A Case Report On Primary Gastric Lymphoma

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Abstract : A case of Primary Non Hodgkin's Lymphoma was diagnosed in 50 years old male patient who presented with a gastric mass. As it can not be differentiated from adenocarcinoma radiologically and progressive nature of the disease, long term follow up of the patient has been advised after the cytological diagnosis. [Panchal N et al NJIRM 2013; 4(4) : 135-137]

Key Words : Non Hodgkin's Lymphoma, Gastric mass, Adenocarcioma.

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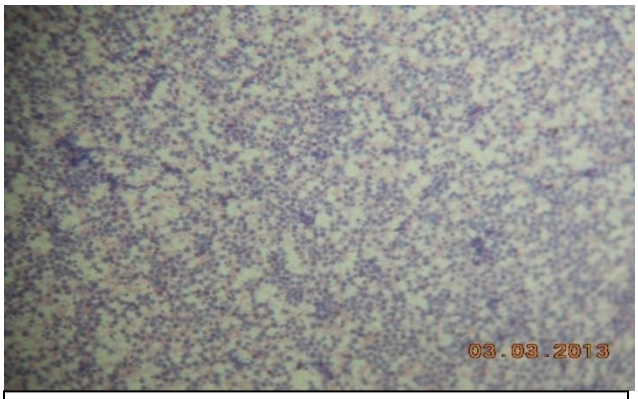
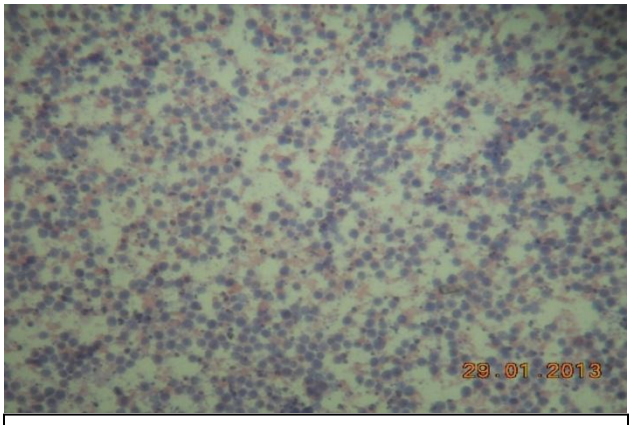
Introduction: The stomach is the most common site for extra nodal lymphoma.[1] According to Dawson gastric lymphoma is considered primary when predominantly stomach is involved, and the intra-abdominal lymphadenopathy, if present, corresponds to the expected lymphatic drainage of stomach.[2] Primary Non Hodgkin's lymphoma of gastrointestinal tract is 41% to 73% times present within the stomach.[3] Primary gastric lymphoma accounts for less than 5% of all gastric cancers.[4] Gastric lymphoma presents in 6th decade of life, rarely occurring under the age of 40 years.[5] It often presents with non specific gastro-intestinal symptoms such as abdominal pain, anorexia, nausea(with or without vomiting), and weight loss or less commonly fatigue, diarrhoea and constipation. Sometimes abdominal mass may be appreciated or may even present with any complication like bleeding, perforation or obstruction. There is a well established relationship of *H. pylori* infection with gastric lymphoma. Majority of the Primary Non Hodgkin's Lymphomas of stomach are B-cell origin. Lymphomas of T-cell origin are very rare in stomach.

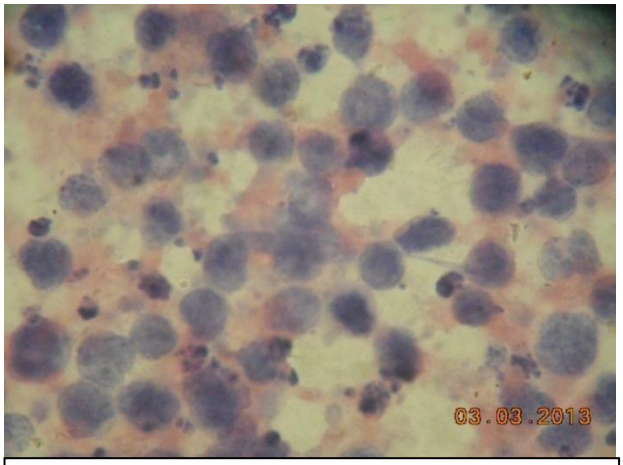
Case Report: A 50 years old male patient presented with complaints of dull intermittent epigastric pain along with weight loss. On systemic examination diffuse not sharply demarcating mass was found in left hypochondrium. No peripheral lymphadenopathy was found. Complete blood count revealed increased WBC count (14,000/cmm) with neutrophilia. Ultrasonography of abdomen showed a circumferential thickening of stomach wall with gross narrowing of lumen suggestive of carcinoma stomach. No Hepatomegaly or spleenomegaly were found. Multiple enlarged lymph nodes were seen at hepatogastric and para aortic region. Multislice CT scan abdomen was suggestive of gastric neoplasm either lymphoma or carcinoma. USG guided fine needle aspiration from gastric mass was done. Microscopy of H & E and Giemsa stained smears revealed high cellularity showing monomorphic lymphoid cells having coarse chromatin and high N:C ratio. Smears also showed few atypical mitoses. Based on these clinical and cytological features a diagnosis of Primary gastric lymphoma was made.



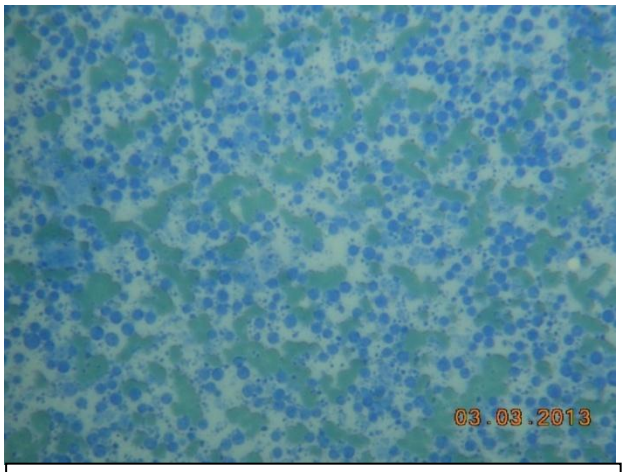
Scanner (4X) view: H & E stain



Scanner (4X) view: H & E stain



Oil Immersion(100X) view: H & E stain



Low Power (10X) view: Giemsa stain

Discussion: Stomach is the commonest site for extra nodal lymphoma in gastrointestinal tract. Otter R *et al* did analysis of 580 patients with Non Hodgkin's lymphoma among which 209 were having primary extra nodal disease. Within this group 96 patients presented with gastrointestinal disease, of which 56% were having disease limited to stomach.[6] Gastric lymphoma has also been seen to occur with disseminated nodal lymphomas. Hermann R *et al* noted gastrointestinal involvement in 46% cases of non hodgkin's lymphoma as an autopsy finding.[7] There are certain risk factors likely to increase the incidence of gastric lymphoma which include *Helicobacter pylori* infection, inflammatory bowel disease, celiac disease, transplant related immunosuppression and HIV infection. Miettinen *et al* demonstrated presence of *H. pylori* colonization in 59% to 98% cases of primary gastric lymphoma in their study.[8] Hussell showed that proliferation of B-cell primary gastric lymphoma is dependent on the activation of T-cells by helicobacter pylori.[9] Parsonnet J *et al* demonstrated that there is a six times increased incidence of Non-Hodgkin's gastric lymphoma with previous *H.pylori* infection.[10]

Radiologically, often it is found as rounded confluent sub mucosal nodules of a superficial spreading lesion, exophytic lesion, ulcerative lesion or more commonly as just a diffuse thickening of a stomach wall as seen in our case. However, it can not differentiate between lymphoma and adenocarcinoma.

Morphological changes like reactive lymphoid follicles, with mucosal atrophy and intestinal metaplasia are seen in the neighbouring mucosa. "Pseudolymphoma" is an inappropriate terminology which was previously designated as a reactive inflammatory response and was considered premalignant condition for gastric lymphoma. But now it is actually considered as a low grade variant of MALT lymphomas.[1]

Clonal population of B cells is the hallmark feature of the gastric lymphoma. It is divided into low grade and high grade varieties. Low grade variety shows centrocyte like cells infiltrating stomach wall with lesser degree of architectural distortion than high grade. Few times high grade variety demonstrate certain areas of low grade within it, further dividing high grade variety into mixed group. There is a widely accepted clinopathological staging criteria based on extent of disease which is called as Modified Ann Arbor classification but it lacks histological grading. According to it stage 1E is when disease is limited to stomach, stage 2E spread regionally within intra-abdominal lymph nodes, stage 3E spread to intra abdominal organs like liver, spleen & stage 4E spread within bone marrow.[11]

There are some specific chromosomal abnormalities found associated with primary gastric lymphoma like trisomy 3 linked with low grade lymphomas where as trisomy 12 & trisomy 18 linked to high grade lymphomas.[12] Isaacson PG *et al* demonstrated that inactivation of a tumour suppressor gene p53 and mutation of proto oncogene *c-myc* lead to development of high grade lymphoma.[13]

Prognosis of gastric lymphoma is better than other gastric tumours[14] because primary gastric lymphoma tends to remain localized for a long time. This has been attributed to its *homing phenomenon* in which the MALT lymphoma cells return to the original mucosal site rather than disseminate elsewhere.

Conclusion: The case here is presented because of the rarity of the lesion and it shows similarity with adenocarcinoma radiologically. Both the conditions are required to be differentiated from each other as treatment modalities and prognosis of the patients greatly differs between two.

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