Hepatocellular Carcinoma Recurrence Presenting as an Abdominal Wall Mass

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INTRODUCTION: A 74 year-old-male with a history of NASH cirrhosis complicated by hepatocellular carcinoma (HCC) underwent orthotopic liver transplantation (OLT) in 5/2013. Surgical margins were negative for carcinoma however there were foci suggestive of vascular invasion. The patient was later found to have atypical areas of recurrence.

CASE DESCRIPTION/METHODS: After OLT the patient was complication-free until he was found to have recurrence in 2017 with an elevated AFP 22.1 and an abdominal MRI which demonstrated a 1.4 x 1.1 cm focus of washout in hepatic segment VII. He was treated with transarterial chemoembolization (TACE). A repeat MRI several months later did not demonstrate this lesion however his AFP continued to increase to 89. One year later his AFP was 378.6 and an MRI pelvis without and with contrast demonstrating a large right anterior lower quadrant mass. He was treated with transarterial chemoembolization (TACE) before OLT may help decrease recurrence2. While most recurrences of HCC after OLT are extracapsular this patient had recurrence within the transplanted liver. Additionally, his latest recurrence as an abdominal wall mass demonstrates an atypical location that providers should be aware of.

REFERENCES
1. Lim KC, Pierce KHC, Allen JC, Chia GS, Lim M, Cheow PC, Chung AYF, Ooi LLP, and Tan SB. Microvascular invasion is one of the most predictive factors for recurrence and is associated with poor survival3. Other factors include positive surgical margins and tumor size. Locoregional therapy (such as TACE) before OLT may help decrease recurrence. While most recurrences of HCC after OLT are extracapsular this patient had recurrence within the transplanted liver. Additionally, his latest recurrence as an abdominal wall mass demonstrates an atypical location that providers should be aware of.


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Rare Case of a Trifecta: CardioHepatoRenal Syndrome

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INTRODUCTION: Cardiorenal syndrome encompasses a bidirectional interaction between heart failure and kidney disease, in which dysfunction of the heart or kidneys can induce dysfunction in the other. CRS presumably occurs due to an inability of the failing heart to sufficiently generate forward flow, leading to prerenal hyperfusion of the kidneys. We present a case of presumed cardiac syndrome with subsequent hypoxic hepatitis complicated by rhabdomyolysis.

CASE DESCRIPTION/METHODS: Our patient was a 71-year-old male with numerous medical comorbidities including HFrEF, chronic kidney disease (CKD), chronic alcohol use, and a recent history of abdominal aortic aneurysm repair. He initially presented with symptoms of shortness of breath, dry cough, and abdominal pain that progressively worsened. Upon admission, the patient’s laboratory and imaging findings supported a diagnosis of acute on chronic congestive heart failure (CHF) exacerbation. He was started on furosemide and carvedilol. Elevated creatinine and creatine kinase (CK) revealed acute renal injury secondary to rhabdomyolysis. He was given a fluid bolus and started on low rate maintenance fluid due to CHF and CKD. Two days later, repeat CMP showed worsening CK levels. Fluids were increased with adjunctive milrinone therapy. Repeat liver enzymes showed worsening liver; fluid resuscitation was discontinued due to volume overload concerns. Days later shortness of breath improved, and liver and renal enzymes stabilized. Milrinone was discontinued. The patient’s course was complicated due to hospital-acquired pneumonia which worsened his liver hypoxia. Eventually the patient died from multi-organ failure.

DISCUSSION: Our patient fits criteria for cardiorenal syndromes (CRHS). The cause for liver shock was likely multifactorial. This case reveals the challenge regarding treatment in patients with CRHS. Fluid administration for rhabdomyolysis is recommended, however our patient was unable to tolerate due to CHF fluid overload. The only established treatment for hypoxic hepatitis involves treating the underlying disease state. Inotropic agents improve cardiac contractility, renal perfusion and preserve hepatic microcirculation and oxygenation. However, in our CHF patient, chronic use of milrinone was contraindicated due to ischemia and arrhythmia concerns. Establishing a better treatment paradigm for patients with cardiorenal syndrome, particularly when complicated by hypoxic hepatitis and rhabdomyolysis, is important.

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A Unique Case of Neutrophilic Dermatitis (Sweet Syndrome) Associated With Primary Sclerosing Cholangitis: Case Report and Literature Review

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INTRODUCTION: We present a unique case of Sweet Syndrome associated with primary sclerosing cholangitis.

CASE DESCRIPTION/METHODS: A 39-year-old woman presented to the emergency department with diffuse, nontender, skin rash and recent urivates. Her inflammatory markers were elevated. Serologic studies showed a positive ANA titer of 1:320 and a p-ANCA titer of 1:80. A full-thickness skin biopsy revealed dense neutrophilic inflammation without evidence of vasculitis. Tissue cultures were negative. She was started on oral corticosteroids with complete resolution. She was found to have an elevated alkaline phosphatase of 677 U/L. Magnetic resonance cholangiopancreatography (MRCP) revealed intrahepatic ductal dilation in an irregular beaded pattern consistent with primary sclerosing cholangitis. She did not have any gastrointestinal symptoms and had a normal previous colonoscopy.

DISCUSSION: Sweet Syndrome is an inflammatory condition characterized by the abrupt eruption of cutaneous lesions. Characteristic findings on histology include prominent edema in the dermis, a dense neutrophilic infiltrate, and endothelial swelling without vasculitis. The most common extra-cutaneous manifestation is urivates. The pathogenesis of Sweet syndrome is unclear but has been postulated to be associated with immune and cytokine dysregulation. Sweet syndrome has been associated with multiple disorders including infections, inflammatory bowel disease (IBD), autoimmune, pregnancy, malignancies, and certain prescription medications. To our knowledge, this is the first described case of Sweet Syndrome associated with primary sclerosing cholangitis in a patient without IBD.