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Enhancing the quality of life of Africans living with a haematological Cancer

Aliyu Bagudo Ibrahim [1,*], Abdulrahman Yakubu [1], Osaro Erhabor [1], Yazid Bello [2], Ibrahim Kalle Kwaifa [1], Festus Uchechukwu Onuigwe [1], Hauwa Ali Buhari [1], Sani Abubakar [3], Gimba Waziri [4], Lukman Haruna [2], Aliyu Garba Bashiru [1] and Mukhtar Yeldu Mainasara [1]

[1] *Department of Haematology, School of Medical Laboratory Sciences, Usmanu Danfodiyo University, Sokoto, Nigeria.*
[2] *Department of Haematology, Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria.*
[3] *Department of Medical Laboratory Science, Faculty of Allied Health Science Pen Resource University, Gombe State, Nigeria.*
[4] *Department of Medical Laboratory Sciences, University of Maiduguri, Borno State, Nigeria.*

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Abstract

Leukaemia, multiple myeloma, and lymphoma are among the blood neoplasms that affect the blood and blood-forming tissues that make up haematological cancers. However, improving the quality of life for Africans who have a haematological malignancy remains difficult due to a variety of issues, including social, health, economic, and family issues. This review's objective was to identify potential strategies for enhancing the quality of life for Africans dealing with haematological cancer. These strategies include detection, diagnosis and current haematological cancer treatments. If carefully followed, these steps might ensure that Africans with blood cancer have a better life expectancy.

Keywords: Quality; Life; Leukaemia; Lymphoma; Myeloma

1 Introduction

A group of tumours known as haematological malignancies are those that develop when lymphatic or bone marrow cells undergo a malignant change (Arber *et al.,* 2014). 5 By using immunologic, cytogenetic, and molecular genetics techniques, they can be divided into myeloid or lymphoid subtypes depending on their origin, and into acute and chronic subtypes depending on their clinical trajectory (Durosinmi, 2013). The leukemias, lymphomas, and myelomas are among these cancers. Around 6.5% of all cancers were reported to be haematological malignancies in the year 2012 (Ferlay *et al.,* 2008). In developed nations, it is the fourth most common cancer to be diagnosed in both men and women (Hoffbrand and Moss, 2011). Haematological malignancies have an unknown cause.But there are some things that have been shown to make them more likely to happen. These factors include chronic bacterial infections like Helicobacter pylori, immunosuppression or immunodeficiency states, radiation, and chemicals like benzenebenzene (Ocheni and Akenóva, 2004; Onwubuya*et al.,* 2015; Akinbami*et al.,* 2014; Hu *et al.,* 2016; Leuraud*et al.,* 2015; Yoon *et al.,* 2018). Infections with viruses include human immunodeficiency virus (HIV), Epstein Barr virus, and human T-lymphotrophic virus. The formation of these malignancies is assumed to be the result of a complicated interplay between these variables and genetic damage in somatic cells brought on by mutations, dysregulated cytokines, and persistent antigenic stimulation(Luca *et al.,* 2016). Haematologic malignancies' occurrence has been proven to vary with age, gender, location, and histologic subtypes, while cure rates can differ with location and may be influenced by early and correct diagnosis, the availability of treatment, and access to care (Okocha*et al.,* 2015; Kagu*et al.,* 2013).

*Corresponding author: Abdulrahman Yakubu

Haematologic cancers place a significant financial, psychological, and medical burden on patients and their families(Statham and Davis, 2018; Fey, 2017). The price of treating these illnesses is very high. Patients with these disorders are treated through out-of-pocket costs, and as a result, they are unable to start, continue, or finish therapy due to financial limitations. Family income is exhausted for those who followed the suggested regimen and succeeded (Zajacova*et al.,* 2015). The stage of the disease at presentation, the type of the disease, other co-morbidities, the timing of presentation, the stage of the disease at presentation, the availability of qualified personnel, the availability of drugs, their cost, and the availability of palliative and supportive medical care can all affect the outcomes in hematologic malignancies (Howel*et al.,* 2019; Dapkevicture*et al.,* 2019).

2 The cause of haematological malignancy

It is largely unknown how genetic abnormalities come together to cause haematological malignancies. For the majority of disorders, the probability of acquiring a malignancy is influenced by both genetic and environmental variables. For instance, single nucleotide polymorphism (SNP) analysis has shown SNPs in germline genes that predispose a person to B-cell acute lymphoblastic leukemia. Some of these genes code for proteins important in B-cell development (B - ALL). But in some instances, neither a genetic predisposition nor environmental effects are readily obvious.

2.1 Inherited factors

Some hereditary illnesses, including Down syndrome (where acute leukemia develops with a 20- to 30-fold increased frequency), Bloom's syndrome, Fanconi'sanemia, ataxia telangiectasia, and others, have a significantly higher prevalence of leukaemia.Wiskott-Aldrich syndrome, Klinefelter's syndrome, and neurofi-bromatosis. Although the genes that predispose to this higher risk are mostly unknown, there is a slight family tendency in diseases such acute myeloid leukemia (AML), CLL, Hodgkin lymphoma, and non-Hodgkin lymphoma (NHL)(Hoffbrand and Moss, 2011).

2.2 Environmental influences

2.2.1 Chemicals

An unusual cause of myelodysplasia or AML is long-term exposure to benzene. Less frequently, some industrial chemicals and solvents can cause leukaemia.

2.2.2 Drugs

The nitrosoureas (BCNU, melphalan, procarbazine, and others) and other alkylating agents(CCNU) predispose to AML, particularly when radiation is added. Etoposide is linked to an increased incidence of secondary leukaemias, notably those involving balanced translocations of the MLL gene at 11q23.

2.2.3 Radiation

Radiation is leukaemogenic, especially when it hits the marrow. An increase in the prevalence of all types of leukemia (except CLL) in survivors of the atom bomb explosions inJapan.

2.2.4 Infection

A genetic predisposition to acute lymphoblastic leukaemia (ALL) may exist in children. Then, some cases of childhood ALL are brought on by genetic abnormalities that take place during foetal development. According to studies done on identical twins, both twins may be born with the same chromosomal defect, such as t (12; 21). Due to the common placental circulation, it is likely that this spontaneously developed in a progenitor cell that was transmitted from one twin to the other. For this first incident, environmental exposure during pregnancy may be significant. Due to a second transformative event that affects the copy counts of multiple genes, including those involved in B-cell development and important for leukaemogenesis, one twin may develop ALL early (for example, at age 4).The other twin is still healthy or might eventually get ALL. About 10% of newborn children have the TEL - AML1 translocation in their blood, although only 1 in 100 of them go on to mature. Epidemiological studies suggest that the second genetic hit0' within the tumour cell may be caused by an aberrant immunological response to infection, while the exact process is unknown. Children who are socially active, especially those who attend early nursery day care, have a lower prevalence of ALL, whereas those who live in more remote areas and who are less exposed to common diseases in infancy are at a higher risk.

2.2.5 Viruses

Numerous haematological malignancies, including several subtypes of lymphoma, are linked to viral infection. Adult T-cell Leukemia/Lymphoma is caused by the retrovirus human T-lymphotrophic virus type I, albeit the majority of those

who contract the virus do not go on to develop the tumour. Nearly all cases of endemic (African) Burkitt lymphoma, post-transplant lymphoproliferative illness (which occurs after immunosuppressive therapy after solid organ transplantation), and a portion of Hodgkin lymphoma patients have Epstein-Barr virus (EBV) infection. Kaposi's sarcoma and primary effusion lymphoma are linked to human herpes virus 8 (also known as the Kaposi's sarcoma-associated virus). Increased incidence of lymphomas at unusual sites, like the central nervous system, is linked to HIV infection.

2.2.6 Bacteria

Infection with *Helicobacter pylori* has been linked to the development of gastric mucosa B-cell (MALT) lymphoma.

2.2.7 Protozoa

The tropics are home to endemic Burkitt lymphoma, especially in places where malaria is prevalent. Malaria is hypothesized to affect the host immune system and increase the risk of developing tumors as a result of EBV infection(Hoffbrand and Moss, 2011).

3 The Haematological Cancer Mechanism

The accumulation of genetic mutations in cellular genes leads to malignant transformation. *Oncogenes and tumour-suppressor genes* make up the two broad categories of genes that play a role in the onset of cancer.

3.1 Oncogenes

Gain-of-function mutations in commonly occurring cellular genes, known as*Proto-oncogenes.* Proto-oncogenes have a role in a number of crucial cellular activities, frequently in the pathway by which signals from the outside world are brought into the cell nucleus and used to activate genes. When proto-oncogenes become more active or take on a new function, oncogenic variants are created. There are several methods for this to happen, including translocation, mutation, and duplication. Contrary to most solid tumors, haematological malignancies exhibit a high frequency of chromosomal translocations, which is one of their remarkable characteristics. Control of apoptosis is regulated by a subset of proto-oncogenes.

3.2 Tyrosine kinases

These enzymes, which phosphorylate proteins on tyrosine residues, are crucial for intracellular signalling and as cell receptors. A significant number of hematological cancers are caused by mutations of them.

3.3 Tumour - suppressor genes

Loss-of-function mutations in tumour suppressor genes, typically through point mutations or deletions, can result in a malignant transformation. The passage of a cell from the G1 phase of the cell cycle into the S phase or from the S phase to the G2 phase and mitosis are both regulated by tumour suppressor genes. P53, which is altered or inactivated in more than 50% of instances of malignant illness, including many haemopoietic tumours, is the most important tumour suppressor gene in human cancer (Hoffbrand and Moss, 2011).

4 Clonal development

It appears that the development of malignant cells involves a number of steps and the accumulation of mutations along many intracellular pathways.Clonal progression is another characteristic of cancer. During the course of the disease's clinical manifestations, the disease frequently acquires novel traits, which may be accompanied by novel genetic alterations. Sub clone selection may take place during therapy or be a sign of progressing illness. Numerous molecular processes can lead to drug resistance. In one illustration, the cells express a protein that actively pumps a variety of medications to the cells' outside (multidrug resistance) (Hoffbrand and Moss, 2011).

5 Current Methods of Detection and Diagnosis of Blood Cancer

There are a number of laboratory techniques for the detection and diagnosis of Haematological cancer which are explained below:

5.1 Complete Blood Count and Blood film Examinations

When a disease is suspected, a complete blood count (CBC) is typically the first step in the potential diagnosis of haematological malignancies. It is conducted using an automated cell counter, incorporating laser technologies and microfluidics, its automatically measures red blood cells (RBCs), white blood cells (WBCs), and platelets in a volume of blood. Under abnormal conditions, the instrument flags the presence/quantification of abnormal blood cells counts, i.e. blast cells or precursors. Abnormal CBC reports must be confirmed by blood film examinations (Gulati *et al.,* 2013). Blood film examination allow for the determination and observation of cell abnormalities, in terms of cell morphology and aberrant staining characteristics under a microscope (Abraham *et al.,* 2010; Lee, 2018).

5.2 Bone Marrow examination and Histocytochemistry

Bone marrow (BM) biopsy/aspirate analyses are two complementary techniques essential for the diagnosis of some haematological malignancies (Cantadori *et al.,* 2019; Riley *et al.,* 2009). During bone marrow collection, a highly specialized needle collects a sample from the bone, which contains a small amount of BM cells and fluid, whereas a bone marrow biopsy collects a tiny piece of the bone. Different types of diagnostic tests canbe applied to these samples, such as morphological examination, cytochemistry, flow cytometry, cytogenetic analyses, polymerase chain reaction (PCR) and fluorescence *in situ* hybridization (FISH). Cytochemistry evaluates the micro-anatomical biochemical components of cells and organelles so constituents can be localized by different chemical components, and potential cell transformation disease dynamics assessed. While this technique is a preliminary step in deciding a definitive diagnosis, its role has become limited as other more advanced techniques have been developed (Rosati and Gabrielli, 2005; Sucic *et al.,* 2008). Immunohistochemistry is a component of the histological process and identifies specific tissue molecules using commercially available antibodies targeting specific proteins implicated in haematological malignancies (Abraham *et al.,* 2010; Matos *et al.,* 2010).

5.3 Flow cytometry

Flow cytometry (immuno-phenotyping) is an advanced cell counting/quantifying technique. The technique generates qualitative and quantitative data on different cell types; it measures physical and chemical properties of a cell, identifies the maturation stage of cells, cell type and cell lineages.It detects, identifies, and quantitates specific haematological markers such as proteins, enzymes, or antigens expressed on cells. Cytometry data provides accurate quantitative information on specific cell types and groups that can help confirm disease status (Mizrahi *et al.,* 2018; Craig and Foon, 2008). A recent study highlighted the relevance of flow cytometry to haematological malignancies. Expression analysis of the surface immunoglobulin (Ig) light chain, which includes kappa (κ) and lambda (λ) chains, provided evidence for malignant B cell tumour proliferation in patients with persistent lymphocytosis (Paiva *et al.,* 2018).

5.4 Whole chromosome techniques

Cytogenetics (conventional cytogenetics, karyotyping, or chromosome analysis), analyzes chromosomes during metaphase growth when chromosomes are becoming more condense, visible, and distinguishable. Chromosome spreads are analysed using differential staining techniques under a microscope, to help identify and quantify chromosomal numbers, abnormal Morphologies, and chromosomal abnormalities implicated in the aetiology of haematological malignancies (Severson, 2013). Fluorescence *in situ* hybridization (FISH) is also used to detect structural chromosomal abnormalities and minimal residual disease (MRD); the detection of small numbers of neoplastic/leukemic cells that remain during or after treatment, when a patient is in a remission state. The technique detects MRD at very sensitive limits since MRD is the main cause of cancer relapse (Severson, 2013; Jehan *et al.,* 2012).

5.5 Molecular techniques

Molecular genetic techniques have a lot of analytical assays, such as Southern blotting, qualitative/quantitative PCR, and real-time polymerase chain reaction (RT-PCR) (Hoffbrand *et al.,* 2015). The use of Southern blotting techniques in the preliminary diagnosis of haematological malignancies is limited, the more molecular techniques, such as ConventionalPCR and RT-PCR are widely used in diagnostic laboratories (Arya *et al.,* 2005). Because of their several advantages over other diagnostics techniques; tiny amounts (ng) of DNA are required, dividing cells are not used for testing, and the techniques detect many chromosomal abnormalities, such as translocations, deletions, duplications and MRD detection (Sabath, 2004; Staudth, 2003).

5.6 Genome approaches

Whole-genome scanning techniques include microarray analysis, comparative genomic hybridization (CGH), and singlenucleotide polymorphism (SNP) arrays (Dugoff *et al.,* 2016; Tarca *et al.,* 2006). The main principle behind microarrays is the hybridization between two DNAstrands (disease sample and the control). The fluorescently labelled

sample sequences bind to the control and generate a signal that depends on the strength of hybridization between strands. The expression patterns of healthy and diseased genes are compared to genes responsible for a particular disease (Govindarajan *et al.,* 2012). Microarray analysis can simultaneously process thousands of genesin one experiment; therefore, it is a convenient, high-throughput molecular technique, with a rapid turnaround. The technique is preferred over the FISH. In terms of CLL, SNP array profiling has contributed to the identification of deletion chromosomal mutations such as del 15q, del 8p, del 22q, and gain of function mutation, such as 20q and 2p (Malek, 2013). CGH detects the gain or loss of important chromosomal fragments harbouring genes involved in a disease, such as haematological malignancies. SNP analyses are used to detect single nucleotide changes (substitution) between two sequences. They are beneficial in genotyping analyses, distinguish between heterozygosity and homozygosity, the detection of gain or loss mutations, and the identification of acquired somatic uniparental disomy (Hoffbrand *et al.,* 2015). Gene expression profiling using microarray analyses have been used to measure changes in gene expression to better understand regulatory mechanisms. Expression patterns of healthy and diseased genes are compared to study genes responsible for the disease (Severson, 2013).

5.7 Sequencing approaches

Next-generation sequencing (NGS) is a high-throughput sequencing method that rapidly sequences DNA or RNA samples. The method has improved our knowledge and understanding of haematological malignancies, particularly for CLL, where additional gene mutations have been detected in *SF3B1, BIRC3, NOTCH1, MYD88, SAMHD1, DDX3X, BIRC3, BRAF,MED12, FBXW7, XPO1, CHD2* and *KLHL6* (Rodriguez-Vicente *et al.,* 2017). NGS uses many different applications such as whole-genome sequencing, whole-exome, and targeted sequencing.

6 Current treatments of haematological malignancies

6.1 Chemotherapy

Chemotherapy means using cell-killing drugs to destroy cancer cells.Chemotherapy is often given directly into a vein. The drugs can travel around the body in the bloodstream and kill the cancer cells. This is called intravenous (IV) chemotherapy. It's also called having an infusion (a drip).Usually, the chemotherapy is in a bag of fluid with a tube coming from it that goes into a vein in your hand, arm or chest. It can take several hours to receive chemotherapy into a vein in this way, or sometimes more than a day. Many people have chemotherapy as an outpatient – that means you come to hospital for treatment and can go home again afterwards.Chemotherapy can also be given as tablets, sometimes as a course of treatment, or sometimes as a more long-term treatment.Whilst chemotherapy can kill cancer cells, it also damages healthy cells in your body. This is what causes the side effects of chemotherapy.

6.2 Stem cell transplant

A stem cell transplant means replacing the stem cells in your body with new, healthy stem cells. Stem cells are cells at an early stage of development. All blood cells start off as stem cells in the bone marrow. Blood cancer happens when something goes wrong with the development of your blood cells and they become cancerous. A stem cell transplant can be a treatment for some blood cancers because it involves destroying the abnormal stem cells that are producing cancerous blood cells, and giving your body new, healthy stem cells that can make healthy blood cells again. This involves having high doses of chemotherapy to destroy your existing stem cells, and then having the transplant to replenish your bone marrow with healthy stem cells. A stem cell transplant is also used to replace your stem cells if you need high doses of chemotherapy to treat your blood cancer, which as a result damages your bone marrow and stem cells. There are two types of stem cell transplant: 1) Autograft/autologous: where your own stem cells are collected and stored, and given back to you later by transplant. 2) Allograft/allogeneic: where someone else's stem cells (a donor's) are used for the transplant.

6.3 Immunotherapy

Immunotherapy is a way of treating cancer that uses your own immune system to attack the cancer. Any cancer treatment that harnesses the immune system to help it work can be classed as an immunotherapy. Some immunotherapy drugs work by triggering your body's own immune system to find and kill cancer cells. The drug attaches itself to a cancer cell, which makes the cancer cell easier to find for your immune system. Your immune system then attacks the cancer cell. An example of an immunotherapy drug that works like this is rituximab. An example of advanced immunotherapy is Chimeric Antigen Receptor **(**CAR-T)therapy, where your own T cells (a type of white blood cell that normally fights infection in the body) are genetically modified to boost their ability to find and kill cancer cells. Examples are Brexucabtagene autoleucel, Axicabtagene Ciloleucel and Tisagenlecleucel.

6.4 Targeted therapies

Targeted therapies are cancer treatments that work by targeting the genetic changes that cancer cells have, which normal cells don't have. There are different types. Some of them may also be called biological therapies. They may be given with chemotherapy or on their own. Some are given into a vein (by a drip), some are injections and some are tablets.

6.4.1 Monoclonal antibodies

Monoclonal antibodies are artificial antibodies that are made in a lab. Once they are in your body, they can find and attach to cancer cells, and kill them. They work by recognising particular proteins on the surface of cancer cells.Monoclonal antibodies work in different ways. Some interfere with signals that a cancer cell needs to survive or divide. Others work by carrying a chemotherapy drug directly to a cancer cell. Some attach to cancer cells to make them more visible to the body's immune system, which can then attack it.Example ofMonoclonal antibody drug is Rituximab.

6.4.2 Cancer Growth Blockers (inhibitors)

Growth factors are chemicals in your body that tell cells what to do or how to grow properly. Cancer growth blockers are drugs that block these signals from affecting cancer cells, so the cancer cells do not grow properly, survive or divide as they otherwise would.Examples are, ibrutinib, bortezomib and types of TKIs.

6.4.3 TKIs (tyrosine kinase inhibitors)

TKIs work by blocking signals sent from tyrosine kinases. Tyrosine kinases are enzymes that send messages to cancer cells telling them to grow and divide. By blocking these signals, TKIs stop the cancer from growing.TKIs are often used to treat CML (chronic myeloid leukaemia). They are tablets that you take daily. Examples are:imatinib, dasatinib, nilotinib, bosutinib and ponatinib

6.5 Radiotherapy

Radiotherapy uses high-energy rays, such as x-rays, to destroy cancer cells. It can be used to treat Hodgkin lymphoma (HL) or non-Hodgkin lymphoma (NHL). The rays are aimed at the part of the body where the cancer cells are, for example particular groups of lymph nodes. During treatment, you will lie on a flat surface with the radiotherapy machine above you. You will not feel anything during the treatment, but high-energy rays will be aimed at the part of your body being treated. This will damage the cancer cells in the area being targeted.

6.6 Surgery

Surgery is rarely used to treat blood cancers, although a small number of people with lymphoma need to have their spleen removed (*blood cancer.org.uk*).

It is everyone's duty to improve the quality of life for Africans who have haematologic cancer. This objective was defined with a thorough framework that can also help lower morbidity and death from non-hematologic malignancies. Improvements in the fight against HIV and other infectious diseases have been made possible by foreign partners' cooperative solutions, which were developed in collaboration with local scientific, governmental, and civil society groups. Cancer programs that are effective must also be "locally developed." What is appropriate, doable, and attainable throughout Africa will be up for discussion. Nevertheless, calls for the eradication of HIV and malaria despite the fact that these two diseases continue to kill millions of people each year have been crucial in enabling current progress against infectious diseases. The following suggestions are essential, in our opinion, and are part of a holistic cancer strategy.

7 Create diagnostics that are both Conventional and cutting-edge for the situation.

Expanding histology, enhancing tissue sampling and processing, and implementing fundamental immunohistochemistry are all urgently needed. However, it is unlikely to be successful to graft pathology systems from developed nations into dissimilar settings without modification. As advocated by the INCTR and Sub-Saharan Africa Lymphoma Consortium, conventional procedures may need to be supplemented, and in some cases even replaced, by diagnostic algorithms, tissue microarrays, and digital microscopy methods appropriate to local conditions (Naresh *et al.,* 2011; Naresh *et al.,* 2011). In addition, distinctive genetic abnormalities and subsequent molecular events are increasingly used to identify malignancies. Due to poor laboratory infrastructure, genotypic and nucleic acid amplification tests are currently not frequently used in Africa. However, PCR assays utilizing dried blood spots have been created especially for use in environments with limited resources to enable HIV diagnosis in infancy, HIV RNA

monitoring, and HIV resistance detection(Rottinghaus *et al.,* 2012). Similar to this, a fully automated, cartridge-based molecular diagnostic system has been created to diagnose tuberculosis and identify drug resistance, enabling earlier start of the best course of treatment (Boehme *et al.,* 2011). These methods are intended for application in environments with dispersed specimen collecting, slow specimen transport, and constrained technical resources in the laboratory. Even in lower-level health centers, they have been successfully adopted, and their cost is acceptable for Africa(Rottinghaus *et al.,* 2012; Boehme *et al.,* 2011). Similar technologies may eventually make early cancer diagnosis, treatment, and response evaluation possible in locations without conventional pathology services or cutting-edge diagnostic imaging, similar to current trends in developed countries.

8 Invest more in human resources to fight cancer in the region

Despite the fact that non-oncologists can treat cancer in a safe and efficient manner, there is an urgent need to address the crucial health professional shortage (Farmer *et al.,* 2010). It is crucial to make investments in a clinical, pharmacy, and laboratory workforce that is experienced in treating cancer patients. International institutions (American Society of Haematology, ASCO, INCTR, NCI), African organizations (West African College of Physicians, African Organization for Research and Training in Cancer), and others have made training initiatives a top priority. For instance, the NCI is now funding 3-year-long pathology training seminars in Kenya to increase regional capability. Programs like the Medical Education Partnership Initiative offer specific financing sources to assist training initiatives. The development of intermediate measures is crucial since creating a committed oncology staff and stopping decades of "brain drain" will both take decades of persistent effort. Progress against infectious diseases has often relied on innovative human resource solutions, such as task shifting, to compensate for physician shortages (Sanne *et al.,* 2010). Videoconferencing and telepathology may also be important approaches, and the suitability of such strategies for cancer care in the region should be further explored.

9 Make cancer medications more cheap and incorporate cutting-edge therapies

The transition from cytotoxic therapy to "targeted" medicines focused on immunophenotypes, mutations, and gene products has been sparked by the molecular age. Monoclonal antibodies, antibody-drug conjugates, small molecule tyrosine kinase inhibitors, immunomodulatory drugs, proteasome inhibitors, and histone deacetylase inhibitors are some of the noncytotoxic medications that have recently been added to the treatments for hematologic malignancies. Even in resource-rich environments, the best use for many of these agents is still being explored, and their cost makes their usage in Africa now unaffordable. They are, however, in many ways the best treatments for the area, cost considerations aside. Some are given orally, eliminating the need for an infusion, and many have more tolerable side effects, such as less myelosuppression and an increased risk of infection when compared with conventional cytotoxic agents. As was previously said, the "newest" WHO essential cancer therapy is 20 years old, and we believe that keeping more recent therapies from patients in Africa for a number of additional decades goes against the guiding principle of health as a human right advocated by both the WHO and the UN. In environments where genetic variations, common comorbid illnesses, nutritional status, interactions with co administered medications, adherence, and other factors may result in efficacy, safety, and cost effectiveness profiles very different from previously studied populations, it is important to develop platforms to study the integration of newer agents. The cornerstone of successful ART in sub-Saharan Africa has been negotiated agreements with manufacturers to offer HIV drugs at cheap cost (typically about 10% of the US retail price), as lead by the Clinton Health Access Initiative (Clinton Health Access Initiative, 2011). Similar tier-based pricing structures may be viable for specific cancer treatments that have been shown to be secure and efficient in the area, particularly if linked to higher rates of long-term cure. Rituximab and imatinib's patents are about to expire, and generic drugs and biosimilar are already being developed and tested in clinical settings.

10 Boost global financing for cancer research and thoroughly evaluate the cost-effectiveness of cancer prevention strategies

South Africa alone accounted for 51% of the $581 million (US) in total regional cancer expenses in 2009, accounting for 5.4% of all new cancer cases worldwide, but just 0.2% of all cancer spending (Beaulieu *et al.,* 2012). In contrast, sub-Saharan Africa spent $2.8 billion (US) on HIV in 2009 (Joint United Nations Program on HIV/AIDS, 2010). Even in industrialized nations, it has been difficult for governments and health systems to sustainably finance contemporary cancer care. Resources are limited in Africa, thus a rational discussion on how to best use and increase available resources is required. This discussion will involve input from patients, doctors, policymakers, economists, and ethicists. Cancer treatment will be predictably more expensive than treating chronic conditions like HIV. For instance, the cost of 6 cycles of CHOP is about $1500 (US). Cost-effectiveness may however, compare favourably with other approved therapies if therapy is followed by decades of event-free survival. For first-line treatment, ART currently costs about

$200 (US), whereas second-line treatment in settings with inadequate resources costs between $500 and $600 (US)(Clinton Health Access Initiative, 2011; Joint United Nations Program on HIV/AIDS, 2010). Future initiatives will include defining ethical regional standards for cost-effective cancer care and submitting cancer control methods to formal cost effectiveness analyses.

11 Utilize current healthcare and research facilities to learn about and treat cancer

International efforts to combat HIV and other infectious diseases have resulted in investments in health care in places with limited pre-existing capacity as well as a strong clinical trial network capable of enrolling patients and keeping track of them in places where this has historically been difficult. Building on recent global health triumphs, it is possible to investigate cancer using this infrastructure. In sub-Saharan Africa, for example, the AIDS Malignancy Consortium (AMC) and AIDS Clinical Trials Group (ACTG) are currently co-sponsoring two HIV-associated KS trials (AMC066-ACTG 5263 and AMC067-ACTG 5264), which are enrolling patients. These networks can be used to study hematologic cancers in patients with and without HIV. Despite the fact that CHOP is the de facto regional standard, sub-Saharan African patients with or without HIV have never had their tolerability, benefit-risk profile, or optimal dosing of the CHOP chemotherapy (cyclophosphamide, doxorubicin, vincristine, and prednisone) for NHL prospectively assessed (Satish *et al.,* 2012). Tens of thousands of patients in the area could receive treatment guidance by answering such urgent clinical concerns through already established networks would provide immediately translatable results to guide treatment for tens of thousands of patients in the region.

12 Conclusion

In conclusion, the quality of life of Africans living with a Haematological cancer can be enhanced through early detection, diagnosis; current treatments options and if the holistic cancer strategy mentioned in the discussion were observed. People might be pessimistic about the chances for controlling haematologic malignancies in Africa given the immense hurdles and quickly growing burden. Nevertheless, despite the fact that there are still many obstacles to overcome and a lot of work to be done, the worldwi de reaction to HIV offers a valuable lesson. As a consequence of persistent international campaigning, cooperation, and investment, there is now increasing optimism and open demands to stop the epidemic in our lifetimes when there was previously existential fear about the fate of entire societies. A similar chance exists today to have a significant impact on haematological cancer prevention in Africa in the years to come. By overcoming this obstacle, we will lessen the suffering of haematological cancer patients in Africa and help many sufferers become long-term survivors.

Compliance with ethical standards

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