Case Report

Burkitt’s Lymphoma: An Unusual Cause of Obstructive Jaundice

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Biliary obstruction secondary to malignancy is a common clinical problem. Rarely, biliary obstruction is due to non-Hodgkin’s lymphoma. Obstructive jaundice in these patients usually presents late in the course of the disease. Burkitt’s lymphoma is a subtype of non-Hodgkin’s lymphoma. Unlike other forms of non-Hodgkin’s lymphoma, Burkitt’s lymphoma presents as an extranodal disease. We present the case of a patient suffering from non-endemic Burkitt’s lymphoma whose initial presenting symptom was obstructive jaundice. [Asian J Surg 2005; 28(4):301–4]

Key Words: bile duct malignancy, Burkitt’s lymphoma, obstructive jaundice

Introduction

Biliary obstruction secondary to malignancy is a common clinical problem. Primary carcinomas of the pancreas, ampulla of Vater, bile ducts or liver are frequently involved. Rarely, biliary obstruction is due to non-Hodgkin’s lymphoma (NHL).1 Obstructive jaundice in these patients usually presents late in the course of the disease. In only 1% of patients with NHL is obstructive jaundice the presenting sign of the disease. NHL cases, in which obstructive jaundice occurs, are histologically diffuse B cell lymphomas.2,3

Obstructive jaundice due to NHL is caused mainly by enlarged lymph nodes compressing the biliary tree. The most common site of obstruction is the liver hilum, followed by the common bile duct (CBD) and peripancreatic area. Unlike more common malignancies causing biliary obstruction, NHL more readily responds to chemotherapy.2–5 Palliation of jaundice and cure are possible, even without a biliary drainage procedure.1

Burkitt’s lymphoma is a subtype of NHL. It is endemic, non-endemic, or HIV-related. Unlike other forms of NHL, Burkitt’s lymphoma presents as an extranodal disease. We present the case of a patient suffering from non-endemic Burkitt’s lymphoma whose initial presenting symptom was obstructive jaundice.

Case report

A 68-year-old man was admitted to the surgical department for investigation of obstructive jaundice. He presented with right upper quadrant abdominal pain, nausea and fatigue, all of which started a week prior to his admission. He also complained of decreased appetite, with no weight loss. The patient’s sclera and skin were jaundiced and, on abdominal examination, a moderate right upper quadrant tenderness was noted and a swollen gallbladder was palpated. The remainder of the physical examination was unremarkable. There was no history of hepatobiliary disease, smoking, alcohol consumption or drug abuse. The patient had a prior history of hypertension but, otherwise, did not suffer from any other diseases.

Initial blood tests in the emergency room revealed a bilirubin level of 10.2 mg/dL, with 7.9 mg/dL being direct. Amylase, lactate dehydrogenase (LDH), liver transaminase,
Total and direct bilirubin levels actually continued rising to 30 mg/dL and 23 mg/dL, respectively. Three days later, another PTC was performed, with the insertion of another internal stent (Figure 2). Again, despite this procedure, the jaundice continued to progress and total and direct bilirubin levels rose to 35 mg/dL and 27 mg/dL, respectively. At this point, separate biliary external–internal drainage of the right and left hepatic ducts was performed, but almost no bile was collected in the drains and there was no clinical or laboratory improvement.

Because of the patient’s clinical deterioration, extreme hyperbilirubinemia and inadequate biliary drainage, palliative surgery was performed on the 19th day of admission. The patient underwent a laparotomy under general anaesthesia, and a malignant process involving the head of the pancreas and hepatoduodenal ligament was found. A swollen gallbladder was removed to allow identification of the CBD within the malignant infiltrate. A drain was inserted into the right biliary system. Only small amounts of bile could be aspirated. The left hepatic bile duct would not allow passage of a separate drain. Still, intraoperative cholangiography demonstrated patency of both biliary systems.

Following this operation, there was still no significant biliary drainage. Bilirubin levels did not decrease. LDH, aspartate aminotransferase, ALP and GGT values were 1,450 U/L, 160 U/L, 200 U/L, and 150 U/L, respectively. The patient continued to deteriorate. He developed hepatic encephalopa-
Obstructive jaundice is a known manifestation of NHL. Few possible mechanisms have been reported to cause jaundice in patients with NHL. Compression of bile ducts by the lymphomatous mass is the most frequent cause, followed by tumor-related haemolysis and malignant liver infiltration. Since NHL-induced obstructive jaundice almost always occurs with advanced disease, it is treated with systemic chemotherapy. Most regimens include anthracyclines, and these usually failed should have raised questions regarding our assumed aetiology and mechanism of jaundice. Repeated failure to alleviate the jaundice by drainage procedures should have prompted the thought that the primary mechanism of jaundice was due to hepatocellular pathology. We believe that in
our case, the cause of the jaundice was secondary liver infiltra-
tion by the lymphoma, leading to fulminant hepatic failure,
although we have no pathological proof of this. The extensive
infiltration of the entire gallbladder wall, however, supports
this assumption. Although primary lymphoma of the liver is
rare, secondary involvement of the liver in patients with NHL
is common and occurs in as many as 50% of patients. Secondary
lymphoma is often diffusely infiltrative and may be difficult
to detect on CT.

Our patient deteriorated rapidly, and perhaps his disease
was so advanced that even chemotherapy would not have
changed the course of his disease. Still, it is crucial that lym-
phoma be taken into consideration early in the management
of patients with malignant obstructive jaundice.

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