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Review Article

Bile Cast Nephropathy: Review of Literature Cases

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Abstract

Renal dysfunction due to high bilirubin levels is called bile cast nephropathy (BCN). Toxic effects of bilirubin and bile acids on the renal tubules occur by tubular epithelial injury, tubular cast formation and hemodynamic changes affecting both the kidney and systemic circulation. The diagnosis of BCN is quite difficult. Because of the bleeding disorder in these patients, renal biopsy is difficult to perform. Light microscopy of urinalysis in patients who cannot undergo renal biopsy may help the diagnosis by means of the presence of bile crystals and bilirubin cast. BCN treatment should focus on lowering the high bilirubin levels and management of underlying disease. But, some patients still need temporary plasmapheresis and hemodialysis for improving renal function.

Keywords: hyperbilirubinemia; bile cast nephropathy; acute kidney injury

Introduction

Bilirubin is formed by the destruction of hemoproteins. With the destruction of the heme molecule, biliverdin is formed, then biliverdin turns into bilirubin with the help of the biliverdin reductase enzyme. The bilirubin formed is unconjugated bilirubin. Unconjugated bilirubin is insoluble in water, does not pass into urine and is not excreted in bile. Bilirubin circulates in plasma bound to albumin. Albumin binding prevents indirect bilirubin diffusion to tissues and all its toxic effects. Bilirubin transported to hepatocytes is conjugated with glucuronic acid with bilirubin uridine-diphosphate glucuronyl transferase enzyme and transforms into direct bilirubin form. Direct bilirubin is soluble in water and excreted in bile. Direct bilirubin entering the bile system is excreted fecally, a small amount is reabsorbed through the terminal ileum and excreted in the urine (1-2).

Bile contains mainly bile salts, bile pigments (bilirubin, biliverdin), cholesterol, phospholipid (lecithin) and plasma electrolytes. Cholesterol concentrated in the gallbladder may collapse in some abnormal conditions and form gallstones. Bile acids are the end products of cholesterol metabolism that play critical physiological roles in the body. Cessation of bile flow and accumulation of toxic bile acids during cholestasis is a clinical complication. If cholestase is not treated, it can lead to irreversible tissue fibrosis and liver failure. Other organs beyond the liver can also be affected by cytotoxic bile acids. The kidneys are among the target organs that are severely affected by bile acids. Events resulting from oxidative stress are well-defined mechanisms for the cytotoxicity of bile acids (3).

Bile cast nephropathy (BCN) is an acute kidney injury seen in patients with liver disease and hyperbilirubinemic patients. Numerous liver diseases in adults can lead to BCN. BCN can be seen during the course of following diseases; cholestasis, biliary cirrhosis, alcoholic cirrhosis, bile duct atresia, nonalcoholic hepatitis, sclerosing cholangitis, hepatotoxic drugs (eg steroids), fulminant autoimmune hepatitis, intrahepatic malignancies, obstructive liver cholestasis, epstein barr virus, non-hodgkin lymphoma with liver involvement, jaundice associated with non-immune hydrops fetalis, jaundice associated with infectious mononucleosis and hemolytic jaundice [4-7]. In **Table 1,** the cases who have published BCN in literature are given.

Age/Sex	Diagnosis	Complaint	Total / direct bilirubin	Treatment	Reference
63/male	DM, HT, CKD, colongiocarcinoma	Jaundice, itching, weight loss	36,1/35mg/dl	HD, transhepatic biliary drainage	5
25/female	Acute liver failure Wilson's disease	Shortness of breath, loss of appetite,	24,4-37,8mg/dl	Exitus	6

		fatigue, jaundice, swelling in the abdomen and feet			
64/male	DM, HT, CKD, chronic liver disease	yellow sclerae, dark urine	19,3/16,2mg/dl	Biliary drainage, HD	**8**
58/male	Hemophagocytic lymphohistiocytosis, liver failure	Jaundice, fatigue, generalized weakness, choluria, acholi	52,7/ >30mg/dL	systemic corticosteroids	**9**
56/male	Anabolic steroid use	Jaundice	39,7mg/dl	High-dose steroid interstitial nephritis was initiated and stopped when pathology appeared, Cholestyramine, HD	**10**
64/male	DM, pancreatic adenocarcinoma	jaundice, dark-colored urine, light-colored stools, weight loss	19,6mg/dl	HD	**11**
54/male	DM HT CKD, osteomyelitis	Jaundice, itching, loss of appetite	19,3mg/dl	HD steroid, ursodeoxycholic acid cholestyramine, plasmapheresis, diuretic	**12**
47/male	acute alcoholic hepatitis	yellowish discoloration of eyes, abdominal distension, reduced appetite, high colored urine	26,5-41,7/13,3-23,4mg/dl	Lactulose, albumin, diuretic, antibiotic, pentoxifylline, TDP L-ornithine aspartate, PD, exitus	**13**
61/male	bladder carcinoma, bilateral retinoblastoma, and multiple malignant melanomas, obstructive cholestasis	fatigue, anorexia, severe jaundice	260mg/dl	HD, Endoscopic retrograde cholangiopancreatography with sphincterotomy and stent insertion was performed	**14**
35/male	Hepatitis A	Nausea, abdominal discomfort, oliguria	10,2/7,9mg/dl	HD, hydration	15
35/male	Anabolic steroid	Jaundice, abdominal pain, vomiting, itching	8,4-19,2/6,13mg/dl	Hydration, ursodeoxycholic acid, albumin, plasma exchange	16
60/male	Flucloxacillin induced cholestatic liver dysfunction	Jaundice, tiredness, dark urine	48,8/>33,9mg/dl	Antibiotic was discontinued.	**17**
41/female	Alcohol use is present	Abdominal bloating, jaundice	23,1/20mg/dl	Hydration, albumin, midodrin HD	18
55/male	Colorectal cancer	jaundice, ascites	42,5/25mg/dl	Supportive therapy	19
61/male	obstructive cholestasis	Jaundice	28,0/15,3mg/dl	HD	**20**
73/male	Cholecystectomy, prostate cancer	Abdominal pain, dark urine, light-colored stools	39,6/29,9mg/dl	Hydration, bile drainage, HD	21
22/male	Hepatitis A	fever, jaundice, loose stools, and vomiting	40/30mg/dl	Hydration antibiotic and HD	22
57/male	Schizophrenia, acute-on-chronic liver failure Paliperidone drug	jaundice	31,3mg/dl	Hydration, albumin, octreotide, midodrin, HD, exitus	23
29/male	Type 1 DM, hepatitis, cirrhosis, AKI from E. coli bacteremia	abdominal pain, jaundice,	23,7mg/dl	Liver transplant, HD	23
61/male	obstructive cholestasis, bile duct stones.	fatigue, anorexia, vomiting, severe jaundice.	32.6 mg/dL	Endoscopic retrograde cholangiopancreatography, cholecystectomy	24
38/male	Epstein-Barr virus	fever, progressive jaundice, cervical lymphadenopathy,	36,1 mg/dl	conservative treatment	25

		hepatomegaly, oliguric			
46/male	Malignant cholangiocarcinoma	jaundice	30 mg/dl	Exitus	26
41/male	Anabolic androgenic steroids	jaundice, fatigue, loss of appetite, weight loss, pruritus, clay-colored stools, dark urine.	47.9 mg/dl	hydroxyzine, cholestyramine, and ursodiol,	27
30/male	Stanozolol induced cholestatic jaundice	jaundice, nausea, vomiting, generalized malaise.	48/28mg/dl	HD	28
43/male	Stanozolol induced cholestatic jaundice	generalized jaundice	49/45mg/dl	HD	28
37/male	MODY	Cholestasis, acute kidney injury	20.1 mg/dl	extracorporeal albumin dialysis, liver–kidney transplantation	29
60/male	flucloxacillin-induced hepatitis	jaundice, generalized fatigue, dark urine.	51.5 />33,9mg/dl)	Medicine was discontinued	30

Table 1: Bile cast nephropathy cases

AKI; acute kidney ınjury, CKD; chronic kidney disease, DM; diabetes mellitus, HD; hemodialysis, HT;hypertension, MODY; maturity onset diabetes of the young

In a patient with jaundice, the etiology of acute kidney injury usually includes hypovolemia, infection, hepatorenal syndrome, and acute tubular necrosis. Severe hyperbilirubinemia (> 20mg / dl) can compromise kidney function and lead to kidney damage. While serum bilirubin levels above 20 mg / dL are required for bilirubin cast formation in acute liver failure with BCN, much lower serum bilirubin levels in chronic liver disease can potentially lead to bilirubin cast formation. Therefore, BCN should still be considered in cirrhotic patients with renal insufficiency, even in the absence of high hyperbilirubinemia. In a study, serum bilirubin levels were found to be normal in many cases with bilirubin cast on histology. Interstitial fibrosis and tubular atrophy were more common in patients with BCN, suggesting that chronic episodic hyperbilirubinemia in cirrhosis leads to long-term kidney damage. Therefore, it has been emphasized that bilirubin levels alone cannot be used as an absolute criterion for suspicion of BCN [4-5, 31].

The mechanism of acute kidney injury in BCN is multifactorial. It is known that bilirubin cast has a direct toxic effect on renal tubule cells with obstruction of tubules. Another mechanism of hyperbilirubinemia is its effect on systemic and renal hemodynamics. It causes splenic and systemic vasodilation, resulting in a low glomerular filtration rate. In addition, bile acids can be directly toxic, and high levels of bile salts in serum have negative inotropic and chronotropic effects. All they together cause a decrease in cardiovascular function and renal hypoperfusion due to combination of peripheral vascular resistance, endotoxemia, hypoalbuminemia and other mechanisms [5,8,11,32].

Bilirubin exerts an oxidative stress on the tubules leading to damage to the tubular cell membranes. This damage can lead to tubular cell hypertrophy in some patients. In Holmes's autopsies, swelling of the tubular epithelium, pigmented cast, hypertrophy, and hyperplasia of the parietal layer of the bowman capsule are shown. Bilirubin also inhibits mitochondrial oxidative phosphorylation, causing further damage to tubular cells. It increases the risk of acute kidney damage in the presence of hypoalbuminemia and acidosis during hyperbilirubinemia. Sulfated bile salts inhibit Na-H- / Na-K / Na-Cl pumps in the proximal tubules and loop of henle, resulting in cellular pH changes that increase bilirubin cast formation and tubular toxicity. Low pH in the distal nephron decreases the solubility of bile, increasing the risk of bilirubin cast nephropathy. When the proximal tubule's bilirubin saturation exceeds normal levels, it causes bilirubin cast formation and tubular occlusion. [11-12,23,32].

The diagnosis of BCN is quite difficult. Because these patients usually have an impaired coagulation profile. Renal biopsy may not be possible due to the high risk of bleeding. The presence of bile crystals and bilirubin cast in urinalysis helps diagnosis. Proximal tubule dysfunction, glucosuria, phosphaturia and microglobunuria can be detected in the laboratory. Low uric acid and phosphorus can be detected in serum. Pigmented bile crystals, natriuresis, beta 2 microglobulinuria and urine concentration defect can be seen in urinalysis. Hyperuricosuria may occur because bilirubin prevents uric acid absorption in the proximal tubules. Potassium loss occurs as a result of tubular injury resulting from bilirubin cast [8,12,32].

Macroscopically, the renal cortex and medulla of patients with hyperbilirubinemia appear yellow due to the presence of bilirubin and bilirubin cast. After formalin fixation of these kidneys, bilirubin turns into biliverdin and its color changes from yellow to green. The green color is more pronounced in the medulla due to the presence of higher cast concentration in distal nephrons. Light microscopy showed intubular yellow-green granular cast, predominantly in the distal tubule and collecting ducts, and minimal bilirubin cast deposition in the proximal tubules. Bilirubin cast consists of epithelial cells and cell-free material. Bilirubin cast can be identified with Hall dye, which detects bilirubin. This dye uses Fouchet's reagent, which converts bilirubin into biliverdine, thus giving it a green color. In these cases, the iron stain is negative (indicates the absence of heme). A spectrum of pathological findings ranging from mild acute tubular damage to epithelial cell swelling and bilirubin cast formation are encountered in kidney biopsies in BCN. BCN is pathologically characterized by renal tubular hypertrophy with the presence of pigmented bile cast in the renal tubular and absence of glomerular pathology. No glomerular abnormalities are detected in light microscopy, immunofluorescence, and electron microscopy[4,12,32-33].

Forty-four patients (23 classified as cirrhotic jaundice, 14 obstructive jaundice, 5 hepatic jaundice, 2 hemolytic jaundice) were included in the study by Van Slambrouck et al. In this study, BCN developed in 24 patients and kidney damage due to other causes developed in 20 patients. In this study, BCN was found in 100% of alcohol-related cirrhosis cases and 50% of combined HCV / alcohol-induced cirrhosis. However, BCN

was not seen in all cases of HCV-related cirrhosis. In patients with BCN, mean total serum bilirubin was 26.2 mg / dl and direct bilirubin was 16.3 mg / dL, while total serum bilirubin was 15.1 mg / dL and direct bilirubin was 9.2 mg / dL in patients without BCN (34). In the study by Foshat et al., autopsies of 94 cases with cirrhosis were examined. BCN was found even in 55% of these cases. 28 of the patients with even BCN were found to be HCV positive (total number of HCV positive cases 49), HCV + alcohol in 17 of the cases (total number of HCV + alcohol 32), and alcoholics in 4 cases (total number of alcoholics 5) were detected. While total bilirubin in cases with even BCN was 10.4 6 ± 12.0, it was observed that the total bilirubin value was 3.5 ± 6 4.3 mg / dl in patients who did not develop it [31].

A total of 127 kidney biopsy specimens were taken in the study conducted by Nayak et al. In this study, BCN was found in 44.8% of the cases (57 cases). BCN was detected in 25/84 (29.7%) of decompensated cirrhosis cases, while acute on chronic liver failure (ACLF) was detected in 32/43 (74.4%) of the patients. In decompensated cirrhosis cases, total bilirubin was 6.2mg / dl (1.0–44.3), while ACLF was 25.8 (5.1–72.8). Bilirubin levels were found to be higher in ACLF cases, which explains the higher rate of BCN. In cases with BCN, total serum bilirubin 27.0 mg / dl (1.5--72.8) and direct bilirubin 16.3 (0.2--45.8) were determined. In patients without BCN, total serum bilirubin level 8.1 (1.0--32.7) and direct bilirubin 2.6 (0.2-- 14.8) were determined. BCN degree; Grade 1+ (1-5 tubular bilirubin cast in distal tubule) in 38.6% of patients, +2 bilirubin cast in distal tubule > 5) in 26.3% of patients and 3+ (multiple bilirubin cast in both distal and proximal tubule) in 35.1% of the patients has been found. In univariate analysis, total bilirubin, direct bilirubin, total leukocyte count, presence of MELD and ACLF were found to be important predictors of BCN in postmortem renal biopsy. In renal biopsy, the best limit for total bilirubin to predict the development of BCN was 14.7 mg / dL and the best limit for direct bilirubin was 6.1 mg / dL [7]

In a prospective study by Mohapatra et al., 110 patients with falciparum malaria complicated by acute kidney injury, jaundice and cerebral malaria were examined for the development of BCN. They found that 20 (18.2%) of these patients had BCN and 15 (13.6%) had hepatorenal syndrome. In cases with BCN, high direct bilirubin (total serum bilirubin 24.7 ± 5.2 and direct bilirubin 14.2 ± 5.3 in cases with BCN, total bilirubin 7.8 ± 3.1 and direct bilirubin 4.2 ± 1.9 in patients without BCN) were detected. Acute kidney damage due to other reasons developed in 75 (68.2%) of the remaining patients. In this study, while mortality was 25% in cases with BCN, it was determined as 10% in those without [35].

There is no accepted treatment guideline for BCN. The aim of treatment is to reduce the bilirubin level and to treat the underlying disease. Treatments for bilirubin reduction can improve kidney function. Percutaneous biliary drainage is one of the treatments aimed at reducing bilirubin. Hemodialysis (MARS, CPFA) and plasmapheresis can be used in the treatment of BCN. Cholestyramine and ursodeoxycholic acid used to lower bile salts are less effective. In addition, lactulose and steroid can be used [11-12].

In conclusion, BCN may develop in most of the diseases that cause bilirubin elevations. BCN was mostly seen the bilirubin level was > 20mg / dl, but it was found to be seen at lower levels It is even emphasized that chronic high bilirubin may also be a cause. Bile cast nephropathy keep in mind in patients with acute kidney injury and hyperbilirubinemia. When BCN is suspected, diagnostic renal biopsy cannot usually be performed due to bleeding disorder. Evaluation of urine by light microscopy in BCN supports the diagnosis of bile crystals and bilirubin cast. Normalization of hyperbilirubinemia and treating underlying disease promptly in the aforementioned patients may improve renal function, but some patients can still need temporary dialysis and plasmapheresis.

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