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Treating Kaposi Wounds in the AIDS Patients

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Review Article

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ABSTRACT

Kaposi's sarcoma is the most perpetual debilitating neoplasm in AIDS, happening in around 10 percent of all peril packs. This study evaluates the effect of intra lesional vinblastine on intraoral Kaposi's sarcoma in 24 HIV-constructive, gay individual folks with 82 injuries. Complete determination happened in right around 70 percent of the cases. In non-HIV-related Kaposi's sarcoma, the contamination is usually confined to as far as possible, however in immunodeficient patients, it is a multifocal systemic infection. The clinical course of the disease shifts among patients, reaching out from a lone or two or three lazy wounds to a commanding diffuse ailment. Impelled Kaposi's sarcoma wounds, usually those on as far as possible, are frequently associated with lymphedema. In this paper, we report an occasion of a patient with an unprecedented sort of AIDS-related Kaposi sarcoma called lymphangiectatic Kaposi's sarcoma.

INTRODUCTION

Kaposi sarcoma (KS) is the most well-known threat found in the setting of HIV disease. Despite the fact that the rate of HIV-tainted patients giving KS as an AIDS-characterizing analysis diminished in the United States from around 30% in the mid-1980s to 15% 10 years after the fact, irrefutably the quantity of cases did not decay over this time period (1-3) as the frequency of KS as an auxiliary AIDS determination expanded from 23% in the 1980s to half in the late 1990s [1]. Treatment with viable antiretroviral treatment (ART) has prompted an emotional decrease in KS frequency. A remedy for ART may decrease the probability of KS by half. In the present time of treatment, KS is by and large recognized as a late appearance of HIV contamination, happening when immunosuppression is serious. In this way, KS-related dreariness and death rates have expanded, especially in sub-Saharan Africa [2].

HIV-infected patients in whom AIDS-related complications have regressed following therapy with HIV protease inhibitors have recently been described [3]. In these circumstances in particular, cutaneous Kaposi's sarcoma (KS) lesions may completely resolve and human herpes virus (HHV)-8 may be cleared from peripheral blood mononuclear cells. In contrast, we describe an HIV-infected man who became dangerously symptomatic with KS shortly after starting protease inhibitor treatment. Reagin and treponemal immunizer tests are very solid in diagnosing auxiliary syphilis [4]. Be that as it may, patients tainted with the human immunodeficiency infection (HIV) react unusually to antigenic incitement and may neglect to create common serologic reactions to contaminations [5]. We report the instance of a HIV-contaminated man with Kaposi sarcoma and auxiliary syphilis whose VDRL test and fluorescent treponemal neutralizer consumed test were more than once nonreactive [6]. Right determination required biopsy of a skin sore with silver recoloring to show spirochetes. Clinicians treating HIV-tainted patients ought to know about the issues of serologic determination of syphilis in these patients. Biopsy tests of fitting tissues and recoloring for spirochetes might be expected to touch base at the right analysis [7].

ANTIRETROVIRAL THERAPY

The visualization connected with some instinctive KS areas (remarkably, that connected with lung KS) has drastically enhanced with the utilization of HAART in mix with chemotherapy [8]. HAART has been accounted for to draw out time to treatment disappointment in KS, and complete abatements of both cutaneous and instinctive KS have been accounted for in patients treated

with HAART alone ^[9]. The reduction of KS seen in patients with AIDS who are treated with HAART is very like the determination of KS seen in patients with iatrogenic KS after treatment with immune suppressors is adjusted or quit, affirming the pioneering way of KS. In patients with AIDS who are treated with HAART, invulnerable rebuilding gives off an impression of being the primary element connected with KS abatement ^[10]. It has been demonstrated that abatement of KS in patients who get a HAART regimen is connected with a huge increment altogether CD4+ cell number. Investigations of cell insusceptibility have shown that KS is connected with an absence of invulnerable reaction to human herpesvirus 8 (HHV-8) epitopes, though HHV-8-tainted patients without KS have a cell reaction to HHV-8 epitopes ^[11]. Notwithstanding, longitudinal studies have demonstrated that HHV-8-particular resistant reclamation is deferred >24 months after the start of HAART. Suggesting that control of KS might be owing to a mix of invulnerable rebuilding and restraint of HIV replication ^[12]. Indeed, in a review study, it was demonstrated that an imperceptible HIV burden was the best marker of KS reaction at 6, 12, and 24 months after the presentation of HAART and that patients with an imperceptible HIV load encountered the best reaction ^[13].

In addition to remission of KS, incomprehensible intensifications of previous KS have been accounted for among extremely immunocompromised patients at the presentation of HAART. Such exacerbations were considered to be manifestations of an immune reconstitution inflammatory syndrome ^[14].

In fact, in addition to KS, HHV-8 contamination has been involved in no less than 2 uncommon lymph proliferative issue: the multicentric type of castle man illness, and essential emission lymphoma ^[15]. Because HHV-8 is embroiled in lymphoma genesis, the overabundance of non-Hodgkin lymphoma among such patients is not totally astounding; the nearness of HHV-8 DNA in all cases and the sorts of non-Hodgkin lymphoma (including 3 instances of primary effusion lymphoma) further represented this speculation ^[16].

The negative effect of doxorubicin itself might be a variable and requires extra studies, since we realize that patients who have been treated with anthracyclins may later create non-Hodgkin lymphoma and leukemia ^[17]. The imperative message passed on by the high number of patients with non-Hodgkin lymphoma reported in this study ought to be the significance of keeping away from unreasonable introduction to chemotherapeutic medications that could support the advancement of non-Hodgkin lymphoma or different neoplasms in populace that are at danger for growing such difficulties ^[18].

For restricted infection, characterized as <10 skin injuries with no proximal edema and no mucosal association, HAART alone ought to be favored, with a strict clinical checking to identify movement of KS, which is uncommon yet has been accounted for in both patients with safe reclamation and patients without resistant rebuilding ^[19]. Adjuvant neighborhood treatments can be utilized to treat patients who experience such movement.

Observing HHV-8 levels in peripheral blood may give intriguing information that can help, not just in taking after the reaction to nonspecific or particular treatment, additionally in recognizing the advancement of non-Hodgkin lymphoma or castle man illness. It has been exhibited that patients with those lymph proliferative issue have abnormal amounts of HHV-8 in this compartment ^[20].

ART Workmanship has substantially affected KS occurrence in asset rich settings, however more consideration is required on truly evaluating this impact with a specific end goal to figure out if extra intercessions are required ^[21]. Developing information from asset constrained areas likewise recommends advantageous effect of ART on KS occurrence, however given the extent of KS in these settings more information are expected to comprehend the broadness and greatness of the impact ^[22].

In a clinic-based cohort, the populace level viability of ART will be overestimated. This is because that the patients investigated in the ART period are efficiently enhanced for those in consideration and are without patients not on ART ^[23]. Likewise, these studies are not legitimately evaluating singular patient-level adequacy since ART use fundamentally was not the indicator variable. Indeed, numerous patients in these studies in the ART period were not really utilizing ART, consequently prompting likely underestimation of the individual patient-level viability of ART ^[24]. It is subsequently in the domain of individual patient-level adequacy of ART that these non-populace based associate studies have their most prominent commitment ^[25]. Given that most are evaluating >70% diminishment in KS occurrence, we can deduce that the genuine quality is likely in this extent ^[26].

The decision of treatment past HAART must be individualized and relies on upon the degree of malady, the nearness and nature of the side effects, the rate of illness movement, and the general remedial objectives. Since its origin, HAART treatment has changed the objective in Kaposi sarcoma treatment from short-term palliation to long haul abatement and control ^[27].

Compelling blend antiretroviral treatment more often than not is included a mix of either a protease inhibitor (PI) or non-nucleoside reverse transcriptase inhibitor (NNRTI) with 2 nucleoside reverse transcriptase inhibitors ^[28]. Some evidence recommends a direct antitumor impact on angio proliferative Kaposi sarcoma-type sores. However without further ado, no level 1 proof backings this clinically. No distinction is clear between PI-based and NNRTI-based antiretroviral regimens regarding reaction of Kaposi sarcoma ^[29]. HAART might be attempted as the sole methodology utilized as a part of non-visceral sickness. For instinctive ailment, chemotherapy might be included. For locally symptomatic ailment, radiation treatment might be presented ^[30].

LOCAL THERAPY

Local therapy is most appropriate for people who require vindication of privately progressed symptomatic illness (e.g.

radiation) or for people who have cosmetically unsuitable sores^[34]. This therapy is also well appropriate for people with huge comorbidities and malady recalcitrant to systemic modalities. It can give better cosmesis, control massive sores that cause dying, torment or, edema, and treat broad skin infection^[32].

TYPES OF LOCAL THERAPIES

Radiation therapy

Radiation therapy is the most generally utilized and powerful nearby treatment. This can whitewash dying, torment, or unattractive injuries^[33]. This might be given as low-voltage (100 kv) photons or electron-beam radiotherapy. Reactions happen in 80-90% of patients. Higher aggregate measurements (40 Gy) results in preferred nearby control over lower dosages (8 Gy or 20 Gy). Electron beam therapy is held for treatment of shallow injuries^[34]. This is generally giving once week after week in 4 Gy fractions. Recurrence might be normal in contiguous, untreated regions, driving a few creators to prescribe stretched out field radiotherapy to influence a higher cure rate. Patients with HIV are more inclined to create radiation-prompted mucositis too and hyperpigmentation, desquamation, and ulceration of treated sores^[35].

Electron beam therapy, which has constrained infiltration past the dermis, is compelling for shallow injuries and for the most part gives great corrective results^[36]. At first, raised sores level, injury size may diminish, and a violaceous sore for the most part changes to a tan-cocoa, hyper pigmented spot. Genital KS connected with agony, trouble with urination, or psychological inconvenience might be all around mitigated with electron shaft treatment without noteworthy danger^[37]. Radiation therapy is compelling in treating oral cavity injuries, but since of the noteworthy danger of radiation-prompted mucositis, this treatment ought to be saved for symptomatic ailment^[38]. To minimize the horribleness of oral pit illumination, patients ought to be forcefully pretreated for nearby parasitic and herpetic diseases, and recommended utilization of an antibacterial mouthwash^[39].

Intralesional chemotherapy

Intralesional infusions with vinblastine have been utilized to treat restricted mucocutaneous infection. In general, littler sores will probably react^[40]. Typically, 0.1 mL of a dilute solution containing 0.2 mg/mL of vinblastine is injected into a lesion using a tuberculin syringe. Repeated injections may be necessary. The injections regularly are agonizing and might be connected with a neighborhood incendiary response^[41]. Most reactions are fractional, and tumor regrowth in 4-6 months is common. The utilization of intralesional interferon (IFN) - alfa (3-5 MU three times each week for 3-4 weeks) accomplishes comparative results. Similarly as with other nearby treatments, skin staining is the principle post therapy^[42].

Intralesional treatment with vinca alkaloids with low-measurement vincristine or vinblastine and additionally bleomycin has been utilized as a part of a restricted mold basically for the exemplary type of Kaposi sarcoma where limited skin illness prevails^[43]. Reactions happen in 60-90% of patients with little in the method for systemic symptoms with term of 4-6 months. Dosing is done at around one-tenth the systemic dosage of medication with 3 to 4 week interims between treatments^[44]. Reactions incorporate changes in pigmentation, swelling, rankling, ulceration, and agony on infusion and additionally limited yet typically transient neuropathic indications^[45]. Since the illness repeats in different territories, its utilization is moderately constrained. Additionally, systemic vinca alkaloid treatment might be similarly successful and cause less restricted skin poisonous quality^[46].

Cryotherapy

Liquid nitrogen connected sufficiently long so it will take the sore around 40 s to defrost has been utilized for cosmetically exasperating injuries, especially of the face. Regularly sores require more than one treatment, and in spite of the fact that reactions are seen, especially in moderately level, little injuries (<2 cm), hypopigmentation for the most part results from treatment^[47].

Cryotherapy has the upside of brief length, insignificant inconvenience, and capacity to be utilized more than once and as a part of blend with different types of treatment. It has restricted entrance and is not perfect for extensive, profound sores^[48].

Topical retinoids

Palliative systemic treatment is demonstrated for symptomatic or life-threatening visceral disease, quickly dynamic mucocutaneous illness with torment or ulceration, and symptomatic lymphedema^[49]. In this setting, couple of dependable appraisals of reaction rate with HAART alone contrasted and consolidated HAART and chemotherapy are accessible^[50]. One trial from South Africa contrasting HAART and HAART and chemotherapy appeared in the purpose to treat patient's 39% reaction in HAART alone contrasted and 66% in HAART in addition to chemotherapy. Additionally, 35% of patient in the HAART arm traversed to require palliative chemotherapy or radiation inside 12 months of randomization. These outcomes bolster that chemotherapy and HAART ought to be utilized together as a part of patients with high tumor volume^[51].

Cytotoxic chemotherapy

Chemotherapy may bring about quick determination of KS-related side effects and in this manner enhance personal satisfaction^[52]. Cytotoxic chemotherapy is demonstrated for patients with broad mucocutaneous KS, quickly dynamic cutaneous illness (more than 10 new sores for each month), symptomatic instinctive malady, aspiratory infection, or broad symptomatic lymphedema^[53]. A wide assortment of chemotherapeutic operators, independently and in mix, has been assessed for the

treatment of KS. The expansive scope of KS reaction rates to single specialists (21-80%) is an aftereffect of contrasts in the adequacy of the operators tried, varieties in the patient populaces treated (counting level of safe capacity, history of earlier sharp diseases, and tumor weight), and absence of institutionalization of the criteria used to organize these patients or to assess their reaction to treatment [54]. When all is said in done, the stage III clinical trials finished following 1990 characterize the study populace and treatment results all the more thoroughly, applying the ACTG organizing and reaction criteria [55].

Investigational therapy

Expanding comprehension of the pathogenesis of AIDS-related KS has prompted examinations concerning an assortment of pathogenesis-based treatments [56]. In spite of the fact that HHV-8 is connected with the greater part of KS injuries, accessible information does not bolster the utilization of antiherpes specialists (foscarnet, ganciclovir, acyclovir and so on) for the treatment of built up KS [57]. Regardless of whether this methodology will be valuable for counteractive action of KS in patients with serologic proof of HHV-8 has yet to be resolved. In the course of the most recent couple of years, inhibitors of both cytokines and angiogenesis have been assessed for the treatment of KS [58]. To date, albeit little quantities of reactions to these biologic treatments have been accounted for, none of these specialists are particularly endorsed for the treatment of KS. It is trusted that further advancement of antiangiogenesis specialists, cytokine inhibitors, or both, will bring about long haul, nontoxic treatment for patients with KS [59].

Therapeutic recommendations

In spite of the fact that KS remains a hopeless tumor for which no settled standard treatment is characterized, general standards of treatment give a premise to balanced clinical basic leadership [60]. The choice of treatment for KS must check the potential advantage and antagonistic impacts of treatment, communications with different pharmaceuticals, and potential effect on basic immunosuppression [61].

Cosmetically disturbing lesions should be treated early and, if limited in number, may be appropriate for local therapy. Radiation therapy is the most effective local therapy [62-69].

Interferon-alfa in mix with antiretroviral operators ought to be considered for the treatment of patients with gradually dynamic or negligibly symptomatic KS [70-77]. The long length of reaction, the restricted danger of low-dosage IFN (1-10 MU/day subcutaneously) either alone or in blend with nonmyelosuppressive antiretroviral specialists, and characteristic antiviral action make IFN-alfa an imperative treatment for KS. This blend ought to be considered for clinically stable patients, even those with CD4 lymphocyte tallies <100 cells/mm³ [78-81].

Systemic chemotherapy should be used for patients with rapidly progressive, symptomatic, or life-threatening KS. Individuals with pulmonary disease or severe lymphedema should receive chemotherapy [82-88].

For patients who require systemic chemotherapy, liposome-epitomized anthracyclines are more compelling and less lethal than ABV. Reaction rates to paclitaxel are high, with adequate poisonous quality [89-95]. This operator is successful in patients who have backslid after earlier anthracycline-based chemotherapy. Patients with previous myelosuppression or people who require other myelosuppressive specialists ought to get state invigorating variables in blend with chemotherapy [96-100].

REFERENCES

1. Chang Y, et al. Identification of herpesvirus-like DNA sequences in AIDS-associated Kaposi's sarcoma. *Science* 1994;226:1865-1869.
2. Rizzieri DA and Liu J. Clearance of HHV-8 from peripheral blood mononuclear cells with a protease inhibitor [letter]. *Lancet* 1997;349:775-776.
3. Murphy M, et al. Regression of AIDS-related Kaposi's sarcoma following treatment with an HIV-1 protease inhibitor [letter]. *AIDS* 1997;11:261-262.
4. Valcuende-Cavero F, et al. Langerhans' cells and lymphocytic infiltrate in AIDS-associated Kaposi's sarcoma. An immunohistological study. *Acta Derm Venereol* 1994;74:183-187.
5. Carr A and Cooper DA Restoration of immunity to chronic hepatitis B infection in HIV-infected patient on protease inhibitor [letter]. *Lancet* 1997;349:995-996.
6. Hymes KB, et al. Kaposi's sarcoma in homosexual men—a report of eight cases. *Lancet*. 1981;2:598-600.
7. Hoover DR, et al. Epidemiologic analysis of Kaposi's sarcoma as an early and later AIDS outcome in homosexual men. *Am J Epidemiol*. 1993;138:266–278.
8. Wabinga HR, et al. Cancer in Kampala, Uganda, in 1989–91: changes in incidence in the era of AIDS. *Int J Cancer*. 1993;54:26-36.
9. Zwahlen M, et al. Mortality of HIV-infected patients starting potent antiretroviral therapy: comparison with the general population in nine industrialized countries. *Int J Epidemiol*. 2009;38:1624-1633.
10. Carrieri MP, et al. Reduced incidence of Kaposi's sarcoma and of systemic non-hodgkin's lymphoma in HIV-infected individuals treated with highly active antiretroviral therapy. *Int J Cancer*. 2003;103:142-144.

11. Clifford GM, et al. Cancer risk in the Swiss HIV cohort study: Associations with immunodeficiency, smoking and highly active antiretroviral therapy. *J Natl Cancer Inst.* 2005;97:425-432.
12. Franceschi S, et al. Kaposi sarcoma incidence in the Swiss HIV cohort study before and after highly active antiretroviral therapy. *Br J Cancer.*2008;99:800-804.
13. Rabkin CS and Yellin F. Cancer incidence in a population with a high prevalence of infection with human immunodeficiency virus type 1. *J Natl Cancer Inst.* 1994;86:1711-1716.
14. Montaner JS, et al. The changing spectrum of AIDS index diseases in Canada. *AIDS.* 1994;8:693-696.
15. Jacobson LP, et al. Impact of potent antiretroviral therapy on the incidence of Kaposi's sarcoma and non-Hodgkin's lymphomas among HIV-1-infected individuals. Multicenter AIDS Cohort Study. *J Acquir Immune Defic Syndr.* 1999;21:34-41.
16. Jones JL, et al. Incidence and trends in Kaposi's sarcoma in the era of effective antiretroviral therapy. *J Acquir Immune Defic Syndr.* 2000;24:270-274.
17. Chokunonga E, et al. Cancer incidence in the African population of Harare, Zimbabwe: Second results from the cancer registry 1993-1995. *Int J Cancer.* 2000;85:54-59.
18. Volberding PA, et al. Effect of chemotherapy for HIV-associated Kaposi's sarcoma on long term survival. Proceedings of the American Society of Clinical Oncology, San Francisco, 1989;11.
19. Holkova B, et al. Effect of highly active antiretroviral therapy on survival in patients with AIDS-associated pulmonary Kaposi's sarcoma treated with chemotherapy. *J Clin Oncol.* 2001;19:3848-3851.
20. Tam HK, et al. Effect of highly active antiretroviral therapy on survival among HIV-infected men with Kaposi sarcoma or non-Hodgkin lymphoma. *Int J Cancer.* 2002;98:916-922.
21. Piedbois P, et al. Radiotherapy in the management of epidemic Kaposi's sarcoma. *Int J Radiat Oncol Biol Phys.* 1994;30:1207-1211.
22. Gao SJ, et al. Seroconversion to antibodies against Kaposi's sarcoma-associated herpes virus-related latent nuclear antigens before the development of Kaposi's sarcoma. *N Engl J Med.* 1996;335:233-241.
23. Dezube BJ. The role of human immunodeficiency virus-I in the pathogenesis of acquired immunodeficiency syndrome-related Kaposi's sarcoma: The importance of an inflammatory and angiogenic milieu. *Semin Oncol.* 2000;27:420-423.
24. Pati S, et al. Antitumorigenic effects of HIV protease inhibitor ritonavir: inhibition of Kaposi sarcoma. *Blood.* 2002;99:3771-3779.
25. Sgadari C, et al. HIV protease inhibitors are potent anti-angiogenic molecules and promote regression of Kaposi sarcoma. *Nat Med.* 2002;8:225-232.
26. Le Bourgeois JP, et al. Radiotherapy in the management of epidemic Kaposi's sarcoma of the oral cavity, the eyelid and the genitals. *Radiother Oncol.* 1994;30:263-266.
27. Nobler MP, et al. The impact of palliative irradiation on the management of patients with acquired immune deficiency syndrome. *J Clin Oncol.* 1987;5:107-112.
28. Chak LY, et al. Radiation therapy for acquired immunodeficiency syndrome-related Kaposi's sarcoma. *J Clin Oncol.* 1988;6:863-867.
29. Hill DR. The role of radiotherapy for epidemic Kaposi's sarcoma. *Semin Oncol.* 1987;14:19-22.
30. Stelzer KJ and Griffin TW. A randomized prospective trial of radiation therapy for AIDS-associated Kaposi's sarcoma. *Int J Radiat Oncol Biol Phys.* 1993;27:1057-1061.
31. Boudreaux AA, et al. Intralesional vinblastine for cutaneous Kaposi's sarcoma associated with acquired immunodeficiency syndrome. A clinical trial to evaluate efficacy and discomfort associated with infection. *J Am Acad Dermatol.* 1993;28:61-65.
32. Luppi M et al. Bone marrow failure associated with human herpes virus 8 infections after transplantation. *N Engl J Med* 2000;343:1378-1385.
33. Schwartz R, et al. Kaposi sarcoma: A continuing conundrum. *Journal of the American Academy of Dermatology.* 2008;59:179-206.
34. Antman K and Chang Y. Kaposi's Sarcoma. *New England Journal of Medicine* 2000;342:1027-1038.
35. Dezube BJ. Clinical presentation and natural history of AIDS-related Kaposi sarcoma. *Hematol Oncol Clin North Am* 1996;10:1023-1029.
36. Schwartz Robert A and Borkovic SP Kaposi's sarcoma presenting in a homosexual man-a new and striking phenomenon. *Ariz Med* 1994;38:902-904.
37. Garay SM, et al. Pulmonary manifestations of Kaposi sarcoma. *Chest* 1987;91:39-43.
38. Ensoli B and Sirianni MC Kaposi's sarcoma pathogenesis: a link between immunology and tumor biology. *Critical Reviews in Oncogenesis (Begell House)* 1998;9:107-124.
39. Ablashi DV, et al. Spectrum of Kaposi's sarcoma-associated herpes virus, or human herpes virus 8, diseases. *Clin Microbiol Rev* 2002;15:439-464.

40. Weninger W, et al. Expression of vascular endothelial growth factor receptor-3 and podoplanin suggest a lymphatic endothelial origin of Kaposi's sarcoma tumor cells. *Lab Invest* 1999;79:243-251.
41. Pauk J, et al. Mucosal shedding of human herpes virus 8 in Men. *New England Journal of Medicine* 2000;343:1369-1377.
42. Danzig JB, et al. Gastrointestinal malignancy in patients with AIDS. *Am J Gastroenterol* 1991;86:715-718.
43. Qunibi W, et al. Serologic association of human herpes virus eight with post-transplant Kaposi sarcoma in Saudi Arabia. *Transplantation* 1998;65:583-585.
44. Olsen SJ, et al. Increasing Kaposi's sarcoma-associated herpes virus seroprevalence with age in a highly Kaposi's sarcoma endemic region, Zambia in 1985. *AIDS* 1998;12:1921-1925.
45. Cook-Mozaffari P, et al. The geographical distribution of Kaposi's sarcoma and of lymphomas in Africa before the AIDS epidemic. *Br J Cancer* 1998;78:1521-1528.
46. Iscovich J, et al. Classic Kaposi's sarcoma in Jews living in Israel, 1961-1989: a population-based incidence study. *AIDS* 1998;12:2067-2072.
47. Kumar P. Classic Kaposi's Sarcoma in Arabs – Widening ethnic involvement. *J Can Res Ther* 2011;7:92–94.
48. Hladik W. Transmission of human herpes virus 8 by blood transfusion. *New England Journal of Medicine* 2006;355:1331-1338.
49. Mesri EA, et al. Kaposi's sarcoma and its associated herpes virus. *Nat rev cancer*. 2010;10:707-719.
50. Lanternier F, et al. Kaposi's sarcoma in HIV-negative men having sex with men. *AIDS*. 2008;22:1163-1168.
51. Suresh K, et al. Pleuropulmonary Kaposi Sarcoma in the setting of immune reactivation. *J Pulm Respir Med* 2016;6:352.
52. Mokale T and Thekiso MD. Oral health awareness of oncology nurses at Charlotte Maxeke Johannesburg Hospital. *J Oral Hyg Health* 2016;4:199.
53. Satyajit P. Overview of xeroderma pigmentosum mutations, prognosis and treatment. *Adv Genet Eng* 2016;5:141.
54. Melé-Ninot G, et al. AIDS-Related Kaposi's sarcoma and associated immune reconstitution inflammatory syndrome. *Clin Microbiol*. 2016;5:236.
55. Patrice GI, et al. Kaposi's Sarcoma in child. A case report. *J Clin Exp Dermatol Res*. 2016;7:317.
56. Corti M. Burkitt's lymphoma associated with HIV Infection. *Clin Microbiol*. 2016;5:232.
57. Marconi B, et al. Neoplastic skin complications in transplant patients: Experience of an Italian multidisciplinary transplant unit. *J Clin Exp Dermatol Res*. 2015;6:295.
58. Jordão C, et al. Disseminated Kaposi's Sarcoma as initial event in AIDS. *J AIDS Clin Res* 2015;6:450.
59. Mohanty S, et al. TAp63alpha induced apoptosis inhibited by Kaposi's Sarcoma herpes virus latency nuclear antigen. *J Carcinog Mutagen*. 2015;6:221.
60. Jalbert E, et al. Stage-IV Kaposi's Sarcoma during abatacept therapy: A case report. *Rheumatology (Sunnyvale)*. 2015;5:144.
61. Yongabi KA, et al. Preliminary report on management of HIV/AIDS-associated opportunistic skin infections with PhytodermaTM, a natural myco-based cream. *J Mol Pharm Org Process Res*. 2014;2:122.
62. Joseph EE, et al. Kaposi Disease in hospitalization: Reflect of accessibility to HAART in African countries? Case of dermatology department of the University Hospital of Treichville (Abidjan-Rci). *J Clin Exp Dermatol Res*. 2014;5:238.
63. Vieira F, et al. Oral Kaposi's Sarcoma In HIV positive patients. a case report and a review of literature. *J AIDS Clin Res*. 2014;5:349.
64. Elfaki MG. Immunosuppression Induced by HIV Infection. *Biol Med*. 2014;6:e111.
65. Ray PE, et al. Expression of a secreted fibroblast growth factor binding protein-1 (FGFBP1) in angioproliferative Kaposi Sarcoma. *J AIDS Clin Res*. 2014;5:309.
66. Hsu YP, et al. Oropharyngeal Kaposi's Sarcoma from an immunocompetent host: A case report. *J Cytol Histol*. 2014;5:254.
67. van Bogaert LJ. Immunostaining by human herpes virus 8 latent nuclear antigen-1 of Kaposi's sarcoma: A potential biomarker of severity of disease? *J Mol Biomark Diagn* 2013;S5:002.
68. Patel M. Human immunodeficiency virus infection and chronic myeloid leukemia: Is there an association? *J Leuk*. 2014;1:e108.
69. Ueda K (2012) Kaposi's Sarcoma-associated herpes virus induced tumorigenesis; how viral oncogenic insults are evaded. *J Blood Lymph* 2:e109.
70. Amare B, et al. Pattern of ocular manifestation of HIV/ AIDS among patients on HAART in ART Clinic of Gondar University Hospital, Northwest Ethiopia. *J Clinic Experiment Ophthalmol*. 2011;2:192.
71. Essadi I, et al. The role of chemotherapy in the treatment of Kaposi's Sarcoma. *J Cancer Sci Ther*. 2011;3:145-148.

72. Goldenitsch E, et al. An uncommon cause of soft tissue mass of the extremities, report of 2 cases and review of literature of cystic echinococcosis. *Primary Health Care*. 2016;6:225.
73. Berezin AE. Is elevated circulating galectin-3 level A predictor of pulmonary artery hypertension development and progression? *Clin Med Biochemistry Open Access*.2016;2:114.
74. Salim X, et al. First case of mesenteric extraosseous osteosarcoma in Australia. *J Oncol Med & Pract*. 2016;1:104.
75. Madoz-Gúrpide J, et al. Proteomic profiling of Ewing Sarcoma reveals a role for TRAF6 in proliferation and ribonucleoproteins/ RNA processing. *J Proteomics Bioinform*. 2016;9:166-175.
76. Goldenitsch E, et al. An uncommon cause of soft tissue mass of the extremities, report of 2 cases and review of literature of cystic echinococcosis. *Primary Health Care*. 2016;6:225.
77. Berezin AE. Is elevated circulating galectin-3 Level A predictor of pulmonary artery hypertension development and progression? *Clin Med Biochemistry Open Access*. 2016;2:114.
78. Tranesh G, et al. Metastatic monophasic synovial sarcoma: A challenging diagnosis on fine needle aspiration with broad differential diagnosis. *Diagn Pathol Open*. 2016;1:114.
79. Gomes MC and Rocha NS. Spontaneous osteosarcoma in dogs: Diagnosis through cytopathological and histopathological assays. *J Cytol Histol*. 2016;7:404.
80. Sébastien P. Peroperative tumoral radial dissemination during cancellous bone graft harvesting: Chondroma of the finger. *J Cytol Histol*. 2016;7:407.
81. Sood N and Sehrawat N. Central primary fibrosarcoma maxilla - A rare presentation. *Otolaryngol (Sunnyvale)*. 2016;6:237.
82. Sreehari S and Balasubramanian B. GIST or not - A unique case of low grade endometrial stromal sarcoma with review of literature. *J Cytol Histol*. 2016;7:414.
83. Semmar A, et al. Laryngeal chondrosarcoma: A case report. *J Clin Case Rep*.2016;6:757.
84. Misra S, et al. Advanced case of rhabdomyosarcoma of orbit mimicking orbital cellulitis. *J Clin Exp Ophthalmol*. 2016;7:533.
85. Stramare R, et al. Imaging features, differential diagnosis and management of leiomyosarcomas: Case series and review of the literature. *J Cancer Sci Ther*. 2016;8:084-091.
86. DeRenzo C and Gottschalk S. Genetically modified T-cell therapy for the treatment of osteosarcoma: An update. *J Clin Cell Immunol*. 2016;7:417.
87. Târcoveanu E, et al. Particularities of primary breast cancer in men. *Journal of Surgery*. 2016;12: 29-35.
88. Shruthi PJ, et al. Nucleolar organizer region count, PCNA and Ki-67 indices are diagnostic markers of malignancy and cell proliferation rate in bovine lymphosarcoma. *J Veterinar Sci Technol*. 2016;7:305.
89. Ilan K, et al. Primary primitive neuroectodermal tumor of the urinary bladder: A case report of a rare pathological entity with a rapidly progressing clinical course. *J Clin Case Rep*. 2016;6:727.
90. Barreca S, et al. Huge liposarcoma of the forefoot: A case report. *J Integr Oncol*. 2016;5:160.
91. Suhag V. Pitfalls in management of childhood tumours. *Int J Sch Cog Psychol*. 2016;3:162.
92. Latheef R and Bali A. Ewing's Sarcoma of uterus - Case report and review of literature. *Gynecol Obstet (Sunnyvale)*. 2016;6:362.
93. Mokale T and Thekiso MD. Oral health awareness of oncology nurses at Charlotte Maxeke Johannesburg Hospital. *J Oral Hyg Health*. 2016;4:199.
94. Viani L, et al. Primitive myeloid sarcoma of the breast: A case report. *J Clin Case Rep*. 2016;6:671.
95. Majethia NK. Lipoblastoma in paediatric tumour-report of two cases. *Med Rep Case Stud*. 2016;1:103.
96. Heissner K, et al. Treatment associated interstitial pulmonary toxicity of temozolomide plus bevacizumab for locally advanced solitary fibrous tumor. *J Pulm Respir Med*. 2016;6:314.
97. Tiong FL. About the rare case of a pelvic primitive neuroectodermal tumor in a 37 year old patient. *J Clin Case Rep*. 2015;5:664.
98. Jiang N, et al. Gas6/Axl inhibits osteosarcoma apoptosis through regulation of apoptosis-related protein Bcl-2. *J Bioanal Biomed*.2015;8:001-008.
99. Kailas KC, et al. Hidradenoma: A skin adnexal tumour, case report and literature review. *J Pat Care*. 2015;1:104.
100. Ticku A and Singh P. Metaplastic carcinoma of breast with osteosarcomatous differentiation: A rare case diagnosed by fine needle aspiration cytology. *J Med Surg Pathol*. 2016;1:103.