Medical Image of the Week: Tumor-Induced Hypoglycemia

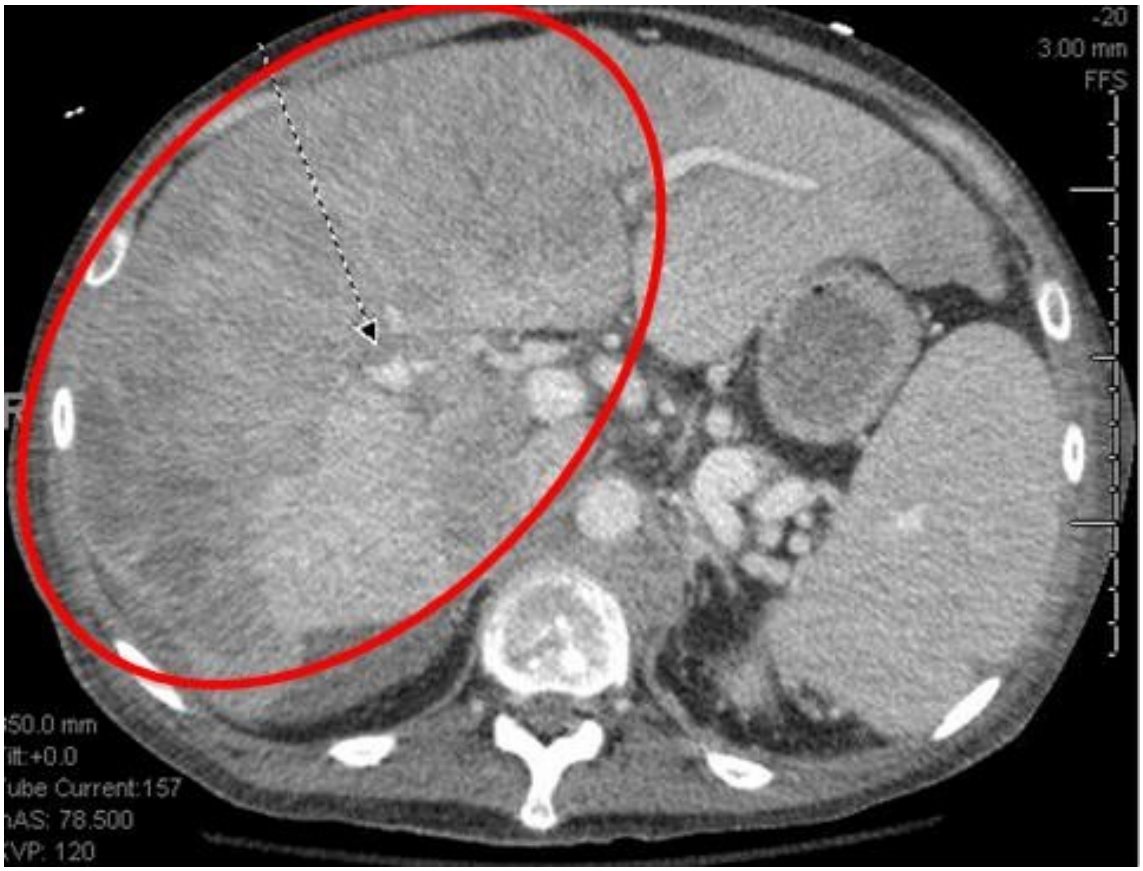

Figure 1. CT of the abdomen with IV contrast (axial image) demonstrating numerous large enhancing liver metastases (red oval) and tumor thrombus in the anterior segment branch of the right portal vein (arrow).

A 39 year-old man with a history of widely metastatic (brain, liver and lung) nonseminomatous germ cell tumor was admitted to the hospital with severe abdominal pain and altered mental status. A CT of the abdomen and pelvis with IV contrast revealed a marked increase in the size of the liver metastases, portal vein tumor thrombus and changes of pseudocirrhosis. There were numerous large heterogeneously enhancing masses within the liver parenchyma with central necrosis (Figure 1).

The patient had significant and sustained hypoglycemia, with the lowest glucose recorded of 30 mg/dl. He required multiple IV doses of 50% dextrose and an infusion of 10% dextrose to maintain a serum glucose level greater than 55 mg/dl. His mental status improved with treatment of the hypoglycemia. The patient decided to pursue a palliative approach to care and was discharged with home hospice services.

Tumor-induced hypoglycemia (TIH) is a paraneoplastic syndrome that is uncommon in clinical practice (1). The more common cause of TIH is insulin hypersecretion in the setting of pancreatic beta-cell tumors. Mechanisms that may lead to TIH without insulin hypersecretion include the hypersecretion by tumors of insulin-like growth factor 2 (IGF2) and it's precursors, insulin-like growth factor 1 (IGF1), somatostatin, and glucagon-like peptide 1. Other pathogenic causes of hypoglycemia include tumor autoimmune hypoglycemia, massive tumor burden, massive tumor liver infiltration, and pituitary or adrenal gland destruction by the tumor. TIH unrelated to pancreatic tumors is called non-islet-cell tumor hypoglycemia (NICTH). The most common cause of NICTH is the hypersecretion of IGF2 and IGF2 precursors by the tumor. This results in increased glucose consumption peripherally and decreased production of glucose in the liver. We did not measure levels of IGF2 in our patient. It was felt the most likely cause of his hypoglycemia was extensive tumor invasion and destruction of the liver due to his advanced disease.

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Reference

1. Iglesias P, Díez JJ. Management of endocrine disease: a clinical update on tumor-induced hypoglycemia. Eur J Endocrinol. 2014;170(4):R147-57. [CrossRef] [PubMed]