MARMARA
MEDICAL JOURNAL

Original Article

https://dergipark.org.tr/tr/pub/marumj

Oncological perspective on Kaposi sarcoma: A single-centre experience

Ali Kaan GUREN[1], Iclal CAKIR[2], Nargiz MAJIDOVA[1], Nadiye SEVER[1], Erkam KOCAASLAN[1], Pinar EREL[1], Yesim AGYOL[1], Abdussamed CELEBI[1], Rukiye ARIKAN[1], Selver ISIK[1], Murat SARI[1], Ozlem ERCELEP[1], Ibrahim Vedat BAYOGLU[1], Osman KOSTEK[1]

[1] Division of Medical Oncology, Department of Internal Medicine, Marmara University School of Medicine, Istanbul, Türkiye.
[2] Department of Internal Medicine, Marmara University School of Medicine, Istanbul, Türkiye.
Corresponding Author: Ali Kaan GUREN
E-mail: alikaanguren@gmail.com

Submitted: 22.02.2024 **Accepted:** 18.07.2024

ABSTRACT

Objective: Kaposi sarcoma (KS) presents as a multifocal angioproliferative disease with distinct four subtypes, including classical, AIDS-related(epidemic), endemic, and iatrogenic. The rarity of the disease and the clinically different characteristics of the subtypes have led to insufficient experience in treatment and follow-up. Based on this, we aim to investigate the clinicopathological features and treatment options of the KS patients treated and followed up in our clinic.

Patients and Methods: The study included 66 patients diagnosed with KS by histopathological examination. KS subtypes, stages, distribution of lesions, chemotherapy regimens and efficacy of chemotherapy regimens were recorded. The efficacies of paclitaxel and pegylated liposomal doxorubicin (PLD) were compared. Kaplan-Meier and Cox regression analyses were used for statistical analyses.

Results: Classical KS was the most common subtype (n=50), followed by AIDS-related (n=14) and iatrogenic (n=2) types. 32 patients received systemic treatment. In different lines, paclitaxel was administered to 32 patients and PLD to 12 patients. Disease control rate (DCR) was 78.1% for paclitaxel and 75% for PLD, while overall response rate (ORR) was 68.7% and 58.3%, respectively.

Conclusion: Paclitaxel and PLD demonstrated efficacy in controlling aggressive KS. However, larger studies are warranted to further validate these findings and optimize chemotherapy strategies.

Keywords: Kaposi sarcoma, Classic Kaposi sarcoma, AIDS-related Kaposi sarcoma, Pegylated liposomal doxorubicin, Paclitaxel

1. INTRODUCTION

Kaposi sarcoma (KS) is a multifocal angioproliferative disease of the vascular endothelium. It is characterised by dark purple and brown coloured lesions that can be seen all over the body [1]. It was first described as idiopathic multipigmented sarcoma of the skin by Moritz Kaposi in 1872 [2]. In the following years, this idiopathic sarcoma was shown to be associated with Kaposi sarcoma herpesvirus/human herpesvirus 8 (KSHV/HHV-8) [3]. KSHV/HHV-8 is thought to activate signalling pathways related to angiogenesis and vascular differentiation [4]. There are 4 subtypes: classical, acquired immunodeficiency syndrome (AIDS)-related (epidemic), endemic and iatrogenic. The classical type is the first type defined and is more common in middle and old age. It generally occurs in the lower extremities and is limited to the skin. It usually has a slow course and is not a mortal disease [5]. AIDS-related type is more common in young males and may have a more aggressive course compared to the classical type. It frequently causes nodal and visceral involvement [6]. The endemic type is the subtype described in sub-Saharan Africa. It can be seen in different spectrum ranging from benign maculonodular forms to aggressive florid forms [7]. Iatrogenic KS is related with immunosuppression and is frequently seen in transplant recipients. In addition to skin involvement, mucosal, nodal and visceral involvement may be observed [8].

Kaposi sarcoma subtypes show different clinicopathological characteristics. Accordingly, there may be differences in primary treatment approaches. In the classical type, solid lesions can be removed surgically. In addition, local treatments such as cryotherapy, radiotherapy, intralesional chemotherapies and electrochemotherapy can also be applied. Conventional chemotherapies such as paclitaxel, pegylated liposomal doxorubicin can be used in cases with aggressive prognosis and diffuse or visceral involvement [9]. In the AIDS-related type,

How to cite this article: Guren AK, Cakir I, Majidova N, et al. Oncological perspective on Kaposi sarcoma: A single-centre experience. Marmara Med J 2025;38(1): 9-14. doi: 10.5472/marumj.1627569

ISSN: 1309-9469

http://doi.org/10.5472/marumj.1627569
Marmara Med J 2025;38(1): 9-14

human immunodeficiency virus (HIV) infection should first be controlled with highly active antiretroviral therapy (HAART) [10]. Conventional chemotherapies are recommended in patients whose local lesions do not regress with HAART treatment, or who have nodal and visceral involvement, or who have life-threatening disease at baseline. Especially, pegylated liposomal doxorubicin and paclitaxel treatments are used [11]. Studies show the endemic type also shows positive responses to chemotherapy and radiotherapy [7]. In the iatrogenic type, it is recommended to reduce the immunosuppression dose as the first step. Systemic treatment may be used in life-threatening situations [8].

Although, KS is rare, its prevalence varies from region to region [12]. The clinically different characteristics of the disease have led to insufficient experience in treatment and follow-up. Treatment decisions in the field of medical oncology are based on retrospective studies in small patient groups and clinical experience. As far as we have seen, there are not enough studies examining the efficacy of systemic chemotherapies. We aimed to determine the clinicopathological characteristics, treatment regimens and response differences between treatment regimens in our patients who were treated and followed up with the diagnosis of KS in our clinic.

2. PATIENTS and METHODS

The study included patients diagnosed with KS who were followed-up at the Marmara University Pendik Training and Research Hospital Medical Oncology Clinic between 01.01.2010 and 31.12.2023. Patients diagnosed with KS only through histopathological examination were included in the study. Patient data were reviewed retrospectively using patient files and electronic information system of our hospital.

Age, gender, date of diagnosis, performance scores, HHV-8 status, HIV status, disease subtype, sites of involvement, stages, local treatments (surgical excision, radiotherapy, cryotherapy), conventional chemotherapies, treatment start and end dates, treatment responses, treatment lines, mortality dates or last outpatient clinic dates were recorded completely. HHV-8 status was determined immunohistochemically. HIV status was determined by antigen/antibody testing and HIV RNA by polymerase chain reaction (PCR). Performance scores were calculated using the Eastern Cooperative Oncology Group Performance Score (ECOG PS). Treatment responses of the patients were evaluated as Complete Response (CR), Partial Response (PR), Stable Disease (SD) and Progressive Disease (PD) according to Response Evaluation Criteria in Solid Tumours (RECIST) version 1.1. Disease Control Rate (DCR) was calculated as the sum of CR, PR and SD, and Objective Response Rate (ORR) was calculated as the sum of CR and PR.

Progression-free survival (PFS) was calculated as the time in months from the patient's first dose of treatment to disease progression or to the date of the last visit if the patient was still receiving treatment. If the patient died while on treatment, the last date was accepted as the date of death. Overall survival (OS) was calculated as the time in months from the first treatment dose until the date of death or until the date of the last visit if the patient was still alive.

Statistical Analysis

SPSS version 22.0 (IBM corp.) was used for all statistics. Categorical variables were calculated using chi-square test. Survival curves were obtained using the Kaplan-Meier method. 95% confidence intervals (CI) were calculated using the Brookmeyer and Crowley method, and survival differences between groups were compared using the log-rank test. During the evaluation of the study data, the conformity of the parameters to normal distribution was evaluated by Shapiro Wilks test. One-way ANOVA test was used for intergroup comparisons of normally distributed parameters. Kruskal-Wallis test was used for intergroup comparisons of parameters that did not show normal distribution. Significance was evaluated at $p<0.05$ level.

3. RESULTS

A total of 81 patients diagnosed with KS were evaluated. 15 patients were excluded due to missing data. The 66 patients included in the study had a histopathological diagnosis of KS. Demographic characteristics, KS subtypes and ECOG performance scores of the patients are summarised in Table I. The mean age of the patients was 62.2 years. The mean age was 68.6 years (min-max 33-86) in the classical KS group and 41.2 years (min-max 22-56) in the AIDS-related KS group. 53 of the patients were male and 13 were female. In the classical KS group, 37 patients were male and 13 were female. In the AIDS-related KS group, all 14 patients were male. HHV-8 results of 60 patients were obtained and 59 patients were HHV-8 positive (98.5%). Of the patients, 50 were classical type, 14 were AIDS-related type and 2 were iatrogenic type. No patient belonging to the endemic type was observed.

Table I. *Basic characteristics of patients*

	Total Patiens (n=66)
Age (min-max)	62.2 (22-86)
Gender (%)	
Male	53 (80.3)
Female	13 (19.7)
HHV-8 (%)	
Positive	59 (98.5)
Negative	1 (1.5)
Unknown	6
Types (%)	
Classic	50 (75.7)
AIDS-related	14 (21.2)
Iatrogenic	2 (3)
Endemic	0 (0)
ECOG PS (%)	
0-1	62 (93.9)
2 and more	4 (6.1)

AIDS: Acquired immunodeficiency syndrome, ECOG PS: Eastern Cooperative Oncology Group Performance Score, HHV-8: Human herpesvirus 8

http://doi.org/10.5472/marumj.1627569
Marmara Med J 2025;38(1): 9-14

The stages of the patients at the time of diagnosis are summarised in Table II. In the classical type, 68% of the patients were in the maculonodular stage, 18% in the infiltrative stage and 7% in the florid stage. In the classical type, no patient was found in the disseminated stage. However, in the AIDS-related type, 21.4% of the patients were in the maculonodular stage, 28.6% in the infiltrative stage, 28.6% in the florid stage and 21.4% in the disseminated stage. When the stages of the patients were compared between subtypes, it was found to be statistically significant (p<0.05).

Table II. *Stage according to involvement*

	Classic (n=50)	AIDS-related (n=14)	Iatrogenic (n=2)	Total (n=66)
Stage (%)				
Maculonodular	34 (68)	3 (21.4)	1 (50)	38 (57.6)
Infiltrative	9 (18)	4 (28.6)	0 (0)	13 (19.7)
Florid	7 (14)	4 (28.6)	0 (0)	11 (16.7)
Disseminated	0 (0)	3 (21.4)	1 (50)	4 (6.1)

AIDS: acquired immunodeficiency syndrome

The distribution of lesions is presented in Table III. Visceral lesions were observed in 8% of patients with the classical type, whereas 57% of patients with the AIDS-related type had visceral involvement (p<0.001; p<0.05). While nodal involvement was observed in 26% of patients with the classical type, nodal involvement was observed in 78.6% of patients with the AIDS-related type (p=0.001; p<0.05). Mucosal involvement was seen in only 1 of 50 patients (2%) in the classical type and in 4 of 14 patients (28.5%) in the AIDS-related type, and this difference was statistically significant (p<0.001; p<0.05). Penile involvement was found in 1 patient in the classical type, whereas oesophageal involvement was seen in 1 patient in the AIDS-related type.

Table III. *Distribution of lesions*

	Classic (n=50)	AIDS-related (n=14)	Iatrogenic (n=2)	Total (n=66)
Skin Lesions (%)				
Lower extremities	45(90)	5 (35.7)	2 %100)	52 (78.7)
Upper extremities	14 (28)	4 (28.5)	0 (0)	18 (36,6)
Trunk	12 (24)	6 (42.8)	0 (0)	18 (36,6)
Head and Neck	8 (16)	2 (14.3)	1 (50)	11 (16.6)
Mucosal Lesions (%)	1 (2)	4 (28.5)	0 (0)	5 (7.5)
Visseral Lesions (%)	4 (8)	8 (57)	1 (50)	13 (19.6)
Liver	1 (2)	3 (21.4)	1 (50)	5 (7.5)
Lungs	2 (4)	3 (21.4)	1 (50)	6 (9.1)
Kidneys	2 (4)	1 (7.2)	0 (0)	3 (4.5)
Surrenals	0 (0)	2 (14.3)	0 (0)	2 %3)
Others	1 (2)	1 (7.2)	1 %50)	3 (4.5)
LAP (%)	13 (26)	11 (78.6)	1 (50)	25 (37.9)
Number of Regions (%)				
One	22 (44)	3 (21.4)	0 (0)	25 (37.9)
Two	16 (32)	4 (28.6)	1 (50)	21 (31.8)
Three	10 (20)	5 (33.3)	0 (0)	15 (22.7)
Four and more	2 (4)	2 (14.3)	1 (50)	5 (7.5)

AIDS: acquired immunodeficiency syndrome, LAP: Lymphadenopathy

While involvement of 3 or more sites was 24% in the classical type, this rate was 47.6% in the AIDS-related type. This difference in the number of involved sites was statistically significant (p=0.041; p<0.05).

Local treatments and interferon treatment were presented in Table IV. Surgical excision was performed in 12 patients (18.2%) and cryotherapy was performed in 27 patients (40.9%). Radiotherapy was applied to 18 (27.3%) patients. Radiotherapy was applied to only 1 site in 7 of these 18 patients, to 2 sites in 3 patients and to 3 or more sites in 8 patients. Radiotherapy fraction number and dose varied according to the number and status of the lesions.

Table IV. *Local, and symptomatic therapies and interferon therapy*

	Classic (n=50)	AIDS-related (n=14)	Iatrogenic (n=2)	Total (n=66)
Surgical Excision (%)	11 (22)	0 (0)	1 (50)	12 (18.2)
Cryotherapy (%)	24 (48)	2 (14.3)	1 (50)	27 (40.9)
Radiotherapy (%)	15 (30)	3 (21.4)	0 (0)	18 (27.3)
Interferon Therapy (%)	7 (14)	0 (0)	0 (0)	7 (10.6)

AIDS: acquired immunodeficiency syndrome

Table V. *Systemic therapy*

	Classic (n=50)	AIDS-related (n=14)	Total (n=66)
First Line (%)			
Paclitaxel	17 (34)	8 (57.1)	25 (37.9)
Liposomal Doxorubicin	3 (6)	2 (14.3)	5 (7.5)
Etoposide	2 (4)	0 (0)	2 (3)
Second Line (%)			
Paclitaxel	2 (4)	2 (14.3)	4 (6)
Liposomal Doxorubicin	3 (6)	1 (7.2)	4 (6)
Etoposide	1 (2)	0 (0)	1 (1.5)
Vinorelbine	1 (2)	0 (0)	1 (1.5)
Pazopanib	1 (2)	0 (0)	1 (1.5)
Third Line (%)			
Paclitaxel	1 (2)	0 (0)	1 (1.5)
Liposomal Doxorubicin	0 (0)	1 (7.2)	1 (1.5)
Vinorelbine	0 (0)	2 (14.3)	2 (3)
Fourth or Higher Line (%)			
Paclitaxel	1 (2)	1 (7.2)	2 (3)
Liposomal Doxorubicin	1 (2)	1 (7.2)	2 (3)
Etoposide	0 (0)	1 (7.2)	1 (1.5)
Vinorelbine	0 (0)	1 (7.2)	1 (1.5)

AIDS: Acquired immunodeficiency syndrome

Paclitaxel, pegylated liposomal doxorubicin, etoposide or vinorelbine were used for conventional chemotherapy treatment sequences (Table V). According to subtypes. 32 of the 66 patients received chemotherapy. All of these 32 patients received IV paclitaxel treatment in different lines (60mg/m2/weekly

or 80-100mg/m2/2 weeks or 135mg/m2/3 weeks). 3 patients received pegylated liposomal doxorubicin as rechallenge and reintroduction in 2 different lines. 12 patients received pegylated liposomal doxorubicin treatment in different lines (20mg/m2/2 weeks or 20-40mg/m2/3 weeks). 2 patients received PLD treatment as rechallenge and reintroduction in 2 different lines. 4 patients received PO etoposide treatment in different lines (50mg/day 7 days of each 14-day cycle). 4 patients also received IV vinorelbine treatment in different lines (30mg/m2/2 weeks). 1 patient received PO pazopanib treatment (800mg/day).

Treatment responses are summarised in Table IV. DCR was 78.1% for paclitaxel and 75% for PLD. ORR was 68.7% for paclitaxel and 58.3% for PLD. There was no statistically significant difference in treatment responses between paclitaxel and PLD (p=959; p>0.5).

Table VI. *Response to treatment*

	Paclitaxel (n=32)	Liposomal Doxorubicin (n=12)
Complete response n (%)	2 (6.2)	1 (8.3)
Partial response n (%)	20 (62.5)	6 (50)
Stable disease n (%)	3 (9.3)	2 (16.6)
Progressive disease n (%)	7 (21.8)	3 (25)
Disease control rate n (%)	25 (78.1)	9 (75)
Objective response rate n (%)	22 (68.7)	7 (58.3)

Median follow-up was 53 months for all patients (IQR 19.75-106). Median PFS for paclitaxel was 27.92 months (95%CI 15.25-40.60) and median PFS for PLD was 25.0 months (95%CI 3.93-46.0). There was no statistically significant difference between the PFS of the 2 treatments (p=0.74; p>0.5). Median OS for paclitaxel was 37.07 months (95%CI 22.86-51.28) and median OS for PLD was 34.55 months (95%CI 7.64-61.47). There was no statistically significant difference between the OS of the 2 treatments (p=0.903; p>0.5). These findings are summarised in Table VII. Kaplan-Meier curves for paclitaxel and PLD and PFS and OS are shown in Figures 1 and 2. The median PFS was 16.75 months (95%CI 4.81-28.68) and OS was 37 months (95%CI 16.33-57.66) in 4 patients receiving oral etoposide.

Table VII. *PFS and OS of paclitaxel and liposomal doxorubicin groups*

PFS	**Median (95%CI)**
Paclitaxel	27.92 (15.25-40.60)
Liposomal Doxorubicin	25.00 (3.93-46.00)
Overall	27.15 (16.44-37.87)
OS	**Median (95%CI)**
Paclitaxel	37.07 (22.86-51.28)
Liposomal Doxorubicin	34.55 (7.64-61.47)
Overall	36.45 (24.053-48.86)

CI: Confidence intervals, PFS: Progression-free survival, OS: Overall survival

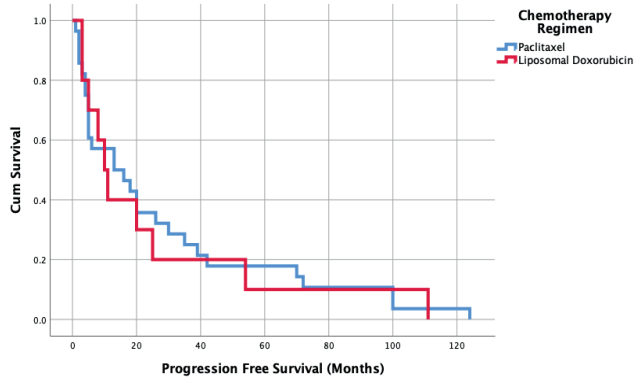

Figure 1. *PFS curves for paclitaxel and liposomal doxorubicin*

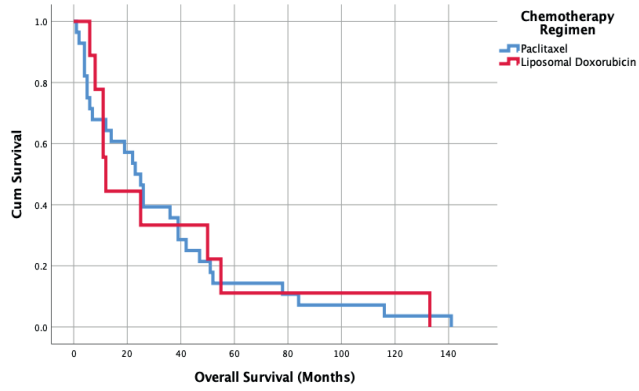

Figure 2. *OS curves for paclitaxel and liposomal doxorubicin*

4. DISCUSSION

Kaposi sarcoma has a clinicopathological heterogeneous presentation. Especially cases with aggressive course, nodal and visceral involvement benefit from systemic chemotherapy. In this study in which we present our real life data, paclitaxel and PLD treatment was used in all steps of the treatment. DCR was 78.1% for paclitaxel and 75% for PLD. ORR was 68.7% for paclitaxel and 58.3% for PLD. These 2 treatment modalities were successful in controlling the disease when used in all steps of treatment and did not provide any superiority over each other.

Paclitaxel has been shown to be effective in the treatment of all subtypes of KS and has been used for a long time [13,14]. In our centre, we administered paclitaxel treatment to all 32 patients who received systemic chemotherapy, 25 of them in the first line. When ORR and DCR rates were analysed independently of the line, the results were found to be consistent with the literature. In the literature, paclitaxel was used in the first line. This showed us that paclitaxel treatment should not be limited

http://doi.org/10.5472/marumj.1627569
Marmara Med J 2025;38(1): 9-14

to the first line and can be used when no response is obtained with other treatment options.

Previous studies have shown the efficacy of PLD treatment [15]. In the study by Colley et al, when PLD was used first, partial response was 41% and stable response was 32% [16]. In our centre, PLD was used in all lines and partial response was 50% and stable response was 16%. PLD, just like paclitaxel, showed us that it provides benefit at all stages of treatment.

In a previous study by Cianfrocca M et al., it was shown that when paclitaxel and PLD treatments were compared, neither treatment was superior to each other [17]. There was no statistically significant difference between PFS, OS, and DCR ORR in the results of our study.

The efficacy of oral etoposide treatment was demonstrated in a study by Evans et al. in patients with AIDS-related KS [18]. We used oral etoposide (PO 50mg/day 7 days of each 14-day cycle) in 4 patients, 3 of which were classical type. All 4 patients had partial response, median PFS was 16.75 months (95%CI 4.81-28.68) and median OS was 37 months. Studies including more patients are needed to confirm these favourable results.

Pazopanib (800mg/day) treatment was used in a patient who showed progression under paclitaxel and PLD, but the treatment was terminated after 3 months due to progression.

The weaknesses of our study were the small number of patients and the different treatment doses (weekly-2-weekly or 3-weekly). Due to the small number of patients, the efficacy of paclitaxel and PLD treatments on separate subgroups could not be clearly analysed.

Conclusion

Kaposi sarcoma has a clinicopathological heterogeneous distribution and there may be differences in primary treatment approaches between subtypes. However, regardless of subtype, paclitaxel and PLD can be used in all patients with uncontrolled, locally aggressive course or nodal and visceral involvement or life-threatening conditions related to KS.

Since, KS is rare, there is a need for randomised, prospective studies involving sufficient number of patients and better demonstration of the efficacy of chemotherapy agents and treatment-related side effects.

Compliance with Ethical Standards

Ethical approval: This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by Marmara University School of Medicine, Istanbul, Turkiye, (approval number: 09.02.2024.172).

Conflict of interest: The authors declare that there is no conflict of interest.

Financial support: This study received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Author contributions: AKG: Writing – reviewing and editing, Writing – original draft, Validation, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation. IC: Methodology, Investigation, Formal analysis, NM: Investigation, Resources, NS: Methodology, Investigation, EK: Software, Resources, PE: Formal analysis, Data curation, YA: Formal analysis, Data curation, AC: Data curation, Funding acquisition, RA: Investigation, Visualization, SI: Formal analysis, Validation, Resources, MS: Data curation, Conceptualization, OE: Validation, Investigation IVB: Validation, Supervision, Conceptualization, OK: Visualization, Supervision, Conceptualization, Project administration. All authors read and approved the final version of the manuscript.

REFERENCES

[1] Dupin N. Update on oncogenesis and therapy for Kaposi sarcoma. Curr Opin Oncol 2020;32:122-8. doi: 10.1097/CCO.000.000.0000000601. PMID: 31815777.

[2] Kaposi Idiopathisches multiples Pigmentsarkom der Haut. Arch. f. Dermat 1872; 4: 265-73. doi.org/10.1007/BF01830024

[3] Chang Y, Cesarman E, Pessin MS, et al. Identification of herpesvirus-like DNA sequences in AIDS-associated Kaposi's sarcoma. Science 1994 ;266:1865-9. doi: 10.1126/science.7997879. PMID: 7997879.

[4] Ganem D, Neill US. A conversation with Don Ganem. J Clin Investi 2014;124:464-5. doi: 10.1172/jci73101.

[5] Hutt MS. Classical and endemic form of Kaposi's sarcoma. A review. Antibiot Chemother (1971). 1983;32:12-7.

[6] Mitsuyasu RT. AIDS-related Kaposi's sarcoma: a review of its pathogenesis and treatment. Blood Rev 1988;2:222-31. doi: 10.1016/0268-960x(88)90011-2.

[7] Stein ME, Spencer D, Ruff P, Lakier R, MacPhail P, Bezwoda WR. Endemic African Kaposi's sarcoma: clinical and therapeutic implications. 10-year experience in the Johannesburg Hospital (1980-1990). Oncology 1994 ;51:63-9. doi: 10.1159/000227312.

[8] Brambilla L, Genovese G, Berti E, et al. Diagnosis and treatment of classic and iatrogenic Kaposi's sarcoma: Italian recommendations. Ital J Dermatol Venerol 2021;156:356-65. doi: 10.23736/S2784-8671.20.06703-6.

[9] Lebbe C, Garbe C, Stratigos AJ, et al. European Dermatology Forum (EDF), the European Association of Dermato-Oncology (EADO) and the European Organisation for Research and Treatment of Cancer (EORTC). Diagnosis and treatment of Kaposi's sarcoma: European consensus-based interdisciplinary guideline (EDF/EADO/EORTC). Eur J Cancer 2019;114:117-27. doi: 10.1016/j.ejca.2018.12.036.

[10] Mangusan RF, Ekwede I, Widell A. CE: HIV-associated Kaposi sarcoma in the combination antiretroviral therapy era. Am J Nurs 2022;122:32-40. doi: 10.1097/01.NAJ.000.090.1848.07128.92.

[11] Ramaswami R, Lurain K, Yarchoan R. Oncologic treatment of HIV-associated Kaposi sarcoma 40 Years on. J Clin Oncol 2022;40:294-306. doi: 10.1200/JCO.21.02040. PMID: 34890242; PMCID: PMC8769148.

[12] Fatahzadeh M. Kaposi sarcoma: review and medical management update. Oral Surg Oral Med Oral Pathol Oral Radiol 2012;113:2-16. doi: 10.1016/j.tripleo.2011.05.011.

[13] Brambilla L, Romanelli A, Bellinvia M, et al. Weekly paclitaxel for advanced aggressive classic Kaposi sarcoma: experience in 17 cases. Br J Dermatol 2008;158:1339-44. doi: 10.1111/j.1365-2133.2008.08517.x.

[14] Saville MW, Lietzau J, Pluda JM, et al. Treatment of HIV-associated Kaposi's sarcoma with paclitaxel. Lancet 1995;346(8966):26-8. doi: 10.1016/s0140-6736(95)92654-2.

[15] Di Lorenzo G, Kreuter A, Di Trolio R, et al. Activity and safety of pegylated liposomal doxorubicin as first-line therapy in the treatment of non-visceral classic Kaposi's sarcoma: a multicenter study. J Invest Dermatol 2008 ;128:1578-80. doi: 10.1038/sj.jid.5701215.

[16] Cooley T, Henry D, Tonda M, Sun S, O'Connell M, Rackoff W. A randomized, double-blind study of pegylated liposomal doxorubicin for the treatment of AIDS-related Kaposi's sarcoma. Oncologist 2007;12:114-23. doi: 10.1634/theoncologist.12-1-114.

[17] Cianfrocca M, Lee S, Von Roenn J, et al. Randomized trial of paclitaxel versus pegylated liposomal doxorubicin for advanced human immunodeficiency virus-associated Kaposi sarcoma: evidence of symptom palliation from chemotherapy. Cancer 2010;116:3969-77. doi: 10.1002/cncr.25362.

[18] Evans SR, Krown SE, Testa MA, Cooley TP, Von Roenn JH. Phase II evaluation of low-dose oral etoposide for the treatment of relapsed or progressive AIDS-related Kaposi's sarcoma: an AIDS Clinical Trials Group clinical study. J Clin Oncol 2002;20:3236-41. doi: 10.1200/JCO.2002.12.038.