St. Joseph's Health ST. JOSEPH'S UNIVERSITY MEDICAL CENTER



Background

- Autoimmune Hepatitis (AIH) is a progressive form of chroi periods of remissions and exacerbations
- Diagnosis includes abnormally high levels of immunoglobut autoantibodies, with female predominance
- Clinical presentation is variable, with a spectrum extending cases to fulminant liver failure
- Presenting symptoms may include abdominal pain, malaise, fatigue, and small joint arthralgia
- We present a case of a 36 YO M with PMH of alcohol dependence and acute pancreatitis who was diagnosed with AIH

Case Description/Methods

<u>A 36-year-old African American male with PMH of alcohol dependence (in</u> remission for 2 yrs), tobacco use, and pancreatitis, presented with epigastric pain

- The patient endorsed non-radiating mid-epigastric abdominal pain 10/10 in severity, associated with NBNB emesis, exacerbated by movement for 2 days
- Vitals: WNL
- Physical Examination: +icteric frenulum, abdominal distension, with liver span 12cm at mid-clavicular line, and absence of fluid wave, shifting dullness, rebound tenderness, or voluntary guarding
- Laboratory studies: pancytopenia, elevated lipase, elevated ALP, elevated AST and ALT with 2:1 ratio, and hyperbilirubinemia [Table 1]
- MRCP showed cirrhotic liver with splenomegaly and varices, as well as with free fluid in the lesser sac along the pancreatic head, duodenum, and right retroperitoneum (compatible with acute pancreatitis) [Figure 2]
- EGD demonstrated findings associated with chronic pancreatitis [Figure 3]
- The patient received IV fluids for pancreatitis
- Additional labs were remarkable for elevated anti-smooth muscle antibody at 26U (ref range: 0-19), ANA positive, with high alpha-1-antitrypsin levels and normal ceruloplasmin levels
- The patient left against medical advice; and was given resources for Hepatology with referral for liver transplant.

References

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Autoimmune Hepatitis presenting with Concomitant Chronic Pancreatitis

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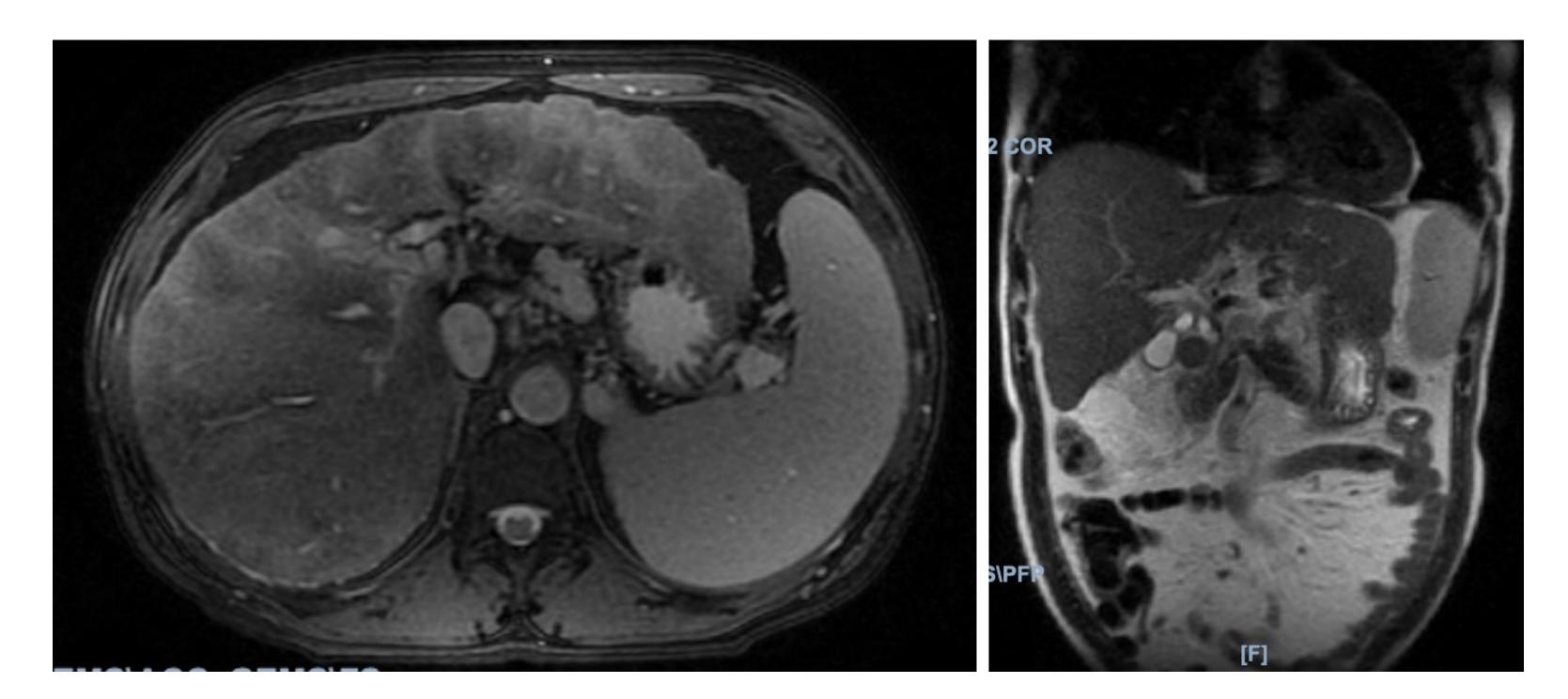
onic hepatitis, with
oulins and multiple
from asymptomatic
fatique, and small

	Results	
Test Name	Reading	Reference Range
WBC	3.9 x10 ³ /mm ³	4.5-11
Hgb	12.6 g/dL	13.5-17.5
Plt	66 k/mm ³	140-440
Lipase	528 U/L	11-82
ALP	129 U/L	34-104
AST	114 U/L	13-39
ALT	47 U/L	7-52
T. Bili	4.9 mg/dL	0.3-1.1

Table 1. Laboratory Studies



Fig 1. Abd US notable for cirrhosis w/ possible cavernous transformation of portal vein, heterogeneity of pancreas, & gallstone sludge



and right retroperitoneum (compatible with acute pancreatitis)

Fig 2. MRCP demonstrating cirrhotic liver w/ splenomegaly & varices, and free fluid in the lesser sac along the pancreatic head, duodenum,



2 Lower Third of the Esophagus



Fig 3. EGD demonstrating grade II & large (>5 mm) esophageal varices w/o evidence of bleeding, with 2 cm hiatal hernia, portal hypertensive gastropathy, and pancreatic parenchymal abnormalities (calcifications, diffuse echogenicity, hypoechoic foci and lobularity), consistent with chronic pancreatitis

- Mechanism of AIH remains poorly understood; however, there is an association between the HLA gene & AIH [1]
- Genetic studies have shown HLA-DRB1*0301 and HLA-DRB1*0401 as primary and 2ndary genotypes susceptible to AIH, as well as genetic variants w/ CARD10 & SH2B3 [2]
- Products secondary to metabolism of ETOH like ETOH-dehydrogenase, malondialdehyde, & acetaldehyde can lead to development of autoAbs [3,4]
- Additional research is indicated to evaluate the relationship between AIH & acute pancreatitis.





Results (continued)



Gastroesophageal Junction



14 CBD close to Panc and Panc head with calcifications

Discussion