Acute necrotizing pancreatitis after transarterial chemoembolization of hepatocellular carcinoma: An usual complication

Dear Editor,

Acute necrotizing pancreatitis following transarterial chemoembolization is an uncommon complication, its incidence being estimated in around 1.7-2 % according to various studies. We report the case of a 60-year-old male patient with a diagnosis of both HBV- and alcohol-related cirrhosis. He underwent a second chemoembolization procedure for a relapsing hepatocellular carcinoma, 12.5 mm in diameter, in segment VIII (he was not eligible for transplantation because of prostate adenocarcinoma). Forty-eight hours after the procedure he presented with epigastric pain radiating to the back and right hypochondrium in association with febricula at 37.8 °C, with lab tests showing serum amylase of 896 mU/ml, C-reactive protein of 122.7 mg/l, total bilirubin of 1.64 mg/dl (direct bilirubin was 0.83 mg/dl), GGT of 147 IU/L, alkaline phosphatase of 154 U/L, and mildly impaired coagulation (PT, 16.5 seconds; INR, 1.47), with normal CBC and transaminases. Abdominal, IV contrast-enhanced tomography revealed several collections: an extrapancreatic one surrounding the head of the pancreas, 50 mm in size, and another one in the uncinate process, 43 mm in size, associated with a complication of acute necrotizing pancreatitis (Fig. 1). Following intestinal rest, hydration, intravenous analgesia, and empiric antibiotic therapy with meropenem 1 g every 8 hours for 7 days, the disease had a favorable evolution, and both amylase and C-reactive protein levels returned to normal. Tomographic follow-up at 3 months showed that collections had shrunk by 50 % in size. Similarly, no uptake was identified by the chemoembolized hepatocellular carcinoma.

Key words: Pancreatitis. Hepatocellular carcinoma. Liver cirrhosis.
Discussion

Transarterial chemoembolization is a first-line therapy for hepatocellular carcinoma. Its mechanism of action consists of compromising the tumor's arterial inflow and instilling a chemotherapy agent (usually doxorubicin) in order to bring about tumor necrosis (1). Following the procedure post-chemoembolization syndrome is commonly experienced, which is characterized by fever, abdominal pain, nausea/vomiting, and occasionally impaired liver function. Unusual complications may develop, such as noted by Xia Jinglin et al. in their study of 1,348 patients, with an incidence of rare complications of 2.68% that ranged from spontaneous carcinoma rupture to hepatic artery occlusion (0.15% and 1.99%); the fact stands out that no acute necrotizing pancreatitis cases was identified in this large series (2). This is an uncommon condition, and studies estimate an incidence of 1.7-2% (clinical pancreatitis) as compared to 40% for biological pancreatitis, defined as hyperamylasemia with no clinical impact (3-5). This complication results from a retrograde injection of the chemotherapeutic, embolizing agent into pancreatic arteries, giving rise to ischemic pancreatitis (6). Amylasemia testing after chemoembolization is controversial -some authors do not recommend its routine use but in patients experiencing severe abdominal pain unrelated to post-chemoembolization syndrome (7,8).

Management in such situation is similar to that of any acute pancreatitis; prophylactic antibiotic use is indicated when severe necrosis or superinfection is present (9). In our case we used meropenem because of the presence of necrotic collections, febricula, and markedly elevated C-reactive protein levels, in addition to cirrhosis-related immune deficiency. Up to 57% of patients may have such collections progressing to pseudocysts; those located at the neck and head should be assessed for drainage or surgical resection when symptomatic (10). In our case all collections evolved satisfactorily with a significant decrease in size, and the patients remained asymptomatic after 3 months of follow-up.

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References