Sinusoidal Obstruction Syndrome: A Known and Often Missed Complication of Oxaliplatin Therapy

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Background

Sinusoidal obstruction syndrome (SOS) is caused by sinusoidal endothelial injury in the setting of hematopoietic cell transplantation, liver irradiation or exposure to chemotherapy such as oxaliplatin and may occur days to weeks following exposure.

SOS can be a life-threatening complication and its presentation may include jaundice, volume overload or hepatic failure.

We present the case of a 32-year-old male with gastroesophageal adenocarcinoma who underwent oxaliplatin-based chemotherapy and developed SOS.

Case report

A 32-year-old male with no significant medical history presented with dysphagia and weight loss. Esophagogastroduodenoscopy showed a mass at the gastroesophageal junction.
Diagnostic laparoscopy showed peritoneal metastasis. Tissue biopsy was consistent with stage 4 gastroesophageal junction adenocarcinoma. Patient was started on 5-fluorouracil (5-FU), oxaliplatin and nivolumab. He developed thrombocytopenia and moderate aminotransferase elevation after 6 cycles of chemotherapy.

Abdominal ultrasound following 10 of 12 cycles of chemotherapy showed portal hypertension. Subsequent abdominal imaging showed worsening ascites (serum ascites to albumin gradient > 1.1), anasarca and splenomegaly.

Transjugular liver biopsy showed elevated portal hepatic venous pressure gradient, focal sinusoidal dilatation and centrizonal ischemic changes suggesting drug induced hepatopathy, and a diagnosis of oxaliplatin induced SOS was made. The patient was not a candidate for defibrotide and was treated conservatively. He continued to have worsening ascites with severe direct hyperbilirubinemia and moderately elevated aminotransferases without biliary obstruction.

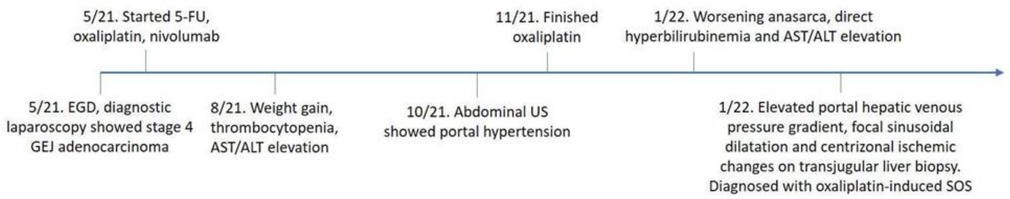

Figure 1. Timeline of events leading up to SOS diagnosis.

Discussion

Oxaliplatin-based chemotherapies are known to cause the potentially life-threatening complication of SOS which is oftentimes missed.

Our patient developed signs and symptoms suggestive of SOS by cycle 10 of oxaliplatin therapy. These changes included thrombocytopenia, elevated liver associated enzymes, volume overload and portal hypertension. The patient went on to complete 12 cycles of oxaliplatin with eventual worsening of his portal hypertension related symptoms and worsening hepatic function.

This case highlights the importance of having a high index of suspicion for the development of SOS with plans to pursue patient-centered discussions regarding alternative chemotherapeutic regimens, instituting prophylactic therapies and timely SOS screening protocols.