Figure 1: Multiple giant molluscum with central one showing crater like depression become basophilic, as seen in our case. Histological variants seen in immunocompromised patients are pseudocystic and polypoidal types.[3] These were not seen in our patient’s biopsy.

Cidofovir (1-3%) cream or ointment [4] and electron beam therapy[5] have been used effectively to treat extensive lesions in the immunosuppressed but are not easily available. Facial mollusca of the giant type and especially those found in HIV-infected patients do not respond well to treatment. Intralesional interferon used in resistant facial mollusca[6] and topical imiquimod[7] were avoided as they were costly.

Therapies targeted at boosting the immune system in the immunocompromised have proven to be most effective in such cases.[8]

Our case had a number of atypical and giant mollusca on the forehead in addition to genital lesions. Some of the lesions on the forehead mimicked keratoacanthomas. They responded satisfactorily when the primary disease was treated with HAART.

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Necrotizing fasciitis in an HIV-infected patient

Sir,
Necrotizing fasciitis is a life-threatening, progressive, rapidly spreading, inflammatory infection of the deep fascia, with secondary necrosis of the subcutaneous tissues and usually associated with the trauma and immunodeficiency. We present a case of necrotizing fasciitis in an HIV-infected patient.
A 27-year-old unmarried male presented with painful raw lesions over the left inguinal region since 3 days. It started with a painful left inguinal swelling associated with high-grade fever followed by the formation of blisters, which ruptured spontaneously to form raw lesions. He was non-diabetic and gave no history of trauma or invasive procedure.

On examination, there was a single large ulcer with necrotic slough on the floor, irregular edges, and the surrounding skin showing ecchymosis [Figure 1]. A differential diagnosis of cutaneous vasculitis, necrotizing fascitis, pyoderma gangrenosum, and pyomyositis was entertained and the patient was investigated.

His hemogram, liver function tests, renal function tests, electrocardiogram, coagulation profile, and X-ray chest were normal. Erythrocyte sedimentation rate was 30 mm at the end of one hour and ELISA for HIV-I was positive. Pus culture grew beta hemolytic streptococci and *Pseudomonas aeruginosa*. Blood culture was normal and CD4 count was 350 cells/µl. Sonography of the abdomen showed external iliac lymphadenopathy and mild splenomegaly. Anti-nuclear antibody and pathergy test were negative. On histopathology, there was a dense infiltrate of polymorphonuclear cells in the dermis and subcutaneous tissue mostly around the blood vessels. Surgical debridement was done and he was started on injectable cefotaxime (1 gm iv 8 hrly), amikacin (500 mg iv 12 hrly), and metronidazole (500 mg iv 8 hrly) along with daily dressing. Split-thickness skin grafting was done with significant healing in 2 weeks’ time [Figure 2].

Necrotizing fasciitis was first described by Wilson.\textsuperscript{[1]} Hospital gangrene, progressive bacterial synergistic gangrene, Fournier’s gangrene, streptococcal gangrene, and flesh-eating bacterial infection are the other terms used. It is of two types, depending on the organisms isolated. Type 1 is polymicrobial, usually caused by aerobic and anaerobic organisms, while Type 2 is caused by Group A β-hemolytic streptococci, either almost always alone or in combination with other species.\textsuperscript{[2]} Our case was of Type 2. The organism enters into the subcutaneous space through a disruption of the overlying skin either by trauma or surgery, or lymphohematogenous spread from a distant site, but rarely infection can occur over healthy skin.\textsuperscript{[3]}

Necrotizing fasciitis has reported cumulative mortality of 34%, with the range being 6 to 76%.\textsuperscript{[4]} In suspected necrotizing fasciitis, a full-thickness biopsy, particularly if combined with more extensive surgery, has been shown to correlate with an improved outcome.\textsuperscript{[5]}

Clinical suspicion of necrotizing fasciitis should be high because early diagnosis and early treatment, including wide excision and debridement, along with antibiotics decrease patient morbidity and mortality in this otherwise poorly-prognostic condition.\textsuperscript{[6]} In immunocompromised host, particularly, it becomes mandatory to biopsy any necrotic cellulitic lesions and to be alert to the possibility of a wide range of bacterial, viral, fungal, and even parasitic infestations.\textsuperscript{[2]}

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Sir,

Lymphedema is the occurrence of chronically swollen extremities or rarely the genitals due to inadequate drainage of interstitial fluid by the lymphatics. Primary lymphedema is uncommon and has female predominance. It may be congenital or familial (Milroy’s disease) or idiopathic appearing either at puberty (precox), or after 35 years of age (tardum).[1,2] Secondary lymphedema caused by obstruction of lymphatic flow due to destruction of lymphatics by various infections is relatively common. In India, secondary lymphedema is synonymous with filariasis.[1] Other causes include surgical removal of lymph nodes or their destruction by radiation fibrosis and malignant cell infiltration.[2]

Genital elephantiasis though uncommon is an important medical problem occurring in the tropics. It causes not only a major physical disability, but also extreme mental anguish. In the majority, filariasis is the cause but bacterial sexually transmitted infections (STIs) like lymphogranuloma venereum and donovanosis form a significant number.[3,4] Other causes of genital elephantiasis like infections and malignancies are very rare.[3,4]

A 45-year-old man, father of four, with swelling of scrotum and penis of about 2 months duration was referred by a surgeon. Onset was sudden and within 2 weeks he developed a large swelling of penis and scrotum. He only had a feeling of heaviness and was depressed due to the embarrassing condition. There was no history of injury, operation, or radiation prior to the onset. He had a large number of pus-filled eruptions on both legs with fever, about 8 weeks prior to the onset. He was treated by a doctor (non-dermatologist) with oral and topical antibiotics. All lesions had healed in 10-15 days leaving behind scars. There was no history of extramarital sexual contact or genital ulcer disease. His wife was apparently healthy.

Clinically, he did not have lymphadenopathy. General and systemic examinations were essentially normal. Genital examination revealed a cold, non-tender, large-curved penis measuring 8 inches in length and 5 inches in circumference, looking like a “saxophone” [Figure 1]. There was no evidence of ulcer, scar, or inflammation. The scrotum was huge and its contents could not be palpated. Both weighed about 1.0 kg. Transillumination test of scrotal swelling was negative. Left side of his lower abdomen and adjacent area of left thigh had extensive postburn scars, but no edema. Both shins and legs had large number of scars without any sign of inflammation.

On the basis of history and clinical findings, a diagnosis of secondary lymphedema of penis and scrotum with “saxophone” deformity was made. In this case, genital lymphedema was secondary to bacterial infection, probably due to Staphylococcus aureus, but peculiarly the lymphedema...