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REFRACTORY PLASMABLASTIC LYMPHOMA OF THE ORAL CAVITY IN A HIV-POSITIVE YOUNG MAN: A DUAL THREAT

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ABSTRACT **Background:** Plasmablastic lymphoma (PBL) is a rare and aggressive subtype of diffuse large B-cell lymphoma strongly associated with HIV infection, typically involving oral mucosa and showing plasmacytic differentiation. Optimum treatment remains undefined. **Case Presentation:** A 31-year-old HIV-positive male presented with a progressively enlarging right cheek mass associated with trismus. Histology and immunohistochemistry confirmed PBL. He received CHOPE plus weekly bortezomib with partial response, subsequently developed refractory disease, and was treated sequentially with CEOP, CODOX-M/IVAC, palliative radiotherapy, and salvage ICE plus bortezomib. **Conclusion:** PBL demonstrates rapid chemoresistance, high relapse burden, and poor outcomes. Proteasome-based combinations, ART optimization, and early consideration of consolidation are essential. Multidisciplinary management remains critical.

KEYWORDS : Plasmablastic Lymphoma, Hiv, Bortezomib, Refractory Lymphoma, Oral Cavity, Salvage Chemotherapy

INTRODUCTION

Plasmablastic lymphoma (PBL) is a rare, high-grade, aggressive lymphoma accounting for approximately 2% of HIV-associated Lymphomas. The disease commonly involves the gastrointestinal tract and oral mucosa, often presenting without lymphadenopathy. Diagnosis is challenging due to morphological overlap with multiple myeloma and overlapping immunophenotype. There is no established standard therapy.

Case Presentation

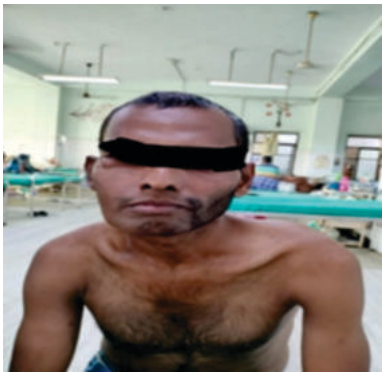
A 31-year-old male, known retroviral disease (RVD) positive, presented with a six-month history of gradually progressive, painless swelling over the right cheek associated with trismus. There were no B symptoms, neurological symptoms, or evidence of systemic swellings.

Past history was significant for HIV and pulmonary tuberculosis diagnosed in October 2019, treated with ATT followed by HAART. He was initially on tenofovir/lamivudine/efavirenz, later switched to tenofovir/lamivudine/dolutegravir. The last known CD4 count prior to symptom onset was 238 cells/mm^3 and viral load was undetectable.

Clinical Examination

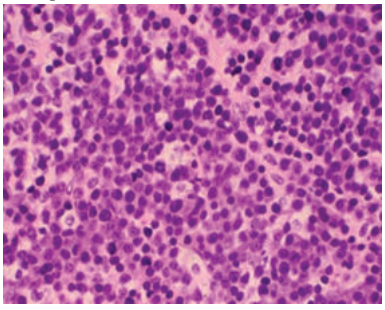
The patient was conscious, oriented, moderately built and nourished, with normal vitals (BP 110/70mmHg, Pulse 85/min). ECOG performance status was 1.
No pallor, edema, clubbing, cyanosis
No generalized lymphadenopathy
No hepatosplenomegaly

Local oral cavity examination revealed a 6×6 cm ulcero-proliferative mass involving the right upper alveolus extending to right buccal mucosa, firm, non-bleeding, with Grade 1 trismus, and no cervical lymphadenopathy.



Investigations

On Histopathology
Tumor cells demonstrated, Moderate amphiphilic/eosinophilic cytoplasm, Eccentric nuclei, Stippled chromatin, Prominent nucleoli, Plasmacytoid and plasmablastic features.



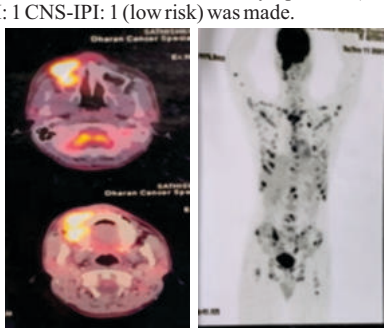
Immunohistochemistry was positive for CD45, Cd79a, CD138, CD38, MUM1, EBV
Ki-67 proliferation index = 40%
Negative for CD3, CD20, CD30, ALK

Bone marrow biopsy showed Hypercellular marrow with sheets of plasmablastic cells, CD138/CD38 positive.

Laboratory values
LDH: 245 U/L (mildly raised)
CBC with modest anemia, Renal and hepatic parameters normal, HBsAg & HCV negative. ECHO, SPEP, Calcium normal.

PET-CT Showed FDG-avid primary lesion with disseminated involvement.

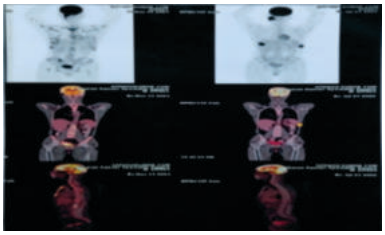
FINAL DIAGNOSIS of Plasmablastic lymphoma (PBL), Lugano Stage IV IPI: 1 CNS-IPI: 1 (low risk) was made.



INITIAL TREATMENT
The patient received CHOPE chemotherapy 6 cycles.

dose of Cyclophosphamide 750 mg/m² D1 Adriamycin 50 mg/m² D1, Vincristine 1.4 mg/m² D1, Prednisolone 100 mg/m² D1-D5, Etoposide 100 mg/m² D1-D3 q21 days with bortezomib 1.3 mg/m² weekly.

End-of-treatment PET-CT showed refractory disease.



The patient defaulted for three months and returned with ECOG 3. He received Second-line palliative intent chemotherapy with CEOP × 3 cycles (Cyclophosphamide, Etoposide, Vincristine, Prednisolone).

Cheek swelling of the patient worsened with new supraclavicular lymphadenopathy. Patient was treated third line with 2 cycles CODOX-M + 2 cycles IVAC, followed by Palliative radiotherapy 39 Gy in 13 fractions to cheek mass, after multidisciplinary tumour board discussion.

Patient progressed and was treated with one cycle of Fourth-line salvage ICE + weekly bortezomib regimen (Ifosfamide 1500 mg/m² D1–3, Carboplatin AUC 5, Etoposide 100 mg/m² D1–3). Patient defaulted further chemotherapy and succumbed after 2 months.

DISCUSSION

PBL is characterized by, Weak/absent B-cell markers (CD19, CD20, PAX-5) Strong plasma cell markers (CD138, CD38, MUM1). High Ki-67 (>80% in most patients), Frequent EBV positivity. Standard CHOP is inadequate because of MYC dysregulation, High proliferative fraction, Chemoresistance. Meta-analysis suggests benefit of dose-adjusted EPOCH over CHOP. Bortezomib may enhance chemosensitivity due to plasma-cell-like biology and has demonstrated of 94%.5-year OS of 63% in small series. Relapsed/refractory PBL has median OS ≈ 3.5 months. ASCT may offer benefit in chemo sensitive disease. Radiotherapy remains as a palliative option.

Outcome

Despite multiple salvage lines, disease progression occurred, consistent with, Rapid chemoresistance and Poor durability of remission. The patient initially showed partial response but developed rapid progression with treatment interruption. Subsequent lines of salvage therapy including CEOP, CODOX-M/IVAC, radiotherapy, and ICE plus bortezomib produced only transient control, reflecting the aggressive biology of PBL. Progression occurred in multiple extranidal regions, signaling aggressive behavior. Overall clinical trajectory remained poor and durability of response was limited.

CONCLUSION

This case reinforces that PBL in HIV-positive patients is characterized by rapid progression, early chemo-resistance, and limited response to salvage regimens. Early dose-intense therapy, treatment compliance, and consideration of stem-cell consolidation when feasible are recommended. Emerging therapeutic options including proteasome inhibitors and immunomodulatory agents warrant further clinical exploration.

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