

CK of 600,000 unit/L had no renal dysfunction (2, 3). Therefore, viral rhabdomyolysis is a diagnosis that needs to be made early and treated aggressively with intravenous fluids due to its unpredictable nature in causing serious complications.

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A Case Series On Fixed Drug Eruptions

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Patient #1: A 60 year old male truck driver with a history of recurrent skin boils presented with painful lesions on the glans penis that started a few days prior. A week ago he was treated with co-trimoxazole for skin abscesses on the left leg. The penile lesions were noted as vesicular eruptions that eventually coalesced and there was absence of discharge, scrotal involvement, or lymphadenopathy. He reported a similar episode one year ago following a course of co-

trimoxazole for skin abscesses. He denied fever, chills, malaise, or oral lesions. He had no drug allergies and was not taking any other drugs. He denied tobacco; illicit drugs, high risk sexual behavior, and rarely had alcohol. On exam, vital signs were stable, edematous and erythematous glans with shallow based tender ulcerations was noted, but otherwise rest of the exam was unremarkable. No other active skin abscesses or lesions. The basic laboratory work-up including blood counts and serum chemistries were normal. HIV Ag/Aby test was non reactive, Nucleic acid amplification test (NAAT) for Gonococci/Chlamydia was negative, RPR was non-reactive, PCR for HSV from the genital lesions was negative, and Hepatitis C Antibody was also negative. Images as below. Infectious diseases team was consulted for suspicion for sexually transmitted diseases and a diagnosis of fixed drug eruptions secondary to co-trimoxazole was made. Dermatology was then consulted and concurred with our diagnosis. Patient was advised not to take this drug ns future.

Patient #1



Patient #2: A 25 year old male with paranoid schizophrenia presented with seizures and painful lesions on the glans penis and scrotum. The patient had a long history of seizures and was recently started on lacosamide a week ago. He had a history of heroin use and had been incarcerated for 8 years. He lived in an assisted living home and reported no sexual activity. He denied fever, chills, malaise, penile discharge or oral lesions. He did not remember any drug allergies and does not take any other medications. His family history was non-contributory. On exam, vital signs were stable, a non-pruritic macular rash on hands, back, and torso was noted, and tender ulcerations with erythematous base were noted on glans penis and scrotum with exudates on the surface (Images Below). The laboratory work-up consisted of a normal blood cell counts and serum chemistries. HIV Ag/Aby was non reactive and Urine NAAT for Gonococci and chlamydia was negative. RPR was non-reactive, HSV-PCR was negative on ulcer swabs and Hepatitis C antibody negative. Infectious diseases consult service arrived at a clinical diagnosis of Fixed drug eruptions from lacosamide. Dermatology was consulted and they concurred with this clinical diagnosis. An alternative antiepileptic was chosen by the primary neurology team.

Patient #2



Discussion:

Fixed drug eruptions typically present as one or more annular erythematous or necrotic lesions, ulcers or patches (0.5-5 cm) as a result of a systemic exposure to a drug. The lesions may last days to weeks and often resolve with residual hyperpigmentation after stopping the offending drug. The most common sites of occurrence include the lip, genitalia, hip, low back/sacrum, or proximal extremity. The lesions may develop any time to two weeks from the initial drug exposure. Fixed drug reactions make up 16-21% of all cutaneous drug eruptions with a male to female ratio of 1:1.1 at a mean age of 30 years. Other common symptoms are pruritus, burning pain and rash. It can rarely result in fever, nausea, diarrhea, abdominal cramps, anorexia, dysuria, and/or malaise. The most common drug classes implicated are analgesics, muscle relaxants, sedatives, anticonvulsants, and antibiotics. The top five drugs implicated, in particular, are penicillins, tetracyclines, sulfonamides, pyrazolones, and barbiturates. The exact pathophysiology is unknown. Most recent research shows that drug acts as a hapten (drug) and preferentially binds to the basal keratinocyte, that then causes release of TNF- α and ICAM1 and induces CD4/CD8 migration. These secrete TNF- α , INF- γ , and IL-15 and result in inflammation. IL-15 is notable for its memory CD8 inducing effects.

The work-up for confirmation of a fixed drug eruption is a skin biopsy, a patch test on the previously affected area, and/or oral provocation of the suspected cause. The lesions will recur at the previously affected areas with re-exposure of the offending agent. Treatment includes systemic antihistamine, topical corticosteroid, avoidance of offending drug, and if infection is suspected: antibiotics and proper wound care. Both of the above patients had the offending drug withdrawn.

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