A Detailed Travel History Solved a Medical Mystery
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INTRODUCTION: Taking a detailed history is often a clinical challenge. Asking the right question related to the presenting symptoms in the key. At the Minneapolis VA, we have encountered an asymptomatic patient who had incidental finding of “portal hypertension” on imaging. Yet, he had an extensive negative workup. At the time of his EGD, a more detailed history was taken during medical records review, the his own history finally solved the mystery.

CASE DESCRIPTION/METHODS: 86 y/o male with incidental findings of “portal hypertension” from a CT scan presented for a diagnostic EGD. A recent radiology report described a cluster of varices near the gastric fundus and a spleen size of 15 cm. Subsequent evaluation by the GI specialty clinic failed to identify a cause; the workup included alcohol use history, medication review, auto-immune markers, viral serologies, genetic testing, metabolic markers, lipid studies, iron studies, tumor markers, liver function tests, clotting factors, and fibroscan. Just prior to EGD, a detailed history revealed a Vietnam deployment in 1970. The patient further described a life threatening event. He was sick for almost a year. The upper endoscopy subsequently showed no varices and no gastropathy.

DISCUSSION: Falciaparum malaria is the most severe form of malarial disease caused by Plasmodium falciparum. This disease is responsible for over 400,000 deaths globally in 2018. Over 228 MILLION cases of malaria were reported by the WORLD HEALTH ORGANIZATION (WHO) in 2018. Plasmodium falciparum is classified as Group 2A carcinogenic by the WHO. This organism is transmitted by infected female anopheline mosquitoes. The eventual multiple organ failure and death is caused by the obstruction of the body’s microcirculation by the organism particles. In this case, the CT findings were caused by temporary pre-sinusoidal portal hypertension until the malaria was cured several months later. As a result, permanent enlarged spleen and permanent dilated veins were discovered on the CT scan several decades later. The primary disease was cured 40 years ago. It is imperative to understand the history, pathophysiology, and the clinical sequelae of common tropical diseases when taking care of patients that travel throughout the world. In today’s western medical practice, we frequently forget the old diseases that still cause wide range of destruction in other parts of the world. We can avoid wasting medical resources by asking the right question.

[2745] Figure 2. Histology of the liver, which shows intravascular infiltration of tumor cells.

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Liver Transaminis in the Setting of HLH
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INTRODUCTION: Hemophagocytic lymphohistiocytosis (HLH) is a syndrome of a severely increased inflammatory response leading to tissue damage. HLH poses a challenge to clinicians as its presentation tends to overlap with conditions such as sepsis, systemic inflammation, cancer. In most cases, HLH is a result of infection, autoimmune disease & malignancy. Our case seeks to highlight the increasing incidence of HLH in the adult population by following a 76 yo male who presented with persistent diarrhea, pancytopenia, liver transaminis which was later diagnosed as HLH.

CASE DESCRIPTION/METHODS: A 76 yo Caucasian male with a past history of CAD, HTN, HLH, C. diff, iron deficiency anemia presented to the hospital with a chief complaint of nausea & diarrhea with an onset of 3 wks prior to presentation. The patient noted that his symptoms first started with chills, body aches, headaches and a subjective fever measured at 103F. However due to persistent watery diarrhea and weakness for 5 days, he was advised to visit our facility. Pt was admitted as he met sepsis criteria and found to be pancytopenic on admission. There was an initial suspicion that pts pancytopenia was secondary to sepsis that may have been caused by C. diff. He was later found to be C. diff negative. He continued treatment for PNA but continued to be pancytopenic. HemOnc was consulted, a bone marrow biopsy was obtained but pts bone marrow did not reveal any underlying pathology. Granul, Restocrit were added with minimal results. His clinical course continued to worsen, now with liver transaminis and clinical decline requiring transfer to the ICU. HLH was suspected after all broad spectrum treatments had failed and pts CT abd/pelv noted hepatomegaly and splenomegaly. With the CT findings, persistent pancytopenia, Ferritin 100865, β elevated IL-6, HLH was suspected. A Liver bx revealed typical Hodgkins lymphoma which was the underlying cause of HLH. Although a clinical diagnosis was made, pts clinical status had rapidly deteriorated prior to starting life-saving measures and the patient succumbed to his illness.

DISCUSSION: This is a case of a 76 year old male who presented with secondary HLH with immune activation in the setting of Hodgkin’s lymphoma and Chronic EBV. HLH is a rapidly progressive condition with a high mortality rate resulting from multisystem failure. Lately, the incidence in adults has been growing and it is important for clinicians consider the disease in the setting of sepsis, worsening clinical picture & multi-organ failure.

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Unusual and Rare Presentation of Hepatitis D
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INTRODUCTION: Hepatitis D virus, also known as the "Delta virus" is a defective virus that requires hepatitis B virus for infection. About 257–291 million people are chronically infected with HBV worldwide, and of those up to 20 million had experienced HDV infection. We present unusual case with histological, serological, virological evidences of chronically active HDV infection which was initially thought to be medication induced.

CASE DESCRIPTION/METHODS: A 34 yo M with PMH of Chronic Hepatitis B infection diagnosed in 2013 has been on Tenofovir Disoproxil (TDF) × 4 years. After getting switched from TDF to Tenofovir Alfenamide (TAF) he had a rise in LFTs: Bilirubin 0.2, AST 126, ALT 327. He denied alcohol use, herbal use, recent travel or other toxic habits. On presentation, VS were within normal limits and complete physical examination was unremarkable. Patient was switched from TAF to TDF as it was thought to be possibly contributing to his abnormal LFTs and a complete liver disease work up was obtained. Laboratory testing revealed TB 0.8, AST 154, ALT 386, ALP 49, INR 1.1, Hepatitis B Ag −, sAb−, eAb−, eAg−, HBV DNA non detected, HDV Ag−, HDV Ab−, HDV PCR 133,000. Ultrasound showed hepatic steatosis. Despite switching back to TDF his LFTs

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[2748] Figure 1. Picture 1
Star: portal inflammation and fibrosis
Arrow: lobular inflammation
Triangle: steatosis