

# [Gastrointestinal involvement: human monocytic ehrlichiosis](https://assignbuster.com/gastrointestinal-involvement-human-monocytic-ehrlichiosis/)

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| Gastrointestinal involvement of human monocytic ehrlichiosis, including a novel finding of elevation of pancreatic lipase. |
| SUMMARY |
| A 57 year-old man presented to the hospital with septic shock. He was found to have human monocytic ehrlichiosis (HME) with constitutional symptoms, as well as multiple gastrointestinal manifestations, including nausea, vomiting, diarrhea, hepatosplenomegaly, and cholestasis. He was also found to have an elevated lipase, suggestive of pancreatitis, although this was most likely secondary to circulatory shock and surrounding inflammation (he had no abdominal pain or radiographic evidence of pancreatitis). He was treated with doxycycline, which resulted in rapid improvement clinically and in terms of his laboratory 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, all of 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 multi-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 lipase 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 June with several days of fever, nausea, vomiting, chills, myalgias, diarrhea, headache and lethargy. He had no abdominal pain. Six days earlier, he presented to the ED of another hospital with fever, and was treated with a course of levofloxacin for possible sinusitis, but experienced no improvement. His past medical history included atrial fibrillation and hypertension. He smoked one pack a day and drank 2-3 beers a week. He resided in rural Southern New Jersey and reported that several of his family members had recently been bitten by ticks, though he does not recall being bit. His blood pressure was initially 60/40 but increased to 106/58 after receiving five litres of intravenous fluid. His temperature was 36. 9 C. Pulse was initially 124 and irregular but normalized with fluid resuscitation. He was alert and oriented to person, place and time. He had dry mucous membranes, a benign abdomen, palpable hepatomegaly 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 (ALT) of 83 U/L (normal 6-45 U/L), a total bilirubin of 1. 4 mg/dL, a direct bilirubin of 0. 9 mg/dL, and a creatinine of 1. 78 mg/dL (normal 0. 60-1. 20 mg/dL). Alkaline phosphatise was normal. His complete blood count with differential was notable for a platelet count of 69, 000 U/L and a 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 and pancrease, and numerous sigmoid diverticula. A small amount of intraperitoneal free fluid was also noted above both inguinal canals. Ultrasonography of the right upper quadrant revealed cholelithiasis but nondilated bile ducts. |
| DIFFERENTIAL DIAGNOSIS If relevant |
| At the top of the differential were the tickborne illnesses. Fulminant leptospirosis (Weil’s disease) was also considered a possibility given his acute non-specific febrile illness and multisystem involvement. Enzyme immunoassay test for antibody to B. Burgdorferi was negative. Rocky mountain spotted fever and leptospirosis serologies were also negative, as was polymerase chain reaction (PCR) for anaplasma and babesia. No malarial parasites or babesia were observed on smear, and blood and urine cultures did not grow any organisms. Finally, E. Chaffeensis DNA was detected by PCR. |
| TREATMENT If relevant |
| The patient was initially treated in the Emergency Department with very broad microbial coverage, including levofloxacin, vancomycin, piperacillin-tazobactam, metronidazole, and doxycycline. He was soon brought to the intensive care unit, where treatment was continued solely with doxycycline. |
| OUTCOME AND FOLLOW-UP |
| Following treatment, he achieved very rapid clinical improvement and was discharged on his sixth hospital day. His creatinine normalized with the administration of intravenous fluid. By the fifth hospital day his AST and ALT had trended down to 46 U/L and 49 U/L, respectively; his total and direct bilirubin had normalized at 0. 6 mg/dL and 0. 2 mg/dL; and his lipase 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 infects mononuclear phagocytes. [1] Over 5496 cases had been reported to the CDC as of 2009, most of which were in the south-central, southeastern and mid-Atlantic United States. It is transmitted by the Lone Star tick ( A. americanum) with white-tailed deer, dogs and coyotes as the reservoir. Most infections happen in the summer months, with the average host being a male in his 50s; men may be infected more due to more occupational and recreational exposure.[1, 2] The fatality rate of HME has hovered around 1-2% in recent years.[3] The most common presenting symptoms are fever, headache, anorexia, myalgia, chills and rigors, nausea and vomiting, and rash, with 40% of cases requiring hospitalization.[2, 4] Other gastrointestinal manifestations are described below. Meningoencephalitis, acute respiratory distress syndrome, myocarditis, septic shock, renal failure and coagulopathies can be present as well.[2] Therefore, in the appropriate setting, rickettsial infections should be considered in any patient who presents with multi-organ failure. Diagnosis is established by clinical suspicion, PCR assays, enzyme immunoassays, and peripheral blood smear. 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, jaundice, nausea, vomiting, diarrhea and even acute abdomen.[5, 6] Gastrointestinal hemorrhage is rare but has been reported.[7] Liver injury occurs as organisms proliferate within hepatocytes and stimulate an immune response, resulting in diffuse hepatitis, formation of 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 severe systemic manifestations such as septic shock and acute respiratory distress syndrome.[2] Cholestasis and cholestatic hepatitis may result from lymphoid infiltration of the sinusoids.[4, 6] Moreover, neutrophilic infiltration of medium-sized bile duct walls has been described.[4]These hepatobiliary lesions result in the commonly observed and characteristic transaminase elevation (80%), as well as less common increases in alkaline phosphatase and 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. The patient had no clinical or radiographical evidence of pancreatitis, and so we are left to 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 chafeensis infection, as these organisms do not infect endothelial cells.[2, 8] Most likely, the elevation in 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 the liver. [9] This may very well have been the case here given the degree of hepatitis and cholestasis evident. Secondly, septic shock and multi-organ failure often lead to lipase elevation, either through pancreatic hypoperfusion itself, in a manner similar to “ shock liver”; or through splanchnic hypoperfusion resulting in mesenteric ischemia and leakage of intra-intestinal pancreatic enzymes through injured submucosa.[10] This case reminds us that in severe sepsis and septic shock, lab abnormalities and organ dysfunction may be due to either the pathogenicity of the organism itself or the systemic or local consequences of infection. |
| LEARNING POINTS/TAKE HOME MESSAGES 3 to 5 bullet points – this is a required field |
| * HME has numerous gastrointestinal manifestations and can cause direct hepatic damage. * Lipase elevation may occur in any critical illness and may result from injury to adjacent organs. * Prompt recognition and treatment of HME is necessary to prevent permanent end-organ damage and death. |

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| REFERENCES Vancouver style (Was the patient involved in a clinical trial? Please reference related articles) |
| 1. Walker DH, Dumler J, Marrie T. Harrison’s Principles of Internal Medicine. New York, NY: McGraw-Hill; 2011. http://accessmedicine. mhmedical. com/content. aspx? bookid= 331&Sectionid= 40726928. (accessed April 14, 2015).  2. Stone JH, Dierberg K, Aram G, Dumler JS. Human monocytic ehrlichiosis. JAMA 2004; 292(18): 2263-70.  3. Centers for Disease Control and Prevention. Ehrlichiosis . http://www. cdc. gov/ehrlichiosis/stats/ (accessed 14 April 2015).  4. Moskovitz M, Fadden R, Min T: Human ehrlichiosis: A rickettsial disease associated with severe cholestasis and multisystemic disease. J Clin Gastroenterol 1991; 13(1): 86–90.  5. Low A, Turett G. Human granulocyticehrlichiosispresenting as acute abdomen in an adult. Clin Infect Dis. 2003; 37(10): 1397 PubMed -8.  6. Zaidi SA, Singer C. Gastrointestinaland hepatic manifestations of tickborne diseases in the United States. Clin Infect Dis. 2002; 34(9): 1206-12.  7. WallaceBJ, BradyG, AckmanDM, et al. Human granulocytic ehrlichiosis in New York . Arch Intern Med 1998 ; 158(7): 769 – 73 .  8. Randall MB, Walker DH. RockyMountainspottedfever. Gastrointestinalandpancreaticlesions and rickettsial infection. Arch Pathol Lab Med. 1984; 108(12): 963-7.  9. Hameed AM, Lam VW, Pleass HC. Significant elevations ofserumlipasenot caused by pancreatitis: a systematic review. HPB (Oxford) 2015; 17(2): 99 PubMed -112.  10. Denz C, Siegel L, Lehmann KJ, Dagorn JC, Fiedler F. Intensive Care Med. Ishyperlipasemiaincritically illpatientsof clinical importance? An observational CT study. 2007; 33(9): 1633-6. Epub 2007 May 12. |