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Original Research Article

CYTOGENETIC EVALUATION OF BURKITT-TYPE TRANSLOCATIONS IN HEMATOLYMPHOID MALIGNANCIES: A 5 YEARS STUDY AT REGIONAL CANCER CENTER

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Abstract

Background: Burkitt lymphoma (BL) is a high grade B-cell non-Hodgkin lymphoma (NHL) occurring in children and adults. The t(8;14)(q24.1;q32) and its variants – the t(2;8)(p12;q24.1) and t(8;22)(q24.1;q11.2) are associated with B-cell neoplasia and result in MYC/Immunoglobulin (IG) gene rearrangement which drive the oncogenesis. The majority of MYC/IG translocation is seen in BL which also is seen in other B cell neoplasms like DLBCL, precursor B cell ALL, and multiple myeloma. Objective: 1.To study the frequency and clinico-pathological features of Burkitt type translocations found in hematolymphoid malignancies. 2. To analyse the additional cytogenetic findings in these cases. **Materials and Methods:** All hematolymphoid cases showing Burkitt type translocations diagnosed from 2016 to 2021 were included in this study. Clinical details were collected from archived case files. Peripheral blood, bone marrow smears, histopathology and IHC slides were reviewed. Bone marrow flowcytometry and cytogenetic analysis was done.**Result:** This study included 55 cases showing Burkitt type translocations, comprising of 43 males and 12 females. We had 33 cases of Burkitt lymphoma (60%), 18 cases of Pre-B – ALL (32.7%), three cases of Multiple Myeloma (5.4%) and one case of CLL (1.8%). Majority of the cases presented with fever (43.6%) followed by abdominal pain and swelling (41.8%). Conventional karyotyping revealed t(8;14)(q24;q32) in 52 (94.5%)cases, t(8;22)(q24;q11) in 2 (3.6%) cases and t(2;8)(p12;q24) in one case. Complex karyotype and additional abnormalities were seen in 12.7% and 47.2% cases respectively. **Conclusion:** Burkitt type translocations are not unique to Burkitt lymphoma can be observed in other hematolymphoid malignancies. A complete clinio-copathological work up is essential for final diagnosis. The frequency and distribution of Burkitt type translocations in our series and association with 1q and 6q abnormalities is similar to the literature. Patient having complex karyotype and additional abnormalities carries a poor prognosis.

INTRODUCTION

Burkitt Lymphoma is an aggressive B-cell NHL originating from mature germinal or post germinal centre B-cells with quick doubling time. BL is also first cancer discovered to be pathologically driven by genetic translocation and the first cancer to be found highly chemosensitive.[1,2] BL is classified into three clinical subtypes – Endemic(eBL), Sporadic(sBL) and HIV-associated BL. Endemic BL occurs most commonly in children living in endemic and hyperendemic areas for malaria infection. The reason explained is malarial parasitemia augments the induction of typical translocation in lymphocytes by EBV involving c-MYC oncogene on chromosome 8 and immunoglobulin (IG) heavy or light chain loci on chromosome 2,14 and 22.[3,4] The eBL classically

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presents with jaw mass with or without abdominal disease showing a high CNS and low bone marrow involvement rate. Also shows that 100% association with EBV. sBL is seen worldwide occurring in childhood and young adults of 30 years median age group.[5] The Abdominal (ileo-caecal)region is the most common site to be involved. Other extranodal sites include bone marrow, ovaries, kidneys, and breasts.

The t(8;14)(q24.1;q32) translocation and its variants t(2;8)(p12;q24.1) and t(8;22)(q24.1;q11.2) involve the MYC gene on chromosome 8 typically with BL and acute lymphoblastic leukemia (ALL-L3).[6] In t(8;14), there is juxtaposition of MYC gene to IGH enhancer leading to its increased transcriptional activity and oncogenic transformation.[7] Variant translocation involves IGK gene on chromosome 2p12 and the IGL gene on chromosome 22q11.2. The 8q breakpoint is centromeric of MYC in t(8;14) and is telomeric in variant translocations. Therefore activation of MYC transpire on derivative of 14 in t(8;14) and on derivative 8 in variant translocations. They can also have auxiliary chromosome rearrangements in 60-70% of cases namely structural abnormality of chromosome 1q and 3q, trisomies 7 and 12 and losses of 6q,13q and 17p.[8] RNA sequencing studies have identified other genes apart from MYC that are seen in BL, such as mutations in TCF3 ,CCND3 and /or ID3.[9]

There is a paucity of information in the literature regarding the clinical significance of Burkitt-type translocations in non- Burkitt hematolymphoid malignancies and the role of additional chromosomal abnormalities in diagnosis and treatment response. Hence, this study was undertaken to evaluate the frequency of Burkitt-type translocations in various hematolymphoid malignancies and the role of additional chromosomal abnormalities.

Aim and Objective

To evaluate the frequency of Burkitt-type translocations seen in hematolymphoid malignancies and correlate with clinico-pathological parameters and analysis of additional cytogenetic abnormalities.

MATERIALSANDMETHODS

All cases whose karyotyping showed Burkitt-type translocations and other associated abnormalities, in the Department of Cytogenetics (Pathology), Kidwai Memorial Institute of Oncology, Bengaluru were included in the study. The study period was from 2016-2021. Reports of the laboratory investigations which included routine hematology and biochemistry such as CBC, and serum LDH were archived from case files. Immunohistochemistry was performed on available biopsy specimens that were formalin-fixed paraffin-embedded tissue using heat-induced epitope retrieval method utilizing the panel of LCA, CD3, CD20, BCL2, BCL6, TdT, and ki-67. Immunophenotyping was done on bone marrow aspirates and aspirated body cavity fluids using 10 color flow cytometry with the panel of markers that included CD45, CD10, CD20, CD23, CD79b, CD5, and surface IgM/D light chains.

For karyotyping, the samples were subjected to 24-48 hours of unstimulated culture. GTG banding as per the standard protocol and analysis of 15-20 metaphases was done. Results were reported using GenASIs software and in accordance with the International System for Human Cytogenetic Nomenclature (ISCN 2020).

RESULTS

There were 55 analyzed cases of Burkitt translocation and structural chromosomal abnormalities. The predominance of male gender (43/55) was noted with male to female ratio of 3.6:1. The Median age was 19 years in the present study about 60%(33/55) were from the pediatric age group and around 40%(22/55) were adult population. Fever (24 cases, 43.6%) was the most common, yet vague symptom, followed by abdominal swelling and pain (23 cases, 41.8%). Head and neck swelling (4 cases, 7.2%) ranks next. Three cases (5.4%)presented with generalized weakness and a single case (1.8%)with involvement of bilateral breasts.

The study population were categorized into four distinct groups based on cytogenetic abnormalities and type of tumor.Thirty-three cases (60%) were diagnosed classically as Burkitt lymphoma, 18 cases (32.7%) were pre B-cell ALL, three cases (5.4%) were multiple myeloma, and a solitary case (1.8%) of chronic lymphocytic lymphoma (CLL).

The median hemoglobin concentration was 9 g/dl and the median WBC count 11. 4 X 10^9/L. The median platelet count was 54 X 10^9/L whereas the median blast count was 84%. The serum LDH was >700 IU/L in 87% of cases.

Now, giving a detailed picture of these 33 cases, 19 (57.5%)cases showed t(8;14)(q24;q32) (Fig 1A) within which one case had an additional marker chromosome and another case showed 11q23 with it.One (3%) case each of t(8;22)(q24;q11) and t(2;8)(p12;q24) translocations was seen. Seven cases (21.2%) had t(8;14)(q24;q32) with duplication of chromosome 1 and two cases (6%) had t(8;14)(q24;q32) with duplication of chromosome 6. The remaining three cases (9%) had complex karyotype. About 18%(6/33) showed bone marrow involvement with immunophenotyping consistent with Burkitt leukemia with the expression of B cell-specific antigen CD19, CD20, CD10 and absence of Tdt and CD34. [Table 1].

Of the 24 cases who received induction chemotherapy, 15 completed induction while 09 cases discontinued or lost to follow-up. Total 09 patients achieved complete remission, 10 cases

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relapsed and 05 died. The median time to relapse or death after the response was 6 months.

There were 18 cases (32.7%) of precursor B cell ALL. Seven cases (38.8%) among them had translocation t(8;14)(q24;q32) of which two cases had additional trisomy1 and trisomy7 respectively. Four cases (22.2%) had t(8;14)(q24;q32) with duplication of chromosome 1, one of them also had der(19). Two cases (11.1%) had t(8;14)(q24;q32) with deletion of chromosome 6. One case with deletion 4 with Trisomy 7. The four cases (22.2%) had complex karyotype. Immunophenotype of all 18 cases are positive for CD34,HLA-DR,CD19,CD10,CD79a,cyCD22 and Negative for Myeloid and T cell markers.[Table 2]. Of the 12 cases who received induction chemotherapy, 04 completed induction while 05 cases discontinued or lost to follow up. Total 04 patients achieved complete remission, 06 cases relapsed and 05 died. The median time to relapse or death after the response was 3 months. Most cases with early relapse or death had additional abnormalities or complex karyotype.

There were three cases(5.45%) of multiple myeloma. One of which showed t(8;22)(q24;q11) was continuous remission at follow up 36 months and the other two cases showed hyperdiploid complex karyotype with t(8;14)(q24;q32). One patient succumbed and the other lost for follow-up. [Table 3]

A solitary case (1.8%) of CLL showed t(8;14)(q24;q32) with a marker chromosome.

Conventional Karyotyping results were divided into simple and complex abnormalities (i.e, more than three structural abnormalities). Among complex karyotype, the most common structural abnormalities were associated with the involvement of chromosome 1in 10 cases (18.18%) followed by chromosome 6 in seven cases (12.72%) and chromosome 7 in six cases (10.9%).FISH was done in 21(38%) cases showing c-MYC rearrangement. [Figure 1B]

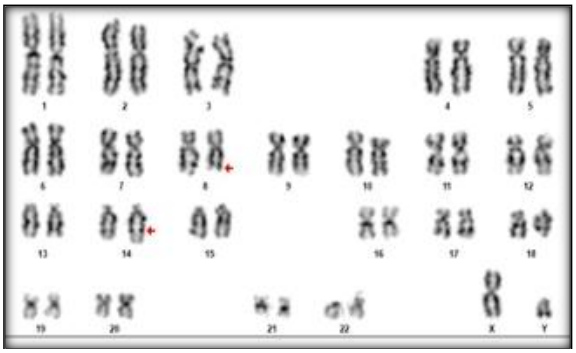

Figure 1A: GTG Banding Karotype,x1000 Objective,46,XY,t(8;14)(q24;q32)

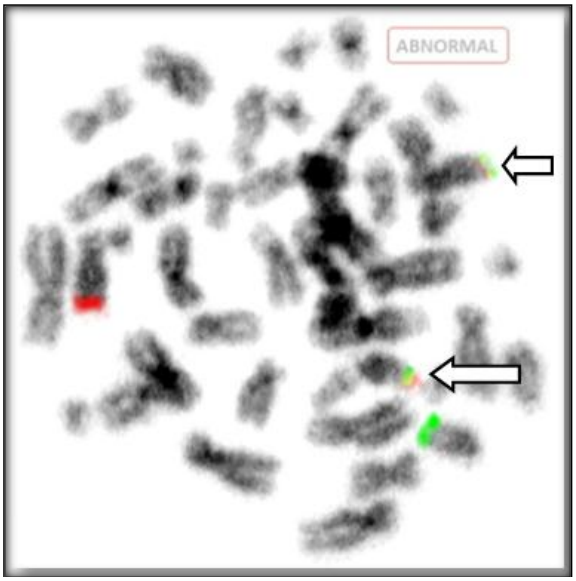

Figure 1B: FISH using Dual color dual fusion probe showed two red-green fusion(IGH/C-MYC -Arrow), one red(C-MYC-8q24) and one green(IGH-14q32) signals suggesting positivity for IGH/C-MYC translocation.

Table 1: Karyotyping in Burkitt lymphoma cases

Sl no	Burkitt Lymphoma
1	46,XY,t(8;14)(q24;q32)(17 cases)
2	47,XX,t(8;14)(q24;q32),+mar
3	46,XY,t(8;14)(q24;q32),add(11)(q23)
4	46,XY,dup(1)(q31q32),t(8;14)(q24;q32)
5	46,XY,dup(1)(q22q32),t(8;14)(q24;q32)/46,XY,t(8;14)(q24;q32)
6	48,XY,dup(1)(q21q32),del(2)(p23),del(3)(q21),+3,t(8;14)(q24;q32),+mar
7	46,XY,dup(1)(q21q25),add(7)(q31),t(8;14)(q24;q32)
8	46,XY,dup(1)(q21q42),del(6)(q23),t(8:14)(q24:q32)add(15)(p11.2)
9	47,XY,dup(1)(q21q24),del(3)(p21),t(8;14)(q24;q32),+mar
10	46,XX,dup(1)(q12q25),t(8;14)(q24;q32)
11	46,XY,del(6)(q21q25),t(8:14)(q24:q32)
12	49,XY,+3+6,del(6)(q13)+7.t(8;14)(q24;q32)
13	46,XX,t(8;14)(q24;q32),der(11)t(7;11)(q11;q23)
14	48,X,del(X)(p32),-4,+5,del(9)(p22)x2,-13,der(14)t(8:14)(q24;q32)r(8)(p22q24),-18,+20,+19,+mar1,+mar2
15	72-84<3n>,XY,+X,+X,+5,t(8;14)(q24;q32) x2,+12,+14,+16,-17,+18
16	46,XY,t(2;8) (p12;q24)
17	45,X,t(X;15)(p22;q22),der(8)t(8:22)(q24;q11),-13,-22,+mar

Table 2: Karyotyping in Precursor B cell ALL

Sl no	Precursor B Cell ALL
1	46,XY,t(8;14)(q24;q32)(5 Cases)
2	47,XY,+1,t(8;14)(q24;q32)

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3	47,XY,+7,t(8;14)(q24;q32)
4	47,XY,dup(1)(q21q32)+7,t(8:14)(q24:q32/47,XY+mar/46,XY
5	46,XY,dup(1)(q21q25),t(8;14)(q24;q32)/46,XY
6	46,XX,dup(1)(q21q22),add(4)(q35),del(6)(q13),t(8;14)(q24;q32)
7	46,XY,t(8;14)(q24;q32), add(17)(p13),der(19)t(1;19)(q23;p13).
8	46,XY,del(6)(q21q25),t(8;14)(q24;q32)
9	46,XY,del(6)(q21q25),t(8;14)(q24;q32)
10	47,XY,del(4)(q31),+7,t(8;14)(q24;q32)
11	46,XY,-3,t(8;14)(q24;q32),-13,+2 mar
12	46,XY,t(7;15)(q32;q15),t(8;14)(q24;q32),add(19)(p13)
13	46,XY,t(8;14)(q24;q32),i(9)(q10),t(9;17)(p13;p13),del(12)(p12)
14	46,XX,t(8;14)(q24;q32),t(14;15)(q32;q22)

Table 3: Karyotyping in Multiple Myeloma cases

Sl no	Mutiple Myeloma
1	46,XY.t(8;22)(q24:q11)
2	52,XY,+X,del(6)(q22),+7,+7,-8,del(8)(q22),der(14),t(8;14)(q24;q32),+11,+3 mar/46,XY
3	49,XX,t(5;12)(q11;q15),+7,t(8;14)(q24;q32),+12,+15/46,XX

Table 4: Comparison of Various studies

Sl no		Pamu PK et al(5)	Agni M et al(6)	Sariban et al(10)	Booth et al(11)	Cavdar et al(12)	Mwanda et al(13)	Present Study
1	Study Period	08 years	12 years	08 years	10 years	22 years	05 years	05 years
2	Number of cases	37	34	100	37	72	1005	55
3	Median Age	31 years	37 years	15 years	5 years	5.9 years	13 years	19 years
4	M:F Ratio	2:1	5.8:1	3.1:1	2.7:1	2:1	1.5:1	3.6:1
5	Site	Bone Marrow-21 Lymph Node-12 CNS-5	Bone Marrow-34	Jaw-28 IleoCaecal-22 CNS-15 Others-35	Mesentric Lymph nodes-29 Bone Marrow-08	Abdomen-44 Jaw -12 Bone Marrow-13 Others-03	Jaw -563 Abdomen-282 Bone Marrow-112 Others-48	Adomen-23 Head and Neck-4 Breast-01 Others-27
6	Chief Complaints	Fever, Anemia, Lymphadenopathy	Not Aviable	Loose tooth,Tooth Ache	Jaw swelling	Jaw swelling	Jaw Swelling	Fever
7	Bone Marrow involvement	67%	100%	23%	32%	56%	39%	47%

DISCUSSION

Burkitt lymphoma is a highly aggressive B cell NHL, with amazing cure rates, if the intensive multidrug regimen of chemotherapy is started well in advance. Its diagnosis is preferentially made with a mature B cell immunophenotype and classic chromosomal translocation involving MYC proto-oncogene on 8q24 and one of the immunoglobulin gene on 14q, 2p or 22.[6] The majority of MYC/IG translocation is seen in BL which also is seen in other B cell neoplasms like DLBCL, precursor B cell ALL, and multiple myeloma. Very rarely the t(8;14) is seen with an additional translocation involving genes such as BCL2 on 18q21, BCL6 on 3q27 or BCL1 on 11q23 (double hit/ triple hit lymphoma).

Angi M et al,[6] reported median age of 37 years and in our study, the overall median age was 19 years (2-61years). Our study median age was somewhat similar to Sariban et al (15yrs).[10] Various clinic pathological parameters such as study period, median age, M:F ratio, site, chief complaints and bone marrow involvement compared to other studies are shown in [Table 4].

The most common translocation being t(8;14) is seen in 75-85% of BL, where t(8;22) and t(2;8) are seen in 10-21% and 3-4% respectively(7,8). Our findings were almost similar with a high percentage of t(8;14) about 95%. The t(8;14) was seen in 94.5%, t(8;22) was seen in 3.6% and t (2;8) was seen in 1.8%. Thirty-three cases (60%) were diagnosed classically as Burkitt lymphoma, 18 cases (32.7%) were pre-B-cell ALL, three cases (5.4%) were multiple myeloma, and a solitary case (1.8%) of chronic lymphocytic lymphoma (CLL). Moorman et al,[14]reported pre B ALL in 19% and Angi M et al,[6] found pre B- ALL only 17% whereas in present study we found pre B-ALL phenotypes in 32.7% and whereas

We had patients with additional abnormalities in 47.2%, complex karyotypes in 12.7%, abnormalities of chromosome 1 were seen in 18.18%, gain of chromosome 7 in 10.9%, and other abnormalities in 20%. In other studies, additional abnormalities have been reported in 60-70% of patients and complex karyotypes in 30-50%.[8,13]1q structural abnormalities were seen in 30%cases, abnormalities of chromosome 13 were seen up to 17%, gains of chromosome 7 and 12 and losses of 6q and 17p are the next common abnormalities.[8,15] Abnormalities of chromosome 1q and trisomies of 7 and 12 are seen as mutually exclusive.[16] As compared to Angi M et al,[6] where they had 91% additional

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abnormalities, 68% Complex Karyotype, and 41% of 1q abnormalities we had less percentage. About 47.2% of cases had additional abnormalities and majority of cases with complex karyotype and additional abnormality had a relapse or death which was similar to the other studies done by Angi M et al,[6] and Mukhtar et al.[17]

t(8;14) in CLL is a rare event, which is thought to be of poor prognosis. The most common aberrations in CLL are trisomy12 and deletions of 13q,11q,6q,14q, and 17p. Immunophenotype positive for CD19,CD20,CD23,CD5,monotypic IgK light chain and negative for CD10. Patients are treated with Bendamustine and combination drugs. If ZAP70 and CD38 are negative they have a poor prognosis. Genomic aberrations are important independent predictors of disease progression and survival. According to the largest series published in 2012, these translocations occur in < 1% of CLL.[18,19]

All the translocations involving MYC/IG have been reported in Multiple Myeloma and signify the progression of the disease. The t(8;14) is more common than variant translocations as compared to other B cell neoplasms. In our study one case of t(8;22) (q24:q11) and the other two cases of t(8;14) were associated with hyperdiploid complex karyotype where as a study done by Angi M et al,[6] showed all three variants translocations in Multiple Myeloma associated with hypodiploid karyotype showing monosomy 13.

CONCLUSION

Burkitt lymphoma is an aggressive non-Hodgkin lymphoma yet has high curable rates. The starry sky morphology is its classical exhibition having monomorphic intermediate-sized lymphoid cells with prominent punched-out vacuoles, positive for lipid vacuoles. It requires aggressive chemotherapy. Burkitt type translocations are not unique to Burkitt lymphoma can be observed in other hematolymphoid malignancies .A complete clinicopathological work up is essential for final diagnosis. As per our knowledge, this is the largest case series from South India. The frequency and distribution of these translocations in our series and association with 1q and 6q abnormalities is similar to the literature. Patient having complex karyotype and additional abnormalities carries a poor prognosis.

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