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Intrahepatic cholestasis accompanied with anastomotic leakage

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Background: Our case is characterized by multifaceted issues and complications. In the past, there were mechanical intestinal obstruction and in resectable splenic flexure tumor, colostomy opening, chemoradiotherapy, postoperative incisional hernia development, fistula development after curative resection and intrahepatic cholestasis. We want to present our case because of the rare complications.

Case: The case was regarding a 68 year old male patient. Two years ago, a mechanical intestinal obstruction was diagnosed and colostomy was opened with in resectable colon splenic flexure tumor. He had received chemoradiotherapy. Incisional herniation from midline and colostomy site within the last three months had grown from day to day and has become a cause of complaints in the patient. The patient applied to our hospital for treatment. Curative resection of the tumor and incisional hernia repair was planned. Splenectomy, curative tumor resection, colostomy closure, anastomosis and incisional hernia repair was performed in the operation of the patient. Postoperatively, the anastomotic fistula developed on the third postoperative day and the fistula was 20 ml/day. The fistula spontaneously closed after two weeks. Elevation of bilirubin level was observed postoperatively. Elevations in ALP, GGT and LDH levels were detected while AST and ALT were at normal values. The parameters were at maximum levels: Bilirubin 16 mg/dl, ALP 961 U/L, LDH 235 U/L, GGT 727 U/L. Intra and extra hepatic bile ducts were normal in MRCP and MRI of the upper abdomen. Intrahepatic cholestasis was diagnosed and ursodeoxycholic acid treatment was started.

Conclusion: It was observed that bilirubin did not increase in the first 24 hours after the treatment of ursodeoxycholic acid treatment and in the following days bilirubin decreased to normal level by about 2 points every day.

Discussion: Production of bile is a vital function for the body. Medications, infections, autoimmune, metabolic or genetic causes and disruption of this function are known as cholestasis. Cholestasis can be divided into intrahepatic and extra hepatic. Bile secretion block may be complete or incomplete. Complete cholestasis occurs in primary parenchymal diseases (intrahepatic cholestasis) or in total obstruction of extra-hepatic bile ducts (extra-hepatic cholestasis). Incomplete obstruction of intrahepatic and/or extra-hepatic bile ducts is the incomplete obstruction of bile secretion. Among the causes of intrahepatic cholestasis are alcoholic liver diseases, amyloidosis, hepatic bacterial abscess, lymphoma, pregnancy, primary bile cirrhosis, primary sclerosing cholangitis, sarcoidosis, severe infections with sepsis, tuberculosis, viral hepatitis, and total parenteral nutrition. We will examine this issue especially since sepsis-associated cholestasis develops in our cases. Jaundice may come from direct bacterial products and/or (either) the host's response to the infection. In addition, specific infections may lead to liver infection by targeting liver directly to hepatitis associated with liver injury. Proinflammatory cytokines and nitric oxide affect cholestasis by affecting hepatocellular and ductal biliary formation. Generally, before cholestasis, sepsis is evident among clinical features. Cytokines are released in response to endotoxemia and bacterial wall lipopolysaccharides. The major cytokines are proinflammatory tumor necrosis factor-alpha (TNF-alpha), interleukin (IL) 1-beta and IL-6. Cholestasis occurs by the interaction between these cytokines and hepatocyte membrane transporters. Jaundice starts a few days after the bacteremia starts. Laboratory findings include elevation of conjugated bilirubin, alkaline phosphatase elevation. A moderate elevation is detected in the aminotransferases.

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