ACUTE THORACIC CORD COMPRESSION DUE TO HIV-ASSOCIATED BURKITT'S LYMPHOMA
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LEARNING OBJECTIVE 1:
Recognize the high risk of non-Hodgkin's lymphoma in a patient with HIV/AIDS.

LEARNING OBJECTIVE 2:
Describe potential serious causes of back pain in a patient with HIV/AIDS.

CASE:
A 46 year-old African-American man with HIV, a CD4 count of 274/mcL and a viral load of 200 copies/mL, on combination antiretroviral therapy (cART), presented with one month of progressive mid-to-low back pain without fevers or night sweats. He had no neurological deficits and was discharged with a diagnosis of musculoskeletal back pain. One week later, he developed lower extremity weakness and numbness. Physical exam demonstrated decreased lower extremity strength, loss of sensation to light touch, proprioception, and pain below the umbilicus, patellar tendon hyperreflexia, bilateral ankle clonus, and decreased rectal sensation. Spine MRI showed extradural soft tissue masses at the T3-T4 and T10-T11 levels with neural foraminal and paraspinal spread with severe canal stenosis at the T3-T4 level suggestive of lymphoma. Intravenous dexamethasone was initiated, followed by emergent laminectomy and epidural mass resection. Pathological exam of the mass showed dense lymphocytic infiltrate. Flow cytometry revealed a monoclonal B-cell population, and FISH studies demonstrated expression of the c-myc oncogene with chromosomal translocation 8q24 consistent with Burkitt's lymphoma (BL). Epstein-Barr virus (EBV) was detected in serum by PCR. CT of the chest, abdomen, and pelvis were negative for lymphadenopathy. Brain MRI showed no parenchymal lesions. Bone marrow biopsy was negative for malignant cells. PET scan showed increased FDG activity within the right trochanter. Systemic and intrathecal chemotherapy were initiated.

DISCUSSION:
Non-Hodgkin's lymphoma (NHL) is an AIDS defining malignancy that is 200-600 times more common in HIV-infected patients compared to the general population. High grade diffuse large B-cell or Burkitt-like lymphomas are more common in people living with HIV and are associated with EBV coinfection. The incidence of NHL has decreased in the cART era. A recent review of 61 cases of NHL in AIDS patients in France showed that the major risks for NHL included both longer and current exposures to a viral load above 500, and a CD4 below 200. Patients commonly present with "B" symptoms, and usual sites of involvement include bone marrow, lungs, abdomen, and CNS. Epidural spinal cord compression occurs in 0.1 to 6.5 percent of NHL patients, either at the time of relapse or as the initial manifestation of NHL. Burkitt's lymphoma (BL) is a highly aggressive, mature B-cell NHL. Endemic variant BL occurs in African children, usually presenting with a mass at the jaw or neck, and is associated with EBV infection. Sporadic BL usually presents with an abdominal mass. Immunodeficiency-related BL is seen in AIDS patients and often involves the lymph nodes, bone marrow, and CNS. Few cases of spinal involvement in adults with BL have been reported in the literature. BL is associated with unique cytogenetic translocations involving the c-myc oncogene. This case demonstrates the potential seriousness of back pain in patients with HIV/AIDS. The differential diagnosis includes cancers that metastasize to bone, compression fracture from osteoporosis, spinal epidural abscess, and NHL including BL. Clinicians should have a lower threshold for imaging HIV-infected patients with back pain when compared to the general population.
A (RING) ENHANCED APPROACH TO HIV AND CENTRAL NERVOUS SYSTEM LESIONS
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LEARNING OBJECTIVE 1:
Understand approach to ring enhancing lesions

LEARNING OBJECTIVE 2:
Recognize ADEM as a cause for CNS disease in HIV patients

CASE:
A 44 year old man with history of HIV presented with 3 weeks of progressive right upper and lower extremity weakness. Symptoms initially began with parasthesias of the fingers and progressed to weakness of right upper extremity and proximal right lower extremity. He endorsed an upper respiratory infection several weeks prior, but denied fevers, neck stiffness, or recent vaccinations. Vitals were temperature 98 F, heart rate 83, and blood pressure 130/80 mmHg. Patient was oriented to person, place, and location. Pupils were equal and reactive, with a midline, and supple neck. Strength was 3/5 throughout the right upper extremity, and 4/5 throughout the right lower extremity. There was decreased sensation to light touch and vibration of both the right upper and lower extremity. Reflexes were 3+ throughout, and Hoffman sign was positive on right hand. Plantar reflexes were down-going bilaterally. CD4 count was 426. Cerebral spinal fluid contained 10 WBC with 92% lymphocytes, and 1 RBC. All serology was negative including VDRL, cryptococcus, JC virus, and toxoplasma. All cultures and gram stains were also negative including AFB and fungal. An MRI revealed three ring-enhancing lesions in the right frontal and parietal lobes along with the left centrum semiovale. MRI also displayed enhancement within the cervical spinal cord.

DISCUSSION:
HIV is encountered commonly in internal medicine. Although rates of CNS disease have decreased with antiretroviral therapy, neurologic complications occur in over 40% of HIV positive patients. The man described above was found to have multiple, ring-enhancing lesions on MRI despite numeric immunocompetence. With enhancing lesions, it is useful to divide the differential diagnosis into 3 categories, which include infection, neoplasm, and demyelination. Further stratifying infection risk based on CD4 will help direct serum and CSF workup. Additional imaging, and even biopsy, may be required for oncologic and demyelinating diagnoses. Imaging and CSF findings in the above case were consistent with a demyelinating process and lead to the diagnosis of acute demyelinating encephalomyelitis (ADEM). ADEM is an inflammatory demyelinating disorder of the CNS usually preceded by an infectious disease or vaccination. ADEM is estimated to occur in 1 per 125,000 people in the US with higher prevalence in children. However, ADEM has been associated with HIV infection. The hypothesis is that the HIV virus causes neuronal damage leading to an inflammatory response with consequent demyelination. Acutely, these lesions will enhance on MRI with contrast as seen in the man described above. His CSF studies revealed a lymphocytosis that was also consistent with ADEM. After ruling out major infectious causes, he was initiated on corticosteroids and was able to return to near baseline level of functioning. Given the potential for recovery with prompt treatment, it is important for the internist to incorporate ADEM into his or her differential when evaluating HIV positive patients with neurologic symptoms.
OPENING PANDORA’S BOX: AN UNFORTUNATE CASE OF INFIDELITY
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LEARNING OBJECTIVE 1:
Illustrate the neurologic manifestations of syphilis

LEARNING OBJECTIVE 2:
Review the challenges of diagnosing HIV and syphilis co-infection

CASE:
A 38 year-old man presented to the hospital complaining of a one-month history of rash. The painful, intensely pruritic rash started on his torso and spread outward until it covered his whole body, including the palms and soles. Two weeks prior to presentation, the patient experienced several days of sore throat, cough, and fever. He also complained of night sweats, headache, fifteen-pound weight loss, pharyngitis and visual disturbances during this time period. The patient had a past medical history of childhood sinus cancer that was treated with surgery. His last HIV test was 6 months prior to admission and was negative. He is sexually active with men and recently left a 6-year relationship after his partner admitted to infidelity. On admission, he was afebrile and normotensive. His physical exam was significant for tonsillar erythema and diffuse and painful lymphadenopathy. An erythematous maculopapular rash extended from the scalp to the feet, including the palms and soles. It was tender to palpation with indistinct borders and was not present on the mucous membranes. Labs were notable for white blood cell count of 11.1x103/mm3, alkaline phosphatase 1165 U/L, ALT 110 U/L, AST 86 IU/L, and bilirubin 0.8 μmol/L. Lumbar puncture was performed which revealed 16 nucleated cells in tube 4 (56% neutrophils) with a normal glucose and protein. RPR was positive with a titer of 1:64. Cerebrospinal fluid (CSF) VDRL was positive with a titer of 1:1. HIV antigen/antibody screening test as well as Western Blot were positive: HIV viral load was 1.2 million copies/mL and CD4 count was 593. Other CSF studies, including herpes simplex virus and cryptococcal antigen, were negative. The patient was treated with continuous IV penicillin G infusion. He was counseled regarding his new diagnosis of HIV and was started on antiretroviral therapy during this admission. His rash resolved and his neurologic symptoms improved.

DISCUSSION:
This case illustrates the challenge of differentiating recent HIV infection from secondary syphilis, as well as the synergistic relationship they have during co-infection. Fifty to ninety percent of patients with HIV have symptoms at presentation. These are usually influenza- or mononucleosis-like syndromes, with fever, rash, myalgias, sore throat, and lymphadenopathy. Less than 20% of patients present with encephalitis or meningitis (1). Even among those with neurologic signs or symptoms, it is not common for these findings to reflect a concomitant opportunistic central nervous system infection. Syphilis increases in incidence, especially in men who have sex with men (MSM). In 2006, 64% of cases of primary and secondary syphilis reported nationwide were among MSM (2). Active syphilis increases the risk of HIV infection 2- to 5- fold (2). The classic manifestation of neurosyphilis is syphilitic meningomyelitis, characterized by slowly progressive lower extremity weakness and paresthesias. The most common form of neurosyphilis, however, is asymptomatic neurosyphilis; patients who are without neurologic symptoms but have evidence of syphilis infection in their CSF. A third presentation of secondary syphilis, syphilitic meningitis, occurs in 5% of patients, usually within the first 12 months of infection and is associated with a variety of neurologic manifestations (3). The similarities between secondary syphilis and recent HIV infection can pose diagnostic challenges for clinicians. As mentioned above, HIV can cause many of the same symptoms as secondary syphilis such as rash, lymphadenopathy, and headache. Laboratory tests may also not be revealing, as both HIV and syphilis can cause changes in CSF studies and co-infection can lead to precipitous increases in viral load as well as rapid decreases in CD4 counts (3, 4). It is essential to understand the presentations of recent HIV infection and syphilis, whether isolated or during co-infection, to avoid missing either diagnosis.

References
LEARNING OBJECTIVE 1:
Recognize atypical presentation of MAC infection in an immunocompromised patient.

LEARNING OBJECTIVE 2: Review management of disseminated MAC.

CASE:
28 year old female with history of AIDS (previously on HAART) and pancytopenia presented as a direct admission for evaluation of elevated liver transaminases. Laboratory findings were significant for AST elevation of 964, ALT of 220, Alkaline phosphatase of 871, CD4 count of 8, WBC count of .5 and platelets of 87. On presentation, patient appeared comfortable and was not complaining of any symptoms. Physical exam and review of systems were unremarkable. Initially, all hepatotoxic drugs were discontinued and Infectious Diseases and Hematology were consulted for further treatment recommendations. Over the course of hospitalization, patient underwent an extensive microbiology work up as well as imaging of the abdomen, the hepatobiliary tree and biopsies of the bone marrow and liver. Results were significant for a positive AFB blood culture. Bone marrow biopsy showed non-necrotizing granulomatous inflammation with acid fast positive organisms and liver biopsy revealed granulomatous hepatitis with acid fast bacilli. Based on biopsy results and positive blood culture, disseminated MAC (DMAC) with multiorgan involvement was thought to be responsible for the pancytopenia as well as the hepatocellular liver injury. Azithromycin, Rifabutin, and Amikacin were chosen as triple therapy for DMAC as the patient had advanced AIDS with a low CD4 count. WBC count increased with Neupogen administration. During hospitalization patient continued to remain asymptomatic, however, her liver function tests significantly improved after the initiation of antibiotics.

DISCUSSION:
Here, we present a patient with disseminated MAC causing granulomatous hepatitis and bone marrow suppression who has remained asymptomatic throughout her illness. DMAC is a life threatening infection caused by either M. avium or M. intracellulare that carries up to a three-fold risk of death in patients with AIDS. MAC infections typically affect the respiratory or GI tract and symptoms include fevers, night sweats, abdominal pain, diarrhea and weight loss. The infection is acquired through inhalation or ingestion of organisms from soil or water and can subsequently spread through the lymphatics in an immunocompromised host. DMAC typically occurs after the CD4 count drops below 50 cells/mm3 and in patients with CD4 counts of less than 10 cells/mm3, the yearly incidence rate has been reported to be as high as 39%. First line agents for treatment are Clarithromycin and Azithromycin followed by Ethambutol and Rifabutin. IDSA guidelines suggest that at least two drugs should be initiated for the treatment of DMAC due to concern for development of resistance. In our patient, however, we elected to use a three drug therapy of Azithromycin, Rifabutin, and Amikacin due to the patient's severe immunocompromised state. Ethambutol was avoided due to patient's history of optic neuritis. Therapy will be continued for at least a year and until the CD4 count remains above 100 cells/mm3 for at least 6 months.