short review

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HIV-triggered lymphoma

Bernd Lorenz Hartmann · Michèle Desiré Atzl

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Summary Treatment outcomes of AIDS-related lymphomas (acquired immune deficiency syndrome, ARL) have improved, nearing those reported for HIV-negative (human immunodeficiency virus) cohorts; recommended treatment protocols are herewith presented. The diagnostic approach to lymphoma in patients with HIV should include screening for and treatment of concomitant infections attributed to HIV-related immunosuppression. Appropriate antiretroviral treatment with adjustments for potential drug interactions should be initiated urgently, and recommended antibiotic, antifungal and antiviral prophylaxis commenced.

Keywords HIV · AIDS · Lymphoma · Treatment · HAART

Take home message

Treatment regimens for lymphoma in patients with HIV (human immunodeficiency virus) are principally similar to those recommended for patients without HIV. By introducing and maintaining appropriate antiretroviral therapy, as well as adding applicable prophylaxis regimens, maximum safety may be achieved.

Introduction

In the era of cART (combination antiretroviral treatment), AIDS-related (acquired immune deficiency syndrome) malignancies still occur with excess prevalence [1, 2], with lymphoma still the leading cause of cancer-related death among patients infected with HIV [3]. On the other hand, treatment outcomes of ARL (AIDS-related lymphomas) have improved, nearing those for reported HIV-negative cohorts [4–6]. We review the current trends in managing lymphoma in patients with HIV infection.

Dr. B. L. Hartmann (✉) · M. D. Atzl
Department of Internal Medicine, Haematology and Medical Oncology, LKH Feldkirch, Feldkirch, Austria
Bernd.Hartmann@lkhf.at

Managing lymphoma in patients with HIV

All patients presenting with lymphoma must be screened for HIV infection. The general management of lymphoma disease in the HIV population and those not infected with HIV are comparable. However, the haematological malignancy in combination with the underlying HIV disease and chemoimmunotherapy will lead to more severe immunosuppression. Historically, due to the fear of worsening the existing immune suppression with concurrent chemotherapy, cortisone treatment and anti-CD20 agents, less intensive chemotherapeutic regimens were used. Implementation of a full lymphoma treatment regimen followed by administration of G-CSF (granulocyte colony-stimulating factor) is now recommended in all HIV-infected patients treated with chemotherapy [7]. Also, specifically referring to the HIV-positive individual, PET (positron emission tomography) results should be interpreted with caution. False-positive results may occur [8, 9], for example with reactive lymphadenopathy in some infective processes like syphilis and tuberculosis.

Specific lymphomas

Diffuse large B-cell lymphoma

Diffuse large B-cell lymphoma (DLBCL) is the most common subtype of lymphoma in adults independent of the HIV-status. It accounts for 31% in HIV-negative [10] and 45% in HIV-positive lymphomas [11]. The

combination of chemotherapy and immunotherapy in the R-CHOP regimen (rituximab, cyclophosphamide, doxorubicin, vincristine and oral prednisone) is standard of care for the immunocompetent patient. In HIV patients, the only phase III trial comparing CHOP vs R-CHOP showed a better complete remission (CR) rate for the addition of rituximab (57.6% vs 47%), but that did not improve the clinical outcome. This was due to an increase in infectious deaths, particularly in those individuals with CD4+ lymphocyte counts less than 50 cells/µl [12]. In severely immunocompromised individuals, one should consider delaying rituximab until some immune recovery has occurred. The R-EPOCH regimen (rituximab, etoposide, prednisone, vincristine, cyclophosphamide and doxorubicin) is highly efficient in ARL patients with CR rates of 73% [13]. Nevertheless, a randomised prospective trial compared R-CHOP with DA (dose-adjusted) R-EPOCH in immunocompetent patients and found no difference in effectivity [14].

Burkitt's lymphoma

Burkitt's lymphoma (BL) represents between 10–35% of HIV-associated non-Hodgkin's lymphoma (NHL) cases [11, 15]. Treatment of BL in HIV-negative patients is guided by the German Multicentre Study Group on Adult Acute Lymphoblastic Leukaemia (GMALL) protocol. It has also been shown to be very effective in the HIV-infected patient, with a CR rate of 80% and a 4-year overall survival (OS) of 72% [16]. Other effective regimens are CODOX-M/IVAC (cyclophosphamide, doxorubicin, vincristine, methotrexate, etoposide, ifosfamide, cytarabine) or HyperCVAD/HD-MTX (hyper-fractionated cyclophosphamide, vincristine, doxorubicin, and dexamethasone plus high-dose methotrexate). CR rates over 90% could be achieved by these regimens; 3-year OS was 77% and 2-year OS was 78% [17, 18]. An alternative regimen is SC-EPOCH-RR (short-course etoposide, prednisone, vincristine, cyclophosphamide, and doxorubicin with dose-dense rituximab), with no progression of disease and OS of 100% and 90% respectively during a follow-up time of 73 months reported [19].

Primary effusion lymphoma

Primary effusion lymphoma (PEL) only accounts for 4% of HIV-associated NHL [20]. Clinically, it presents as a malignant lymphomatous effusion in body cavities like the pleural space, peritoneal cavity or pericardium. Diagnosis is made by identifying malignant cells, characterizing by immune phenotyping, and finally detecting the presence of pathognomonic HHV8 (human herpesvirus 8) infection. PEL cells may express CD45, CD30 and CD38, but have no typical B or T cell markers. Epstein–Barr virus (EBV) co-infection is commonly diagnosed (60–90% of cases), although its role in the pathogenesis of PEL is not clear [21, 22].

There have been no randomised controlled trials. Data regarding the use of (DA) EPOCH can be extrapolated from the study of the National Cancer Institute with AIDS-associated aggressive lymphoma [23]. A case report for this regimen in combination with ART describes tolerability and effectivity [24]. In a retrospective analysis, a CHOP-like regimen was evaluated in eight patients, omitting prednisone to prevent exacerbation of Kaposi sarcoma. A CR rate of 42% and median OS of 6 months was described [25]. PEL is rarely CD20 positive; when it is expressed, treatment should incorporate rituximab [26, 27].

Relapse is frequent and expected within 6–8 months after first-line therapy [4]. When this occurs, autologous stem cell transplantation may be considered [28, 29].

Targeted therapy strategies are under investigation. These include bortezomib, a proteasome inhibitor, brentuximab vedotin (BV), a CD30 antibody, daratumumab, an antibody targeting CD38 [30–32], and antiviral agents valgancyclovir [33] and cidofovir [34]. For unfit patients, a palliative talc pleurodesis may avoid repeated thoracentesis.

Hodgkin's lymphoma

A German multicentre study of 108 patients with HIV infection and Hodgkin's lymphoma (HL) showed that stage- and risk-adapted treatment is feasible and effective [35]. In this study, patients with early favourable HL, defined as Ann Arbor stage IA/B or IIA/B without risk factors (large mediastinal tumour, extranodal involvement, three or more lymph node areas involved), received two courses of doxorubicin, bleomycin, vinblastine, and dacarbazine (ABVD) plus 30 Gy of involved-field (IF) radiation. Patients with early unfavourable HL (stage IA/B or IIA/B and at least one risk factor) received four courses of BEACOPP baseline (bleomycin, etoposide, adriamycin, cyclophosphamide, oncovin, procarbazine, prednisone) plus 30 Gy of IF radiotherapy to sites of initial bulky disease (those at least 5 cm in diameter), or residual tumour of 2 cm in diameter. However, the German Hodgkin Study Group HD11 trial, which excluded HIV-infected patients, found four courses of ABVD plus 30 Gy IF radiation equal to 4 cycles of BEACOPP and 30 Gy IF radiotherapy in early unfavourable HL, and was associated with less toxicity [36]. It is not known if this can be extrapolated for populations with HIV infection.

For advanced HL, treatment entailed six to eight courses of BEACOPP baseline. After the completion of chemotherapy, sites of initial bulky disease (those at least 5 cm in diameter) and residual tumour larger than 2.5 cm in diameter received 30 Gy of irradiation. The 2-year overall survival rate was 90.7% with no significant difference between early favourable (95.7%), early unfavourable (100%), and advanced-stage HL

(86.8%). Eleven patients (11%) died, and treatment-related mortality was 5.6% [35].

BV and the programmed cell death protein 1 (PD-1) blocker nivolumab are approved for the treatment of HL and reported equally active and feasible in HIV-infected HL patients [37, 38]. Serious drug interactions between BV and ritonavir, cobicistat or other CYP3A inhibitors can be expected.

Plasmablastic lymphoma

Plasmablastic lymphoma (PBL) accounts for less than 3–12% of all HIV-related lymphomas [11]. The expression of CD38, CD138, IRF4/MUM1 and the lack of B-cell markers like CD19 und CD20 is characteristic [39], but the cell of origin has not been identified [40]. This rare subtype of DLBCL is found, but not limited to HIV-infected and otherwise immunocompromised persons [41]. CHOP or CHOP-like regimens are most widely used. A report from a single centre in abstract form suggested superiority of EPOCH to CHOP in OS (17 months vs 7 months respectively) [42]. Bortezomib in combination with chemotherapy shows improved effectiveness in combination with a CHOP or EPOCH regimen [43, 44].

Autologous stem cell transplantation

In the era of highly active antiretroviral therapy, autologous transplantation can be done safely, with similar incidence of relapse, overall survival, and progression-free survival, compared to that achieved in HIV-negative patients. There may, however, be a slightly higher rate of early non-relapse mortality associated with bacterial infections [45, 46].

Managing HIV in the lymphoma patient

Evidence supports concomitant treatment of HIV infection during treatment of lymphoma and has become standard of care. Patients who still take antiretroviral combinations that include zidovudine should be switched to a non-myelosuppressive combination, as newer regimens have little cytotoxicity. There is seldom reason to delay or interrupt ART, as even difficulty in swallowing would allow switching to tablets that may be crushed. Potential drug interactions should be reviewed, and necessary ART changes made to avoid these: one can expect to have to avoid strong CYP3A4 inducers such as cobicistat and ritonavir. Viral load testing should be repeated at ART switch, and resistance testing done if the HIV viral load exceeds 700–1000 copies HIV-RNA/ml (this may differ depending on which laboratory is used). For patients on a failing regimen, switching may be urgent and an experienced treater from the infectious diseases team can advise on the most appropriate regimen based on a full treatment history.

Concurrent to staging of the lymphoma, it is pertinent to screen additionally for infections associated with advanced immunosuppression, for example pneumocystis pneumonia (PCP) and mycobacterial infection. These must be diagnosed and promptly treated. Furthermore, routine serological screening for viral hepatitis, syphilis and CMV (cytomegalovirus), as well as cryptococcal antigen and aspergillus antigen is strongly recommended.

For patients who are hepatitis B surface antigen positive and/or hepatitis B core antibody positive, an ART regimen also active against hepatitis B—emtricitabine or lamivudine plus tenofovirdisoproxil—should be selected. Discontinuation of these agents may lead to an acute exacerbation of hepatitis B. Also, rituximab may induce hepatitis B reactivation; therefore, monitoring the HBV-DNA levels ideally once a month is recommended [47].

All patients should be started on at least prophylactic treatment for PCP and herpesvirus reactivation. Trimethoprim–sulfamethoxazole (TMP-SMX), taken as one double-strength tablet daily, is recommended as effective prophylaxis against PCP, also providing protection against toxoplasmosis. Acyclovir, valacyclovir or famciclovir is indicated for the prevention of Herpes zoster and Herpes simplex reactivation, not only in advanced immunosuppression, but also, and especially during exposure to immunochemotherapy. Further prophylaxis may be indicated against Mycobacterium tuberculosis: baseline screening with interferon-gamma release assays (IGRAs), such as the QuantiFERON-TB GOLD test, can be performed but should be interpreted with caution, as false negativity may occur in the setting of lymphopenia. If reactive, a standard regimen such as isoniazid (INH) 300 mg with pyridoxine daily for a duration of 9 months can be used.

Normally, primary prophylaxis for mucosal candidiasis is not recommended in the lymphoma patient: however, in our opinion, in the HIV-positive lymphoma patient periods of severe lymphopenia are often complicated by oropharyngeal, oesophageal, and vulvovaginal manifestations and warrants fluconazole prophylaxis. If the treating physician decides against primary candidiasis prophylaxis, routine screening for cryptococcal antigen at HIV/lymphoma diagnosis is recommended and if positive, fluconazole prophylaxis should be started [48]. At times of prolonged neutropenia, antibiotic prophylaxis with ciprofloxacin or levofloxacin must also be added for the period of cytopenia, as is recommended for individuals without HIV infection.

Conclusion

Treatment outcomes for individuals with ARL have improved, nearing those for reported HIV-negative cohorts. DLBCL is the most prevalent ARL, followed by Burkitt's lymphoma. Appropriate treatment strate-

gies are well described and if appropriate antiretroviral therapy is instituted and combined with prophylaxis against AIDS-related complications, comparable safety of treatment can be achieved.

Conflict of interest B.L. Hartmann and M.D. Atzl declare that they have no competing interests.

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