Annals of Case Reports
McClairen C and Mandaliya R. Ann Case Rep: 8: 1153
www.doi.org/10.29011/2574-7754.101153
www.gavinpublishers.com

Case Report

Case of Cholangiogram Causing Acute Liver Injury Disease

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Citation: McClairen C, Mandaliya R (2023) Case of Cholangiogram Causing Acute Liver Injury Disease. Ann Case Report. 8: 1153. DOI:10.29011/2574-7754.101153

Received: 29 January 2023, **Accepted:** 02 February 2023, **Published:** 06 February 2023

Abstract

There have been prior cases of endoscopic retrograde cholangiopancreatography resulting in a very rare complication of prolonged cholestatic pattern. We present a 49-year-old Hispanic female with a history of type 2 diabetes and chronic kidney disease who had prolonged acute hepatocellular liver injury after undergoing a cholangiogram using Omnipaque contrast. She was discharged home and presented again with the chief complaint of pruritus. Serological and imaging workup was unremarkable. With symptomatic management, her pruritus resolved. On outpatient follow-up, her elevated transaminases resolved. About 10 cases of cholestatic liver injury have been reported after ERCP, however our case is unique being predominantly hepatocellular injury.

Introduction

Acute liver injury following endoscopic retrograde cholangiopancreatography (ERCP) is a rare occurrence. There have been rare cases reporting cholestatic liver injury after ERCP (3-6). We report a case of hepatocellular liver injury after the use of contrast during an intraoperative cholangiogram without biliary etiology noted on follow-up ERCP.

Case Report

A 49-year-old Hispanic female with a history of type 2 diabetes and chronic kidney disease presented to the hospital with midepigastric abdominal tenderness. She was found to have acute cholecystitis on the right upper quadrant ultrasound. On admission, the liver enzymes were completely normal. She subsequently underwent laparoscopic cholecystectomy with intraoperative cholangiogram that was suggestive of a retained stone. During the procedure, 10mL of contrast agent Iohexol (Omnipaque) was used. Laboratory the day after surgery revealed elevated liver function test with total bilirubin 1.8 mg/dL (normal< 1.1 mg/dL), alkaline phosphatase (ALP) 195 IU/L (normal <118 IU/L), aspartate transaminase (AST) 530 IU/L (normal <46), and alanine transaminase (ALT) 627 (normal <43) (Figure 1). She underwent ERCP that revealed an edematous major papilla. Cholangiogram showed mildly dilated common bile duct. Sphincterotomy was performed with removal of the retained stone. A balloon sphincteroplasty was also performed. Two days later she presented to the hospital for persistent itching. She denied any abdominal pain. Blood chemistries on readmission revealed total bilirubin 2.1 mg/dL, direct bilirubin 1.3 mg/dL (normal < 0.2 mg/dL), ALP 252 IU/L, AST 158 IU/L, ALT 404 IU/L (Figure 1). She had a right upper quadrant ultrasound and CT abdomen and pelvis consistent with surgical changes status post cholecystectomy with no acute findings. MRCP revealed no intrahepatic or extrahepatic biliary ductal dilation or choledocolithiasis. Further endoscopic interventions were not deemed necessary at this time. Workup of hepatitis A, B, and C were unremarkable. Her liver functions gradually up trended throughout the hospitalization, but, due to improvement of her symptoms with symptomatic management, she was discharged home to follow-up with a primary care physician and surgery. Two weeks later at an outpatient follow-up with gastroenterology, she denied abdominal pain and endorsed resolution of itching. Repeat liver function panel revealed ALP 335 IU/L with down trending of AST and ALT. Two months later, she followed up with her primary care physician for an annual physical with laboratory revealing liver function within normal limits.

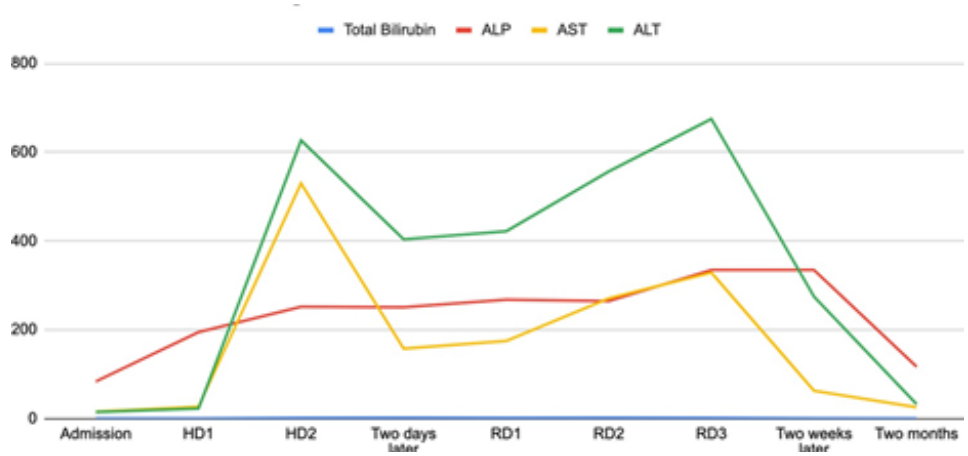

Figure 1: Liver function tests during clinical course, Liver function tests over the hospitalization, readmission, two-week outpatient follow-up, and two-month outpatient follow-up reveal fluctuating elevation in transaminases after cholangiogram performed on day of admission while total bilirubin remains within normal limits. ERCP occurred on hospital day 1. HD- Hospital day, RD- Readmission day, ALP- Alkaline phosphatase, AST- Aspartate transaminases, ALT- Alanine transaminase.

No.	Age/Sex	T-bilirubin, mg/dl	Peak days	2nd ERCP	Contrast agent	Treatment	References
1	48/M	12.5 -> 27	7	yes	Meglumine amidotrizoate (Angiografin)	Prednisolone 30 mg/d, 12 d	6
2	36/M	7.5 -> 26	8	yes	Meglumine amidotrizoate (Angiografin)	Prednisonlone 30 mg/d, 14 d	6
3	51/F	4.9 -> 6.8	21	yes	Not mentioned	UDCA 15 mg/kg prednisolone	7
4	51/M	6.7 -> 12.6	6	no	Not mentioned	–	7
5	73/M	18.3 -> 28.7	10	yes	Not mentioned	UDCA 15 mg/kg prednisolone 40 mg/d, plasmapharesis	3
6	54/F	16.2 -> 45.3	28	yes	Hypaque	UDCA 15 mg/kg	4
7	39/F	4.6 -> 8.8	11	no	Non-ionized contrast	UDCA 15 mg/kg prednisolone 50 mg/d	8
8	56/M	9.6 -> 18	15	yes	Iobitridol (Xenetix)	–	1
9	68/M	10.2 -> 35.2	21	no	Sodium and meglumine ioxitalamate, (Telebrix 35)	UDCA 15 mg/kg	5
10	49/F	1.3 -> 2.1	5	no	Omnipaque	–	

Table 1: Reported cases of cholestasis after endoscopic retrograde cholangiopancreatography. ERCP= endoscopic retrograde cholangiopancreatography, UDCA= ursodeoxycholic acid.

Discussion

There have been cases of drug induced liver disease, revealing cholestatic pattern (Table 1), in the setting of contrast agents after undergoing ERCP [1,2]. In these cases, iodinated contrasts, similar to the contrast used in our patient, were used while other cases have shown drug induced liver injury possibly after antibiotic use during ERCP [3,4]. When patients experienced severe pruritus, there was improvement with ursodeoxycholic acid [3], prednisone [3,4], or complete resolution with plasmapheresis [3]. Resolution of elevated transaminases with time has been noted without further intervention [1]. In all, there have been a total of about 10 cases that have revealed cholestatic liver injury in the setting of ERCP that resulted in a benign course with complete resolution of liver function and good outcomes [2,5]. The mechanism of liver injury has been speculated but overall remains unknown. Some have postulated liver injury being idiosyncratic [5]. It is also thought that the high pressure the contrast material is being infused under can create disruption of the canalicular membrane leading to hepatotoxicity [6]. Direct liver toxicity may also be in the setting of systemic distribution of the contrast from the bile duct [1]. To determine etiology of the disease, a "re-challenge" test (repeating ERCP with contrast) has been done to determine if further contrast load replicates liver injury similar to prior contrast load [1,3]. Even though those cases provided the evidence they were looking for, there has been the argument to avoid the "rechallenge test" and order imaging instead to avoid worsening of patient symptoms [2,4]. In one case, a liver biopsy was done to confirm the diagnosis that was consistent with drug induced liver injury [1], but this is one case compared to the others that determined diagnosis without the procedure. In our patient, they developed a prolonged hepatocellular liver injury (Table 1, Case 10), rather than cholestatic as seen in prior cases, after undergoing intraoperative cholangiogram using iodinated-contrast Omnipaque. Omnipaque is a low osmolar contrast agent hypothesized to decrease the incidence of contrast-induced drug induced liver injury [2]. Furthermore, she underwent

endoscopic intervention early in the course of her up trending liver function with continued gradual worsening of liver function after removal of stone via ERCP [7,8]. On readmission, with new onset pruritus, viral and mechanical etiologies were excluded confidently via laboratory and imaging studies. Of note, she did have resolution of symptoms along with liver function tests noted on serial laboratory the further out on the timeline she was from cholangiogram. We believe that while prior cases have shown a contrast-induced cholestatic pattern liver injury subsequently after cholangiogram, it is also important to consider a contrast-induced hepatocellular pattern in patients with no other etiology of liver injury. Furthermore, it is important to reimage prior to further endoscopic intervention when re-evaluating worsening liver function. Overall, patients who do experience cholangiogram contrast induced liver injury have a higher likelihood of a good prognosis with symptomatic management and allowing time for the liver to recover from injury.

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