

Unusual Multivisceral Extranodal Involvement of Aggressive B-Cell Lymphoma: A Case Series and Literature Review

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Abstract

Background: Aggressive B-cell lymphomas may present with extranodal involvement; however, simultaneous involvement of multiple unusual extranodal sites is uncommon and may mimic disseminated solid malignancies, resulting in diagnostic challenges. We report three cases of aggressive B-cell lymphoma with unusual multivisceral extranodal involvement. **Case presentation:** Three women aged 26, 34, and 28 years presented with advanced aggressive B-cell lymphomas involving multiple extranodal sites. Two patients were living with HIV. Cases 1 and 2 were diagnosed with diffuse large B-cell lymphoma (DLBCL), with germinal center B-cell-like and non-germinal center B-cell-like immunophenotypes, respectively. Case 3 had morphological and cytological findings strongly supportive of Burkitt lymphoma; however, immunohistochemical characterization and MYC rearrangement analysis were unavailable, precluding complete classification according to current WHO-HAEM5/ICC criteria. Extranodal involvement included the pleura, pericardium, liver, stomach, bilateral breasts, kidneys, gallbladder, inferior vena cava, latero-uterine region, and Waldeyer's ring. Cases 1 and 2 received eight cycles of R-CHOP with prophylactic intrathecal chemotherapy, whereas Case 3 received an LMB02-based regimen and completed two R-COPADM induction courses. At the August 12, 2026 data cut-off, Cases 1 and 2 were in complete remission, with no residual disease on post-treatment computed tomography, and remained under clinical follow-up. Case 3 had completed induction and was awaiting CYM1 consolidation; no formal imaging-based response assess-

ment had yet been performed. **Conclusion:** Multivisceral extranodal involvement of aggressive B-cell lymphomas may closely mimic metastatic solid malignancies. Early tissue sampling combined with appropriate immunophenotypic characterization is essential for establishing the diagnosis. These cases also highlight the diagnostic challenges encountered when advanced molecular and immunophenotypic investigations are not readily available in resource-limited settings.

Keywords

Aggressive B-Cell Lymphoma, Diffuse Large B-Cell Lymphoma, Burkitt Lymphoma, Extranodal Involvement, Multivisceral Involvement, HIV

1. Introduction

Aggressive B-cell lymphomas represent a heterogeneous group of hematologic malignancies characterized by rapid proliferation and a high potential for progression. Among them, diffuse large B-cell lymphoma (DLBCL) is the most common subtype of adult non-Hodgkin lymphoma (NHL), whereas Burkitt lymphoma (BL) represents a particularly aggressive form characterized by a very high proliferative activity [1].

Extranodal involvement is a common clinical presentation of aggressive B-cell lymphomas. In diffuse large B-cell lymphoma, approximately 40% - 60% of patients present with extranodal disease at diagnosis according to published series [2].

The most frequent sites of involvement include the gastrointestinal tract, bone, central nervous system, and bone marrow [3].

However, involvement of sites such as the breast, adrenal glands, liver, genital organs, lungs, and pleura remains uncommon. Their simultaneous occurrence in the same patient is even rarer and may constitute a genuine diagnostic, prognostic, and therapeutic challenge. Several studies have suggested that the multiplicity of extranodal sites may be associated with more aggressive disease forms and an unfavorable prognosis [4].

In this context, we report a series of three cases of aggressive B-cell lymphoma presenting with unusual multivisceral involvement.

2. Literature Search Strategy

A literature search was conducted in PubMed/MEDLINE and Google Scholar up to August 12, 2026. The search strategy combined the terms “diffuse large B-cell lymphoma”, “Burkitt lymphoma”, “aggressive B-cell lymphoma”, “extranodal lymphoma”, “multivisceral involvement”, “multifocal extranodal lymphoma”, “breast lymphoma”, “pleural lymphoma”, “renal lymphoma”, and “HIV-associated lymphoma”. Original articles, case reports, case series, and relevant reviews published

in English or French and describing aggressive B-cell lymphomas with unusual or multiple extranodal organ involvement were considered eligible. Titles and abstracts were initially screened for relevance, followed by full-text assessment of potentially eligible publications. Reference lists of selected articles were also manually screened to identify additional relevant reports.

3. Cases Presentations

The epidemiological characteristics of the patients are depicted in **Table 1**. The median age at diagnosis was 28 years (range, 26 to 34 years). The gender distribution for the entire population was 3 females. We observed that the patients of case 2 and 3 were infected with HIV. the patient in Case 2 had been diagnosed with HIV-1 infection more than three years earlier and was receiving antiretroviral therapy with dolutegravir, tenofovir, and lamivudine (DTG/TDF/3TC). At the time of lymphoma diagnosis, her CD4+ T-cell count was 260 cells/mm³ and her HIV viral load was 100 copies/mL. EBV serology (VCA-IgM, VCA-IgG, and EBNA-IgG) and blood EBV PCR were negative. Tumor EBER-ISH was not performed because this molecular testing was not accessible in our setting at the time of diagnosis.

The patient in Case 3 had been diagnosed with HIV-1 infection approximately one month before the lymphoma diagnosis and was also receiving antiretroviral therapy with dolutegravir, tenofovir, and lamivudine (DTG/TDF/3TC). At the time of lymphoma diagnosis, her CD4+ T-cell count was 210 cells/mm³ and her HIV viral load was 150 copies/mL. EBV serology (VCA-IgM, VCA-IgG, and EBNA-IgG) and blood EBV PCR were negative. Tumor EBER-ISH was not performed because this molecular testing was not accessible in our setting at the time of diagnosis.

Table 1. The characteristics of the cases.

Characteristics	Case 1	Case 2	Case 3
Age	26	34	28
Sex	Female	Female	Female
Occupation	Student	Trader	Trader
Medical history	Uninvestigated epigastric pain	HIV-1 infection	HIV-1 infection
Duration since HIV diagnosis	Not concerned	>3 years	Approximately 1 month
CD4+ T-cell count at lymphoma diagnosis	Not concerned	260 cells/mm ³	210 cells/mm ³
HIV viral load at lymphoma diagnosis	Not concerned	100 copies/mL	150 copies/mL
ART at lymphoma diagnosis	Not concerned	Yes - DTG/TDF/3TC	Yes - DTG/TDF/3TC

Continued

EBV serology	Not performed	Negative (VCA-IgM, VCA-IgG, EBNA-IgG)	Negative (VCA-IgM, VCA-IgG, EBNA-IgG)
Blood EBV PCR	Not performed	Negative	Negative
Tumor EBER-ISH	Not performed	Not performed	Not performed
Site	Pleural and pericardial involvement Asymmetric bilateral submandibular lymphadenopathy hepatic involvement	gastric mucosal, Bilateral breast and bilateral renal involvement with infiltration of the gallbladder and inferior vena cava, associated with a right lateral uterine mass	Left laterocervical mass and right breast involvement with abdominal lymphadenopathy Waldeyer's ring involvement
Symptoms	Dyspnea, unexplained fever, drenching night sweats and unexplained weight loss of >10% of body weight within the previous 6 months	Abdominal pain, dysphagia, lower limb edema, melena, unexplained fever, drenching night sweats and unexplained weight loss of >10% of body weight within the previous 6 months	Laterocervical pain, dysphonia Breast mass, unexplained fever, drenching night sweats and unexplained weight loss of >10% of body weight within the previous 6 months
Diagnostic specimen	Axillary lymph node	Renal biopsy	cervical lymph node
Histopathological examination	Suggested either DLBCL or Hodgkin lymphoma	Suggested either an undifferentiated carcinoma or non-Hodgkin lymphoma (NHL)	Suggested a Burkitt lymphoma
Immunohistochemistry examination	CD20 + CD10 + BCL2 + CD3 – CD30 – CD15 – BCL6 – Ki67 = 70%	CD20+ CD10 – BCL2 – BCL6 – MUM1 – Ki67 = 40%	Not performed
MYC rearrangement	Not assessed	Not assessed	Not assessed
Diagnosis	Germinal Center B-cell-DLBCL	Non-Germinal Center B-cell-DLBCL	Burkitt lymphoma
Cytologically confirmed sites	Pleural fluid + Asymmetric bilateral submandibular lymphadenopathy	Bilateral breast mass	Right breast mass
Histologically confirmed sites	Axillary lymph node	Renal biopsy and gastric mucosal biopsy	cervical lymph node
Imaging-inferred extranodal sites and treatment response	hepatic pericardial involvement	Infiltration of the gallbladder and inferior vena cava + a right lateral uterine mass	involvement with abdominal lymphadenopathy + Waldeyer's ring involvement

The clinical characteristics of the patients are depicted in **Table 1**. The patient in case 1 mainly presented with dyspnea as the primary symptom. The diagnosis was established through an axillary lymph node biopsy. Morphological findings of the axillary lymph node biopsy revealed a diffuse malignant lymphoid proliferation composed of large atypical lymphoid cells with moderately abundant cytoplasm and large, irregular, occasionally prominent nucleolated nuclei. These large cells were distributed within a polymorphous inflammatory background consisting predominantly of small lymphocytes and scattered histiocytes. The morphological features were consistent with a large-cell lymphoma but were insufficient to reliably distinguish diffuse large B-cell lymphoma from Hodgkin lymphoma. Immunohistochemical analysis was therefore required for definitive classification. Immunohistochemical analysis demonstrated strong expression of CD20 and CD10 by the neoplastic cells, with BCL2 expressed in 70%, while CD3, CD15, CD30, MUM1 and BCL6 were negative. The Ki-67 proliferation index was approximately 70%. Overall, the immunophenotypic findings supported the diagnosis of diffuse large B-cell lymphoma (DLBCL), with a germinal center B-cell-like (GCB) immunophenotype according to the Hans algorithm. The molecular testing for MYC, BCL2, and BCL6 rearrangements, were not performed because these investigations were not accessible in our setting at the time of diagnosis.

Cytological examination of the pleural fluid revealed the presence of malignant cells. The moderate pericardial effusion was not amenable to percutaneous drainage.

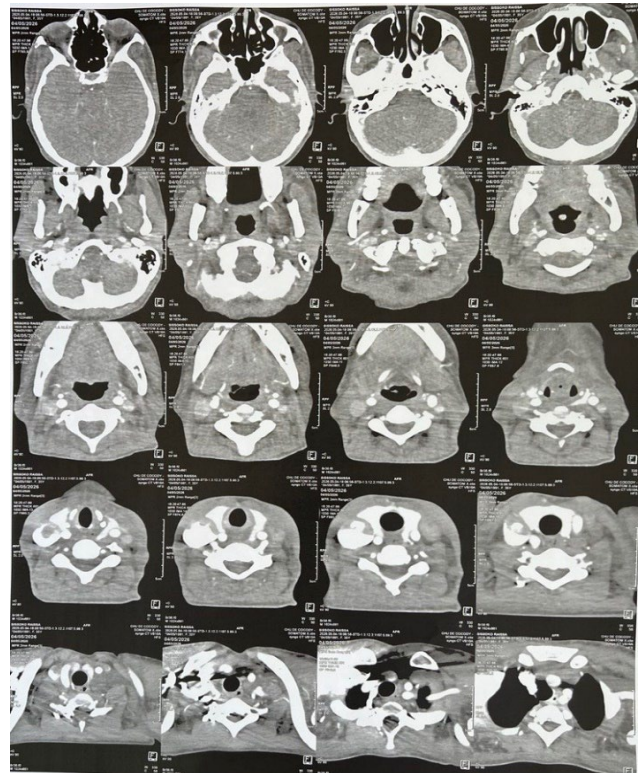
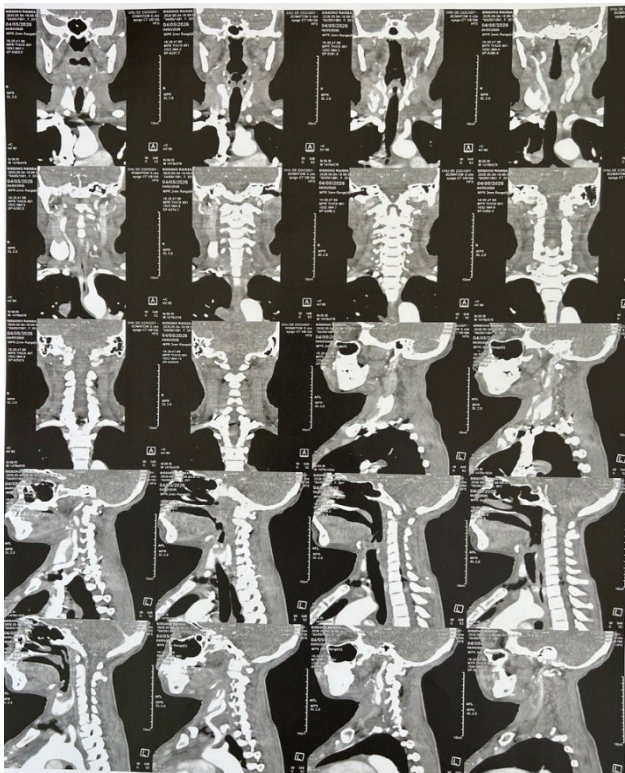
The patient in the case 2 presented with a “frozen abdomen” related to the multiple abdominal masses (**Figure 1** & **Figure 2**). Morphological findings of the renal biopsy showed a diffuse proliferation of large atypical cells arranged predominantly in sheets. The tumor cells exhibited marked nuclear pleomorphism, irregular nuclear contours, vesicular chromatin, prominent nucleoli, and moderate amounts of cytoplasm. No definite glandular or squamous differentiation was identified on routine hematoxylin and eosin staining. Based on morphology alone, the findings were suggestive of a poorly differentiated malignant neoplasm, with the main differential diagnoses including undifferentiated carcinoma and non-Hodgkin lymphoma. Immunohistochemical studies were required for definitive lineage assignment and classification. Immunohistochemical analysis demonstrated diffuse CD20 expression by the neoplastic cells, supporting B-cell lineage. The tumor cells were negative for CD10, BCL2, BCL6, and MUM1/IRF4. The Ki-67 proliferation index was approximately 40%. CD45, MYC and pancytokeratin (AE1/AE3) were not assessed. Overall, the morphologic and immunophenotypic findings supported the diagnosis of diffuse large B-cell lymphoma (DLBCL). FISH analysis for MYC, BCL2, and BCL6 rearrangements was also not performed because these molecular investigations were not available in our setting at the time of diagnosis.

Case 3 had cytological (**Figure 3**) and histopathological findings strongly supportive of Burkitt lymphoma. Histological examination of the cervical lymph node biopsy revealed a diffuse, monomorphic proliferation of medium-sized atypical

lymphoid cells with round nuclei, finely clumped chromatin, multiple small basophilic nucleoli, and deeply basophilic cytoplasm containing occasional cytoplasmic vacuoles. Numerous mitotic figures and apoptotic bodies were present, reflecting a very high proliferative activity. Numerous tangible-body macrophages containing apoptotic debris were scattered throughout the tumor, producing the characteristic “starry-sky” appearance. These morphological features were highly suggestive of Burkitt lymphoma. However, immunohistochemical characterization was not performed, and MYC rearrangement analysis by FISH was unavailable in our setting at the time of diagnosis. Therefore, although the morphological and cytological findings strongly supported Burkitt lymphoma, complete classification according to current WHO-HAEM5/ICC criteria could not be established [5] [6].



Figure 1. The frozen abdomen and Bilateral breast masses of case 2.



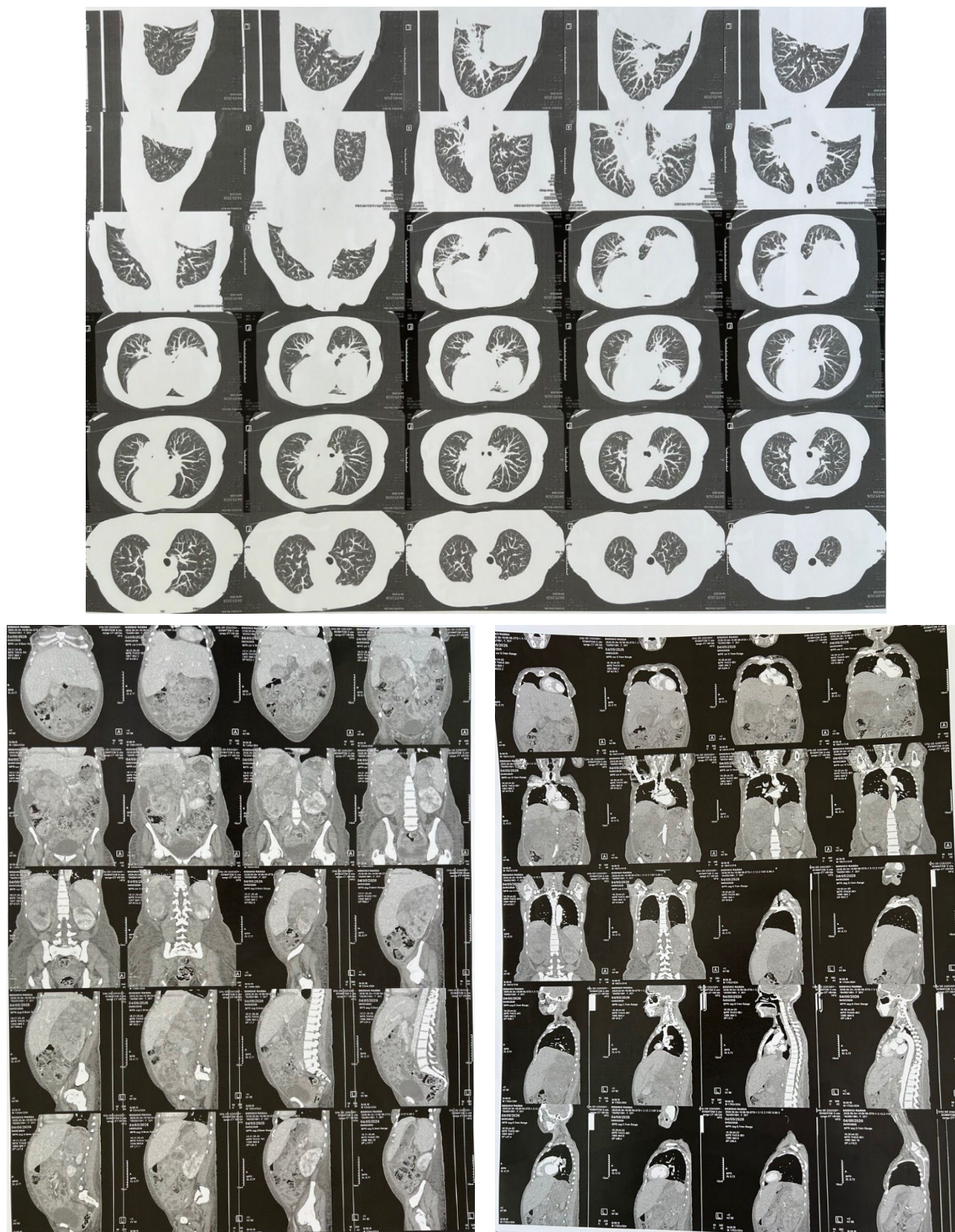


Figure 2. CT scan and the abdominopelvic MRI of case 2 showing Bilateral breast and bilateral renal involvement with infiltration of the gallbladder and inferior vena cava, associated with a right lateral uterine mass.

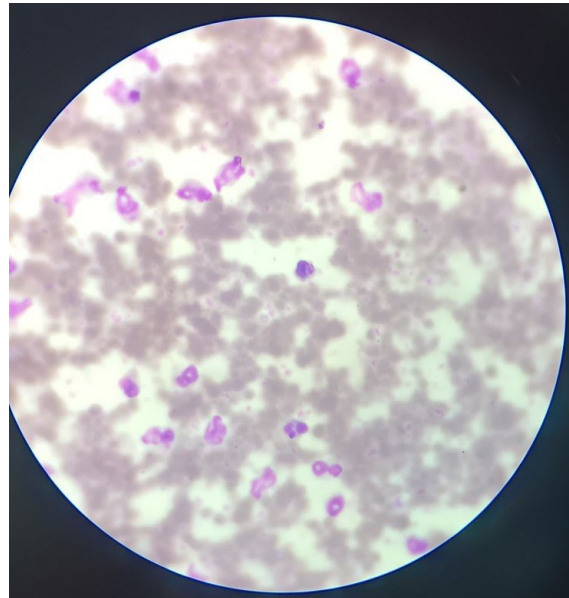


Figure 3. Breast fine-needle aspiration cytology of case 3 showed a lymphoid proliferation composed of vacuolated lymphoblasts.

Table 2. Patient baseline clinical, laboratory, staging and prognostic characteristics.

Baseline staging	Case 1	Case 2	Case 3
ECOG performance status	II	III	II
Bone marrow assessment (by bone marrow aspirate)	50% lymphocytes, lymphoma involvement not confirmed	No morphologic evidence of lymphoma on marrow aspirate	38% lymphocytes and 4% vacuolated lymphoblasts cells, lymphoma involvement not confirmed
CNS assessment	CSF cytology negative	CSF cytology negative	CSF cytology negative
Ann Arbor stage	Stage IV	Stage IV	Not reported
Murphy/St Jude stage	Not reported	Not reported	Stage III
B symptoms (unexplained fever, drenching night sweats and unexplained weight loss of >10% of body weight within the previous 6 months)	Yes	Yes	Yes
LDH, U/L (ULN: 489 U/L)	556	1665	1592
β 2-microglobulin, mg/L (0.70 - 2.40)	4.0	5.6	Not available
International Prognostic Index	4/5 (High risk)	4/5 (High risk)	Not reported
BL-IPI	Not reported	Not reported	2/4 (High risk)
LMB risk group	Not reported	Not reported	group B

Bone marrow assessment was limited to morphologic examination of aspirates shown in **Table 2**; neither bone marrow biopsy nor immunophenotyping of marrow cells was performed. In Case 1, the aspirate contained 50% lymphocytes, but their lymphomatous nature could not be established. In Case 2, no morphologic evidence of lymphoma was reported on the marrow aspirate. In Case 3, the aspirate contained 38% lymphocytes and 4% vacuolated blast-like cells. Without confirmatory immunophenotyping, marrow involvement by Burkitt lymphoma could not be established. Cerebrospinal fluid cytology was negative in all three cases.

Case 3 was assigned to group B of an LMB02-based regimen because of stage III disease, with negative cerebrospinal fluid cytology and no confirmed bone marrow involvement. She completed two R-COPADM induction courses. Both included rituximab 375 mg/m² on days 0 and 6, prednisolone 60 mg/m² on days 1 - 5 followed by a three-day taper, doxorubicin 60 mg/m² on day 2, and high-dose methotrexate 3 g/m² on day 1 with folinic acid rescue. Vincristine 1.4 mg/m² was given on day 1 of R-COPADM1 and on days 1 and 6 of R-COPADM2. Cyclophosphamide was given on days 2 - 4 at 500 mg/m²/day in the first course and 1000 mg/m²/day in the second. Intrathecal methotrexate 15 mg plus hydrocortisone hemisuccinate was scheduled on days 2 and 6 of each course for CNS prophylaxis (4 administrations actually given). At the August 12, 2026 data cut-off, induction was complete and the patient was awaiting CYM1 consolidation. No formal imaging-based response assessment had yet been performed. The first response assessment was planned by CT after CYM1.

Cases 1 and 2 each completed eight 21-day cycles of R-CHOP. Each cycle comprised intravenous rituximab 375 mg/m², cyclophosphamide 750 mg/m², doxorubicin 50 mg/m², and vincristine 1.4 mg/m² on day 1, together with oral prednisone 40 mg/m² on days 1 - 5. Both patients also received four administrations of prophylactic intrathecal chemotherapy comprising methotrexate 15 mg, cytarabine 40 mg, and hydrocortisone hemisuccinate 20 mg. CNS prophylaxis was administered because the treating team considered both patients to be at high risk of CNS involvement; however, the specific criteria used to define this high-risk status were not documented in the available medical records. Case 1 additionally underwent repeated therapeutic pleural drainage followed by talc pleurodesis.

At the August 12, 2026 data cut-off, Cases 1 and 2 were in complete remission following R-CHOP. In both patients, the initial symptoms had resolved on physical examination, and post-treatment cervical, thoracic, abdominal, and pelvic CT showed no residual disease. The CT examinations were performed on March 13, 2026, for Case 1 and June 8, 2026, for Case 2. The corresponding β 2-microglobulin levels were 2.00 mg/L and 2.38 mg/L, respectively. Both patients remained under clinical follow-up every three months.

4. Discussion

Pleural and pericardial involvement observed in our first patient represents an

uncommon presentation of diffuse large B-cell lymphoma (DLBCL). Although extranodal disease is identified in approximately 40% of patients with DLBCL, serosal involvement remains relatively rare and generally reflects advanced disseminated disease [7].

In our patient, cytological examination of the pleural fluid provided an early diagnostic clue before histopathological confirmation from the lymph node biopsy. This finding highlights the diagnostic value of cytological analysis of serous effusions in aggressive B-cell lymphomas, particularly when tissue biopsy is delayed or technically challenging.

Pleural involvement presents as an important extranodal site for DLBCL, which is also found to be associated with overall survival [8].

Despite presenting with Ann Arbor stage IV disease and an International Prognostic Index (IPI) score of 4/5, the patient achieved complete remission following eight cycles of R-CHOP combined with repeated therapeutic pleural drainage, prophylactic intrathecal chemotherapy, and talc pleurodesis. This favorable outcome illustrates that long-term remission remains achievable even in disseminated disease when prompt diagnosis and multidisciplinary management are implemented. This is consistent with the case reported by Shah SH *et al.* [7].

Our second patient presented with the most remarkable pattern of extranodal dissemination in this series. Simultaneous involvement of the stomach, both breasts, kidneys, gallbladder, inferior vena cava, and the latero-uterine region represents an exceptionally rare clinical presentation. Only a limited number of similar cases have been reported in the literature. One of the closest descriptions is that reported by Li *et al.* [9], who described multifocal extranodal DLBCL involving the stomach, bilateral breasts, pancreas, adrenal glands, ovaries, and bone, with subsequent central nervous system dissemination. In our patient, the extensive abdominal disease associated with multiple visceral masses initially suggested metastatic carcinoma. This observation emphasizes that aggressive B-cell lymphomas should always be considered in the differential diagnosis of rapidly progressive multiorgan masses, particularly in young patients. The final diagnosis was established only after immunohistochemical analysis, underscoring the essential role of immunophenotyping in differentiating lymphoma from poorly differentiated carcinoma.

Our third patient had histopathological findings strongly supportive of Burkitt lymphoma, although complete classification according to current WHO-HAEM5/ICC criteria could not be established because immunohistochemical and MYC rearrangement studies were unavailable [5] [6]. The disease involved Waldeyer's ring, the breast, and abdominal lymph nodes. Burkitt lymphoma is one of the most rapidly proliferating human malignancies and is characterized by MYC deregulation and an extremely high proliferative index. Although the sporadic form most commonly involves the abdomen, breast involvement remains uncommon and is usually associated with disseminated disease. This is consistent with the case reported by Lee HJ *et al.* [10] who described a very rare case of secondary

breast Burkitt lymphoma.

Burkitt lymphoma is a rare and highly aggressive form of B-cell NHL. It is characterized by the translocation and deregulation of the MYC gene on chromosome 8 and has the potential to involve multiple organ systems [1]. Burkitt lymphoma has three subtypes (sporadic, endemic, and immunodeficiency-associated), each demonstrating different epidemiology, risk factors, and clinical presentations. In this case, the immunodeficiency-associated subtype was considered. According to Judith A. Ferry: Immunodeficiency-associated BL is mostly commonly seen in HIV-positive patients, but also may be seen in allograft recipients and patients with congenital immunodeficiency. HIV patients with BL tend to have relatively higher CD4 counts (>200 cells/ μ L) and the majority demonstrate EBV positivity. Our patient had higher CD4 counts (>200 cells/ μ L) and demonstrated EBV serology (VCA-IgM, VCA-IgG, and EBNA-IgG) and blood EBV PCR negativity. Tumor EBER-ISH was not performed [11].

In our patient, the coexistence of Waldeyer's ring and breast involvement reflects the highly aggressive biological behavior of Burkitt lymphoma and justified prompt initiation of intensive immunochemotherapy according to the LMB protocol.

Beyond the individual characteristics of each case, several common features deserve emphasis.

All three patients in our series were young women, with a median age of 28 years, and two were living with HIV. Given the small sample size and descriptive nature of this case series, no inference can be made regarding an association between age, sex, HIV infection, and the observed pattern of extranodal involvement. Nevertheless, HIV infection is a well-established risk factor for aggressive B-cell lymphomas. B-cell aggressive NHL was included in the 1985 revised case definition of AIDS as one of three AIDS-defining cancer categories [12]. and, currently, it is the most common hematological malignancy in PWH [13].

Human immunodeficiency virus is not known to have direct oncogenic effects, but HIV infection causes several indirect effects, which are implicated in lymphomagenesis. Recent evidence suggests that HIV may contribute to lymphomagenesis by acting directly on B-cells, as a critical microenvironmental modifier [14]. In a systemic background of immunodeficiency, B-cells receive an uncontrolled chronic activation through persistent antigenic stimulation, HIV CD40 ligand (CD40L), and HIV-encoded proteins, such as gp120, p17, and TAT [14] [15]. Moreover, HIV infection leads to a loss of immune surveillance because of depletion of T lymphocytes, and reactivated oncogenic viral infection, such as Epstein-Barr virus (EBV), human herpesvirus 8 (HHV8), and chronic antigen stimulation mediated by other viral co-infections, including hepatitis B and C viruses [16].

Second, all patients presented with advanced-stage disease and multiple extranodal sites of involvement. Previous studies have demonstrated that the number of extranodal sites is associated with an increased risk of disease progression and poorer survival, reflecting a high tumor burden and aggressive biological behavior

[7].

Finally, our case series highlights the diagnostic challenges posed by multivisceral aggressive B-cell lymphomas. Therefore, early tissue biopsy combined with immunohistochemistry remains essential for establishing the correct diagnosis. Furthermore, access to molecular testing, including MYC, BCL2 and BCL6 rearrangement analysis, as well as 18F-FDG PET/CT for accurate staging and treatment response assessment, remains a major challenge, particularly in resource-limited settings.

5. Conclusions

This case series highlights the remarkable clinical heterogeneity of aggressive B-cell lymphomas and emphasizes that extensive multivisceral extranodal involvement, although uncommon, may represent the initial presentation of these highly aggressive malignancies. The coexistence of rare extranodal sites—including the breast, pleura, pericardium, stomach, kidneys, Waldeyer's ring, and female pelvic organs—can closely mimic metastatic solid tumors, leading to diagnostic delay.

Our observations underscore the pivotal role of early tissue biopsy combined with immunohistochemistry in establishing an accurate diagnosis and guiding appropriate treatment. Despite advanced-stage disease, timely initiation of standardized immunochemotherapy may result in favorable clinical outcomes. Finally, this series highlights the need to consider aggressive B-cell lymphoma in the differential diagnosis of unexplained multiorgan masses, particularly in young patients and those living with HIV, and reinforces the importance of improving access to advanced diagnostic techniques in resource-limited settings.

6. Limitation

This case series has several limitations. First, the small number of patients limits the generalizability of our observations and does not allow firm conclusions to be drawn regarding the frequency, prognostic significance, or outcomes of multivisceral extranodal involvement in aggressive B-cell lymphomas. Second, the series includes different histological subtypes, namely diffuse large B-cell lymphoma and Burkitt lymphoma, which differ in their biological behavior and therapeutic management. Third, histopathological confirmation was not obtained from every extranodal site. However, the distribution of the lesions, their radiological characteristics and their response to lymphoma-directed therapy strongly supported multivisceral lymphomatous involvement. In addition, comprehensive molecular characterization, including MYC, BCL2, and BCL6 rearrangement analyses, was not available for all patients.

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Ethical Consideration and Consent

This case series did not involve any experimental intervention. Patient confidentiality was maintained throughout the preparation of the manuscript. Written informed consent for publication of clinical information and accompanying images was obtained from each of the three adult patients. According to the institutional policy of Cocody University Hospital, formal ethics committee approval was not required for the publication of this anonymized case series.

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Author Contributions

Kouakou Boidy, Kouamé Norman Isaac Klébair, and Kamara Ismaël were involved in the clinical management of the patients and contributed to drafting the manuscript.

Atiméré Yao Nicaise, Kouakou Affoué N'guessan Jeanne Ines, Djeket Ruth, Assohou-Koffi Hiabba Emmanuela, and Kouadio Atta Yao Emmanuel contributed to the critical revision of the manuscript.

Koffi Kouassi Gustave, Professor of Clinical Hematology and Head of the Department of Clinical Hematology at Cocody University Hospital, provided scientific supervision and critically reviewed the manuscript.

All authors read and approved the final version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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