International Society of Gastrointestinal Oncology
2009 Gastrointestinal Oncology Conference
October 1–3, 2009

ABSTRACTS SELECTED FOR POSTER PRESENTATIONS

Pancreatic Cancer

abstr 0919

Gastric Large Cell Neuroendocrine Carcinoma

Tarun Rustagi, MD[1], Todd J. Alekshun, MD[2]
[1]Department of Internal Medicine, University of Connecticut Health Center, Farmington, CT; [2]Division of Hematology and Oncology, Hartford Hospital, Hartford, CT

Case: A 63-year-old male presented with unintentional weight loss of 20 pounds over a 4-month duration. He reported loss of appetite, intermittent post-prandial nausea, bloating and early satiety. He also complained of dyspepsia and had been treated for reflux during the previous 2 years. He denied vomiting, dysphagia, odynophagia, abdominal pain, melena, hematochezia, or alterations in bowel habits. Additionally, he denied fevers, night sweats, cough, or dyspnea. He quit smoking 25 years ago, and denied alcohol use. His past medical history was significant for basal cell carcinoma treated with local curative therapy and he was without recurrence on surveillance. Pertinent family history included a paternal uncle with lung cancer at the age of 74. Physical examination was unremarkable except for occult heme-positive stools. Laboratory evaluation revealed elevated liver enzymes (ALT-112, AST-81, AlkPhos-364). CT scan of the chest, abdomen and pelvis showed diffuse heterogeneous liver with extensive nodularity, raising the concern for metastases. Serum tumor-markers: PSA, CEA, CA 19-9, and AFP were all within normal limits. Screening colonoscopy was normal, but esophagogastroduodenoscopy revealed a malignant-appearing ulcerative lesion involving the gastro-esophageal junction and gastric cardia. Pathology confirmed an invasive gastric large cell neuroendocrine carcinoma. Ultrasound-guided fine needle aspiration of a hepatic lesion revealed malignant cells with cytologic features consistent with large-cell type carcinoma and positive immunostaining for synaptophysin favoring neuroendocrine differentiation. A PET-CT demonstrated intense diffuse FDG uptake of the liver, suggesting diffuse hepatic parenchymal infiltration by tumor. There were multiple foci of intense osseous FDG uptake with corresponding osteolytic lesions seen on CT scan. The remaining intra-abdominal and intra-thoracic structures were unremarkable. The patient will receive palliative systemic therapy with a platinum-based regimen.

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Discussion: In the current WHO classification of gastric tumors, only carcinoids and small cell carcinomas are included in the neuroendocrine (NE) tumor category. However, a new pathologic entity has recently been described by Jiang et al. High-grade gastric NE carcinomas of non-small cell type have been tentatively named large cell neuroendocrine carcinomas (LCNEC). Morphologically, these tumors differ from both carcinoids and small cell carcinomas, and are confirmed immunohistochemically using NE markers, chromogranin-A and synaptophysin. Gastric LCNEC is currently defined if > 50% of tumor cells demonstrate positivity for chromogranin-A and/or synaptophysin.

LCNECs account for < 1.5% of all gastric cancers. LCNECs, which had been previously diagnosed as adenocarcinomas (ACs) are highly malignant and portend a significantly worse prognosis than ACs. Reported 5-year survival rates for LCNECs and ACs have are 31.1% and 69.3%, respectively. At the time of presentation, 70-75% of LCNECs have lymph node metastases and 5-10% also have metastases to liver. Clinical management of gastric LCNECs has not been clearly defined given the rarity of the malignancy and limited experience in its management. An overall regression rate of 67% was achieved in a small series of poorly differentiated gastroenteropancreatic NE carcinomas treated with cisplatin and etoposide.