A Rare case of cholestasis

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Abstract:
Cholestatic disease is a rare initial presentation of Non Hodgkin lymphoma. Lymphomatous infiltration and extr hepatic obstruction occur more commonly in Non Hodgkin Lymphoma than in Hodgkin Lymphoma. 16 to 43 of patients with Non Hodgkin Lymphoma have liver involvement. We present this case report of a patient who presented with progressive intrahepatic cholestasis which turned out to be secondary to infiltration of the liver by Non Hodgkin lymphoma.

Keyword:
cholestasis, Non Hodgkin lymphoma

A 28 yr old male patient, presented with pruritus and clay colored stools for 10 months, jaundice for 6 months, malaise for 4 months, with significant loss of appetite and loss of weight for 4 months. Symptoms were progressive in nature. There was no history of abdominal pain, arthralgia or taking hepatotoxic drugs, with no past history of viral hepatitis, abdominal surgery, high risk sexual behavior or tuberculosis. Patient is not an alcoholic and is not a smoker or substance abuser. Clinical examination revealed that patient is thin built and icteric, with scratch marks all over the body. No generalized lymphadenopathy. Mild hepatomegaly was noted. Patient was previously evaluated extensively, elsewhere for the above mentioned complaints and had undergone extensive work up including CT chest and abdomen. Investigations: Total Count-8,200 cells/mm³. Differential Count-Polymorphs 70%, Lymphocytes-28%, ESR-80mm at 1 hr, Platelet count-3,42,000 cells/mm³. Renal Function Test was within normal limits. Viral markers for hepatitis B and C were negative. HIV status negative. Liver Function Test-Total Bilirubin-10.95mg/dl, Direct-8.04mg/dl, Indirect-2.91mg/dl, SGOT-75 IU/L, SGPT-46 IU/L, GGT-221 IU/L, SAP-627IU/L, Total Protein 8 gm/dl, Albumin 4.6 gm/dl. serum calcium - 9 mgs%. serum LDH - 510 IU/L. AMA Negative, ANA Negative, Ultrasonogram of Abdomen showed Hepatomegaly, upper gastrointestinal endoscopy revealed normal study.
CT Thorax-Enlarged Lymph nodes In Subcarinal Paraesophageal, Para Aortic Nodes. CT Abdomen revealed periportal, peripancreatic node enlargement. Peripheral smear-normal study. Bone marrow aspiration cytology-normal study. CT guided FNAC of periportal nodes- smear showed clusters of epithelial cells, no lymph node material visualized. EUS guided biopsy of peripancreatic node – smear showed normal lymphoid histology. Liver biopsy – portal tract shows dense lymphoid cell infiltrate with creeping periportal inflammation and focal necrosis of periportal hepatocytes. Bile duct show minimal regeneration. The hepatocytes show numerous foci of focal necrosis. Rest of the hepatocytes show marked cholestasis and bile duct plugging. Sparse lymphoid cells within the portal tract, in sinusoids and around focal necrosis show atypia and appear larger with features of lymphoblast in an occasional cell.

**HISTOPATHOLOGY**

Impression: Intrahepatic cholestasis with diffuse atypical lymphoid cell infiltrate suggestive of lymphoma.

**Immunohistochemistry of liver biopsy specimen:**
- T-cell (CD 3) +++ in all lymphoid cells within the lobule and periportal area.
- B-cell (CD 20) occasional scattered single cell positive.
- Ki 67: increased, CD 30 negative.
IMMUNOHISTOCHEMISTRY

IMPRESSION: Intra Hepatic cholestasis With Diffuse atypical Lymphoid cell Infiltrate predominantly TCell Type. Features are suggestive of T-cell Lymphoma.

Final diagnosis: NON HODGKIN LYMPHOMA (T-cell lymphoma) Patient was offered chemo radiation as per medical oncologist opinion. However patient refused to undergo treatment and decided to undergo native treatment.

Discussion:
The Non Hodgkin Lymphomas are a large and diffuse group of malignancies involving mutations of B and T cells that essentially include all lymphomas other than Hodgin Lymphoma. Compared with HodginLymphoma, this type of lymphoma is more common, it spreads in a less-contiguous fashion, and it usually affects patients over the age of 60 years. Hepatic manifestations of Lymphoma like lymphomatous infiltration and extrahepatic obstruction occur more commonly in Non Hodgkin Lymphoma than in Hodgin Lymphoma; 16% to 43% of patients with Non Hodgkin Lymphoma have liver involvement. Infiltrating disease is found more often in low-grade lymphomas than in those that are high-grade. Mild to moderate increases in alkaline phosphatase level and hepatomegaly commonly occur in Non Hodgkin Lymphoma whether or not there is lymphomatous hepatic involvement. Acute liver failure can also occur in Non Hodgkin Lymphoma. The mechanism by which this occurs is sudden ischemia related to massive infiltration of the sinusoids or replacement of liver parenchyma by malignant cells. Although the prognosis is poor, there have been reports of successful treatment with immediate initiation of chemotherapy. Percutaneous liver biopsies have been found to be of value in detecting hepatic involvement with lymphoma, and if such specimens are properly processed, immunotyping can be performed to better characterize the phenotype of the malignant cells. However, quantity of tissue obtained appears very important in determining diagnostic sensitivity, with biopsy at laparotomy being superior to either blind percutaneous or laparoscopic biopsies in obtaining a diagnosis of hepatic infiltration by non- Hodgin's lymphoma. When evaluated by percutaneous liver biopsy, 16% to 26% of patients with non-Hodgin's lymphomas are found to have liver infiltration with significantly higher percentages found to have hepatic involvement when evaluated by laparoscopy. In both Hodgin's and non-Hodgin's lymphomas, the majority of infiltrative lesions are portal in location.

References:


