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Escalate and Snowball-Burkitt s Lymphoma

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Introduction

Burkitt's lymphoma is an aggressive, rapidly progressive, non-Hodgkin B cell lymphoma occurring within humans. Burkitt's lymphoma is primarily associated with genetic rearrangement of c-MYC proto-oncogene, commonly encountered within germinal centre derived B lymphocytes. Characteristically, Burkitt's lymphoma demonstrates chromosomal translocation t(8; 14) between c-MYC situated upon chromosome 8q24 and IGH discerned upon chromosome 14q32.

Burkitt's lymphoma is additionally designated as Burkitt's tumour, malignant lymphoma, undi erentiated, Burkitt's type or malignant lymphoma, small non-cleaved, Burkitt's type [1, 2].

Burkitt's lymphoma delineates diverse categories as endemic, sporadic and immunode ciency related subtype. e lymphoma demonstrates a male predominance [1, 2]. Endemic subtype is encountered within young children preponderantly of African descent, is comprehensively (100%) associated with Epstein Barr virus (EBV) [illegible]

Sporadic subtype exempli es lesions within gastrointestinal tract, especially ileocecal junction, head and neck wherein lymphoma occurs within lymph nodes, pharynx, tonsils or sinuses. Jaw lesions are exceptional. Bone marrow is commonly involved which confers an unfavourable prognostic outcome.

- Immunode ciency associated subtype is associated with

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20-Oct-2022, Manuscript No: JCD-22-77848, 22-Oct-2022, PreQC No: JCD-22-77848(PQ), 27-Oct-2022, QC No: JCD-22-77848, 29-Oct-2022, Manuscript No: JCD-22-77848(R), 04-Nov-2022, DOI: 10.4172/2476-2253.1000162

Bajaj A (2022) Escalate and Snowball-Burkitt's Lymphoma. J Cancer Diagn 6: 162.

aggressive B cell lymphomas, karyotype of Burkitt's lymphoma appears uncomplicated [1, 2].

In situ hybridization (ISH) exempli es Epstein Barr virus encoding region (EBER) within Burkitt's lymphoma secondary to EBV infection. Fluorescent in situ hybridization (FISH) can be adopted for ascertaining c-MYC translocation [1, 2]. Polymerase chain reaction can be employed to detect monoclonal immunoglobulin genetic rearrangements [1, 2].

Tumefaction is rapidly progressive with a doubling time of 24 hours to 48 hours. Cogent clinical symptoms appear within weeks of disease onset [1, 2].

Distinct subtypes of Burkitt's lymphoma delineate variable presenting signs and symptoms designated as

- Endemic subtype manifests with tumefaction con ned to jaw or orbital region. Periorbital oedema is observed within ~50% individuals.
- Sporadic subtype enunciates pain, abdominal tumefaction or intussusception. Gastrointestinal haemorrhage occurs within ~80% instances.
- Immunode ciency associated subtype delineates abdominal symptoms and pancytopenia [1, 2].

Cytological examination exempli es a hyper-cellular smear

International Paediatric Non-Hodgkin's Lymphoma Staging System (IPNHLSS) categorizes paediatric lymphomas as

- Stage I composed lymphoma con ned to singular lymph node region as cervical, axillary or inguinal or a singular extra-nodal site with absent regional lymph node metastasis. Mediastinal or abdominal cavity tumefaction is absent.

- Stage II where lymphoma is absent within mediastinum and ~tumefaction appears within a singular site and incriminates adjacent nodes as cervical, axillary or inguinal. ~tumefaction incriminates > singular lymph node group and is situated on one side of diaphragm ~lymphoma commences within gastrointestinal tract along with or devoid of incrimination of adjacent lymph nodes and is amenable to surgical resection.

- Stage III is described where ~lymphoma commences within thoracic cavity as thymus, mediastinal lymph nodes or pleura ~lymphoma commences within abdominal cavity, disseminates and is un-amenable to complete surgical eradication ~lymphoma commences within vertebral column or paravertebral region along with incrimination of diverse sites ~lymphoma emerges within > singular extra-nodal site, is con ned to dual sides of diaphragm and may occur within bones or cutis ~ lymphoma incriminates >singular regional lymph node group and appears con ned to dual sides of diaphragm ~lymphoma emerges as a singular focus within bone and incriminates adjacent lymph nodes or extra-nodal site.

- Stage IV is comprised of lymphoma initially discerned within central nervous system as brain or spinal cord and/or bone marrow (replacement of >25% of bone marrow with neoplastic cells or 'blasts' is categorized as acute lymphoblastic leukaemia) [2, 5].

Stage I and stage II lymphomas con gure 'limited stage' disease. Stage III and stage IV lymphomas articulate 'advanced stage' disease [2, 5].

Burkitt's lymphoma is immune reactive to CD45, pan B cell markers as CD19, CD20, CD22, CD79a, PAX5 and germinal centre markers as CD10, BCL6. Ki67 proliferative index is ~ 100% [5, 6]. Majority of neoplastic cells depict MYC protein. Oil red O stain is employed to highlight intra-cytoplasmic lipid vacuoles [5, 6].

Burkitt's lymphoma is immune non-reactive to T cell markers as CD2, CD3, CD5, CD7. Neoplasm is immune non-reactive to BCL2 or TdT.

Burkitt's lymphoma requires segregation from neoplasms such as high grade B cell lymphoma with MYC and BCL2 or BCL6 genetic rearrangements, high grade B cell lymphoma not otherwise speci ed (NOS), blastoid variant of mantle cell lymphoma, di use large B cell lymphoma not otherwise speci ed (NOS), B lymphoblastic lymphoma, Burkitt-like lymphoma with 11q alteration or plasmablastic lymphoma. Pertinent karyotyping or uorescent in situ hybridization (FISH) can be employed for detection of c-MYC chromosomal translocation and appears con rmatory for Burkitt's lymphoma [5, 6].

Additionally, ~ 10% of typical Burkitt's lymphoma may be devoid of c-MYC genetic translocation, discernible by cytogenetics or uorescent in situ hybridization (FISH) [5, 6]. Peripheral blood examination delineates neoplastic cells imbued with an attenuated peripheral basophilic cytoplasm with several lipid vacuoles and spherical to elliptical nuclei [5, 6]. Elevated levels of serum lactate dehydrogenase (LDH) may occur, indicative of inferior prognostic outcomes [5, 6].

e aggressive Burkitt's lymphoma can be alleviated with diverse chemotherapeutic regimens such as R-CODOX-M / R-IVAC (rituximab, cyclophosphamide, vincristine, doxorubicin, methotrexate, ifosfamide, etoposide, cytarabine) or R-hyper-CVAD / R-MA (rituximab, cyclophosphamide, vincristine, doxorubicin, dexamethasone, methotrexate, cytarabine) [5, 6]. Intractable instances can be subjected to high intensity chemotherapy comprised of DA-R-EPOCH (rituximab, etoposide, prednisone, vincristine, cyclophosphamide, doxorubicin, and methotrexate).

Intra-thecal prophylaxis for treating central nervous system lymphoma with methotrexate along with or devoid of cytarabine is a component of aforesaid regimen [5, 6]. Relapsing or Burkitt's lymphoma unresponsive to aforesaid regimen can be treated with hematopoietic stem cell transplant [5, 6]. A paucity of pertinent randomized clinical trials is observed. With precise therapy, 5 year event free survival in children is ~ 90% whereas adults depict a 4 year event free survival at ~ 85% [5, 6].

Burkitt's lymphoma concordant with controlled HIV infection demonstrates superior event free survival. Disease occurring within lower socioeconomic zones delineates decimated survival. Extent of