxic agents, albumin infusions, and corticosteroids for confirmed alcoholic hepatitis. Despite initial improvement in both liver and kidney function (creatinine 0.99 mg/dl and bilirubin 16.99 mg/dl by day 12), the patient continued to have a normal anion gap metabolic acidosis and persistent hypokalaemia.

Urine studies performed after the normalization of renal function revealed a urinary potassium of 51 mmol/l, a urinary chloride of 30 mmol/l, a urinary sodium <20 mmol/l,

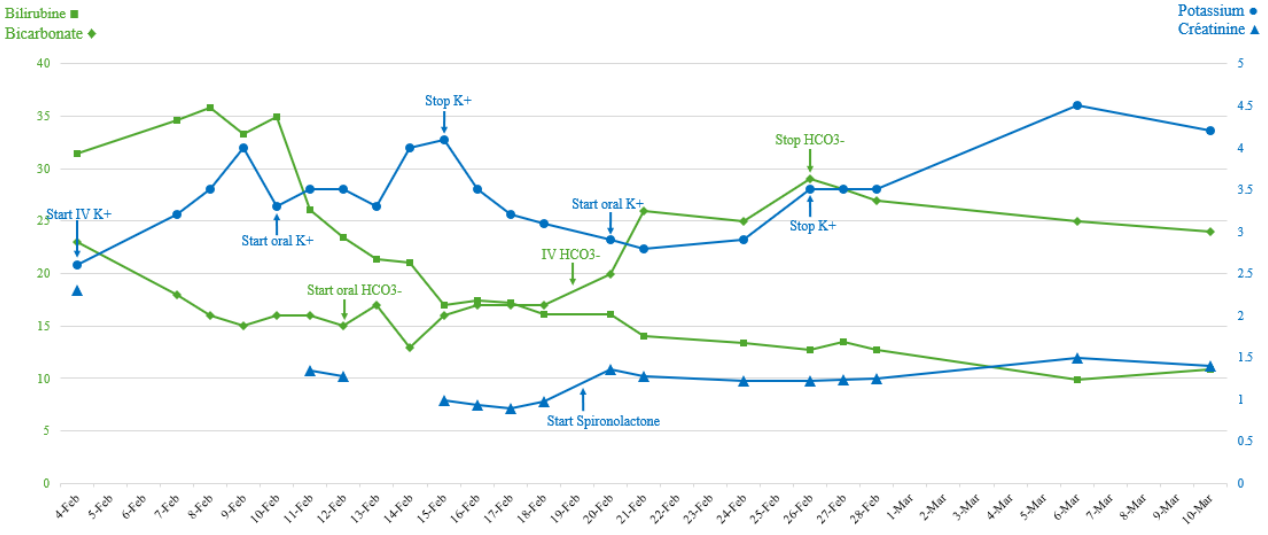


Figure 1. Evolution of renal and hepatic markers during bile cast nephropathy (BCN) and distal renal tubular acidosis (dRTA). Bilirubin and bicarbonate are shown on the primary Y-axis; Potassium and creatinine are shown on the secondary Y-axis. Treatment modifications are indicated on the chart. Missing creatinine values are due to interference from high bilirubin levels with the local laboratory's assay method. Values on February 11th and 12th were obtained from an external laboratory using a different measurement technique.

Date	Bilirubin (mg/dl)	Creatinine (mg/dl)	Bicarbonate (mmol/l) |	Potassium (mmol/l)
4 Feb	31.4	2.3	23	2.6
7 Feb	34.57	-	18	3.2
8 Feb	35.84	-	16	3.5
9 Feb	**33.3**	-	15	4
10 Feb	34.89	-	16	3.3
11 Feb	26.06	1.34	16	3.5
12 Feb	23.43	1.28	15	3.5
13 Feb	21.42	-	17	3.3
14 Feb	21.08	-	13	4
15 Feb	16.99	0.99	16	4.1
16 Feb	17.38	0.94	17	3.5
17 Feb	17.17	0.9	17	3.2
18 Feb	16.16	**0.97**	17	3.1
20 Feb	16.09	1.36	20	2.9
21 Feb	14.02	1.27	26	2.8
24 Feb	13.39	1.22	25	2.9
26 Feb	12.72	1.22	**29**	**3.5**
27 Feb	13.44	1.23	28	3.5
28 Feb	12.76	1.25	27	3.5
6 Mar	9.9	1.5	25	4.5
10 Mar	10.9	1.4	24	4.2

Table 1. Daily laboratory values during the course of bile cast nephropathy (BCN) and distal renal tubular acidosis (dRTA).
Bold values indicate clinically significant time points: Bilirubin on February 9th corresponds to the initiation of methylprednisolone. Creatinine on February 18th marks the addition of spironolactone, which may explain the subsequent transient rise. Bicarbonate on February 26th, and potassium on the same day, represent the discontinuation of bicarbonate and potassium supplementation, respectively. Creatinine values on February 11th and 12th were measured by an external laboratory using a different method due to interference from elevated bilirubin levels.

and a microalbuminuria of 16.7 mg/l. The urinary anion gap (UAG) was positive, and the transtubular potassium gradient (TTKG) was > 7, suggestive of ongoing renal potassium loss - findings consistent with dRTA.

Treatment included intravenous potassium and bicarbonates supplementation, later transitioned to oral formulations. Spironolactone was added both for volume management and its potassium-sparing effects. The patient's electrolyte and acid-base status stabilized during the stay. At discharge (February 28th 2025), potassium and bicarbonate levels were normalized. Daily laboratory values during the course of BCN and dRTA are reported in *Table 1*. The evolution of renal and hepatic markers is shown in *Fig. 1*. Outpatient labs on days 6 and 10 post-discharge remained stable.

DISCUSSION

This case demonstrates a rare biphasic renal complication in the context of cholestatic liver injury. BCN occurs due to the nephrotoxic effects of bile acids and bilirubin[4]. Although recovery is usually gradual, our patient developed a delayed and unexpected complication: distal renal tubular acidosis.

dRTA is typically associated with autoimmune diseases or hereditary disorders but can occur due to acquired tubular injury[5]. We hypothesize that bile-induced toxicity caused subclinical tubular dysfunction, particularly affecting the intercalated cells of the distal nephron[6]. As renal filtration normalized, a defect in distal acidification became clinically apparent, leading to persistent metabolic acidosis and hypokalemia[6].

To our knowledge, this is one of the first reported cases of dRTA arising during the recovery phase of BCN. While a renal biopsy was not performed, the diagnosis was supported by clinical, biochemical, and temporal correlations. Awareness of this potential complication is important, as failure to recognize and treat dRTA may lead to ongoing electrolyte derangements and delays in recovery.

CONCLUSION

BCN is a rare but important cause of AKI in the setting of severe hyperbilirubinemia. Although recovery of renal function is often anticipated with resolution of liver dysfunction, clinicians should remain alert to secondary tubular complications such asdRTA. Early recognition and treatment of these disturbances are essential to optimizing outcomes.

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