Bone Marrow Transplantation (2020) 55:110–116
https://doi.org/10.1038/s41409-019-0644-8

ARTICLE

Incidence and outcome of Kaposi sarcoma after hematopoietic stem cell transplantation: a retrospective analysis and a review of the literature, on behalf of infectious diseases working party of EBMT

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Received: 10 May 2019 / Revised: 18 July 2019 / Accepted: 24 July 2019 / Published online: 21 August 2019

Abstract

The incidence, the clinical characteristics, and the outcome of Kaposi sarcoma (KS) in patients after hematopoietic stem cell transplantation (HSCT) were assessed. During the period 1987–2018, 13 cases of KS were diagnosed, 3 females and 10 males, median age of 50 years, median time from HSCT of 7 months. KS had an incidence of 0.17% in allogeneic and 0.05% in autologous HSCT. HHV-8 was documented in eight of nine tumor tissue samples assessed. The organ involvement was: skin in nine, lymph nodes in six, oral cavity in four, and visceral in three patients, respectively; seven patients had >1 organ involved. Five patients had immunosuppression withdrawn, whereas four and three patients received radiotherapy and chemotherapy, respectively. Eight patients are alive (median follow-up 48 months, range 5–128), whereas five patients died after a median time of 8 months from the diagnosis of KS. However, no death was caused by KS. We conclude that the incidence of KS after HSCT is very low. Although KS can be managed with the reduction of immunosuppression, visceral forms may require chemotherapy and/or radiotherapy. The low prevalence of KS indicates that screening for HHV-8 serology and surveillance for HHV-8 viremia are not indicated in HSCT patients.

Introduction

Kaposi sarcoma (KS) is an angioproliferative neoplastic disease described for the first time by Kaposi in 1871, which rose to the fore in 1981 when the epidemic increase of cases among young homosexual men contributed to define the syndrome of acquired immunodeficiency caused by human immunodeficiency virus [1]. Subsequently, in 1994, a potent oncogenic DNA virus was found in KS tumor cells, which was surnamed Kaposi Sarcoma Herpes Virus (KSHV) or human herpes virus 8 (HHV-8) [2]. The worldwide prevalence of KS is variable and follows the

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prevalence of HHV-8 infection, this being higher and involving more than 50% of population in central Africa where KS is the second more frequent pediatric tumor after Burkitt lymphoma [1]. In developed countries with low prevalence of HHV-8 infection, KS occurs mainly in patients who are severely immunocompromised especially after solid organ transplant (SOT) where the risk to develop KS is 400–500 folds higher than that of general population [3]. Also hematopoietic stem cell transplant (HSCT) is risk factor for KS considering that HHV-8 infection has a prevalence up to 15% but until now only few cases were reported [4]. The aim of this retrospective study was to assess the incidence, the clinical characteristics, and the outcome of KS in patients after HSCT among EBMT centers: moreover, a summary of published literature is presented and discussed.

Methods

The study was approved by scientific board of Infectious Diseases Working Party of EBMT. The cases of KS were identified on the EBMT patient data base (ProMISe); moreover, a letter was sent on two-round basis (November 2017 and April 2018) to all 569 EBMT centers inviting to notify any proven case of KS. The main demographic and clinical characteristics of KS cases (sex, age, underlying disease, type of transplant, type of conditioning, engraftment, therapy of KS, survival, and causes of death) were retrieved from of ProMISe or, if lacking, by a specific case report form sent to participating centers. Descriptive statistics was used by medians, means, and percentages. Considering the rarity of KS and the possibility to underdiagnose KS in less experienced centers that did not ever report cases, the incidence was calculated only for the centers that had cases. The incidence was estimated considering the period from 2004 to 2017 that comprised all cases reported but one. The data presented are as July, 31st 2018.

Moreover, a literature search was performed on PubMed using the key words KS and HSCT (last access as 31st March 2019).

Results

The response rate of centers was 74/569 (13%). Fourteen centers reported 17 patients with KS who were diagnosed from 2004 to 2017, but one case, that was diagnosed in 1987. Four of 17 patients were excluded from the analysis because they had KS diagnosed before undergoing HSCT. Table 1 shows the main demographic and clinical characteristics of the 13 patients with KS after HSCT. Only two of them were previously reported [5, 6]. They were three females and ten males affected by acute leukemia (6), severe aplastic anemia (3), lymphoma (3), and myelodysplastic syndrome (1), who developed KS after allogeneic HSCT in ten cases (sibling donor 4, volunteer unrelated donor 3, related mismatched donor 2) and autologous HSCT in three cases. Two patients had an HIV-related lymphoma (patients 11 and 12 in Table 1). The median age at KS diagnosis was 50 years (range 1–61). The interval of time between HSCT and the diagnosis of KS was 7 months (range 2.7–61). The search for HHV-8 in tumor tissue was done in nine cases and HHV-8 was documented in eight out of nine. The search for HHV-8 in the blood by PCR was performed in four KS patients and in three of them it was positive although viral load was not specified. Considering the number of transplant procedures performed in the participating centers in the period from 2004 to 2017 where 12 of 13 KS cases were diagnosed, the incidence of KS was 0.17% in allogeneic transplantations (9/5345), 0.05% in autologous transplantations (3/5857), and 0.11% (12/11202) in the whole group. The organ involvement was: skin in nine patients, lymph nodes in six patients, oral cavity (gingiva, tongue, oral mucosa, Waldeyer ring, or pharynx) in four patients, and visceral (lung 2, stomach–oesophagus 1) in three patients. Seven patients had two or more organs or apparatus involved. Five patients had immunosuppression withdrawn, four patients received radiotherapy (one of them combined with chemotherapy), and three patients received chemotherapy (doxorubicin 1, vinblastine 1, doxorubicin–bleomycin–vincristine 1). Five patients received also antiviral treatment: ganciclovir and interferon 1, ganciclovir and foscarnet 2, foscarnet and acyclovir 1, ganciclovir 1.

Eight patients (62%) are alive after a median follow-up of 48 months, range 13–128, whereas five patients (38%) died at a median time of 8 months, range 0.5–12. The cause of death was infection in three patients, and relapse/progression of underlying disease in two patients. One patient died during treatment for KS for bacterial sepsis (patient 6), whereas two patients with HIV-related lymphoma had a progression of the underlying disease after KS (patients 11 and 12).

Literature search

As 31st March 2019, 19 cases of KS after HSCT were found on PubMed search [4–22]. Table 2 summarizes the main demographic and clinical data. They were 14 males and 5 females, median age 46 years (range 7–69), affected by malignant lymphoprolipherative or myeloproliferative diseases in 15 cases and non malignant diseases in 4 cases who received an allogenic HSCT in 16 cases and an autologous HSCT in 3 cases (2 purged). KS was diagnosed at a median time from HSCT of 7 months (range 3–27) and most of the allogenic HSCT patients were on

Table 1 Summary of cases Kaposi sarcoma retrieved from EBMT data base

Patient number	Age (years)/ sex	Underlying disease	Type of donor HSCT	Conditioning regimen	GVHD, score, time from HSCT	Onset of KS after HSCT (months)	Sites involved	Therapy of KS, time from HSCT	Antivirals	Outcome, follow-up (months from HSCT),
1	16/M	SAA	MUD	Fludara-TT-L-PHAM	Acute: 0–I, chronic: no	9.4	Lymph nodes	R/W IS	no	Alive, 128
2	35/F	AML	Allo-sib	TBI, Cy	Acute: 0–I, +14 days, chronic: limited, +13 months	8.4	Skin, palate/oral mucosa, GI tract	RT	GCV, IFN	Died of interstitial pneumonitis, 8
3	7/F	ALL	Allo-sib	TBI, etoposide	Acute: II–IV, +16 days, chronic: no	4.8	Lymph nodes, pharynx, oral mucosa, Waldeyer ring	R/W IS	no	Alive, 13
4	1/M	MDS	Allo-sib	BU, Cy,	Acute: 0–I chronic: no	61.2	Skin, lymph nodes	n.r	no	Alive, 79
5	57/F	ALL	Allo-sib	Fludara, L-PHAM	Acute: II–IV, +23 days, chronic: extensive, + 7 months	5.6	skin	n.r	Fos, ACV	Alive, 108
6	43/M	AML	Related MM	Fludara-TT-L-PHAM, ATG	Acute: II–IV, +170 days Chronic: extensive, +6 months	3	Skin	Doxo, VCR, Bleo, RT	GCV, Fos	Died of sepsis, 8
7	52/M	SAA	Allo-sib	BU, Fludara	Acute: 0–I, chronic: no	26.3	Skin, lymph nodes, lungs	RT	no	Alive, 47
8	56/M	SAA	MUD	Fludara, ATG	Acute: 0–I chronic: no	4.8	Gums	R/W IS	no	Died of *Pseudomonas sp* sepsis, 0.5
9	11/M	AML	Related MM	Cyclophoshamide,	Acute: II–IV, +228 days, chronic: extensive, n.a.	15.7	Skin, lungs, bone marrow	R/W IS, Doxo	GCV	Alive, 125
10	61/M	Lymphoma	auto	Ara-C, etoposide, BCNU, L-PHAM	n.a.	n.r.	skin	n.r	no	Alive, 48
11	60/M	Lymphoma (HIV positive)	auto	Ara-C, CCNU, etoposide, L-PHAM	n.a.	12.6	Skin, lymph nodes	VBL	no	Died of lymphoma, 12
12	50/M	Lymphoma (HIV positive)	auto	Ara-C, CCNU, etoposide, L-PHAM	n.a.	2.7	Skin	R /W IS	GCV, FOS	Died of lymphoma, 7
13	53/M	ALL	MUD	TBI, Cy, Fludara, ATG	Acute: II–IV, +97 days chronic: no	7.4	Neck lymph nodes, tongue	RT	No	Alive, 33

HSCT hematopoietic stem cell transplantation, *GVHD* graft versus host disease, *KS* Kaposi sarcoma, *SAA* severe aplastic anemia, *AML* acute myeloid leukemia, *ALL* acute lymphoblastic leukemia, *MDS* myelodysplastic syndrome, *HIV* human immunodeficiency virus, *BU* busulfan, *Fludara* fludarabine, *Cy* cyclophosphamide, L-PHAM, melfalan, *TT* Thiotepa, CCNU, lomustine, BCNU, carmustine, *TBI* total body irradiation, *ATG* antithymocyte globulin, *allo-sib* sibling allogeneic transplant, *MM* mismatched, *MUD* marrow unrelated donor, *IS* immunosuppression, *R/W IS* reduction or withdrawal of immunosuppression, *Doxo* doxorubicin, *VCR* vincristine, *bleo* bleomycin, *VBL* vinblastine, *RT* radiotherapy, *Fos* foscarnet, *GCV* ganciclovir, *ACV* aciclovir, *n.r* not reported, *n.a* not applicable

Table 2 Summary of cases Kaposi sarcoma diagnosed after hematopoietic stem cell transplantation reported in the medical literature

Author, year (Reference)	Age (years) /sex	Diagnosis	Type of HSCT	Onset KS after HSCT (months)	Sites involved	Immunosuppressive therapy at KS diagnosis	GVHD at diagnosis of KS	Therapy of KS	Response to therapy for KS	Outcome, follow-up time from KS
Porta, 1991 [7]	7/M	NHL	Auto (purged)	5	Supraclavicular and mediastinum LN	None	n.a.	IFN, excision, RT	Responded	Alive, 13 months
Helg, 1994 [8]	49/F	ID	Allo	8	Skin	steroids	No	Withdrawal IS, RT	Responded	Died of AML, n.k.
Gluckman, 1995 [9]	N.R./M	AML	Allo (T-depleted)	8	Diffuse[a]	n.r.	Chronic	IFN, RT, DLI	Progressed	Died of KS, 7 months
Vivancos, 1996 (10)	55/M	MM	Auto (purged)	4.5	Diffuse[a]	n.a.	n.a.	Bleomycin, etoposide, IFN	Progressed	Died of KS, n.k.
Erer, 1997 [11]	26/M	MD	Allo	24	Skin	Cyclosporin, steroids	Chronic	Withdrawal IS	Responded	Alive, >60 months
De Medeiros, 2000 [12]	46/F	CML	Allo	27	Skin	Prednisone, azathioprine	Chronic	Withdrawal IS, RT thalidomide, topical retinoids	Responded	Alive, >6 months
Tamariz-Martel, 2000 [13]	7/F	SCD	Allo	9	Diffuse[a] (skin, oral mucosa, tongue, cervical lymph nodes)	Cyclosporin, steroids, ATG	Chronic	Withdrawal IS	Progressed	Died of KS, 4.9 months
Palencia, 2003 [14]	55/F	AML	Allo	9	skin	Cyclosporin, steroids	Chronic	Withdrawal IS	responded	Alive, 6 months
Bruno, 2006 [15]	62/M	MM	Allo	3	Skin	Cyclosporin, steroids	Acute	Withdrawal IS	Responded	Alive, 5 months
Marco de F, 2010 [16]	57/M	AML	Allo	19	Diffuse (skin oral mucosa)	Cyclosporin, tacrolimus, steroids, ECP	Chronic	Switch to rapamycin	Responded	Alive, 2 months
Sala, 2011 [5]	10/M	AML	Allo	15	Diffuse[a] (multiple lymph node sites, tonsils, oral mucosa, lungs)	Cyclosporin, azathioprine, steroids	Chronic	withdrawal IS, pegylated doxorubicin	Responded	Alive, 34 months
Ye, 2011 [17]	33/M	SAA	Allo	6.6	Diffuse[a] (multiple lymph node and skin sites, exophthalmos	Cyclosporin	No, but graft rejection	Excision	Progressed	Died of KS, 1 month
Avivi, 2011 [18]	46/M	AML	Allo (T-depleted)	6	Diffuse[a] (multiple skin sites, oropharynx, lungs, liver)	Steroids	Chronic	Withdrawal IS, IFN, doxorubicin	Progressed	Died of KS, 1 month
Abbas, 2012 [19]	8/M	AML	Allo	8	Diffuse[a] (skin, gingival, lungs)	Cyclosporin, steroids	Chronic	Withdrawal IS	Responded	Alive, 12.5 months
Deauna-Limayo, 2013 [4]	69/M	AML	Allo	9	Lymph nodes (multiple sites)	Tacrolimus, steroids	Chronic	Withdrawal IS	Responded	Alive, 13 months
Guo, 2013 [20]	13/M	ALL	Allo	7	Right eye (cornea, conjunctiva)	n.r.	n.r.	Excision, transplantation of cornea and sclera	Responded	Alive, >6 months
Heyrman, 2016 [6]	61/M	LNH	2nd Auto	n.r.	Multiple skin sites	n.a.	n.a	Wait and see	Responded with the increase of CD4 + count	Alive, 9 months
Innes, 2017 [21]	58/M	PTCL	Allo	22	Skin (multiple sites)	Steroids	Chronic	Chemotherapy (not specified)	Progressed	Died of KS and sepsis, n.k.
Ramzi, 2018 [22]	44/F	AML	Allo	4	Skin	Cyclosporin	no	Withdrawal IS, cryotherapy	Responded	Alive, 32 months

HSCT hematopoietic stem cell transplantation, *KS* Kaposi sarcoma, *GVHD* graft versus host disease, *AML* acute myeloid leukemia, *ID* immunodeficiency, *LNH* non-Hodgkin Lymphoma, *MD* microdrepanocytosis, *SAA* severe aplastic anemia, *SCD* sickle cell disease, *CML* chronic myeloid leukemia, *MM* multiple myeloma, *ALL* acute lymphoblastic leukemia, *PTCL* peripheral T cell lymphoma, *IS* immunosuppression, *IFN* interferon, *RT* radiotherapy, *n.a* not applicable, *n.r.* not reported, *n.k* not known

[a]Diffuse: >1 organ or apparatus or visceral involvement

immunosuppressive therapy for acute or chronic GVHD. Diffuse involvement defined as >1 organ or apparatus or visceral involvement was present in nine patients. The treatment was based on withdrawal of immunosuppression in 11 cases, chemotherapy in 4 cases, radiotherapy in 4 cases, excision in 2 cases, and other various measures (cryotherapy, thalidomide, interferon, donor lymphocyte infusion, conversion to rapamycin). KS responded to the treatment in 13 of 19 cases and 12 of them were alive at the time of the publication of the reports whereas 7 patients died, and in 6 of them the cause of death was the progression of KS.

Discussion

This is the first comprehensive study aiming at defining in detail the epidemiology of HHV-8 related KS in HSCT recipients. Although the response rate was slightly inferior than other surveys conducted by IDWP [23], the results confirm that KS is a very rare complication after HSCT. In fact, only 13 cases of KS were diagnosed and recorded in the registry over a large period of observation spanning from 1987 to 2017. Considering the period 2004–2017, the incidence of KS in the centers that had cases was only 0.05% for autologous and 0.17% for allogeneic HSCT, respectively. These findings are in line with the fact the only 19 cases of KS after HSCT were published so far, all as single case reports. KS occurrence is strictly related with HHV-8 infection in HIV patients and this association is confirmed by the oncogenic properties of HHV-8 in vitro studies and by its association in KS in SOT patients. In this study, HHV-8 in tumor tissue was detected in almost all patients in whom the search was performed. Despite that, no precise risk factors were identified for KS after HSCT although most of the cases diagnosed after allogenic HSCT occurred in patient severely immunodepressed due to treatment of chronic GVHD; moreover, the three cases described after autologous HSCT included two purged autologous HSCT and a case after a second autologous HSCT in a patient heavily treated [6]. The fact that CMV infection of previously HHV-8 infected human fibroblasts can reactivate the viral lytic cycle of HHV-8 in vitro and that HHV-8 viremia and KS can develop together with CMV infection suggests a potential role of CMV especially if combined with immunosuppression for GVHD prophylaxis [15]. Besides KS, HHV-8 infection has been associated anecdotally with other transplant complications, such as fever, hepatitis, skin rash, and bone marrow failure as other herpes virus [24] but the real prevalence of these events is still a matter of investigation. Currently, there is no indication to use HHV-8 viremia to guide a preemptive modification of immunosuppression [25]. Previous studies showed that the seropositivity for HHV-8 is 9.5% in blood donors, 14.5% in bone marrow donors, 10% in transplant recipient and that seroconversion occurs in 15% of pre-HSCT seronegative recipients [26, 27]. In SOT setting, the seroprevalence for HHV-8 was 18% in recipients and 4% in donors, being the difference influenced by geographical origin aerea and age. In the same study, HHV-8 viremia was found in 25% of seronegative recipient with seropositive donor (HHV-8 primary infection) and in 2.1% of seropositive recipient with seronegative donor (HHV-8 reactivation) whereas in seropositive SOT recipients, the overall incidence of KS was 2.1% [28]. Due to the variable and in general low seroprevalence for HHV-8 in European countries and the very rare occurrence of KS in HSCT patients, this study confirms previous issued guidelines that did not recommend the serological testing of the donor/recipient pair and the prospective monitoring of HHV-8 viral load in the first months after transplant as it is recommended for other herpes virus such as CMV and EBV [25]. Conversely, the determination of HHV-8 viremia can be useful in patients with suspected skin, lymphadenopathy, mucosal, or deep organ lesions together with biopsy, or to follow the response to KS therapy.

The main therapeutic measure for KS is the withdrawal of immunosuppression both for superficial and visceral involvement in order to allow the host immune recovery whereas small, limited, or superficial lesions may benefit of complete surgical excision. The treatment with chemotherapy or radiotherapy is usually considered in a patient with diffuse visceral involvement or not responsive to withdrawal of immunosuppression because of the risk of inducing an excess of marrow and mucosal toxicity in an HSCT patient. In patients with overt GVHD or at higher risk of GVHD flare, who need the continuation of immunosuppression the switching from calcineurin inhibitors (CNIs) such as tacrolimus and cyclosporine to sirolimus may represent an option. In fact, CNIs favor KS progression by upregulation of vascular endothelial growth factor, whereas sirolimus has antiangiogenic properties by inhibiting mammalian target of rapamycin. There are case reports in SOT and HSCT patients showing that CNI/sirolimus conversion was effective in inducing the regression of KS [4, 15]. Five KS patients of our study received also antiviral therapy with ganciclovir, foscarnet, or interferon. Cidofovir, a potent inhibitor of herpes virus DNA polymerase, was effective in resolving KS lesions in HIV and non-HIV patients but not in all cases [29, 30]. The fact that KS tumor cells are latently infected by HHV-8 and do not express lytic genes may explain the lack of efficacy of antivirals as sole treatment for HHV-8-KS [31]. In our series, including seven patients with multiple site involvement and three patients with visceral involvement, only one death was related to KS treatment (bacterial sepsis in patient 6, while after chemotherapy for KS) whereas two patients with

HIV-related lymphoma had a progression of the underlying disease after KS diagnosis. This outcome is better than that reported in previously where five of seven patients with visceral involvement died of KS [4]. The predominance of KS forms limited to skin and lymph nodes and the treatment of visceral forms with chemotherapy or radiotherapy may explain this favorable outcome.

In conclusion, the incidence of KS after HSCT is very low and most of cases can be managed with the reduction of immunosuppression or, alternatively, with the conversion from CNIs to sirolimus but the visceral forms may require chemotherapy alone or even combined with radiotherapy. HHV-8 latent infection is implicated in driving the development of KS after HSCT but the low prevalence of infection suggests that HHV-8 serology and viremia are indicated only in suspected cases.

Funding P.J. (Pilsen Czech Republic) was supported by the grant AZV NV18–03–00277 of the Ministry of Health.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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