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OBSTRUCTIVE JAUNDICE AS THE FIRST CLUE TO SYSTEMIC DIFFUSE LARGE B-CELL LYMPHOMA: A DIAGNOSTIC CHALLENGE

Gastroenterology

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ABSTRACT

Obstructive jaundice is most frequently caused by pancreaticobiliary malignancies or gallstones. However, lymphoma presenting initially as obstructive jaundice remains rare and diagnostically challenging. We describe a 58-year-old male who presented with jaundice, pruritus, and constitutional symptoms, along with dysphagia and systemic lymphadenopathy. Imaging revealed biliary obstruction due to periportal lymphadenopathy and an ulcerated lesion at the gastroesophageal junction (GEJ). Histopathology, complemented by immunohistochemistry, established a diagnosis of diffuse large B-cell lymphoma (DLBCL). Biliary decompression and systemic chemotherapy led to a significant clinical and radiological response. This case highlights the importance of considering lymphoma in the differential diagnosis of obstructive jaundice, the utility of EUS-guided sampling, and the role of timely chemotherapy for both local and systemic disease control.

KEYWORDS

Obstructive jaundice, Diffuse large B-cell lymphoma, EUS-guided biopsy, Biliary obstruction

INTRODUCTION

Obstructive jaundice is most commonly associated with gallstones or primary hepatopancreatobiliary malignancies, whereas lymphomas account for less than 2% of malignant biliary obstruction cases [1]. Non-Hodgkin lymphoma (NHL) involving the hepatobiliary system typically arises as part of widespread disease rather than as a primary lesion. Diffuse large B-cell lymphoma (DLBCL) is the most common NHL subtype [2]. Rarely, DLBCL may initially present as obstructive jaundice, sometimes leading to diagnostic delays [3, 4]. Timely tissue diagnosis and systemic therapy can significantly improve outcomes [5, 6].We report a diagnostically complex case where obstructive jaundice was the first clue to an underlying systemic lymphoma.

Case Report

A 58-year-old male presented with a one-month history of progressive jaundice, generalized pruritus, right upper quadrant pain, and vomiting. He also reported dysphagia to solids for two months, described as a retrosternal sticking sensation, and a four-month history of significant constitutional symptoms, including anorexia and unintentional weight loss.

On physical examination, he had marked pallor and icterus with multiple excoriation marks on his abdomen. Palpable, non-tender, firm, and mobile lymphadenopathy was noted in the left supraclavicular and left axillary regions, with the largest node measuring approximately 2 cm.

Laboratory investigations revealed normocytic, normochromic anemia (Hemoglobin: 9.2 gm/dL) and a cholestatic pattern of liver injury (Total Bilirubin: 31.48 mg/dL, Direct Bilirubin: 15.75 mg/dL, ALP: 574 U/L, GGT: 312 U/L). Inflammatory markers were elevated (ESR: 78 mm/hr, CRP: 24 mg/L). Viral markers for hepatitis and HIV were non-reactive.

An initial abdominal ultrasonogram (USG) showed mild hepato-splenomegaly, bilobar intrahepatic biliary radical dilatation (IHBRD), and a dilated common bile duct (CBD) measuring 11 mm. USG of the axilla noted bilateral nodes with loss of fatty hilum, the largest measuring 3.8×2.2 cm. A Contrast-Enhanced Computed Tomography (CECT) scan of the abdomen and pelvis confirmed the significant IHBRD and dilated proximal CBD, identifying a mid-CBD narrowing due to extrinsic compression by a large periportal lymph node (2.9×1.8 cm). The scan also revealed widespread, well-defined, homogeneous lymphadenopathy involving the porta hepatis, peripancreatic, perisplenic, retroperitoneal, and pelvic regions. At the GE junction, circumferential wall thickening with heterogeneous post-contrast enhancement was observed (Figure 1).

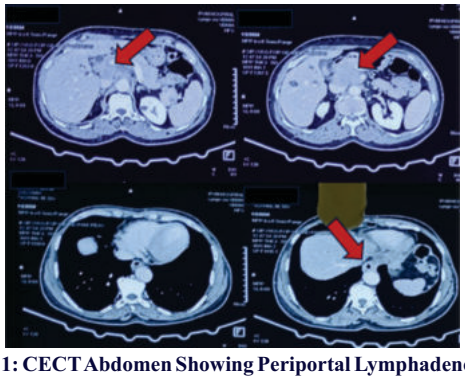

Figure 1: CECT Abdomen Showing Periportal Lymphadenopathy and Thickening in GE Junction

Upper gastrointestinal endoscopy identified a 2×2 cm ulcer at the GE junction extending into the cardia, consistent with a Siewert class II tumor. The ulcer had irregular margins, raised edges, and a yellowish base (Figure 2). Multiple biopsies were taken, and the initial histopathological impression was a poorly differentiated carcinoma, given the sheets of atypical cells amidst extensive necrosis.

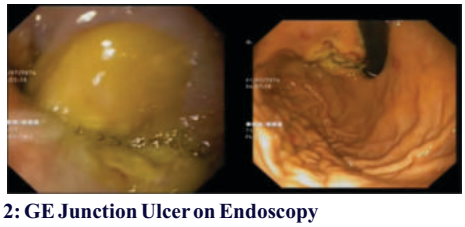

Figure 2: GE Junction Ulcer on Endoscopy

To further investigate the biliary obstruction and obtain diagnostic tissue, an Endoscopic Ultrasound (EUS) was performed. EUS confirmed a dilated CBD (11 mm) compressed posteriorly by a 2.8×1.5 cm hypoechoic, homogenous periportal lymph node (Figure 3). EUS-

guided fine-needle biopsy (FNB) of this nodal mass was performed, along with a fine-needle aspiration cytology (FNAC) from a palpable axillary lymph node. Both specimens revealed a monomorphic population of large atypical lymphoid cells with pleomorphic nuclei, coarse chromatin, and distinct nucleoli (Figure 4).

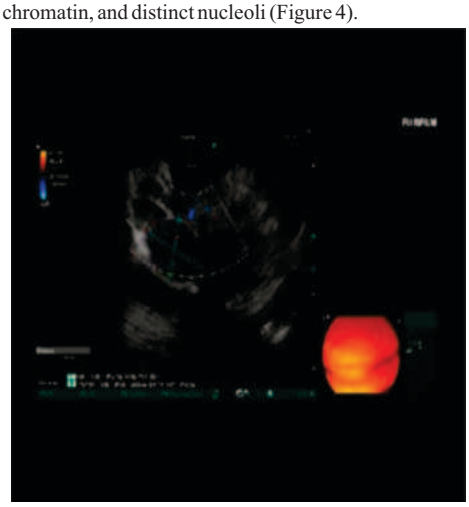

Figure 3: EUS Showing Periportal Lymph Nodes

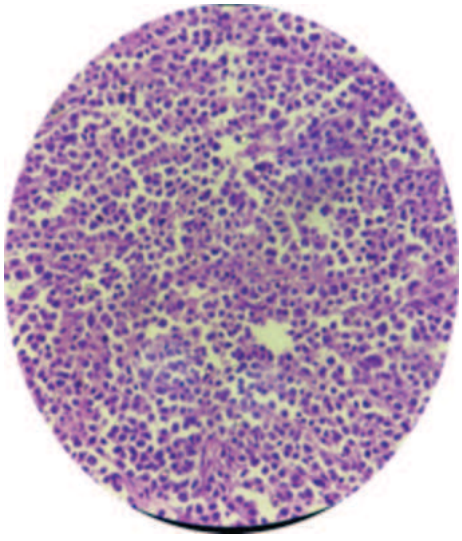

Figure 4: Atypical Lymphoid Cells

This discordance prompted a comprehensive IHC panel on both the GE junction and nodal biopsy samples. The atypical cells in all specimens were strongly positive for LCA (CD45) and CD20, and positive for Bcl-2 (Figure 5). Crucially, they were negative for Pan-Cytokeratin (PCK). This IHC profile conclusively ruled out carcinoma and established a definitive diagnosis of Diffuse Large B-Cell Lymphoma (DLBCL).

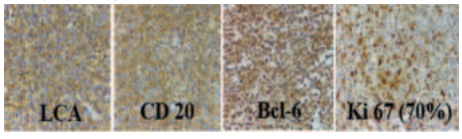

Figure 5: IHC

For management of the severe jaundice, the patient underwent an Endoscopic Retrograde Cholangiopancreatography (ERCP). A cholangiogram revealed an irregular narrowing of the mid-distal CBD from extrinsic compression (Figure 6). A 10 Fr × 10 cm plastic biliary stent was successfully placed, leading to a rapid decline in serum bilirubin from 31.48 mg/dL to 1.7 mg/dL over 3 weeks.

A staging PET-CT scan showed FDG-avid disease at the GE junction and in widespread lymph nodes above and below the diaphragm (Figure 7). A bone marrow aspiration and biopsy were negative for lymphomatous involvement. The final diagnosis was Stage IV DLBCL (Lugano Classification).

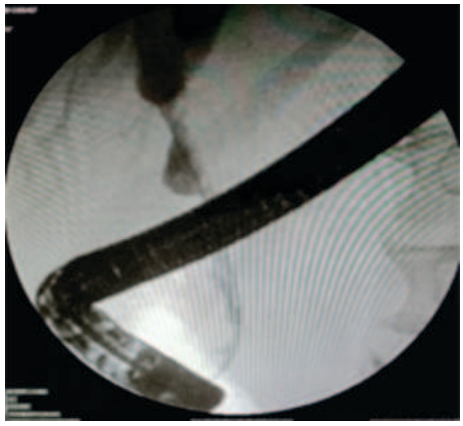

Figure 6: ERCP

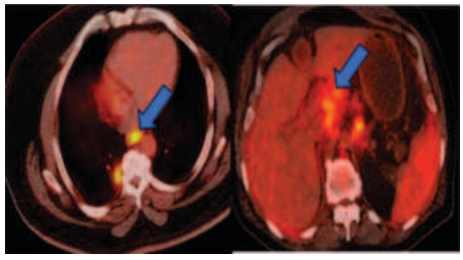

Figure 7: PET CT

The patient was initiated on R-CHOP chemotherapy. He tolerated the treatment well and showed a remarkable response. After two cycles, his dysphagia, pain, and B-symptoms had completely resolved. A follow-up endoscopy after six cycles of chemotherapy showed near-complete healing of the GE junction ulcer, with only residual scar tissue (Figure 8). A post-treatment PET scan confirmed a significant metabolic response with an interval reduction in the size and avidity of the widespread lymphadenopathy. The biliary stent was removed following confirmation of resolved extrinsic compression.

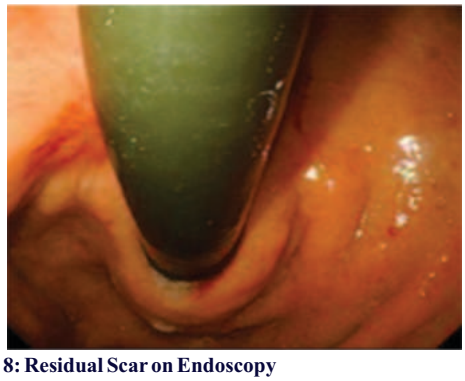

Figure 8: Residual Scar on Endoscopy

DISCUSSION

DLBCL is the most frequent subtype of NHL [2], representing 30–40% of cases globally [7]. Jaundice in lymphoma patients may result from several mechanisms: direct hepatic infiltration [8], external compression of the extrahepatic bile ducts by enlarged lymph nodes [9], intrahepatic cholestasis, drug-induced toxic hepatitis, or tumor-related hemolysis [10]. Among these, the most common cause in non-Hodgkin lymphoma (NHL) is extrahepatic biliary obstruction secondary to nodal compression [8]. Notably, NHL accounts for only about 1–2% of all cases of malignant biliary obstruction [3].

Although jaundice is usually a late complication of NHL, it can rarely present at disease onset. Rosenberg et al. (1961) described jaundice in 159 of 1,269 NHL patients, with only three presenting initially with obstructive jaundice [10]. A retrospective series by Ravindra et al. reported nine patients (six adults, three children) whose first manifestation of NHL was obstructive jaundice [4]. Similarly, Ödemiş et al. (2007) identified seven NHL cases among 1,123 patients with malignant biliary obstruction over a six-year period [3]. Due to its rarity, NHL is often overlooked in the initial differential diagnosis of obstructive jaundice.

The site of obstruction most frequently involves the hepatic hilum or peripancreatic head region. This predilection is thought to relate to reduced mobility of the bile ducts in these regions, making them more susceptible to compression from enlarged lymph nodes [8].

In this case, the co-existing ulcerative GEJ lesion suggested a primary adenocarcinoma (supported by initial morphology), illustrating the diagnostic pitfall. However, EUS-FNB from lymph nodes, together with IHC, confirmed lymphoma. EUS-FNB has a reported diagnostic accuracy exceeding 85% for retroperitoneal and periportal lesions [11, 12].

The patient's high NCCN-IPI score reflected advanced age, elevated LDH, stage IV disease, extranodal GI involvement, and ECOG PS 1. Early death in NHL correlates with poor prognostic factors [15]. Notably, studies suggest patients presenting initially with obstructive jaundice may achieve better survival than those developing jaundice later [4,13,14].

Despite these high-risk features, early biliary decompression by ERCP stenting reduced bilirubin rapidly, facilitating timely initiation of R-CHOP chemotherapy. Therapeutically, the mainstay is systemic chemotherapy, with R-CHOP achieving complete response rates up to 60–70% [4, 14]. Biliary decompression is often required pre-chemotherapy in high bilirubin states, using ERCP or PTBD [2, 5, 6]. The role of biliary drainage procedures before initiating chemotherapy in lymphoma patients presenting with obstructive jaundice remains uncertain. Various interventions, including external–internal drainage, stent insertion, or even laparotomy, have been employed to relieve biliary obstruction [1]. While these measures can alleviate obstruction and associated symptoms, they do not address the underlying malignancy and may potentially delay the start of systemic therapy. Current recommendations suggest biliary stenting primarily in patients who have symptomatic obstruction or develop infectious complications like cholangitis [14].

A retrospective study by Ross et al. at MD Anderson Cancer Center evaluated 35 patients with lymphoma-related obstructive jaundice who underwent ERCP or percutaneous biliary drainage between 2002 and 2008. Among them, bilirubin levels normalized in 29 of 33 evaluable patients, and complete resolution of biliary strictures was observed in 12 cases following stenting or drainage [6].

Importantly, unlike adenocarcinoma, lymphoma responds dramatically to chemotherapy, often obviating the need for surgery [3, 4, 6]. Our patient's bilirubin fell rapidly after stenting, facilitating timely R-CHOP.

Similar cases by Fidias et al [14]. and Ravindra et al [4]. highlighted the benefit of early biliary drainage and prompt chemotherapy, which improves prognosis. Prognosis in NHL with biliary obstruction depends on age, stage, and chemotherapy response; early mortality can be high without timely intervention [16].

CONCLUSION

Obstructive jaundice rarely heralds underlying lymphoma but should always be considered, especially in the presence of systemic lymphadenopathy and constitutional symptoms. This case illustrates the diagnostic challenge when extranodal lesions mimic carcinoma and underscores the utility of EUS-guided sampling and IHC. Early biliary decompression followed by chemotherapy can achieve rapid disease control and excellent symptomatic relief.

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