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A misdiagnosed case of cancer leading to delayed HIV diagnosis and B-cell lymphoma: a lesson on the importance of timely HIV testing

Bogusz Jan Aksak-Wąs[1, A] ✉, Daniel Chober[1, B], Laura Lesiewska[1], Jagna Gordziejczyk-Łabuda[2, C], Miłosz Parczewski[1, D]

[1] Pomeranian Medical University in Szczecin, Department of Infectious, Tropical Diseases and Immune Deficiency, Arkońska 4, 71-455 Szczecin, Poland
[2] Independent Public Provincial Hospital in Szczecin, Department of Hematology, Arkońska 4, 71-455 Szczecin, Poland

[A] ORCID: 0000-0003-2092-3737; [B] ORCID: 0000-0002-6775-4152; [C] ORCID: 0009-0006-4930-0073; [D] ORCID: 0000-0003-1869-1084

✉ bogusz.aw@gmail.com

ABSTRACT

Introduction: This case report describes a serious complication of delayed human immunodeficiency virus (HIV) diagnosis in a patient initially misdiagnosed with palliative-stage cancer and subsequently diagnosed with B-cell lymphoma. This situation highlights the diagnostic challenges and treatment implications following HIV diagnosis, and underscores the critical importance of accurate and timely HIV detection in oncology patients.

Case presentation: A 39-year-old man was initially diagnosed with lung cancer based on computed tomography and histopathologic findings. He was treated with 2 lines of chemotherapy over an 8-month period, the neoplastic lung lesions progressed, and he was referred to palliative care. Two years after his initial diagnosis, during the management of an actively bleeding gastric tumor, he was diagnosed with HIV and referred to an infectious disease unit. Immediate antiretroviral treatment was initiated after diagnosis, and further evaluation confirmed the presence of B-cell lymphoma. A retrospective review suggested that the initial cancer diagnosis was likely misinterpreted and was in fact lymphoma, a malignancy commonly associated with HIV. Lymphoma treatment was initiated, and after 1 year, the patient achieved HIV-1 virologic suppression and near-complete remission of all lesions.

Conclusions: This case highlights the consequences of delayed HIV diagnosis, which can significantly alter treatment course and patient prognosis. The initial misdiagnosis of cancer and subsequent ineffective treatment highlights the importance of HIV testing, particularly in patients presenting with malignancy. It also highlights the role of multidisciplinary collaboration, the need for prompt initiation of antiretroviral therapy, and the efficacy of targeted anticancer therapies for HIV-associated malignancies.

Keywords: human immunodeficiency virus (HIV), malignancy, diffuse large B-cell lymphoma (DLBCL), acquired immunodeficiency syndrome (AIDS), lung cancer.

INTRODUCTION

Early diagnosis of human immunodeficiency virus (HIV) infection is of paramount importance for both individual patient care and public health [1]. Timely identification of HIV allows the initiation of antiretroviral therapy (ART), which improves patient outcomes, reduces morbidity and mortality, and decreases the risk of onward transmission [2]. However, despite advances in diagnostic tools, awareness-raising campaigns, and numerous initiatives aimed at reducing late diagnosis, delayed HIV diagnosis remains a significant challenge in clinical practice [3, 4].

Delayed diagnosis of HIV infection can lead to significant complications, not only related to the progression to acquired immunodeficiency syndrome (AIDS), but also to an increased risk of diseases associated with chronic inflammatory dysfunction, such as cardiovascular or metabolic diseases, as well as malignancies [5]. The World Health Organization (WHO) recommends routine HIV screening for all individuals at increased risk of infection [6]. These recommendations are confirmed by both the European AIDS Clinical Society (EACS) [7] and the Polish AIDS Society (PTN AIDS) [8], as well as other national recommendations. Indicator disease-based HIV testing may allow for earlier HIV diagnosis, effectively allowing clinicians to focus on the clinical picture without inquiring about risk behaviors, to the benefit of individual and public health [9]. Despite numerous efforts to improve HIV diagnosis in the early stages of the disease, the European Centre for Disease Prevention and Control (ECDC) estimates that 1 in 7 people with HIV are unaware of their infection [10], underscoring the continued need for vigilance in clinical practice. In addition, late diagnosis of HIV in migrant populations is a significant public health concern, as it can lead to delayed initiation of treatment, increased risk of transmission, and poorer health outcomes. Cultural and linguistic barriers, limited access to health care, and fear of stigma or discrimination may contribute to delayed diagnosis in these communities [11, 12].

In some cases, HIV infection may present with non-specific symptoms or manifestations that mimic other diseases, including malignancies [13]. For example, B-cell lymphomas are a group of malignancies commonly associated with HIV infection and are associated with profound immunologic dysfunction and low CD4 lymphocyte counts [14]. B-cell lymphomas are

also among the diseases that define AIDS illness and therefore may often be a manifestation of a profound immune deficiency of the host. The clinical presentation of these lymphomas can vary, ranging from localized masses to systemic symptoms such as fever, night sweats, and weight loss [15]. In patients with a high clinical suspicion of HIV, it is important to rule out the possibility of an HIV-associated malignancy before making a definitive diagnosis of another disease. Data from a French cohort showed that the incidence of lymphoma in the HIV population decreased significantly after the widespread introduction of antiretroviral treatment: 86.0–42.9 per 10,000 person-years [14].

In people living with HIV, the incidence of malignancies is significantly increased, with AIDS-defining malignancies (ADMs) and non-AIDS-defining malignancies (NADMs) having different profiles. AIDS-defining malignancies, which include Kaposi's sarcoma (up to several thousand times more common), non-Hodgkin's lymphoma (NHL; 70–200 times more common), and invasive cervical cancer (about 5–6 times more common in women with HIV), serve as indicators of progression to AIDS in people with untreated or poorly controlled HIV infection. Conversely, NADMs such as Hodgkin's lymphoma (5–15 times more common), lung cancer (3–9 times more common), and anal cancer (28–60 times more common) have an increased incidence in the era of effective ART, reflecting both increased life expectancy and ongoing immunologic challenges in HIV-infected individuals. The management of these malignancies is complex, influenced by the interplay of anticancer therapies, ART, and altered pharmacokinetics in the HIV-positive population.

In this case report, we present a patient who was diagnosed with cancer, underwent chemotherapy, was referred to palliative care due to disease progression, and was diagnosed with HIV after a suspected secondary cancer – B-cell lymphoma [16]. The diagnosis of cancer in patients with undiagnosed HIV infection can have significant consequences. Inappropriate treatment and delayed initiation of ART may further exacerbate immune dysfunction, increasing the risk of opportunistic infections and other complications. In addition, anticancer therapies may have limited efficacy in the context of uncontrolled HIV infection, leading to suboptimal treatment outcomes [17]. In such cases, ART should be administered promptly and should take precedence over anticancer therapies [7].

This case report highlights the clinical implications of delayed HIV diagnosis and underscores the importance of considering HIV infection in the differential diagnosis of patients presenting with symptoms suggestive of malignancy [18]. We aimed to emphasize the critical role of thorough clinical evaluation, adherence to diagnostic guidelines, and the necessity of HIV testing in patients with known risk factors for HIV infection. We also discuss the clinical management of such cases, exploring the importance of multidisciplinary collaboration, the need for prompt initiation of ART, and the role of targeted anticancer therapies in HIV-associated malignancies [19].

CASE REPORT – COMPLEX PATH TO HIV DIAGNOSIS

Initial cancer diagnosis

A 39-year-old previously healthy Romanian male, permanent resident in Poland, was admitted to the Department of Pulmonology because of deterioration of general condition and fever (3 weeks before admission). No significant disease exposure history worked physically, with no history of stimulant, alcohol, cigarette, or other substance use. Sexual history was not obtained. The epidemiological history was insignificant regarding tuberculosis, no information was collected regarding the injecting drug use or risky sexual behavior. Body mass index (BMI) was 23.8 kg/m² (normal range). Chest radiography revealed multiple disseminated nodules up to 10 mm in diameter, with intrapulmonary and subpleural locations in both lungs (Fig. 1). On lung computed tomography (CT), multiple metastatic foci, most likely originating from the testicle, were described as a differential diagnosis of sarcoidosis.

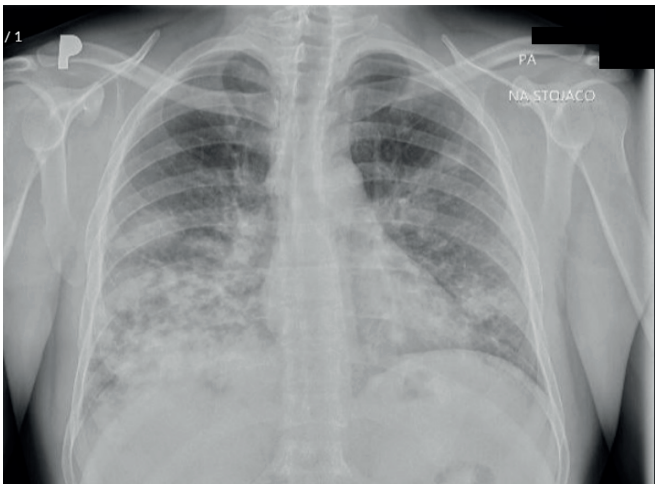

FIGURE 1. Radiograph showing numerous scattered nodules in both lungs, up to 10 mm in diameter, with intrapulmonary and subpleural locations

Multiple disseminated nodules up to 10 mm in diameter, located intrapulmonary and subpleural, were observed in both lungs. These lesions were most intense in the right lower lobe, where the nodules coalesced into larger conglomerates. In addition, confluent ground-glass nodules were observed in the right lower lobe, with associated stroma thickening at the base of the lobe and changes in budding tree type.

Further diagnostic testing did not reveal any testicular lesions or other significant pathology. Fine-needle aspiration biopsy of the supraclavicular node was performed, but only the elements of the reactive lymph node were identified. Tuberculosis was excluded by direct acid-fast bacilli (AFB) testing and sputum molecular assay (RIF-TB, GenXpert). A complete blood count showed no thrombocytopenia or lymphopenia. Bronchoscopy showed mild mucosal thickening on the right side of the S7 spur. In the histopathologic specimen, probabiliter adenocarcinomatosae were identified from cellulae neoplasmaticae malignae;

probabiliter adenocarcinomatosae was initiated based on this result. The patient received 4 cycles of carboplatin, paclitaxel, and dexamethasone (over the course of 4 months) and due to further dissemination of lesions after 6 months of observation, the patient was qualified for 2nd line therapy with 4 cycles of carboplatin, pemetrexed and dexamethasone (over the course of 4 months). Further progression of the neoplastic lung lesions was observed (Fig. 2), which was the reason for discontinuation of anticancer treatment and referral to palliative care 14 months after the initial diagnosis of cancer.

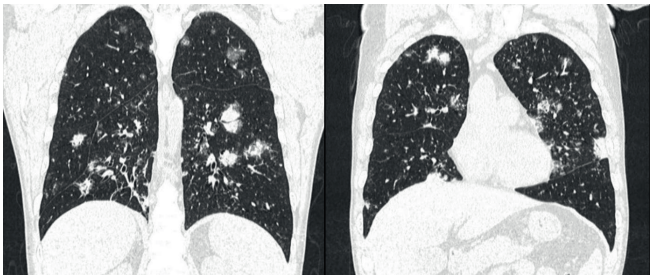

FIGURE 2. Chest radiograph showing advanced stage of pulmonary infiltration with numerous opacities in the lung fields

The chest radiograph shows an advanced stage of pulmonary infiltration with multiple opacities within the pulmonary fields. These opacities vary in size up to 29 mm. Some of these findings were not seen on previous imaging, while others have decreased in size since the previous study. The interstitial pattern is markedly increased, suggesting an interstitial lung disease process. The costophrenic angles were clear, suggesting the absence of pleural effusion. Diaphragmatic contours were smooth and well-delineated, suggesting the absence of diaphragmatic abnormalities. The mediastinum does not appear distended, suggesting no mediastinal mass or lymphadenopathy. Cardiothoracic imaging shows no cardiovascular abnormalities, suggesting a normative cardiologic profile.

From HIV diagnosis onward

Two years after the initial diagnosis of cancer, the patient was referred to the ID department of the Independent Public Provincial Hospital in Szczecin, Poland, following a positive immunoenzymatic HIV test performed during the management of an actively bleeding gastric tumor (the indication for HIV testing was mild leukopenia, oral candidiasis, history of mediastinal lymphadenopathy, and atypical course of cancer in a relatively young patient). On admission, the patient was clinically stable, and oral candidiasis was the only clinically detectable abnormality. Major laboratory abnormalities included normocytic anemia 10.0 g/dL (normal range 14–17 g/dL) and leukopenia 3.77 thousand/μL (normal range 4–10 thousand/μL). Human immunodeficiency virus infection was confirmed by immunoblot assay (Geenius™ Bio-RAID; positive bands for p31, gp160, p24, gp41, and gp 140). Human immunodeficiency virus viral load at diagnosis was 1,150,000 (6.06 log) copies/mL, lymphocyte CD4+ was 95 cells/μL (7%), CD4+/CD8+ was 0.07. The patient was diagnosed late (so-called late presentation into care), which is a diagnosis with a baseline CD4+ lymphocyte count <350/μL or an AIDS-defining illness at the time of diagnosis, with the exclusion of early infection using the RITA algorithm nor a negative HIV test result within the preceding 24 months [20]. Genotypic drug resistance testing (Viroseq 2.8, Abbot Diagnostics) revealed no drug resistance mutations and confirmed HIV-1 subtype A6. Antiretroviral treatment with BIC/F/TAF was started immediately. Serologic tests for toxoplasmosis and hepatitis B and C were negative.

A follow-up gastroscopic examination confirmed an extensive tumor located on the posterior gastric wall, occupying ½ of the circumference, encapsulated, with a central depression covered by necrosis. A protruding vessel was noted in the central part, which was injected with an adrenaline solution (Fig. 3). Esophageal candidiasis was also observed. Histopathological examination of the tumor tissue showed fragments of gastric mucosa with B-cell lymphoma infiltration. Immunochemical staining was as follows: CD20+, PAX5, Bcl6+, Ki67+ 70% of cells, MUM1+ in some cells, c-Myc+ <10% of cells, CD10 (inconclusive), Bcl2 nucleolar reaction, confirming infiltration with diffuse large B-cell lymphoma (DLBCL). Abdominal CT scan showed infiltration of the kidney, spleen, adrenal gland, and liver (Fig. 4).

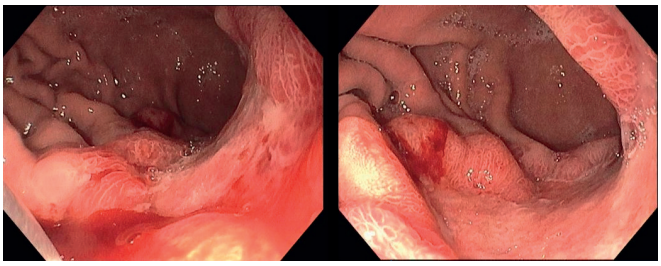

FIGURE 3. Gastroscopy – foci of glycogen acanthosis are visible in the esophagus; on the lesser curvature, there is an extensive infiltration descending to the posterior wall of the stomach, covering ½ of the circumference, with a central depression covered with necrosis – raising suspicion of a neoplastic process

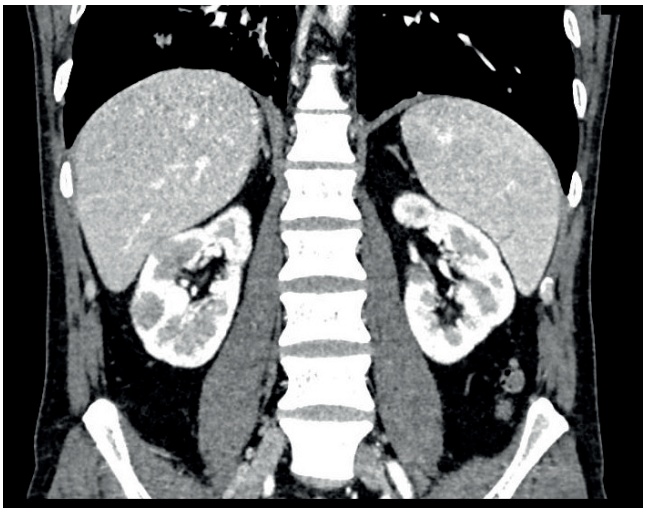

FIGURE 4. Radiological image of the lesions

In radiologic description: in segment 8 of the liver a hypodense lesion of 8 mm was observed, isodense in the equilibrium phase. In addition, a nodule of 8 mm diameter was observed in the right adrenal body. In the parenchyma of both kidneys, there were numerous changes of diameter up to 23 mm of atypical character, changes were isodense to the parenchyma in the native phase and hypodense in the dynamic examination. In the spleen, several small focal lesions of low hypodensity were observed in the venous phase of the examination. Generalized abdominal lymphadenopathy was also observed.

A fine-needle aspiration biopsy of the parapleural lesions was performed to confirm the previous diagnosis of lung cancer, but no neoplastic cells were found. Differential diagnoses included testing for bartonellosis, aspergillosis, pneumocystosis, Kaposi's sarcoma-associated herpesvirus (KSHV), and tuberculosis, all of which were negative. Treatment for DLBCL was initiated according to the standard protocol, modified by the addition of a high dose of methotrexate due to possible central nervous system involvement (3 R-CHOP cycles followed by 5 HdMTX/R-CHOP cycles), without major complications. Further evaluation revealed multiple disseminated hyperintense areas in the subcortical white matter on brain magnetic resonance imaging (MRI). Cerebrospinal fluid molecular testing revealed no *Cryptococcus neoformans*, human polyomavirus 2, John Cunningham virus (JCV), *Aspergillus fumigatus*, or tuberculosis genetic material. Magnetic resonance imaging of the brain revealed lymphoma dissemination or inflammatory foci (Fig. 5).

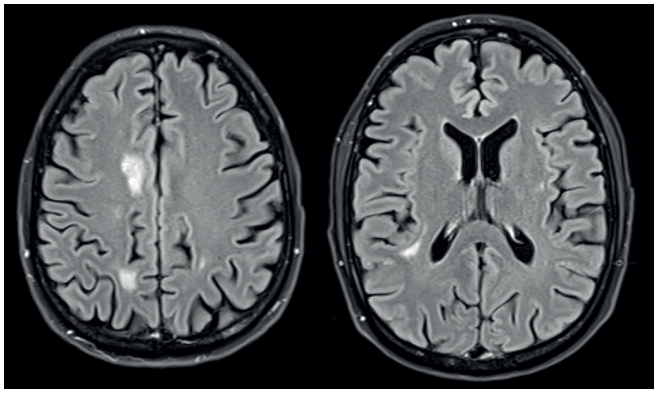

FIGURE 5. Flair and T2 sequences with slight features of diffusion restriction, areas of contrast enhancement; the largest change was observed in the area of the cingulate gyrus on the right side, dimensions 21 x 14 mm, and dissemination of lymphoma or inflammatory foci was suspected

CASE RESOLUTION

The follow-up PET-CT showed partial remission of the lung changes, and a control MRI of the brain showed regression of the lymphoma lesions. One year after HIV diagnosis, the patient's condition was stable, with good general health and well-being. During the 12-month follow-up, the patient gained 10 kg in weight (BMI: 27.5 kg/m^2 – overweight). The patient denied shortness of breath, fever, or pain. Human immunodeficiency virus-1 virologic suppression was achieved after 12 weeks of antiretroviral treatment, and HIV viremia remained suppressed with lymphocyte CD4+ stable at 95 cell/μL (5.7%) CD4/CD8:0.06, no immunological reconstruction was seen.

DISCUSSION

This case is a cautionary tale about the importance of timely diagnosis and the need to test all patients with cancer, including those with NADMs, for HIV. The consequences of delayed diagnosis in this case were disastrous – the patient may have been treated with inappropriate chemotherapy, with tumor progression and inappropriate referral to palliative care. The diagnosis of HIV was made as part of routine diagnostics in the ID department. This case highlights the importance of timely and appropriate HIV testing and the consequences of delays [21].

The initial diagnosis of lung cancer in our patient was based primarily on histopathologic examination (*cellulae neoplasmaticae malignae*, *probabiliter adenocarcinomatosae*) and radiologic findings on CT scans; however, proper immunochemistry was not performed. Even these early CT scans showed mediastinal lymphadenopathy, which should have prompted HIV testing. Only after 2 years of delayed diagnosis, palliative chemotherapy, and gastrointestinal tumor bleeding was the diagnosis of AIDS and B-cell lymphoma confirmed as the underlying clinical conditions.

The prevalence of HIV infection in Poland is relatively low compared to other countries, but there is still a significant proportion of undiagnosed individuals. Men who have sex with men (MSM) represent a high-risk population and are estimated to account for almost half of new HIV infections [21, 22]. Late presentation to care among people living with HIV remains a critical issue with significant implications for both individual health outcomes and public health efforts. In Europe, a significant proportion of HIV-infected individuals are late presenters, with estimates suggesting that approx. 30–50% of newly diagnosed individuals present with a CD4+ count below 350 cells/mm^3 or an AIDS-defining condition at the time of diagnosis, excluding recent HIV acquisition. This scenario is mirrored in Poland, where recent data indicate that approx. 40–50% of new HIV diagnoses fall into the category of late presentation [21, 23]. The consequences of such late presentation are multiple, including increased morbidity and mortality, reduced immunologic reconstitution [24, 25], and increased potential for HIV transmission within the population. This scenario underscores the need for improved screening strategies, public awareness campaigns, and accessible testing facilities to facilitate earlier diagnosis and initiation of treatment.

The diagnosis of HIV-associated malignancies can also be challenging due to their atypical presentation and aggressive clinical course. The most common HIV-associated malignancies include Kaposi sarcoma, NHL, and invasive cervical cancer. In 2001–2005 US data, there were 765 cases per year of Kaposi sarcoma, 1194 cases per year of NHL, and 106 cases per year of invasive cervical cancer in the AIDS population [26]. Diffuse large B-cell lymphoma,

a subtype of NHL, is one of the most commonly observed lymphomas in HIV-infected individuals, accounting for 50% of all NHL cases [27]. The clinical presentation of DLBCL is highly variable and may include pulmonary involvement.

The initial misdiagnosis of lung cancer in this patient underscores the need for clinical care and awareness in cases with atypical and typical presentations. The management of HIV-associated B-cell lymphoma is complex and requires the integration of ART and chemotherapy [28]. The rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone (R-CHOP) regimen is the standard first-line treatment for DLBCL in both HIV-positive and HIV-negative patients. In our patient, the combination of ART and R-CHOP chemotherapy resulted in a favorable clinical response as evidenced by tumor shrinkage and achievement of an undetectable HIV viral load. Immune reconstitution may be delayed in similar cases, both because of advanced HIV disease at diagnosis and, most importantly, because of the immunosuppressive effects of both required and unnecessary chemotherapies. Strategies to improve immune reconstitution in patients with HIV-related malignancies, such as the use of adjunctive immunomodulatory agents, remain an area of ongoing research [17].

In conclusion, this case report serves as a cautionary tale for clinicians, reminding us to maintain a high level of clinical vigilance for HIV infection in patients presenting with symptoms suggestive of malignancy or other conditions [29]. Early diagnosis and appropriate management of HIV infection can significantly improve patient outcomes, reduce the risk of complications, and prevent misdiagnosis of other conditions. By highlighting the potential consequences of delayed HIV diagnosis and cancer misdiagnosis, we hope to contribute to a better understanding of the importance of timely and accurate clinical assessment in the management of these complex patients [30].

REFERENCES

1. Branson B. Current HIV epidemiology and revised recommendations for HIV testing in health-care settings. J Med Virol 2007;79 Suppl 1:S6-10.
2. Cohen MS, Chen YQ, McCauley M, Gamble T, Hosseinipour MC, Kumarasamy N, et al. Prevention of HIV-1 infection with early antiretroviral therapy. N Engl J Med 2011;365(6):493-505.
3. Tucker JD, Bien CH, Peeling RW. Point-of-care testing for sexually transmitted infections: recent advances and implications for disease control. Curr Opin Infect Dis 2013;26(1):73-9.
4. Parczewski M, Rockstroh JK. Late HIV diagnosis: Where we stand and the way forward. HIV Med 2022;23(11):1115-7.
5. Palella FJ Jr, Delaney KM, Moorman AC, Loveless MO, Fuhrer J, Satten GA, et al. Declining morbidity and mortality among patients with advanced human immunodeficiency virus infection. HIV Outpatient Study Investigators. N Engl J Med 1998;338(13):853-60.
6. Guidelines on HIV Self-Testing and Partner Notification: Supplement to Consolidated Guidelines on HIV Testing Services. Geneva: World Health Organization; 2016.
7. EACS Guidelines 2022. https://www.eacsociety.org/media/guidelines-11.1_final_09-10.pdf (21.05.2023).
8. Polish AIDS Society Recommendations. https://www.ptnaids.pl/index.php?option=com_content&view=article&id=20%3Arekomendacje-polskiego-towarzystwa-naukowego-aids&catid=14%3Arekomendacje&Itemid=24&lang=en (3.12.2023).
9. Bull L, Rayment M. HIV-indicator-condition-driven HIV testing: clinically effective but still rarely implemented. Clin Med (Lond) 2016;16(2):175-9.
10. HIV/AIDS surveillance in Europe 2015. European Centre for Disease Prevention and Control. https://www.ecdc.europa.eu/en/publications-data/hivaids-surveillance-europe-2015 (18.06.2023).
11. Suchacz MM, Krankowska D, Cybula A, Kamerys J, Jabłonowska E, Rozpłochowski B, et al. Delayed HIV diagnosis during the COVID-19 pandemic in Poland: A call for targeted HIV testing for those under suspicion of SARS-CoV-2. HIV Med 2022;23(11):1173-83.
12. Kostaki EG, Limnaios S, Adamis G, Xylomenos G, Chini M, Mangafas N, et al. Estimation of the determinants for HIV late presentation using the traditional definition and molecular clock-inferred dates: Evidence that older age, heterosexual risk group and more recent diagnosis are prognostic factors. HIV Med 2022;23(11):1143-52.
13. Gopal S, Patel MR, Yanik EL, Cole SR, Achenbach CJ, Napravnik S, et al. Temporal trends in presentation and survival for HIV-associated lymphoma in the antiretroviral therapy era. J Natl Cancer Inst 2013;105(16):1221-9.
14. Besson C, Goubar A, Gabarre J, Rozenbaum W, Pialoux G, Châtelet FP, et al. Changes in AIDS-related lymphoma since the era of highly active antiretroviral therapy. Blood 2001;98(8):2339-44.
15. Castillo JJ, Bibas M, Miranda RN. The biology and treatment of plasmablastic lymphoma. Blood 2015;125(15):2323-30.
16. Powles T, Imami N, Nelson M, Gazzard BG, Bower M. Effects of combination chemotherapy and highly active antiretroviral therapy on immune parameters in HIV-1 associated lymphoma. AIDS 2002;16(4):531-6.
17. Barta SK, Xue X, Wang D, Tamari R, Lee JY, Mounier N, et al. Treatment factors affecting outcomes in HIV-associated non-Hodgkin lymphomas: a pooled analysis of 1546 patients. Blood 2013;122(19):3251-62.
18. Mounier N, Spina M, Gabarre J, Raphael M, Rizzardini G, Golfier JB, et al. AIDS-related non-Hodgkin lymphoma: final analysis of 485 patients treated with risk-adapted intensive chemotherapy. Blood 2006;107(10):3832-40.
19. Grulich AE, van Leeuwen MT, Falster MO, Vajdic CM. Incidence of cancers in people with HIV/AIDS compared with immunosuppressed transplant recipients: a meta-analysis. Lancet 2007;370(9581):59-67.
20. Kirwan PD, Croxford S, Aghaizu A, Murphy G, Tosswill J, Brown AE, et al. Re-assessing the late HIV diagnosis surveillance definition in the era of increased and frequent testing. HIV Med 2022;23(11):1127-42.
21. Aksak-Wąs BJ, Urbańska A, Leszczyszyn-Pynka M, Chober D, Parczewski M. Clinical parameters, selected HLA and chemokine gene variants associated with late presentation into care of people living with HIV/AIDS. Infect Genet Evol 2022;97:105180.
22. Mocroft A, Lundgren J, Sabin ML, Monforte Ad, Brockmeyer N, Casabona J, et al. Risk factors and outcomes for late presentation for HIV-positive persons in Europe: results from the Collaboration of Observational HIV Epidemiological Research Europe Study (COHERE). PLoS Med 2013;10(9):e1001510.
23. Jabłonowska E, Szetela B, Bielecki M, Horban A, Bociąga-Jasik M, Mularska E, et al. Acquired immune deficiency syndrome (AIDS) and late presentation in Poland – data from Test and Keep in Care (TAK) Polska project. HIV Med 2021;22(5):387-96.
24. Aksak-Wąs BJ, Urbańska A, Scheibe K, Serwin K, Leszczyszyn-Pynka M, Rafalska-Kosior M, et al. Factors influencing immune restoration in people living with HIV/AIDS. J Clin Med 2022;11(7):1887.
25. Aksak-Wąs BJ, Kowalska JD, Ząbek P, Serwin K, Rafalska-Kosior M, Gołąb J, et al. Immune restoration affects 10-year survival in people living with HIV/AIDS. HIV Med 2023;24(3):325-34.
26. Yarchoan R, Uldrick TS. HIV-associated cancers and related diseases. N Engl J Med 2018;378(11):1029-41.
27. Berhan A, Bayleyegn B, Getaneh Z. HIV/AIDS associated lymphoma: Review. Blood Lymphat Cancer 2022;12:31-45.
28. Antinori A, Cingolani A, Alba L, Ammassari A, Serraino D, Palmieri F, et al. Better response to chemotherapy and prolonged survival in AIDS-related lymphomas responding to highly active antiretroviral therapy. AIDS 2001;15(12):1483-91.
29. Engels EA, Biggar RJ, Hall HI, Cross H, Crutchfield A, Finch JL, et al. Cancer risk in people infected with human immunodeficiency virus in the United States. Int J Cancer 2008;123(1):187-94.
30. Clifford GM, Polesel J, Rickenbach M, Dal Maso L, Keiser O, Kofler A, 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(6):425-32.