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Histopathological and dermoscopic findings of Kaposi sarcoma in an HIV-tuberculosis co-infected patient

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Abstract
HIV-associated Kaposi's sarcoma is one of the AIDS-defining conditions, indicating an advanced immune-suppressed state. AIDS-related Kaposi sarcoma has rarely been reported, despite India having a high prevalence of HIV-AIDS. Here, we report a rare case of an HIV-tuberculosis co-infected Indian heterosexual male patient along with its histopathological and dermoscopic correlation. This report also targets the necessity of early diagnosis of KS in PLHA cases to start prompt treatment in managing such cases.

Keywords: HIV-associated Kaposi's sarcoma, AIDS-defining condition, immune suppression

Introduction
Kaposi sarcoma (KS) is a multifocal, endothelial proliferation caused by human herpesvirus 8 (HHV-8), most often with cutaneous involvement and with or without visceral extension. AIDS-related Kaposi sarcoma has rarely been reported, despite India having a high prevalence of HIV-AIDS. About 25 such cases exist in published literature from India [1]. In immunocompetent individuals, the disease usually manifests more in the distal parts of limbs, whereas in immunosuppressed individuals KS behaves like a multifocal systemic disease. We report a case of KS in a heterosexual male with HIV- Tuberculosis co-infection from India who presented with widespread lesions; also focusing on the histopathological and dermoscopic correlation of KS.

Case Report
A 28-year-old unmarried heterosexual HIV seropositive male presented to our OPD with complaints of multiple asymptomatic red raised lesions since 7 months present all over the body i.e. face, trunk, upper and lower extremities, similar lesions were also present on his lip and palate. The lesions first appeared on the lower limbs and then had a cephalic progression. When the patient presented to us, he was already taking anti-tubercular therapy (3FDC's - HRZ) for abdominal Koch's, which he had developed 8 months back. He was initially started on ATT (4FDC's- HRZE), but after beginning ATT, the patient developed jaundice. He was treated with ethambutol and streptomycin until his jaundice improved, and subsequently put on three drug regimes for 5 months.
Cutaneous examination showed multiple red-brown colored papules coalescing to form plaques ranging from 0.5 cm in diameter to as big as 15cm in widest diameter, on the face, trunk, and upper & lower extremities present symmetrically on both sides (Fig.1A, 1B & 1C). His face appeared swollen, his limbs were normal though. He also had multiple purplish-red nodules and plaques over the hard palate & few lesions over the soft palate as well (Fig.1D). Post-auricular lymph nodes were also enlarged. Systemic examination was normal.
A clinical diagnosis of Kaposi sarcoma was considered. A biopsy was taken from a new lesion on the back. HPE showed infiltration of collagen by the proliferation of blood vessels and spindloid fibroblast-like cells, infiltration of eccrine sweat glands and erector pili muscles by blood vessels, extravasation of RBCs and hemosiderin-laden macrophages were also seen (Fig.2 A & B). These findings were characteristic of Kaposi Sarcoma.

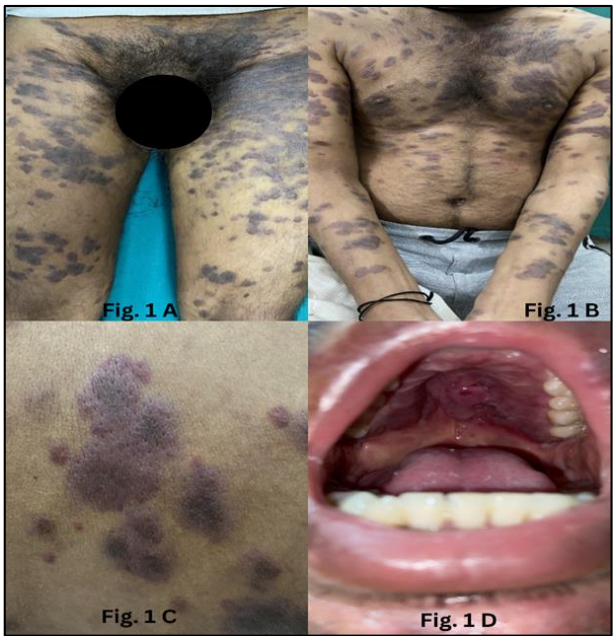

Fig 1: A, B, C & D- Clinical presentation: Multiple erythematous and violaceous papules and plaques over limbs, face and neck and trunk. Purplish red nodules over the palate.

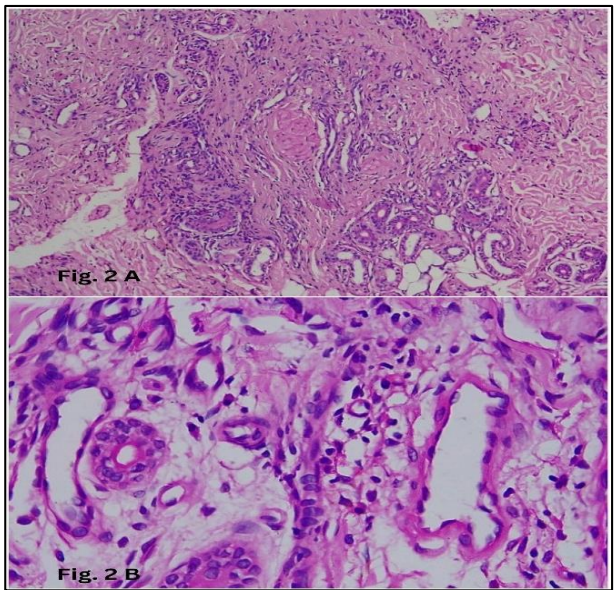

Fig 2: A &B- Histopathology: H& E stain 40 X & 100 X respectively; showing infiltration of collagen by the proliferation of blood vessels and spindloid fibroblast-like cells, infiltration of eccrine sweat glands and erector pili muscles by blood vessels, extravasation of RBCs and hemosiderin-laden macrophages.

His Routine laboratory investigations were as follows

1. Hb: 9.7 gm/dl	5. B. Urea: 21 mg %	9. S. bilirubin: 1.2 mg%	HIV 1: Positive HIV 2: Negative
2. TLC 12000/ cumm	6. S. creatinine: 0.81 mg %	10. ALP: 134 U/L	HBsAg: Non-reactive
3. RBC: 3.4 lakhs/cumm	7. ALT: 46 U/L	11. T. protein: 8.3 g/l	Anti- HBC: Non-reactive
4. Platelets: 1.7 lakhs/cumm	8. AST: 58 U/L	12. RBS: 88 mg/dl	Mantoux: Positive CD4 counts 164/mm^3

Dermoscopic Examination (3rd Gen Dermlite DL200 Hybrid, Polarised, 10 X) revealed, structureless areas with bluish-red discoloration, studded by areas of violaceous globules consistent with a study done by Hu *et al* on the same that showed bluish-reddish coloration in 84% of lesions.[8] The bluish/violaceous structureless areas correspond to the excessive proliferation of blood vessels as HHV- 8 being a lymph-vascular trophic virus. Also, the red areas correspond to extravasation of RBCs & hemosiderin-laden macrophages.

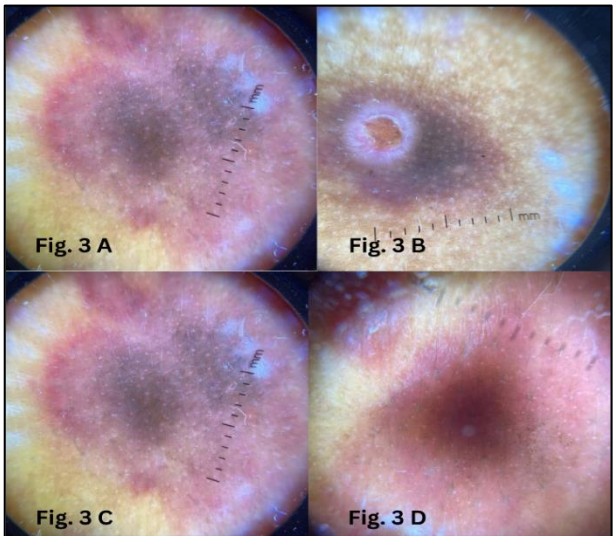

Fig 3: A, B, C & D- Dermoscopy; 3rd Gen Dermlite DL200 Hybrid, Polarised, 10 X, revealed, structureless areas with bluish-red discoloration, studded by areas of violaceous globules.

Considering the history, Clinical features, laboratory, and histopathological findings, a working diagnosis of AIDS-related Kaposi's sarcoma was made.

As per NACO guidelines for the treatment of HIV- TB co-infection, the patient was put on Highly active antiretroviral therapy (HAART) to be continued with 3FDC (ATT), which he was already taking for 5 months.

Discussion

KS is a multifocal, endothelial proliferation caused by HHV-8. Co-infection of HIV and HHV-8 increases the oncogenic potential of HHV-8 [2]. It is the most common malignancy associated with HIV [3] and yet there is a paucity of Indian case reports of KS literature [4]. This may be attributed to the low prevalence of HHV 8 [5]. Only about 25 cases have been reported in India with the maximum cases being reported from Mumbai.

HIV infection fuels the TB epidemic in several ways. HIV infection promotes progression to active TB in people with recently acquired as well as latent TB. HIV infection is the most powerful known risk factor for reactivation of latent TB infection to active disease manifestation.

According to the Integrated Training Module for HIV/TB Collaborative Activities 2015, the annual risk of developing TB in persons living with HIV (PLHIV) who are co-infected with Mycobacterium tuberculosis ranges from 5% to 15%. Up to 60% of PLHIV develop active TB during their lifetime compared to about 10% of HIV-negative individuals.

HIV infection increases the rate of recurrent TB, which may be due to either endogenous reactivation (true relapse) or

exogenous re-infection.

AIDS-associated KS, frequently reported among homosexual men, presents as asymptomatic violaceous macules, papules, and nodules, with initial lesions presenting on the face and trunk. The lesions over the extremities occasionally coalesce to constricting plaques, forming an armor-like plate. This may impair the drainage of extremities and cause lymphedema. In addition, the lower leg lesions are more likely to become secondarily infected. AIDS-associated KS in contrast to classical KS, is characterized by rapid clinical course, multifocal dissemination, and rapid internal organ involvement. Oral mucosa is more frequently involved [6].

Dermoscopy is a routine noninvasive investigation in dermatology practice. The dermoscopic features of KS s[8] are scaling, bluish-red discoloration, multicolored areas, lacuna, and brown globules. Multicolored areas (or rainbow effect) are the circumscribed structure-less areas seen in KS and are believed to be due to the diffraction phenomenon when white light is split into various wavelengths while passing through closely arranged slit-like vascular channels [7].

In our patient, dermoscopy of the KS skin lesion showed bluish-red discoloration of the plaques, which was suggestive of vascular pathology. In a study done by Hu *et al* [8] on 141 KS lesions (seven patients), bluish-red coloration was the most common dermoscopic pattern seen in 84% of the lesions. The other findings are rainbow effect seen in 36% of the lesions, followed by scaly surface (29%), and brown globules (15%) [7].

Our patient had widespread cutaneous and extensive oral involvement, CD4 count >150 cells/mm^3 and had co-infection with TB. Hence, according to the AIDS Clinical Trials Group, our patient was staged as T1 L1S1 [9]. Treatment options in such cases include liposomal anthracyclines (doxorubicin and daunorubicin), paclitaxel, and interferon alpha [10].

We report this case due to rarity of KS cases reported in the Indian literature. We also want to demonstrate the dermoscopic findings of KS. Survival with AIDS is negatively affected by fatal infections, so the incidence of cancers in people living with HIV/AIDS (PLHA) in India is low. With improving care of HIV and better management of infections, especially tuberculosis, the longer survival of PLHA in India will likely to increase the importance of cancer as a clinical problem in India.

Conflict of Interest

Not available

Financial Support

Not available

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