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Cytogenetic analysis of 100 consecutive newly diagnosed cases of acute lymphoblastic leukemia in Rio de Janeiro

Maria Luiza Macedo Silva[a]*, Maria Helena Ornellas de Souza[a,b], Raul C. Ribeiro[c], Marcelo Gerardin Poirot Land[d], Alice Maria Boulhosa de Azevedo[b,d], Felippe Vasconcelos[a], Luize Otero[a], Zilton Vasconcelos[a], Luiz Fernando Bouzas[a], Eliane Abdelhay[e]

[a]*Laboratorio de Citogenetica, Centro de Transplante de Medula Óssea (CEMO), Instituto Nacional de Cancer (INCA), Rio de Janeiro, Brazil*
[b]*Hospital Universitário Pedro Ernesto (HUPE) de Universidade Estadual de Rio de Janeiro (UERJ), Rio de Janeiro, Brazil*
[c]*St. Jude Children's Research Hospital, Memphis, TN, USA*
[d]*Instituto de Puericultura e Pediatria Martagão Gesteira (IPPMG) of Universidade Federal of Rio de Janeiro (UFRJ), Rio de Janeiro, Brazil*
[e]*Instituto de Biofísica Carlos Chagas Filho (IBCCF) of Universidade Federal of Rio de Janeiro (UFRJ), Rio de Janeiro, Brazil*

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Abstract We report the cytogenetic analysis of newly diagnosed Brazilian children with acute lymphocytic leukemia (ALL). We investigated 100 ALL cases from four different institutions in Rio de Janeiro. The frequency of chromosomal abnormalities was 92.3%. The karyotype profile and recurrent abnormalities found in this study do not differ essentially from those described by other groups. Although the Brazilian population is usually the product of different ethnic groups, our results show that the frequency of each recurrent abnormality is similar to that found in populations without our degree of diverse ethnic composition. Hence, our results suggest that childhood ALL in Brazil has the same biological features as that in developed countries, supporting the use of similar treatment protocols. We can therefore expect to reach the same survival rates in the coming years, depending possibly on the efficacy of the support therapy and extent of social assistance.

1. Introduction

Childhood acute lymphoblastic leukemia (ALL) is a neoplastic disease characterized by clonal proliferation of lymphoid precursors that have lost the normal capacity to differentiate. Cytogenetic analysis of leukemic blasts cells contributes important prognostic information, in combination with other prognostic factors (usually age and white blood count at the diagnosis), for risk group classification of these patients. For the past two decades, clinical trials for ALL treatment have been based on the risk of relapse calculated at the diagnosis using mainly these three classes of factors [1].

Hitherto, only a few reports in our country have dealt with the cytogenetics of ALL, describing isolated and unusual cases [2]. For this reason, the karyotypic pattern of the childhood ALL in our population was unknown. We here report the cytogenetic analyses of 100 consecutive newly diagnosed Brazilian children with ALL collected from four institutions in Rio de Janeiro city specialized in the treatment of ALL. Our primary aim is to describe the frequency of chromosomal abnormalities in this disease.

2. Materials and methods

2.1. Patients

From September 1991 to May 1995, 100 children (ages 3 months to 18 years) with newly diagnosed ALL were consecutively admitted to four institutions of Rio de Janeiro (Brazil), specialized in the treatment of this disease. The institutions were: (1) Instituto Nacional do Câncer (INCA-RJ) (50 patients); (2) Instituto de Puericultura e Pediatria Martagão Gesteira (IPPMG-UFRJ) (34 patients); (3) Hospital Universitário Pedro Ernesto (HUPE-UERJ) (12 patients); and (4) Hospital Raul Sertã (4 patients). The chromosomal and immunophenotypic analyses of bone marrow and/or peripheral blood were conducted in the Cytogenetic Laboratory of the Bone Marrow Transplantation Unit of Instituto Nacional do Câncer (INCA-RJ). The cytochemical and

* Corresponding author. Laboratorio de Citogenetica, Centro de Transplante de Medula Óssea (CEMO), Instituto Nacional de Cancer (INCA), Praca Cruz Vermelha 23, 20 230-130, Rio de Janeiro, Brazil. Tel.: +55-21-5066701.
E-mail address: luizamacedo@hotmail.com (M.L.M. Silva).

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morphological diagnoses were performed in the children's original institutions.

2.2. Chromosomal and immunophenotypic studies

The chromosome studies were performed on bone marrow and/or peripheral blood cells according to Testa et al. [3]. Banding was performed according to Seabright [4] and the chromosome identified and arranged according to the International System for Human Cytogenetic Nomenclature [ISCN 1995] [5]. The karyotype profile was determined by the analysis of at least 20 metaphases using an optical microscopy. A case was classified as abnormal when at least two metaphases have the same structural aberration and the same chromosome again, and when at least three metaphases have the same loss. The presence of normal cells side by side with abnormal ones was used as a criterion to exclude a constitutional chromosomal abnormality. When the patient cells showed 100% of abnormalities with a non-recurring or a X /Y chromosomal abnormality, the exclusion of a constitutional anomaly was made by karyotypic analysis of the peripheral blood [6], after a complete remission had been obtained.

Only samples containing more than 60% of blastic cells were used for immunophenotypic analysis. Surface immunoglobulin (sIg) was usually identified in direct immunofluorescence, using fluoresceinated goat F(ab)'2 anti-human Ig. The presence of intracytoplasmatic Ig (cIg) was investigated by direct immunofluorescence on fixed cytospin smears. All other membrane molecules were identified using monoclonal antibodies and indirect immunofluorescence with fluoresceinated goat anti-mouse Ig serum as a second step reagent. The immunologic panel consisted of HLA-DR, CD1, CD2, CD3, CD4, CD7, CD8, CD10, CD14, CD15, CD19, CD20, CD22, CD33, and CD34 (Becton & Dickinson). Fluorescence activity was analyzed in a FacsCan (fluorescence activated cell analyses cell quest software, San Jose, CA) Becton & Dickinson flow cytometer.

3. Results

In this study, samples from 91 patients (91%) were adequate for cytogenetic analysis. In nine patients, the chromosomal analysis could not be performed due to lack of metaphases or poor mitotic index. The cytogenetic analysis of the 91 samples showed seven cases (7.7%) with a normal karyotype and 84 cases (92.3%) with chromosomal abnormalities. We detected hyperdiploidy in 35 (38.5%), pseudodiploidy in 43 (47.2%), and hypodiploidy in six of the cases (6.6%). In Fig. 1 we present the distribution of the 91 cases classified according to the cytogenetic, immunophenotypic, and morphologic characteristics.

3.1. Chromosomal abnormalities in non-B non–T-ALL (N = 69)

3.1.1. Hyperdiploidy (N = 32)

We found 32 cases of hyperdiploidy. In 18 cases (56%), the patients had only numerical changes. The chromosomal

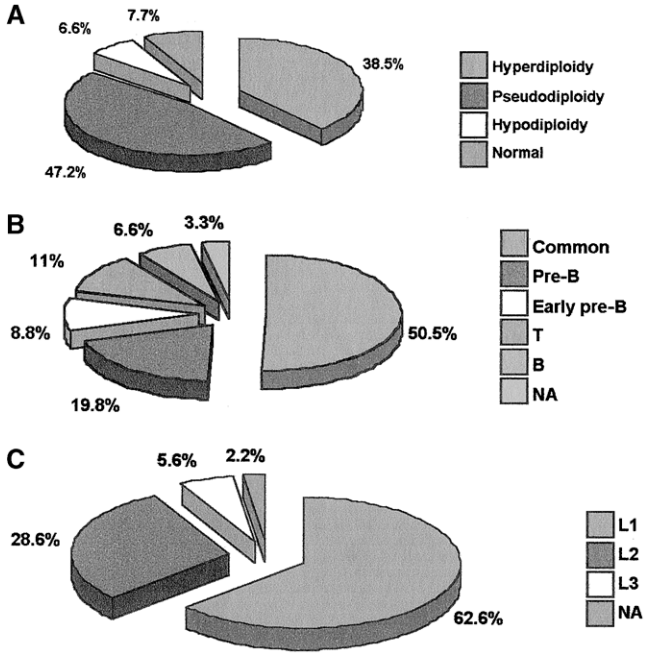

Fig. 1.The distribution of 91 cases according to (A) cytogenetic ploidy, (B) immunophenotype (the immunophenotype of three patients was not available [NA]), and (C) morphologic characteristics (the morphology of two patients was not available [NA]).

number ranged from 47 to 92 (median = 52 chromosomes). In decreasing order, the chromosomes most frequently involved were 21 (trisomy in three and tetrasomy in 20 cases), X (trisomy in 21 and tetrasomy in two cases), 6 (trisomy in 21 cases), 18 (trisomy in 21 cases and tetrasomy in one case), 17 (trisomy in 20 cases), 14 (trisomy in 13 and tetrasomy in five cases), 4 (trisomy in 13 cases), 10 (trisomy in 13 cases), 5 (trisomy in 10 cases), 12 (trisomy in eight cases), 8 (trisomy in seven cases), 11 (trisomy in five cases), 16 (trisomy in five cases), 7 (trisomy in one case), 20 (trisomy in five cases), and 2 (trisomy in five cases). Chromosomes 1, 3, 9, 13, 15, 19, and 22 were not involved in trisomy in any karyotype (Table 1).

Structural abnormalities in this group were observed in 34.4% of the cases (11 patients, including one with a constitutional abnormality). The types of abnormalities were duplications, translocations, additions, and isochromosomes. Trisomy or duplication of the q arm of chromosome 1 was the most frequent structural abnormality.

3.1.2. Hypodiploidy (N = 6)

The chromosomal number was less than 46 in six patients (45 in five patients, 44 in one patient). The chromosomes involved in monosomy were X, 8 (two cases), 6, 21, 7, 1, 11, 12, and 15. In this group, one case had trisomy 14 and two markers. We noted structural abnormalities in all cases, including three patients with complex karyotypes. These abnormalities were: 1) addition [add(12)(p13) in two cases, add(2)(p25), and add(16)(q24)]; 2) a dicentric chromosome, dic(7;12)(p11;p12); 3) an isochromosome, i(9)(q10); 4) deletions [del(6)(q21q23) and del(12)(p12)];

Table 1
Clinical, morphologic, immunophenotypic, and cytogenetic findings of 84 children with chromosomal abnormalities

Case no.	Initials	Age	Sex	EG	WBC × 10^9/L	FAB type	Immunophenotype	Karyotype
Hyperdiploid non-B non-T ALL								
1	NMMM	3	F	W	20	L2	Common	47,XX,+10,t(11;21)(q13;q21)[15]/46,XX[5]
2	ALNDS	5	F	W	480	L2	Common	47,XX,+8[17]/46,XX[4]
3	MJMS	1y/6mo	F	Non-W	5.4	L2	Common	47,XX,+D[3]/46,XX[20]
4	ESJ	16	F	Non-W	31	L2	Common	47,XX,+X[15]/47,XX,+X,i(17)(q10)[5]/ 47,XX,+X,dup(1)(q21q32)[6]/46,XX[15]
5	TTAM	2	M	Non-W	34	L1	Common	47,XY,+16[16]/46,XY[5]
6	JD	6	F	W	3	L2	Unknown	49,XX,+X,+14,+21[25]/46,XX[40]
7	PRA	9	F	W	12.9	L1	Common	51,XX,+X,+4,+6,+18,+21[8]/46,XX[40]
8	ACA	1y/10mo	F	W	4.7	L1	Common	52,XX,+X,+4,+5,+8,+21,+21[12]/46,XX[7]
9	MT	5	F	W	6.4	L1	Common	53,XX,+X,+6,+14,+17,+18,+21,+21[30]/46,XX[6]
10	JPT	2	F	Non-W	9.5	L1	Common	53,XX,+X,+4,+6,+14,+17,+18,+21[10]/46,XX[20]
11	CBFS	3	M	W	3	L1	Common	53,XY,+X,+6,+10,+17,+18,+21,+21[37]/46,XY[10]
12	JVR	2	M	W	15.3	L1	Pre-B	54,XY,+X,+5,+6,+14,+17,+18,+21,+21[20]/46,XX[10]
13	ESS	8	M	Non-W	25.6	L2	Common	54,XY,+X,dup(1)(q21q32),+4,+6,+10,+12,+18,+21,+21 [36]/46,XY[5]
14	JM	5	F	Non-W	42.2	L1	Pre-B	54,XY,+X,dup(1)(q21q32),+5,+6,+7,+14,+17,+18,+21 [20]/46,XY[10]
15	JGS	1y/6mo	F	Non-W	13.5	L1	Common	55,XX,+X,+4,+6,+10,+14,+17,+18,+21,+21[10]/ 46,XX[2]
16	TCD	14	F	Non-W	4	L2	Common	55,XX,+X,+4,+6,+10,+14,+i(17)(q10),+18,add(19)(p13), +21,+21[10]/56,XX,+X,dup(1)(q21q32),+4,+6,+8, +10,+14,+17,+18,+21[4]/46,XX[5]
17	KRSC	3	F	W	4.8	L1	Unknown	55,XX,+X,+4,+6,+10,+add(12)(q24),+17,+18,+21,+21 [32]/46,XX[15]
18	DC	3	F	Non-W	43.6	L1	Common	55,XX,+X,+6,+12,+14,+14,+17,+18,+21,+21[15]/ 46,XX[4]
19	DCL	2	F	W	13.5	L1	Common	55,XX,+X,+6,+8,t(9;15)(p22;q15)c,+14,+14,+17,+18, +21,+21[33]/46,XX,t(9;15)(p22;q15)[10]
20	DPP	1y/9mo	M	Non-W	2.8	L1	Pre-B	55,XY,+X,+4,+6,+10,+14,+17,+18,+21,+21[9]/ 55,XY,+X,dup(1)(q21q32),idem[19]/55,idem,trp(1) (q11∼q42::q11∼q32::q11∼qter),[8]/46,XY[5]
21	BSFC	9	M	Non-W	18	L1	Common	55,XY,+X,+5,+6,+10,+14,+17,+18,+21,+21[4]/ 46,XY[6]
22	LR	1y/11mo	M	Non-W	30.9	L1	Common	55,XY,+X,dup(1)(q12q32),+5,+6,+10,+14,+17,+18,+21, +21[38]/46,XY[9]
23	DMC	13	F	W	0.5	L1	Pre-B	57,XX,+X,+4,+6,t(7;8)(p22;q21),+11,+12,+14,+16, +17,+18,+21,+21[10]/46,XX[8]
24	JCMR	2	F	W	13.9	L2	Common	57,XX,+X,t(2;14)(p13;q32),+5,+6,+8,+10,+14,+16, +17,+18,+21,+21[38]/46,XX[11]
25	JS	4	F	Non-W	10.7	L1	Common	58,XX,+X,+4,+5,+6,+8,+10,+14,+14,+17,+18,+21, +21[30]/46,XX[10]
26	OMVS	3	F	W	3.4	L1	Common	58,XX,+X,trp(1)(q11∼q42::q11∼q32::q11∼qter),+5,+6, +8,+10,+11,+12,+14,+17,+18,+21,+21[14]/ 59,idem,+14[7]/46,XX[8]
27	FME	1y/11mo	M	W	13.8	Unknown	Common	59,XY,+X,+X,+4,+6,+11,+12,+14,+14,+16,+17,+18, +21,+21[10]/46,XY[6]
28	AL	2	M	W	14.7	L1	Pre-B	61,X,+X,−Y,dup(1)(q12q32),+2,+4,+5,+6,+8,+10,+1 1,+12,+14,+17,+18,+20,+21,+21,+mar[10]/62, idem,+16[9]/46,XY[6]
29	APBS	5	M	Non-W	12	L1	Early pre-B	62,XY,+X,+X,+4,+5,+6,+8,+10,+11,+12,+14,+16, +17,+18,+18,+21,+21[33]/46,XY[11]
30	LMA	3	F	W	5.2	L1	Common	64,XXX,−1,−2,−3,−C,−D,−D,+G[9]/46,XX[5]
31	JCS	4	F	Non-W	3.9	L1	Pre-B	94,XXXX,+2mar[12]/46,XX[10]
32	EAS	11	M	Non-W	3.9	L1	Common	94,XXYY,−?6,+mar[15]/46,XY[10]

(Continued)

Table 1
(Continued)

Case no.	Initials	Age	Sex	EG	WBC × 10^9/L	FAB type	Immunophenotype	Karyotype
Hypodiploid non-B non-T ALL								
33	AG	2	M	Non-W	3.4	L2	Pre-B	44,XY,−1,−11,−12,−15,+2ar[4]/46,XY[10]/46,XX[5]
34	BLG	9	F	W	43	L1	Common	45,X,−X,add(12)(p13)[22]/46,XX[6]
35	TBS	3	F	W	69.4	L2	Pre-B	45,XX,−6,der(19)t(1;19)(q23;p13)[10]/46,XX[5]
36	SSA	11	F	W	33.6	L1	Early pre-B	45,XX,−8,−21,+14,add(12)(p13)[40]/46,XX[7]
37	PSC	6	F	W	2.3	L2	Pre-B	45,XX,t(2;14)(p11;q32),−7,dic(7;12)(p11;p12),i(9)(q10)
38	EA	5	M	W	3.8	L1	Pre-B	45,XY,t(1;4)(q32;q35),add(2)(p25),−8,del(6)(q21q23), del(12)(p12),add(16)(q24)[40]/46,XY[10]
Pseudodiploid non-B non-T ALL								
39	FP	6	M	W	4.5	L2	Common	46,X,−Y,+8,der(16),t(1;16)(q21;q24)[14]/46,XY[10]
40	MMG	6	F	W	3.5	L1	Pre-B	46,XX,t(10;17)(p15;q21)[24]/46,XX[3]
41	VCSC	4	F	Non-W	27.5	L1	Common	46,XX,add(1)(p3?6),t(13;15)(q31;q21)[5]/46,XX[2]
42	SCB	8	F	W	5.5	L1	Common	46,XX,add(17)(p13)[5]/46,XX[45]
43	ACS	4mo	F	W	20	L1	Early pre-B	46,XX,del(11)(q23)[10]/46,XX[25]
44	RRB	2	F	W	180	L1	Early pre-B	46,XX,del(11)(q23)[15]/46,XX[10]
45	SO	3	F	W	48	L1	Common	46,XX,del(5)(p14)[5]/46,XX[21]
46	CSB	1y/1mo	F	W	106	L2	Pre-B	46,XX,del(9)(p13),−13,+mar[30]
47	ACPS	3	F	Non-W	26	L1	Pre-B	46,XX,der(19)t(1;19)(q23;p13)[23]/46,XX3]
48	OAM	4	F	W	8.1	L1	Pre-B	46,XX,i(9)(q10)[5]/47,XX,+X,i(9)(q10)[11]/46,XX9]
49	MBC	1y/8mo	F	W	51.5	L1	Early pre-B	46,XX,i(9)(q10)[8]/46,XX[3]
50	BSS	2	F	Non-W	47.2	L1	Pre-B	46,XX,i(9)(q10),der(19)t(1;19)(q23;p13)[21]/46,XX[11]
51	DM	3mo	F	Non-W	120	L1	Early pre-B	46,XX,t(4;11)(q21;q23)[22]/46,XX[2]
52	EJS	15	F	Non-W	48	L1	Pre-B CD10-	46,XX,t(4;11)(q21;q23)[24]/46,XX[4]
53	LATB	6	F	W	180	L2	Early pre-B	46,XX,t(4;11)(q21;q23)[24]/46,XX[4]
54	EG	3	F	W	1.2	L1	Common	46,XX,t(X;9)(p11;q34)[3]/46,XX[10]
55	ROF	17	M	W	2.6	L1	Common	46,XY,−19,add(12)(p13),+mar[26]/46,XY[8]
56	BAMR	3	M	Non-W	6.8	L1	Common	46,XY,−4,−8,add(17)(p13),+2mar[23]/46,XY26]
57	TBP	5	M	W	158	L1	Common	46,XY,add(X)(p22)[22]/46,XY[6]
58	ASR	3	M	W	5.7	L1	Common	46,XY,del(10)(p13)[4]/46,XY[10]
59	DRAA	6	M	W	1.5	L1	Early pre-B	46,XY,del(11)(q23)[12]/46,XY[4]
60	JOC	15	M	Non-W	49.4	L1	Common	46,XY,del(11)(q23)[6]/46,XY22]
61	WMS	4	M	Non-W	5.1	L1	Common	46,XY,del(6)(q15q21)[7]/46,XY[4]
62	CMP	7	M	Non-W	3.9	L2	Unknown	46,XY,del(7)(q32)[4]/46,XY[11]
63	LGP	10	M	Non-W	1.2	L1	Common	46,XY,i(9)(q10)[13]/46,XY[2]
64	FAAP	1y/10mo	M	W	78.6	L2	Pre-B	46,XY,t(11;19)(q23;p13)[17]/46,XY[15]
65	TCL	5	M	Non-W	10	L3	Common	46,XY,t(12;21)(p12;q11)[15]/46,XY[5]
66	GHT	7	M	Non-W	6.1	L2	Common	46,XY,t(9;15)(q34;q15)[43]/46,XY[6]
67	LFAV	3	M	W	21.7	L1	Common	46,XY,t(9;22)(q34;q11)[19]/46,XY[28]
68	MLC	8	M	W	6.5	L1	Common	46,XY,t(9;22)(q34;q11),−14,−21,+2mar[12]/46,XX[6]
69	NO	18	M	Non-W	3.6	Unknown	Common	47,XY,dup(1)(q32q41),i(9)(q10),+21c[5]/47,XY,−8, +5,i(9)(q10),+21c[6]/47,XY,add(3)(q38),−8,+5,i(9) (q10),+21c[4]/48,XY,add(3)(q38),−8,+5,i(9)(q10), +i(9)(q10),+21c[3]/47,XY,+21c[10]
B-ALL								
70	AAP	9	M	Non-W	11.7	L3	B	47,XY,+X?c,t(8;14)(q24;q32)[3]/47,XY,+X?c[47]
71	ARS	4	F	Non-W	6.5	L3	B	93,XXXX,i(9)(q10),+mar[15]/46,XX[35]
72	JCC	9	F	W	14	L3	B	47,XX,add(6)(q26),add(11)(q23),+12[30]/46,XX[3]
73	VFML	3	F	W	15.6	L3	B	46,XX,t(8;14)(q24;q32)[22]/46,XX[8]
74	LSG	11	M	W	11.5	L2	B	46,XY,t(8;14)(q24;q32),add(11)(q23),add(13)(q34)[21]/ 46,XY[5]
75	MSR	11	F	W	4.6	L2	B	46,XX,t(8;14)(q24;q32),der(15),t(1;15)(q21;p13)[26]/ 46,XX[8]
T-ALL								
76	ASM	9	M	W	250	L1	T	46,XY,t(4;7)(p1?1;p1?2)[6]/46,XY[2]
77	DMS	7	F	Non-W	182	L2	T	46,XX,t(11;14)(p13;q11)[30]
78	GBS	11	M	W	192	L2	T	46,XY,del(4)(q12),i(14)(q10)[20]/46,XY[10]
79	JHOS	9	M	Non-W	38.8	L2	T	46,XY,del(9)(p21)[25]
80	JP	11	M	Non-W	120	L1	T	46,XY,del(3)(q21),del(6)(q21)[22]/46,XY[7]
81	MXC	16	M	Non-W	34.8	L2	T	46,XY,del(9)(p13)[26]/47,XY,del(9)(p13),+21[7]/ 46,XY[2]
82	REA	6	F	Non-W	134	L1	T	46,XX,t(6;7)(p21;q35)[20]/46,XX[10]
83	RSR	16	M	W	700	L2	T	46,XY,t(9;22)(q34;q11)[20]/46,XY[10]
84	TPF	8	M	W	105	L1	T	46,XY,del(5)(p13)[3]/46,XY[44]

Abbreviations: F, female; FAB, French–American–British classification; mo, months; M, male; y, years.

and 5) translocations [t(1;4)(q32;q35), t(2;14)(p11;q32), and der(19)t(1;19)(q23;p13)] (Table 1).

3.1.3. Pseudodiploidy (N = 31)

Thirty-one patients showed a pseudodiploid karyotype. Most frequently, the abnormalities involved chromosomes 9 and 11. Eight patients (25.8%) had abnormalities involving the 11q23 region, which included three patients with t(4;11)(q21;q23), one patient with t(11;19)(q23;q21), and four patients with del(11)(q23). We detected anomalies involving chromosome 9 in 10 patients (32.2%). An i(9)(q10) was present in five patients. In three cases, this was the only abnormality and, in the other two cases, it was present as a secondary change.

The t(9;22)(q34;q11) was detected in two patients (6.4%). Anomalies involving chromosome 1 were present in five cases (16.1%). These abnormalities included a dup(1)(q21q32) associated with i(9)(q10), a der(16)t(1;16)(q21;q24) together with −Y and +8. One of the cases had an add(1)(p3?6) together with other translocations and two cases had a t(1;19)(q23;q13), one of which was associated with an i(9)(q10). We detected anomalies involving chromosome 12 in two patients: a t(12;21)(p12;q11) and an add(12)(p13) together with −19 and a marker chromosome. Further anomalies were present at a very low frequency or were nonspecific for ALL (Table 1).

3.2. Chromosomal abnormalities in B-type ALL (N = 6)

Six patients (6.6%) had B-type ALL. Four cases showed 46 chromosomes, one case 47 chromosomes, and the other a hypertetraploid complement. Four patients had the t(8;14)(q24;q32). In three of these patients, this translocation was associated with other chromosomal anomalies such as der(15)t(1;15)(q21;p13), add(11)(q23), add(13)(q34), and a constitutional trisomy X. We could not find the t(8;14)(q24;q32) or its variations in two case of B-ALL, which instead showed the following karyotypes: 47,XX,add(6)(q26),add(11)(q23),+12, and 93,XXXX,i(9)(q10),+mar, respectively (Table 1).

3.3. Chromosomal abnormalities in T-ALL (N = 9)

Eleven of the cases (11%) were classified as T-ALL. It was impossible to obtain mitoses for analysis in one patient. Nine cases had chromosomal abnormalities (90%) and one had a normal karyotype (10%). The structural anomalies included deletions, translocations, and isochromosomes. Some of them have rarely been described in this subtype, such as an i(14)(q10) associated with a del(4)(q12) [7] and t(9;22)(q34;q11) [8] (Table 1).

4. Discussion

Prospective chromosomal ALL studies have shown abnormalities in 55% to 94% of the cases [9–15]. Secker-Walker et al. [9] analyzed 123 children with ALL and detected abnormal karyotypes in 55% of them, whereas Williams et al. [10] observed abnormal karyotypes in 94% of 116 children. According to Secker-Walker et al. [9], the high level of anomalies detected by Williams et al. [10] could be explained by the fact that this was a unicentric study, where the technical approaches were always the same. In the opinion of Secker-Walker et al., such results would be very difficult to achieve in multicentric studies. Our data, although derived from a multicentric study, are similar to those of Williams et al. [10] inasmuch as we found abnormal karyotypes in 92.3% of 100 patients from several hospitals in Rio de Janeiro. This high level of abnormalities in our study is probably due to the utilization of the technique described by Testa et al. [3]. Consequently, we would like to suggest the use of this technique as a good choice for multicentric studies.

The karyotype profiles and recurrent abnormalities found in this study do not essentially differ from those described by other groups. We found the t(4;11)(q21;q23) in 4.3% of the non-B non-T ALL patients, t(9;22)(q34;q11) in 3.8% of 78 patients with non-B non-T ALL (two cases) and T-ALL (one case), and t(1;19) in 12.5% of the pre-B ALL patients. We attributed the high percentage of the last translocation to the small size of our sample. We found 12p abnormalities in association with hypodiploidy. The 12p abnormalities have been related to the t(12;21)(p13;q22), but we are unable to perform FISH to demonstrate this relation in our study.

Some rare abnormalities in T-ALL were identified as i(14)(q10) [7] and Ph^+ T-ALL [8]. The i(14)(q10) was the second case described in the literature and appears to be associated with good prognosis. Remarkably, the "de novo" Ph^+ T-ALL case showed the expression of a p210 protein, and was associated with a very aggressive evolution and with a poor clinical outcome [8]. In addition, we identified two cases of B-type ALL with chromosomal abnormalities other than the common t(8;14)(q24;q32) and its variants. It is possible that the molecular rearrangement between *c-MYC* and Ig chain genes was present only at the molecular level.

Although the Brazilian population is usually the product of different ethnic groups, our results show that the frequency of each recurrent abnormality is similar to those found in populations without our degree of ethnic composition. Magalhães et al. [16] reached a similar conclusion by demonstrating a frequency of 20% of *TEL-AML1* fusion in Brazilian children. A very similar frequency of this fusion was found in the US and Europe. Likewise, Ornellas et al. [17] indicated that the immunophenotype characteristics of their ALL cases were more related to age at diagnosis than to the putative ethnic group of the patients.

Hence, the combination of these results suggests that ALL in Brazilian children have the same biological features as those in developed countries, supporting the use of similar treatment protocols. We can, therefore, expect to reach the same survival rates in the coming years, depending possibly on the efficacy of the support therapy and the extent of social assistance.

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