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Simultaneous occurrence of obstructive jaundice and protein-losing enteropathy in Hodgkin's lymphoma

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Protein-losing enteropathy (PLE) and obstructive jaundice as initial findings of Hodgkin's lymphoma are extremely rare in childhood. The diagnosis of PLE and obstructive cholestasis is critical because early diagnosis and treatment are important for improving survival in patients with Hodgkin's lymphoma.

PLE is a rare entity characterized by a loss of serum proteins into the gastrointestinal (GI) tract resulting in hypoproteinemia, which can be involuted by edema, ascites, pleural and pericardial effusions, and malnutrition (1).

The pathophysiology of this disorder is directly related to excessive permeation of plasma proteins into the lumen of the GI tract (2). Such patients have edema and documented hypoalbuminemia but without clinical or biochemical evidence of liver or renal disease. The clearest evidence of protein-losing enteropathy is if one detects a serum protein in the stool that is not secreted, digested, or reabsorbed in the GI tract (3).

In this letter, we describe a 14-year-old boy with simultaneous occurrence of PLE and obstructive jaundice. He presented with fever and non-tender bilateral cervical lymphadenopathy. Abdominopelvic computed tomography (CT) for abdominal distension showed multiple hypoechoic para-aortic lymphadenopathy with a compression effect on the biliary system, leading to obstructive jaundice with severe ascites.

Direct bilirubin: 17.3 mg/dL, total protein, 4.9 g/dL; albumin, 2.8 g/dL; α-fetoprotein, 1.93 ng/ml; stool alpha-1 anti trypsin (A1AT) 110 mg/ml (normal range for serum: 100-190 mg/dL and for stool ≤54 mg/dL) and serum A1AT 85 mg/ml and A1AT Clearance: = 50 mL/24 hours (normal range: ≤27 mL/24 hours). Analysis of the peritoneal fluid showed a non infectious exudative ascites fluid. Complete blood cell (CBC) and liver transaminases, renal function tests and coagulation profile were mildly abnormal and bilateral bone marrow aspiration and biopsy failed to reveal any pathologic finding.

Three established types of tests have been used for evaluating PLE.

1- Intravenous administration of a radiolabeled substrate followed by the determination of radioactivity in the feces. 2- Nuclear scintigraphy, not only for diagnosis, but also to identify potential regional or localized areas of protein loss. 3- Direct measurement of endogenous proteins such as Alpha-1 Antitrypsin(A1-AT) in the feces (4).

Fecal A1AT levels and A1AT clearance are used for diagnosis and follow-up care in patients with protein-losing enteropathy (5).

Hodgkin's Lymphoma (HL) presenting as peritoneal carcinomatosis has rarely been reported. Only in 1-2% of adults does Non-Hodgkin lymphoma (NHL) with aggressive presentation have biliary obstruction as the main manifestation. A review of the literature revealed only 3 cases where primary diffuse large B-cell lymphoma (DLBL) in the head of the pancreas induced biliary stenosis and obstructive jaundice, though PLE as the sole manifestation of intestinal NHL had been reported previously by Iranikhah *et al* (6). This

manifestation has not been described in patients with HL before the present report. From 1958 until 2005, only 8 cases of HL presenting with obstructive jaundice were reported in PUBMED.

In conclusion, obstructive jaundice with peritoneal carcinomatosis and PLE are rare forms of presentation carrying a poor prognosis and warranting aggressive and timely intervention.

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