Gastrointestinal involvement: human monocytic ehrlichiosis

Gastrointestinal involvement of human monocytic ehrlichiosis, including a novel find
elevation of pancreatic lipase.

SUMMARY

A 57 year-old man presented to the hospital with septic shock. He was found to have
monocytic ehrlichiosis (HME) with constitutional symptoms, as well as multiple
gastrointestinal manifestations, including nausea, vomiting, diarrhea, hepatosplenor
and cholestasis. He was also found to have an elevated lipase, suggestive of pancrea
although this was most likely secondary to circulatory shock and surrounding inflam
(he had no abdominal pain or radiographic evidence of pancreatitis). He was treated
doxycycline, which resulted in rapid improvement clinically and in terms of his labora
values.

BACKGROUND *Why you think this case is important – why did you write it up?*

This case reminds us that HME can have numerous gastrointestinal manifestations, a
which can present in a single patient. Prompt recognition and treatment is necessary
because this is a life-threatening disease with a high mortality rate if untreated. A m
organ process, HME can result in circulatory shock. This is the first case to report an
elevation in pancreatic lipase in association with HME. This further reminds us that li
elevation can be seen in a variety of conditions beside pancreatitis.

CASE PRESENTATION *Presenting features, medical/social/family history*

A 57 year-old man presented to the Emergency Department (ED) of our hospital in Ju
several days of fever, nausea, vomiting, chills, myalgias, diarrhea, headache and leth
He had no abdominal pain. Six days earlier, he presented to the ED of another hospi

fever, and was treated with a course of levofloxacin for possible sinusitis, but experie
improvement. His past medical history included atrial fibrillation and hypertension. H
smoked one pack a day and drank 2-3 beers a week. He resided in rural Southern Ne
and reported that several of his family members had recently been bitten by ticks, th
does not recall being bit. His blood pressure was initially 60/40 but increased to 106/
receiving five litres of intravenous fluid. His temperature was 36. 9 C. Pulse was initia
and irregular but normalized with fluid resuscitation. He was alert and oriented to pe
place and time. He had dry mucous membranes, a benign abdomen, palpable hepat
with a smooth liver edge, and no visible rash or insect bites.

INVESTIGATIONS *If relevant*

Lab abnormalities included a lipase of 1772 U/L (normal 16-63 U/L), an aspartate aminotransferase (AST) of 110 U/L (normal 10-35 U/L), an alanine aminotransferase 83 U/L (normal 6-45 U/L), a total bilirubin of 1. 4 mg/dL, a direct bilirubin of 0. 9 mg/d creatinine of 1. 78 mg/dL (normal 0. 60-1. 20 mg/dL). Alkaline phosphatise was norm complete blood count with differential was notable for a platelet count of 69, 000 U/L bandemia of 37%.

Computed tomography of the abdomen with oral contrast was obtained to rule out pancreatitis. It revealed enlargement of the liver and spleen, a normal gallbladder ar pancrease, and numerous sigmoid diverticula. A small amount of intraperitoneal free was also noted above both inguinal canals. Ultrasonography of the right upper quadr revealed cholelithiasis but nondilated bile ducts.

DIFFERENTIAL DIAGNOSIS *If relevant*

At the top of the differential were the tickborne illnesses. Fulminant leptospirosis (We

disease) was also considered a possibility given his acute non-specific febrile illness multisystem involvement. Enzyme immunoassay test for antibody to *B. Burgdorferi* negative. Rocky mountain spotted fever and leptospirosis serologies were also negat was polymerase chain reaction (PCR) for anaplasma and babesia. No malarial parasi babesia were observed on smear, and blood and urine cultures did not grow any org Finally, *E. Chaffeensis* DNA was detected by PCR.

TREATMENT *If relevant*

The patient was initially treated in the Emergency Department with very broad micro coverage, including levofloxacin, vancomycin, piperacillin-tazobactam, metronidazol doxycycline. He was soon brought to the intensive care unit, where treatment was c solely with doxycycline.

OUTCOME AND FOLLOW-UP

Following treatment, he achieved very rapid clinical improvement and was discharge sixth hospital day. His creatinine normalized with the administration of intravenous f the fifth hospital day his AST and ALT had trended down to 46 U/L and 49 U/L, respec his total and direct bilirubin had normalized at 0. 6 mg/dL and 0. 2 mg/dL; and his lip almost came down to normal at 84 U/L.

DISCUSSION *Include a very brief review of similar published cases*

HME is caused by *E. Chafeensis* , an obligately intracellular organism that primarily i mononuclear phagocytes. [1] Over 5496 cases had been reported to the CDC as of 2 most of which were in the south-central, southeastern and mid-Atlantic United State transmitted by the Lone Star tick (*A. americanum)* with white-tailed deer, dogs and

as the reservoir. Most infections happen in the summer months, with the average ho
a male in his 50s; men may be infected more due to more occupational and recreati
exposure.[1, 2] The fatality rate of HME has hovered around 1-2% in recent years.[3]
most common presenting symptoms are fever, headache, anorexia, myalgia, chills a
rigors, nausea and vomiting, and rash, with 40% of cases requiring hospitalization.[2
Other gastrointestinal manifestations are described below. Meningoencephalitis, acu
respiratory distress syndrome, myocarditis, septic shock, renal failure and coagulopa
can be present as well.[2] Therefore, in the appropriate setting, rickettsial infections
be considered in any patient who presents with multi-organ failure. Diagnosis is esta
by clinical suspicion, PCR assays, enzyme immunoassays, and peripheral blood smea
However, peripheral blood smear may be low yield, with only 3% of HME patients
demonstrating the characteristic intracytoplasmic morulae.[2]

Gastrointestinal involvement of HME includes hepatosplenomegaly, cholestasis, jaun
nausea, vomiting, diarrhea and even acute abdomen.[5, 6] Gastrointestinal hemorrh
rare but has been reported.[7] Liver injury occurs as organisms proliferate within
hepatocytes and stimulate an immune response, resulting in diffuse hepatitis, forma
noncaseating granulomas, and even focal necrosis.[7] Macrophage-rich
inflammatoryinfiltrates are commonly observed in the liver; indeed, activation of
macrophages may beresponsible not only for local liver inflammation, but also for se
systemic manifestations such as septic shock and acute respiratory distress syndrom
Cholestasis and cholestatic hepatitis may result from lymphoid infiltration of the sinu
6] Moreover, neutrophilic infiltration of medium-sized bile duct walls has been descri
[4]These hepatobiliary lesions result in the commonly observed and characteristic
transaminase elevation (80%), as well as less common increases in alkaline phospha
bilirubin; other lab abnormalities associated with HME include leukopenia (61%) and

thrombocytopenia (73%).[1] Resolution of hepatosplenomegaly parallels overall improvement with appropriate antibiotic therapy.[6]

This case is is the first to report an elevation in lipase in association with severe HME patient had no clinical or radiographical evidence of pancreatitis, and so we are left t wander about the cause of this lab abnormality. *R rickettsii* has been known to cause pancreatic injury through vasculitis; however, vasculitis is not a feature of *E chafeen* infection, as these organisms do not infect endothelial cells.[2, 8] Most likely, the ele lipase was the result of two mechanisms. First, the pancreas may be susceptible to inflammation in surrounding intra-abdominal organs, including the bililary tree and t [9] This may very well have been the case here given the degree of hepatitis and ch evident. Secondly, septic shock and multi-organ failure often lead to lipase elevation through pancreatic hypoperfusion itself, in a manner similar to “ shock liver”; or thro splanchnic hypoperfusion resulting in mesenteric ischemia and leakage of intra-intes pancreatic enzymes through injured submucosa.[10] This case reminds us that in se sepsis and septic shock, lab abnormalities and organ dysfunction may be due to eith pathogenicity of the organism itself or the systemic or local consequences of infectio

LEARNING POINTS/TAKE HOME MESSAGES *3 to 5 bullet points – this is a required fiel*

- HME has numerous gastrointestinal manifestations and can cause direct hepat damage.
- Lipase elevation may occur in any critical illness and may result from injury to organs.
- Prompt recognition and treatment of HME is necessary to prevent permanent e organ damage and death.

REFERENCES *Vancouver style (Was the patient involved in a clinical trial? Please refe related articles)*

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