



Metastatic Pancreatic Adenocarcinoma to a Lymph Node with Diffuse Large B Cell Lymphoma: A Case Report and Review of the Literature

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Abstract

Metastatic carcinoma to a lymph node with high grade lymphoma is very rare, particularly when high grade lymphoma is the first time diagnosis. Here we report a case where metastatic pancreatic ductal adenocarcinoma was found in a retroperitoneal lymph node with diffuse large B cell lymphoma (DLBCL). Histologically, the metastatic adenocarcinoma is located mainly in the subcapsular region. The lymph node architecture is diffusely effaced by proliferation of medium to large atypical lymphoid cells with prominent nucleoli, eosinophilic cytoplasm and eccentric nuclei. Immunohistochemical studies revealed these neoplastic lymphocytes are consistent with diffuse large B-cell lymphoma with immunoblastic morphology. Literatures on concomitant metastatic carcinoma with lymphoma in the same lymph node were reviewed. The significance and underline biological mechanisms were also discussed.

Keywords

Concomitant metastatic pancreatic ductal adenocarcinoma, Lymph node, Diffuse large B cell lymphoma

Introduction

The occurrence of multiple primary malignant tumors in the same individual is well reported [1,2], whereas coexistence of two malignancies in the same organ/site is uncommon [3,4]. The second malignancy may be unexpectedly discovered during the investigation for previous malignant tumor [5]. It has been reported previously that patients with small cell lymphoma/chronic lymphocytic leukemia (SLL/CLL), have an increased risk of developing other malignancies, particularly squamous-cell carcinoma [6-8]. However, metastasis of adenocarcinoma to a lymph node harboring diffuse large B cell lymphoma (DLBCL) is extremely rare. Here we report a case that DLBCL was unexpectedly discovered in a patient's retroperitoneal lymph node with metastatic moderately differentiated pancreatic ductal adenocarcinoma.

Case Presentation

A 79-year-old Caucasian male presented with chest pruritus, darkening urine and clay-colored stools for 2 months. This diabetic

patient has family history of pancreatic cancer (father). Accompanying symptoms included depression, dizziness, dry mouth and recent weight loss of 6.8 kg. There was no fever or night sweats. Computer tomography showed a 1.9 cm mass in the head of pancreas, and several less than 1 cm lymph nodes in the peripancreatic aortocaval celiac. No significant adenopathy was identified in chest and pelvis. Magnetic resonance cholangiopancreatography (MRCP) showed dilatation of both the common bile duct and the pancreatic duct, suggesting a lesion of pancreatic head. Endoscopic ultrasound (EUS) guided fine-needle aspiration (FNA) biopsies of the pancreatic mass revealed adenocarcinoma. Pancreaticoduodenectomy was performed with feeding jejunostomy. During the surgery, a bulky lymph node, 2.5 cm in size was identified, which is directly behind the C-loop of the duodenum and is within 1 cm to common bile duct margin. This lymph node was mobilized and dissection of regional lymph nodes was performed. Grossly, within the pancreatic head, there is an ill defined tan to white mass, 4 x 3 x 3 cm adjacent to common bile duct and duodenum. Tumor is confined to the pancreatic parenchyma. Histological examination reveals an infiltrating moderately differentiated pancreatic ductal adenocarcinoma (Figure 1). Examination of regional lymph nodes shows metastatic adenocarcinoma in 8 out of 18 lymph nodes, and among the 8 positive lymph nodes, one also displays features of diffuse large B cell lymphoma. As illustrated in figure 2, metastatic adenocarcinoma was seen predominantly in the subcapsular area (Figure 1). In addition, the architecture of the lymph node is diffusely effaced by proliferation of medium to large atypical lymphoid cells with prominent nucleoli, eosinophilic cytoplasm and some with eccentric nuclei. Small reactive lymphocytes are seen in the background. Few residual follicles are also noted (Figure 2). The neoplastic lymphocytes are positive for CD45, CD20, Pax-5, CD79a, Bcl-2, MUM-1, CD43 (Figure 2), and negative for CD10, Bcl-6, CD30, CD3, CD4, CD7, CD8, TIA, Granzyme-B, CD138, kappa, lambda and EBV-LMP. CD5 immunostain shows focal weak staining of neoplastic lymphoid cells. CD23 and CD21 highlight the residual follicular dendritic cell meshwork. The MIB-1 proliferation index is approximately 60-70%. The overall morphological features and immunostain profiles are consistent with DLBCL with immunoblastic morphology. Pathological stage of pT2N1M0 for pancreatic adenocarcinoma was assigned with clinical stage of IIB. The anatomic stage/prognostic group for

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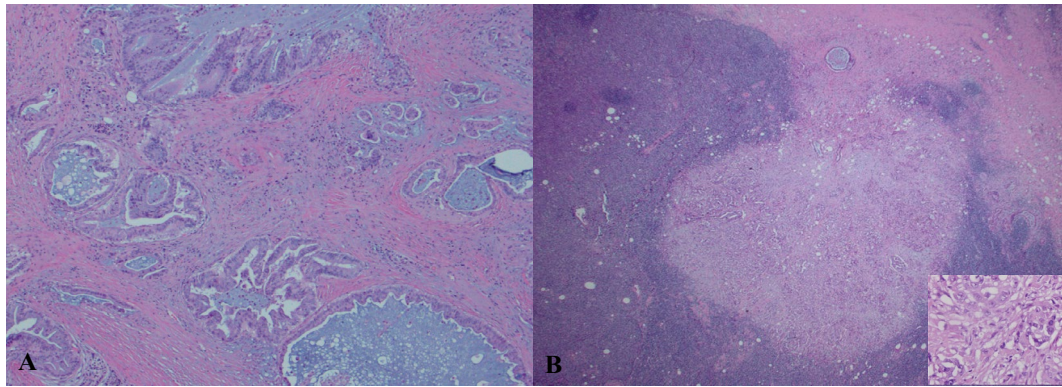


Figure 1: Pancreatic ductal adenocarcinoma (A) and its metastasis to a retroperitoneal lymph node (B). H & E stain, A: 40 x and B: 40 x with insert 200 x.

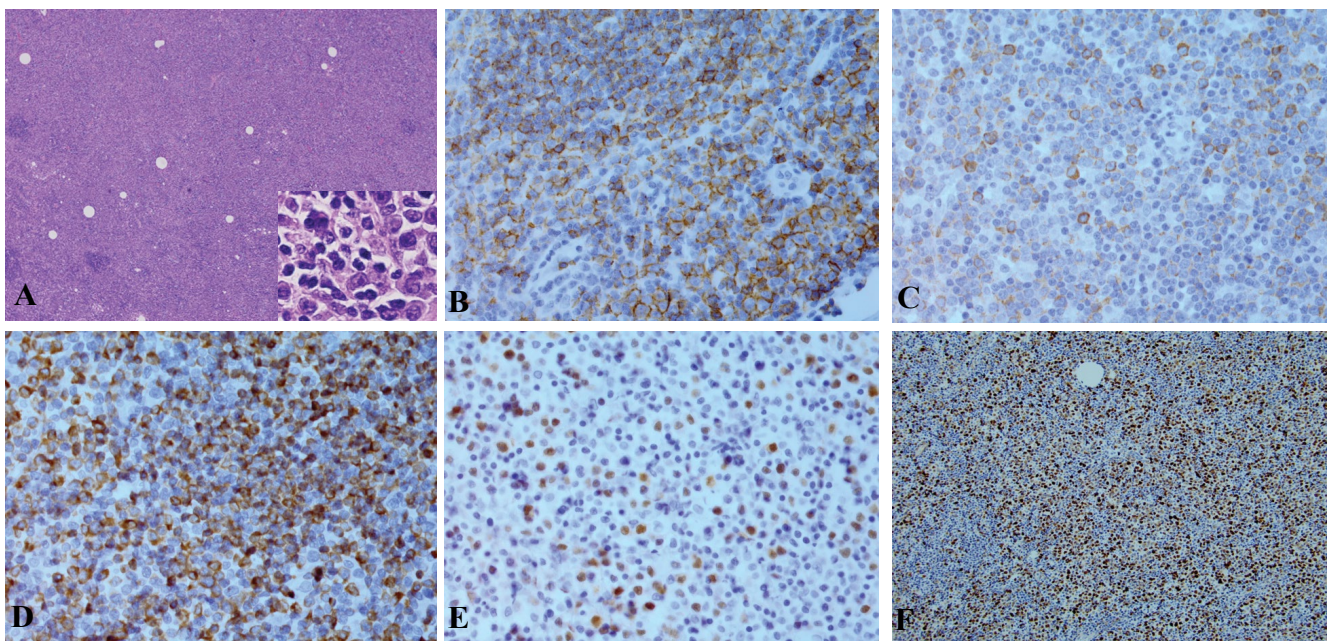


Figure 2: DLBCL of a lymph node with metastatic pancreatic adenocarcinoma. A: H&E stain, 40 x with insert 200 x, B-F: Immunostains for CD20 (B), CD79a (C), BCL-2 (D), MUM-1 (E) and Ki67 (F). B-E, 200 x, F, 40 x.

DLBCL is stage I. The patient was given adjuvant chemotherapy for pancreatic adenocarcinoma with gemcitabine, 5-FU and leucovorin. After 10 cycles of chemotherapy, peritoneal recurrence of metastatic adenocarcinoma was identified. Although Leucovorin and 5-FU were added in the treatment regiment, progress of disease was not controlled. Fifteen months after Whipple procedure, the patient was transferred to hospice care. No treatment was given for DLBCL post surgery.

Discussion

Collision tumors are rare entities defined by presence of two neoplasms of distinct origin found in a single anatomic location [9,10]. The mechanism of the second primary malignancy development is unclear. There are multiple hypotheses among the pathogenic mechanisms to explain the increased risk of second tumor, such as mutagenic effects of radiation and chemotherapy, genetic predisposition, and environmental factors [6]. In our case, metastatic pancreatic ductal adenocarcinoma and DLBCL were identified in the same lymph node. DLBCL is an aggressive lymphoma and can involve the whole lymphatic system. However, the DLBCL was unexpectedly identified in a retroperitoneal lymph node and not found in other site/organ in this patient. It has been speculated that DLBCL may increase the risk for developing other non-hematological malignancies [11-13].

Pancreatic cancer is the fourth leading cause of cancer death in the United States, and it has the worst prognosis of any major tumor type.

There are some risk factors of developing pancreatic cancer, such as family history [14], cigarette smoking [15], type 2 diabetes [16], increasing body mass index [17], and heavy alcohol consumption [18]. There is also 20-40% higher incidence of malignancy, such as pancreatic carcinoma and non-Hodgkin's lymphoma, in type 2 diabetes patients [19]. In our case, patient's family history of pancreatic cancer and his diabetes may be contributing factors to the development of pancreatic carcinoma. It is not clear whether the microenvironment created by the first malignancy in lymph node potentiates development of the second malignancy. More studies need to be done to further our understanding of the mechanisms for this rare collision of malignant tumors.

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