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Doxycycline-Induced Acute Pancreatitis: A Likely Source Rarely Seen

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Doxycycline Induced Acute Pancreatitis:

A likely source rarely seen

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Introduction

The reported annual incidence of acute pancreatitis in the United States ranges from 4.9 to 35 per 100,000 populations [1]. The diagnosis of drug-induced acute pancreatitis compared to the others causes of acute pancreatitis (AP) is estimated between 1.4 and 2%. As this is a rare etiology of acute pancreatitis, the diagnosis is often difficult to establish. [2]. Drug-induced pancreatitis is classified (Class I-IV) based on the number of cases reported, demonstration of a consistent latency period (time from initiation of drug to development of pancreatitis), and reaction with rechallenge. Doxycycline is a class I drug (At least 1 case report with positive re-challenge, excluding all other causes) which has the greatest potential for causing acute pancreatitis [3]. Despite this association the clinical evidence on doxycycline-induced pancreatitis is sparse as proving the association with a particular drug may not always be straightforward, even in suspected cases [4]. In this case, we report an episode of an acute pancreatitis where well-known etiologies such as alcohol, hypertriglyceridemia, hypercalcemia, gallstones, and other drugs were ruled out and patient was deemed to have doxycycline induced acute pancreatitis (DIAP)

Case Description

A female in her early 70's with a past medical history of a previous cholecystectomy, and bullous pemphigoid who presented to the Emergency Department (ED) of RMCBP with one-day history of epigastric pain. She does admit to eating a chicken salad sandwich then subsequently having nausea with multiple episodes of non-projectile emesis. She denies previous history of pancreatitis, heavy alcohol consumption or binge drinking. She has been on Zocor for many years without any recent dosage changes. Patient did not exhibit fevers, chills, hematemesis, hemoptysis, chest pain, shortness of breath, hematochezia or melena. Upon further questioning, she does admit to a recent addition of doxycycline by her dermatologist about one month ago which she took twice daily to treat her bullous pemphigoid. Physical examination revealed an elderly patient in no acute distress; vital signs were within normal limits. Vital signs were within normal limit.

Case continued

Abdominal examination was positive for mild epigastric tenderness, soft with no guarding, normal bowel sounds, no distention or hepatosplenomegaly noted. The results of the laboratory studies included a white-cell count of 28,100 mm³, with 92.9% neutrophils, the blood urea nitrogen level was 25 mg/dL, the serum creatinine level was 1.33 mg/dL and the sodium level was 138 meq/L, blood glucose 253, HA1c 7%, Lipase 6204. Abdominal and pelvis tomography obtain revealed peri-pancreatic fat stranding and fluid suggestive of acute pancreatitis. A Magnetic Retrograde Cholangio-Pancreatography (MRCP) was obtained which revealed diffuse peri-pancreatic edema and inflammatory changes with free fluid surrounding the pancreatic head and body, perihepatic and perisplenic regions compatible with patient's known history of pancreatitis (Figure 2, Red arrow). Furthermore, no extrahepatic biliary dilation, choledocholithiasis or choledochal filling defects observed to suggest biliary tree stone (Figure 2, Blue arrow). After a review of numerous literature of drug implicated in DIP as well as ruling out other causes of acute pancreatitis we determined that our patient had doxycycline-induced pancreatitis. Doxycycline was held immediately. She was started on normal saline IV fluids and broad spectrum antibiotics. The following day the patients' abdominal pain began improving. Oral feedings were started as patient was no longer nauseous and the rest of his hospital course was uneventful. Patient was discharged 6 days after admission.

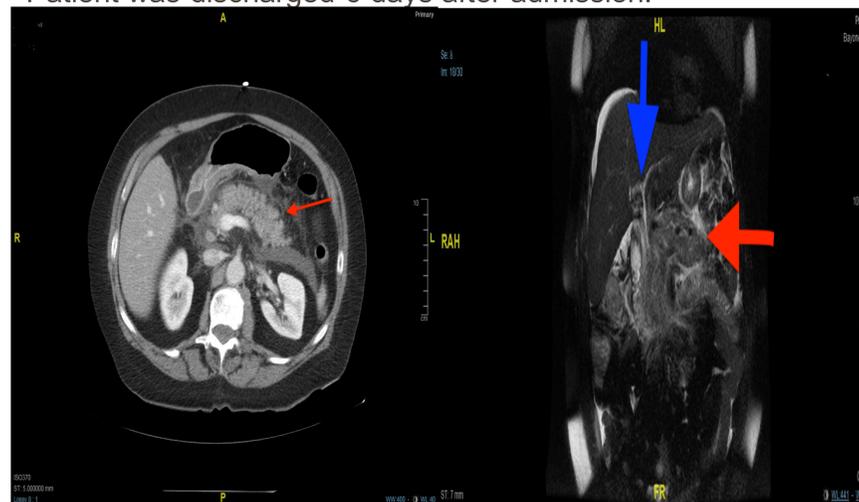


Figure 1

Figure 2

Discussion

Tetracycline has long been designated as a causative agent in acute pancreatitis. Unfortunately, there has only been 5 reports of DIP following doxycycline either used in monotherapy or in combination therapy [5,6,7]. Interestingly all five previous cases involved a female patient as in this case which is most likely coincidental. Nevertheless, reports of doxycycline as the sole source of DIP comparable to our case have only been reported once [8]. The rate of onset of symptoms after administration of drugs can be seen almost immediately to about 1 month (like in our case) owing to the variability of onset seen in DIP. What makes our case unique is that this would be the second documented DIP developed secondary to doxycycline as the sole etiology [9,10,11]. Between 1968 and 1993 the World Health Organization (WHO) linked over 525 different drugs in the etiology of DIP [12].

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