

CASE REPORT

Plasmapheresis and corticosteroid treatment for persistent jaundice after successful drainage of common bile duct stones by endoscopic retrograde cholangiography

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Abstract

Prolonged cholestasis is a very rare complication of endoscopic retrograde cholangiography (ERC). Only few cases with this complication are reported in the English literature. We report persisting cholestatic jaundice in a 73-year old man after successful therapeutic ERC for choledocholithiasis. Serologic tests for viral and autoimmune hepatitis were all negative. A second-look ERC was normal also. He denied any medication except for prophylaxis given intravenous 1 g ceftriaxone prior to the ERC procedure. After an unsuccessful trial with ursodeoxycholic acid and cholestyramine for 2 wk, this case was efficiently treated with corticosteroids and plasmapheresis. His cholestatic enzymes became normal and intense pruritis quickly resolved after this treatment which lasted during his follow-up period. We discussed the possible mechanisms and treatment alternatives of intrahepatic cholestasis associated with the ERC procedure.

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Key words: Endoscopic retrograde cholangiography; Cholestasis; Plasmapheresis

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INTRODUCTION

The main complications of endoscopic retrograde

cholangiography (ERC) and sphincterotomy are bleeding, pancreatitis, perforation and cholangitis^[1,2]. The wide variety of miscellaneous complications of diagnostic and therapeutic ERC have also been described in the endoscopic literature, usually as single case reports, including splenic trauma, portal venous air, gall-stone ileus, intra-abdominal hemorrhage from injury to the gastropiploic artery, bleeding from bile duct varices and necrotizing fasciitis^[3-8]. Similarly, prolonged cholestasis is a very rare complication of ERC^[9,10] and a concern for a retained common bile duct stone, inadequate biliary drainage or another cause of liver disease. We describe a case of prolonged cholestatic jaundice with intense pruritis following successful therapeutic ERC in the absence of a retained stone, inadequate biliary drainage or unrelated cause of liver disease. This case gave a prompt response to corticosteroid treatment and plasmapheresis.

CASE REPORT

A 73-year-old male was admitted with complaints of epigastric pain and jaundice for the last few days. Physical examination was normal except for the apparent icterus. His laboratory results indicated elevated serum bilirubin levels (total: 18.3 mg/dL, direct fraction: 12.9 mg/dL), high alanine aminotransferase (ALT: 153 U/L, normal up to 40 U/L), alkaline phosphatase (ALP: 349 U/L, normal up to 130 U/L) and gammaglutamyl transpeptidase (GGT: 272 U/L, normal up to 40 U/L). An upper abdominal ultrasonography revealed dilated biliary tree and calculi of various sizes with associated acoustic shadows in the gall bladder. He underwent ERC procedure. Half an hour prior to ERC, a prophylaxis with 1 g intravenous ceftriaxone was done. During the procedure, a cholangiography revealed multiple filling defects consistent with stones and debris in the dilated common bile duct. A standard sphincterotomy was performed with gallstones and debris obstructing the common bile duct removed endoscopically. Control with balloon occlusion cholangiography was normal. However, during the days following ERC, a progressive increase of total and conjugated bilirubins (27.3 mg/dL and 15.5 mg/dL, respectively) associated with intense pruritis was noted. Nevertheless, the patient had no fever and his leukocyte count was within the normal range. ERC was repeated due to concern for a retained stone and cholangiography obtained at that time was normal. To ascertain bile duct drains well enough, we

placed a plastic biliary stent into the common bile duct. However, the insertion of biliary stent did not improve his jaundice. Three days after the second ERC, the patient continued to have intense pruritus and elevated cholestatic biochemical tests. Ten days later, his total and direct bilirubin levels were 28.7 mg/dL and 17.2 mg/dL, respectively and alkaline phosphatase 743 U/L. An upper abdominal ultrasound was unrevealing. Hepatitis B surface antigen and anti-hepatitis C virus antibody, anti-nuclear and anti-mitochondrial antibodies were all negative. There was no improvement in his pruritus and blood tests despite treatment with ursodeoxycholic acid (15 mg/kg per day) and cholestyramine (12 g/d) for 2 wk. He refused to undergo a liver biopsy. Cholestyramine and ursodeoxycholic acid were stopped and we started on treatment with prednisone (40 mg/d). Although his serum bilirubin (10.7 mg/dL) and ALP (181 U/L) levels gradually declined, his annoying pruritus continued. Biliary stent was removed endoscopically and plasmapheresis was performed. After plasmapheresis, pruritus promptly declined. Prednisone was stopped. His liver function tests were normal within 1 mo and remained stable after a further 4 mo period of follow-up.

DISCUSSION

Prolonged cholestasis is a rare complication of ERC^[3,4]. Our patient developed prolonged cholestasis following successful drainage of the bile ducts with ERC procedure. Viral, mechanical, and immunologic etiologies were excluded confidently in the present case. The only possible risk factor regarding the etiology of cholestasis aggravated after ERC was the administration of ceftriaxone before the procedure.

The cause of prolonged cholestasis after a successful ERC is unclear. Four similar patients have been reported previously, three of them recovered completely after treatment with glucocorticoids^[3,4]. Some speculated that it might have been caused by allergy to the radio-contrast material or antibiotics used during ERC leading to impaired secretion of bilirubin^[4]. Others also postulated that the radio-contrast material used may be the responsible factor for cholestatic injury after ERC as these materials are capable of acting toxically on hepatocytes with disruption of the canalicular plasma membrane^[3]. Thus, it may be reasonable to treat such patients with ursodeoxycholic acid and glucocorticoids. If we cannot get a symptomatic control of such complaints in these cases, plasmapheresis can be an optimal alternative treatment modality which has been successfully used to treat intractable pruritus associated with cholestatic diseases^[11,12]. Our patient's complaints abruptly improved soon after plasmapheresis sessions. Although liver biopsy examination would be

valuable to make a differential diagnosis better in the setting of intrahepatic cholestasis, our patient refused to undergo liver biopsy procedure. In the differential diagnosis of refractory cholestasis in this case, stent-induced cholangitis or cholestasis should also be evaluated. However, our case had no classic signs of stent-induced cholangitis, including fever and right upper quadrant pain. Moreover, sequential treatments with ursodeoxycholic acid/cholestyramine and prednisone which were partially successful in our case might not be useful in the setting of cholestasis or cholangitis associated with the presence of biliary stent.

We believe that intrahepatic cholestasis should be considered in the differential diagnosis of persistent jaundice after ERC when a retained common bile duct stone and other causes of liver disease are excluded. Plasmapheresis can be a therapeutic choice in these patients with persistent pruritus and biochemical abnormalities despite treatment with ursodeoxycholic acid, cholestyramine and glucocorticoids.

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