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Review Article

Leukaemia in children

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ABSTRACT

Leukemia, albeit an uncommon ailment, surpasses a reason for death from huge numbers of the intense transferable disorder due to its deadly character. It is described by far reaching, quick, and confused multiplication of leukocytes. In India, leukemia is the most widely recognized youth malignant growth with a relative extent fluctuating somewhere in the range of 25% and 40% and keeps on being the biggest supporter of malignancy related mortality in children.

1. Introduction

Leukemia is a harm with scattered multiplication of young or shoot cells of the bone marrow, which supplant the ordinary marrow components and tend to aggregate in different tissues of the body.[1] Leukemia was first distinguished by scientists, Virchow and Bennet in the year 1845.[2] European doctors in the nineteenth century were the soonest spectators of patients who had extraordinarily expanded white cell tallies. The expression "Weisses Blut" or "white blood" developed as an assignment to this issue. Afterward, the term leukemia, which is, got from the Greek word "leukos," signifying "white," and "haima," which means blood was utilized to show the disease.[3] Leukemia, albeit an uncommon illness, surpasses a reason for demise from a large number of the intense transferable ailments as a result of its lethal character[4] It is described by far reaching, fast, and messy multiplication of leukocytes and their antecedent and the nearness of youthful leukocytes in the blood regularly in extremely huge numbers unexceptionally sooner or later during the course of the disease.[5] Leukemias are normally classifi ed concurring to their clinical conduct (intense or constant) or histogenesis (myeloid or lymphocytic/lymphoblastic). Henceforth, there are four primary kinds of leukemia, in particular incessant lymphocytic leukemia (CLL), constant myelogenous leukemia (CML), intense lymphocytic leukemia (ALL), and intense myelogenous leukemia (AML). As per the Leukemia and Lymphoma Society, USA, there were roughly 13,410 new instances of AML, 5,200 new instances of ALL, 4570 instances of CML and 15,110 instances of CLL analyzed in the year 2007-2008 in USA. Once more, this general public has revealed in the year 2010-2011 that blood tumors would represent 9.0% of the 1,529,560 new malignant growth cases analyzed in the US this year. Leukemia alone[6] involves 27.5% of malignant growths influencing the youngsters matured 0-19 years in United States. It further states that each 4 min, one individual in the United States is determined to have a blood malignant growth. Indeed, even in Britain the second biggest supporter of mortality from youth malignant growth is leukemia.[7]

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In India, leukemia is the most widely recognized youth malignant growth with a relative extent changing somewhere in the range of 25% and 40% also, keeps on being the biggest supporter of cancerrelated mortality in children.[8] Sixty to 85% everything being equal detailed are intense lymphoblastic leukemia. Revealed yearly frequency of ALL is around 9-10 cases for each 100,000 populace in childhood.[9] Compared to the created world, the science of ALL seems distinctive in India, with a higher extent of T-cell ALL (20-half as contrasted with 10-20% in the created world), hypodiploidy what's more, translocations t(1;19), t(9;22), and t(4;11), the entirety of which add to a less fortunate anticipation of this leukemia.[10–13]

1.1. Etiology

1.1.1. Genetic basis

On the whole, chromosomal translocations happen normally. It is felt that most translocations happen during pre-birth advancement. These translocations cause a adjustment of qualities, which thusly to change of proto-oncogene into an oncogene. The oncogene causes leukemia either by animating cell division or by hindering the customized cell demise called apoptosis. A translocation can enact a proto-oncogene by two various components.[14] An increasingly visit occasion is a merger of two qualities to shape a combination quality that produces anomalous illusory protein instigating leukemia. As an model, translocation t(1; 19) in ALL makes the combination of E2A (immunoglobulin enhancer-restricting components E12/ E47) and PBX1 (pre-B-cell leukemia translation factor 1) qualities. In the E2A-PBX1 combination, protein transactivating areas of E2A are joined to the DNA-restricting space of PBX1, which modifies the transcriptional properties of the PBX1 translation factor.[15,16] Inactivation of a tumor silencer quality is another occasion that may start leukemia. Tumor silencer qualities are basic for typical cell improvement, and they forestall carcinogenesis. Not many tumor silencer qualities have been announced in intense leukemias. Screening for chromosomal areas with loss of heterozygosity is one approach to follow novel tumor silencer qualities. In youth ALL, short arms of chromosomes 9 and 12 (in around 30-40% and 25-30% of the patients, individually) are the locales that most much of the time show loss of heterozygosity.[17,18]

The other system by which a translocation causes leukemia is move of a regularly calm interpretation factor quality to the area of dynamic advertiser or enhancer components, which quicken the capacity of the quality. For model, in translocations t(8;14), t(2;8), and t(8;22) in Burkitt leukemia, the quality encoding the MYC translation factor is presented to the enhancer components of an immunoglobulin quality. These enhancer components cause overexpression of the MYC quality, which is significant in the guideline of cell division and cell death.[19] Further portrayal of these qualities uncovered that they are regularly included straightforwardly or in a roundabout way in the turn of events and homeostasis of typical platelets, and that unusual protein results of combination qualities made by specifi c translocations and reversals can deregulate multiplication, separation or modified cell demise (apoptosis) of platelet precursors.[20,21]

1.2. Risk Factor

1.2.1. Ethnicity

Indian populace being multicultural and multiethnic have saved their genetic stock in light of the standing framework what's more, intra rank marriage necessity. Hindus (counting Sikhs, Buddhists, Jains) comprise 85% of the populace, what's more, 15% strict minorities are included Muslims what's more, Christians. The records of leukemia include 86.5% of Hindus and rest for different religions which are in agreement with their population.[22]

1.2.2. Blood Group

The huge nearness of AML has been seen in a wide range of blood gatherings however not with different sorts of leukemias. Information have demonstrated a signifi cant relationship of ABO blood gatherings and various malignant growths like duodenal ulcer,[23] gastric cancers[24] and so on. Macmohan et al. have indicated a propensity of leukemia to happen less much of the time in people of gathering O than in people of gathering B and AB[25] However, Modak et al. invalidates the idea of propensity of a specific blood bunch toward leukemia.[26]

1.2.3. Sex differences

Mens show a higher hazard for all types of leukemia with the by and large proportion of 1:8.1. There is no clarification for females to be shielded against leukemia.[27] In an examination from Haryana by Kumar et al., there were 70.2% kids and 29.8% grown-up patients of ALL in which male to female proportion was 2.03:1.[28]

1.2.4. Geographic

It has been recommended that T-cell ALL prevails in monetarily hindered zones, yet with urbanization, industrialization, and expanding prosperity rate of ALL have increased.[29] ALL is accounted for to be the most visit in the south[30] and moderate in the East, West,[31] and focal India.[32] Interestingly, the occurrence of ALL is lesser in east India.[33] Just as Northern areas.[34] However, this has be resolved if this is a valid contrast or an enrollment ancient rarity (disease enlistment in the North East began in 2003).

1.2.5. Age

The age circulation of offspring of ALL in created nations shows an exceptionally checked early top among 2 and 5 years, trailed by a little top somewhere in the range of 11 and 15 years what's more, the middle age of 4 years.[35–38] There has been a steady increment in the rate of ALL in the previous 25 years.[39]

1.2.6. Ionizing radiation

Ionizing radiation is considered as a known reason for ALL. The hazard is additionally higher for those uncovered at an prior age[40] and optional leukemias in the people rewarded by radiotherapy.[41] Radiation from atomic force plants[42] and X-beam assessments of pregnant ladies might be related with expanded danger of adolescence ALL.[42] Postnatal introduction of newborn children for demonstrative X-beam expanded the hazard by 60%.[43] and was related with of ALL, specifi cally B-cell ALL however no AML or T-cell ALL.[44]

1.2.7. Pesticides

Home utilization of various different pesticides put the youngsters into danger of building up ALL. Introduction of anticipating moms to solvents, paints, or thinners expanded the danger of ALL in kids. The dad's introduction to plastics before origination was additionally connected with more serious hazard. Besides, time of presentation is a significant factor.[45]

1.2.8. Electromagnetic field

Youngsters living close to high voltage power establishments were bound to be found to have leukemia than other children.[46] One late investigation found that danger of leukemia was raised when introduction to electromagnetic field was steady over the term of the pregnancy and in cases where the plan of the water framework in the home prompted "ground flows" from associations between plumbing pipes and the establishing for the electricity.[47]

1.3. Diagnosis

Everything is determined to have clinical history, physical assessment, fringe blood spreads, bone marrow biopsy, cytogenetics, and immunophenotyping. The higher the white platelet (WBC) checks, the more regrettable the prognosis.[48] Pathological assessment, cytogenetics (in specific for the nearness of Philadelphia chromosome), what's more, immunophenotyping build up whether leukemia is myeloblastic (neutrophils, eosinophils, or basophils) or lymphoblastic (B lymphocytes or T lymphocytes) and distinguish the cell surface antigens communicated by the tumor cells. RNA testing can set up how forceful the infection is, various changes have been related with shorter or on the other hand longer endurance. Clinical imaging can discover metastasis to different organs generally the lung, liver, spleen, lymph nodes, mind, kidneys, and regenerative organs.[49,50]

1.3.1. Peripheral blood smear

Microscopic assessment of leukemia influenced tissue appears diffuse penetration and demolition of the ordinary host tissue by sheets of inadequately separated cells with either myelomoncytic attributes or lymphoid highlights. Impact cells are seen on the blood smear in lion's share of cases.[51]

1.3.2. Bone marrow biopsy

Bone marrow biopsy is regularly acted related with the fringe blood smear since certain patients may experience through an aleukemic stage in which atypical cells are missing from the circulation.[52]

1.3.3. Immunophenotyping

The phases of ALL incorporate early pre-B ALL, regular ALL, Pre-B-cell ALL, develop B-cell ALL (Burkitt leukemia), pre-T-cell ALL, and develop T-cell ALL.[53] B-and T-cell lymphoblastic leukemia cells express surface antigens that equal their individual ancestry improvements. Antecedent B-cell ALL phones regularly express CD10, CD19, and CD34 on their surface along, with atomic terminal deoxynucleotide transferase (TdT), while forerunner T-cell ALL cells usually express CD2, CD3, CD7, CD34, and TdT.[54] In an investigation by Bayram et al. the most much of the time distinguished fi ve antigens were I2, CD10, CD41, CD2, and CD7/CD19 at the hour of conclusion and CD41, I2, CD10, CD19, and CD2 at the hour of backslide. Stream cytometric examinations uncovered that antigen levels decided at the hour of conclusion expanded or diminished by 10% at the hour of relapse.[55] CD19 is additionally communicated on the most punctual B-antecedent lymphocytes that are dangerously changed altogether.

1.3.4. Cytogenetics

Numerous specialized troubles make it hard to pick up data for chromosomal discoveries altogether. Chromosome concentrates in ALL show poor morphology; chromosomes will in general spread inadequately, and seem obscured what's more, fluffy with ill defined edges, making banding examines testing or even impossible.[56,57] Williams distinguished clonal karyotypic variations from the norm in 94% to 98% of cases of ALL.[58] most of instances of ALL illustrate an anomalous karyotype, either in chromosome number (ploidy) or as auxiliary changes, for example, translocations, reversals or cancellations. These progressions were identified in just 50% of ALL patients in the fi rst banding studies.[59] Upgrades in spreading and banding procedures have brought about

higher paces of location, and most concentrates currently report chromosomal changes in 60-85% of ALL cases.[60–63] The Third International Workshop on Chromosomes in Leukemia found most of cytogenetic changes in instances of B antecedent ALL, with just 39% happening in T-cell ALL.[64,65] Most investigations on karyotypic variations from the norm and their clinical significance have been acted in youth ALL. Grown-up ALL indicated nonrandom chromosomal variations from the norm like those found in youth ALL, however their circulation and their organic signifi cance were unique. In any case, in grown-up ALL the job of cytogenetics in tolerant the executives has generally been focused on the nearness of the Philadelphia (Ph) chromosome which as a rule emerges from t(9;22)(q34;q11.2) and results in BCR-ABL fusion. Among the few changes, ploidy appropriation also, repetitive translocation related with explicit morphology and immunophenotype are well-recognized in ALL.

2. Treatment

Leukemia is generally rewarded with chemotherapy, light, or then again bone marrow transplantation. Chemotherapy and radiotherapy are commonly cytotoxic for quickly duplicating dangerous cells, yet in addition adversely sway the creation of ordinary hemopoietic and secretary cells as these don't separate among ordinary and dangerous cells. This side impact regularly brings about resistant concealment and diminished discharges in the body. The foundational sequelae subsequently of this medicine or radiation can likewise prompt a number of oral and dental confusions. The patient with malignant growth faces an attack on oral wellbeing from both the ailment and the treatment alternatives. The immediate and aberrant sick impacts to the oral cavity are related with the turn of events of ulcerative, hemorrhagic, or irresistible complications.[66]

3. Conclusion

In India, youngster wellbeing is a need medical problem, and we are advancing toward lessening contamination related adolescence passings. In any case, youth malignancy isn't yet a significant zone of core interest, furthermore, it isn't satisfactory to disregard these youngsters as they have an improving probability of fix with fitting treatment.

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None.

5. Conflict of Interest

None.

References

1. Ferretti GA, Ash RC, Brown AT, Largent BM, Kaplan A, Lillich TT, et al. Chlorhexidine for prophylaxis against oral infections and associated complications in patients receiving bone marrow transplants. *J Am Dent Assoc* . 1987;114(4):461–7.
2. Genc A, Atalay T, Gedikoglu G, Kar BZ, Kullu S. Leukemic children: Clinical and histopathological gingival lesions. *J Clin Pediatr Dent*. 1998;22:253–6.
3. Subramaniam P, Babu KG, Nagarathna J. Oral Manifestations In Acute Lymphoblastic Leukemic Children Under Chemotherapy. *J Clin Pediatr Dent* . 2008;32(4):319–24.
4. Meadors GF. Epidemiology of Leukemia: PHR Review. *Public Health Rep* . 1956;71(2):103–8.
5. Forkner CE. Leukemia and Allied Disorders. New York: Macmillan Co; 1938. p. 333.
6. Ries LA, Percy CL, Bunin GR, Ries L, Smith M, Gurney JG. SEER Pediatric monograph. In: and others, editor. Cancer incidence and survival among children and adolescents: United States SEER program 1975-1999 [NIH (Pub. No. 99-4649]. Bethesda (MD): National Cancer Institute, SEER program; 1999; 1999. Available from: //www.seer.cancer.gov.
7. Stiller C. Childhood Cancer in Britain: Incidence Survival, Mortality. Oxford: Oxford University Press; 2007.
8. Ribera JM, Oriol A. Acute Lymphoblastic Leukemia in Adolescents and Young Adults. *Hematol Oncol Clin North Am* . 2009;23(5):1033–42.
9. Amare P, Gladstone B, Varghese C, Pai S, Advani S. Clinical signifi cance of cytogenetic fi ndings at diagnosis and in remission in childhood and adult acute lymphoblastic leukemia: Experience from India. *Cancer Genet Cytogenet*. 1999;110:44–53.
10. Magrath I, Shanta V, Advani S, Adde M, Arya LS, Banavali S, et al. Treatment of acute lymphoblastic leukaemia in countries with limited resources; lessons from use of a single protocol in India over a twenty year peroid. *Eur J Cancer* . 2005;41(11):1570–83.
11. Rajalekshmy KR, Abitha AR, Pramila R, Gnanasagar T, Maitreyan V, Shanta V, et al. Immunophenotyping of acute lymphoblastic leukaemia in Madras, India. *Leuk Res* . 1994;18(3):183–90.
12. Siraj AK, Kamat S, Gutiérrez MI, Banavali S, Timpson G, Sazawal S, et al. Frequencies of the major subgroups of precursor B-cell acute lymphoblastic leukemia in Indian children differ from the West. *Leukemia*. 2003;17(6):1192–3.
13. Escalon EA. Acute lymhocytic leukemia in childhood. *Int Pediatr*. 1999;4:83–9.
14. Smith MA, Seibel NL, Altekruse SF, Ries LAG, Melbert DL, O'Leary M, et al. Outcomes for Children and Adolescents With Cancer: Challenges for the Twenty-First Century. *J Clin Oncol*. 2010;28(15):2625–34.
15. Lu Q, Wright DD, Kamps MP. Fusion with E2A converts the Pbx1 homeodomain protein into a constitutive transcriptional activator in human leukemias carrying the t(1;19) translocation. *Mol Cell Biol* . 1994;14(6):3938–48.
16. Hunger SP, Lu X, Devidas M, Camitta BM, Gaynon PS, Winick NJ, et al. Improved Survival for Children and Adolescents With Acute Lymphoblastic Leukemia Between 1990 and 2005: A Report From the Children's Oncology Group. *J Clin Oncol* . 2012;30(14):1663–9.
17. Baccichet A, Benachenhou N, Couture F, Leclerc JM, Sinnett D. Microsatellite instability in childhood T cell acute lymphoblastic leukemia. *Leukemia*. 1997;11(6):797–802.
18. Moorman AV, Richards SM, Martineau M, Cheung KL, Robinson HM, Jalali GR, et al. Outcome heterogeneity in childhood high-hyperdiploid acute lymphoblastic leukemia. *Blood*. 2003;102:2756–62.
19. Knudson AG. Chasing the Cancer Demon. *Annu Rev Genet* . 2000;34(1):1–19.
20. Besson C, Gonin C, Brebion A, Delaunay C, Panelatti G, Plumelle Y, et al. Incidence of hematological malignancies in Martinique, French West Indies, overrepresentation of multiple myeloma and adult T cell leukemia/lymphoma. *Leukemia*. 2001;15(5):828–31.
21. Yanada M, Jinnai I, Takeuchi J, Ueda T, Miyawaki S, Tsuzuki M, et al. Clinical features and outcome of T-lineage acute lymphoblastic leukemia in adults: A low initial white blood cell count, as well

as a high count predict decreased survival rates. *Leukemia Res*. 2007;31(7):907–14.
22. Rathee R, Kamal N, Kumar A, Vashist M, Yadav R. Dermatoglyphic PaĴ erns of Acute Leukemia Patients. *Int Res J Biological Sci*. 2014;3:90–3.
23. Taub JW, Konrad MA, Ge Y, Naber JM, Scott JS, Matherly LH, et al. High frequency of leukemic clones in newborn screening blood samples of children with B-precursor acute lymphoblastic leukemia. *Blood*. 2002;99(8):2992–6.
24. Greaves MF, Maia AT, Wiemels JL, Ford AM. Leukemia in twins: lessons in natural history. *Blood*. 2003;102(7):2321–33.
25. Greaves M. In utero origins of childhood leukaemia. *Early Hum Dev*. 2005;81:123–9.
26. Modak H, Kulkarni SS, Kadakol GS, Hiremath SV, Patil BR, Hallikeri U, et al. Prevalence and risk of leukemia in the multi-ethnic population of North Karnataka. *Asian Pac J Cancer Prev*. 2011;12:671–5.
27. Clarke CA, Edwards JW, Haddock DRW, Howel-Evans AW, McConnell RB, Sheppard PM, et al. ABO Blood Groups and Secretor Character in Duodenal Ulcer. *BMJ*. 1956;2(4995):725–31.
28. Roberts JAF. Blood Groups and Susceptibility to Disease. *Br J Prev Soc Med* . 1957;11(3):107–25.
29. Macmahon B, Folusiak JC. Leukemia and ABO blood group. *Am J Hum Genet*. 1958;10:287–93.
30. Jackson N, Menon BS, Zarina W, Zawawi N, Naing NN. Why is acute leukemia more common in males? A possible sex-determined risk linked to the ABO blood group genes. *Ann Hematol* . 1999;78(5):233–6.
31. Kumar A, Rathee R, Vashist M, Neelkamal N, Singh S, Gupta S, et al. Acute Lymphocytic Leukemia: An epidemiological and Hematological Study from Haryana. *Biosci Biotechnol Res Asia*. 2012;9(2):813–7.
32. Ramot B, Magrath I. Hypothesis: The environment is a major determinant of the immunological sub-type of lymphoma and acute lymphoblastic leukaemia in children. *Br J Haematol*. 1982;50(2):183–9.
33. Varghese PR, Elayidom NB, Joseph CD, Kumar S. Epidemiological observations on leukemia in Kerala (A study of 1016 cases over three years). *Indian J Haematol*. 1984;2:15–7.
34. Advani SH, Jussawalla DJ, Rao DN, Gangadharan P, Y PAS. A study of 1126 leukaemia cases - Epidemiologic and end result analysis. *Indian J Cancer*. 1979;16:8–17.
35. Pradhan PK, Tiwari SK, Dabke AT, Agarwal S. PaĴ ern of leukaemia in Raipur (Madhya Pradesh) - An analysis of 162 cases. *Indian J Cancer*. 1982;19:20–3.
36. Rani S, Beohar PC, Mohanty TK, Mathur MD. Leukaemic pattern in Delhi - A ten year study of 490 cases. *Indian J Cancer*. 1982;19:81–6.
37. Hanson MR, Mulvihill JJ. Epidemiology of child-blood cancer. Cancer in the Young. NewYork: Masson; 1980. p. 3–12.
38. Draper GJ, Kroll ME, Stiller CA. Childhood cancer. *Cancer Surv*. 1994;19(20):493–17.
39. Gurney JG, Davis S, Severson RK, Fang JY, Ross JA, Robison LL, et al. Trends in cancer incidence among children in the U.S. *Cancer*. 1996;78(3):532–41.
40. Holmes L, Hossain J, desVignes Kendrick M, Opara F. Sex Variability in Pediatric Leukemia Survival: Large Cohort Evidence. *ISRN Oncol*. 2012;2012:1–9.
41. Schmitz-Feuerhake I, Dannheim B, Heimers A, Oberheitmann B, Schröder H, Ziggel H, et al. Leukemia in the proximity of a German boilingwater nuclear reactor: Evidence of population exposure by chromosome studies and environmental radioactivity. *Environ Health Perspect*. 1997;105(6):1499–504.
42. Doll R, Wakeford R. Risk of childhood cancer from fetal irradiation. *Br J Radiol* . 1997;70(830):130–9.
43. Infante-Rivard C, Fortier I, Olson E. Markers of infection, breast-feeding and childhood acute lymphoblastic leukaemia. *Br J Cancer* . 2000;83(11):1559–64.
44. Bartley K, Metayer C, Selvin S, Ducore J. Buffl er P. Diagnostic X-rays and risk of childhood leukaemia. *Int J Epidemiol*. 2010;39:1628–37.
45. Shu XO, Linet MS, Steinbuch M, Wen WQ, Buckley JD, Neglia JP, et al. Breast-Feeding and Risk of Childhood Acute Leukemia. *J Natl Cancer Inst* . 1999;91(20):1765–72.
46. Feychting M, Schulgen G, Olsen JH, Ahlbom A. Magnetic fields and childhood cancer—a pooled analysis of two Scandinavian studies. *Eur J Cancer* . 1995;31(12):2035–9.
47. Wertheimer N, Savitz DA, Leeper E. Childhood cancer in relation to indicators of magnetic fields from ground current sources. *Bioelectromagnetics*. 1995;16(2):86–96.
48. Toledano SR, Lange BJ. Ataxia-telangiectasia and acute lymphoblastic leukemia. *Cancer*. 1980;45(7):1675–8.
49. Shaw MP, Eden OB, Grace E, Ellis PM. Acute Lymphoblastic Leukemia and Klinefelter's Syndrome. *Pediatr Hematol Oncol*. 1992;9(1):81–5.
50. Mertens AC, Wen W, Davies SM, Steinbuch M, Buckley JD, Potter JD, et al. Congenital abnormalities in children with acute leukemia: A report from the Children's Cancer Group. *J Pediatr* . 1998;133(5):617–23.
51. Chessells JM. Down's syndrome and acute lymphoblastic leukaemia: clinical features and response to treatment. *Arch Dis Child* . 2001;85(4):321–5.
52. Greaves MF, Colman SM, Beard ME, Bradstock K, Cabrera ME, Chen PM, et al. Geographical distribution of acute lymphoblastic leukaemia subtypes: Second report of the collaborative group study. *Leukemia*. 1993;7:27–34.
53. Greaves MF. Aetiology of acute leukaemia. *Lancet*. 1997;349:344–9.
54. Kinlen LJ. Infection and childhood leukaemia near nuclear sites. *Lancet*. 1998;9:237–9.
55. Silva NJD, Perel Y, Méchinaud F, Plouvier E, Gandemer V, Lutz P, et al. Infectious diseases in the first year of life, perinatal characteristics and childhood acute leukaemia. *Br J Cancer*. 2004;90(1):139–45.
56. Roman E, Simpson J, Ansell P, Kinsey S, Mitchell C, McKinney P, et al. Childhood Acute Lymphoblastic Leukemia and Infections in the First Year of Life: A Report from the United Kingdom Childhood Cancer Study. *Am J Epidemiol*. 2006;165(5):496–504.
57. Lombardi L, Newcomb EW, Dalla-Favera R. Pathogenesis of Burkitt lymphoma: Expression of an activated c-myc oncogene causes the tumorigenic conversion of EBV-infected human B lymphoblasts. *Cell*. 1987;49(2):161–70.
58. Gessain A, Mahieux R, de Thé G. Genetic Variability and Molecular Epidemiology of Human and Simian T Cell Leukemia/Lymphoma Virus Type I. *Mel Infect* . 1996;13:132–45.
59. Williams CK. Some biological and epidemiological characteristics of human leukaemia in Africans. *IARC Sci Publ*. 1984;p. 687–712.
60. Yunis JJ. Recurrent chromosomal defects are found in most patients with acute nonlymphocytic leukemia. *Cancer Genet Cytogenet* . 1984;11(2):125–37.
61. Heim S, Békàssy AN, Garwicz S, Heldrup J, Ersson UK, Mandahl N, et al. Bone marrow karyotypes in 94 children with acute leukemia. *Eur J Haematol*. 1990;44:227–33.
62. Neville B, Damm D, Allen C, Bouquot J. Oral and Maxillofacial Pathology. Philadelphia: Saunders: Elsevier; 2009.
63. Bene MC, Castoldi G, Knapp W, Ludwig WD, Matutes E, Orfao A, et al. Proposals for the immunological classifi cation of acute leukemias. European Group for the Immunological Characterization of Leukemias (EGIL). *Leukemia*. 1995;9:1783–6.
64. Pui CH, Jeha S. New therapeutic strategies for the treatment of acute lymphoblastic leukaemia. *Nat Rev Drug Discov* . 2007;6(2):149–65.
65. Bayram I, Erbey F, Kömür M, Kibar F, Tanyeli A. Flow cytometry results at diagnosis and relapse in childhood acute lymphoblastic leukemia. *Asian Pac J Cancer Prev*. 2010;11:1321–1325.
66. Elman FM. The 3rd international workshop on chromosomes in leukemia. *Cancer Genet Cytogenet*. 1980;4:96–104.

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