INTRODUCTION: Acute pancreatitis (AP) and acute aortic dissection (AAD) are medical emergencies which must be promptly recognized to avoid the development of life-threatening complications. Both of these diseases can present with chest or epigastric pain which can radiate to the back, thus early suspicion based on clinical presentation and risk factors is essential. We present the case of a classic AP later incidentally discovered to be AAD.

CASE DESCRIPTION/METHODS: A 56-year-old man with a history of alcohol abuse presented with 1 day of diffuse abdominal pain, nausea, and vomiting. His lipase was 3909 U/L and creatinine 2.19 mg/dL and was diagnosed as acute alcoholic pancreatitis with acute kidney injury. A non-contrast computed tomography (NCCT) scan of the abdomen showed aortic calcifications. He received 3.8 liters of fluids after which he developed acute respiratory distress requiring intubation. A workup for ECMO was initiated given the suspicion of acute respiratory distress syndrome due to pancreatitis which revealed an AAD with severe aortic regurgitation on transthoracic echocardiography. CT angiogram showed type A AAD (Figure 1) involving the aortic root, ascending aorta, descending aorta, suprarenal and infrarenal abdominal aorta with a patent celiac axis (Figure 2), SMA, and IMA. The patient underwent type A dissection repair with mechanical aortic valve replacement and survived the acute event. His AP resolved and he was discharged home with appropriate follow up.

DISCUSSION: AP is rarely associated with AAD with only 11 documented cases, the exact mechanism not fully elucidated. AAD involving arteries to the pancreas is thought to cause hyperperfusion leading to ischemic injury. The pancreas has a rich collateral circulation which may be the reason there was no radiographic evidence of pancreatitis in our patient. In suspected cases of pancreatic necrosis and AAD, cholestrol emboli are thought to be the culprit well known to cause end-organ damage. Our patient’s respiratory distress was from cardiogenic pulmonary edema due to aortic regurgitation and aggressive fluid resuscitations for suspected pancreatitis. An NCCT was performed due to the ARD in our patient which can miss subtle findings of AAD, therefore, a contrast-enhanced study is recommended. We hypothesize that if our patient was not assessed for ECMO, the finding of AAD would have been a diagnostic challenge. AP secondary to AAD is rare but a high index of suspicion is required for diagnosis.

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[Image 57x287 to 289x438]  

Figure 1. Computer tomography angiogram of the chest. Demonstrating a Type A Aortic Dissection involving the Ascending and Descending Aorta.