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Prognostic significance of chromosome analysis in de novo acute myeloid leukemia (AML)

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Summary. Between 1981 and 1986 cytogenetic studies of bone marrow and/or blood cells in 139 patients with de novo acute myeloid leukemia (AML) were performed. The overall incidence of chromosomal aberrations was 53%, and this was not significantly influenced by sex, age nor the FAB classification. The aberrations most often found were: complex anomalies ($n = 13$), t(8;21) ($n = 10$), trisomy 8 ($n = 9$), monosomy 7 ($n = 6$), monosomy 5, 5q-, trisomy 11, 12p- ($n = 4$) and trisomy 6, 11q-, inv [16] ($n = 3$). The prognostic significance of chromosomal findings was evaluated in 112 patients treated by combination chemotherapy. The chromosomal status NN, AN, AA did neither significantly influence complete remission rate (NN: 68%, AN: 71%, AA: 60%) nor mean survival (NN: 24, AN: 26.6, AA: 35.6 months). On the other hand, certain types of chromosomal anomalies were of prognostic value. CR was obtained in all 10 patients with t(8;21) but only in 2 out of 9 patients with complex aberrations. Median duration of CR in patients with t(8;21) was significantly longer than in patients with a normal karyotype (30 vs 7 months).

Key words: Cytogenetic studies – AML – Prognostic value

Introduction

Using a metaphase technique, chromosomal anomalies in bone marrow cells are found in about 50% of the patients with de novo acute myeloid leukemia [13, 15]. Since the first report of Sakurai and Sandberg in 1973 [36], indicating a better prognosis for patients with a normal karyotype (NN) or a mixture of normal and abnormal mitoses (AN) than for patients with only abnormal mitoses (AA), several other large cytogenetic studies [5, 7, 13, 17, 25, 26, 31] confirmed these prognostic findings. On the other hand, some reports deny such a prognostic value of the chromosomal status NN, AN, AA [8, 19, 28, 29]. In more recent studies [30, 43] the prognostic value of the types of chromosomal aberrations rather than the NN-AN-AA classification was emphasized. Yunis et al. [43] reported that complex aberrations predicted a poor prognosis, inversion [16] a good prognosis, and trisomy 8 an intermediate prognosis. The aim of this report is to reevaluate the prognostic significance of the karyotype in 112 patients with de novo acute myeloid leukemia treated by combination chemotherapy.

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Materials and methods

Patients, diagnosis and treatment

Between January 1981 and December 1986, 139 patients with de novo acute myeloid leukemia (AML) were studied. More than 90% of the patients were clinically followed at four departments (Department of Hematology, St. Georg Hospital, Hamburg; Department of Oncology and Hematology, University Clinic, Hamburg; Department of Hematology, Pediatric University Clinic, Hamburg; Department of Hematology and Oncology, Pediatric University Clinic, Münster). In none of the patients a myelodysplastic syndrome preceding AML nor prior chemo- and/or radiotherapy for a primary malignancy was known. Diagnosis of AML and classification according to the FAB nomenclature [3] were based on peripheral blood and bone marrow slides stained with May-Grünwald-Giemsa and cytochemical reactions for myeloperoxidase, naphthol AS-D acetate esterase and periodic acid phosphatase (PAS).

In 27 patients no specific treatment was prescribed because of advanced age, poor performance status or heavy comorbidity. The decision not to give treatment was made by the responsible clinician who had no knowledge of the patients karyotype. The prognostic significance of chromosomal findings in AML was evaluated in 112 patients treated by aggressive combination chemotherapy. The treatment protocols used were (Table 1): TAD regimen (6-thioguanine, cytosine arabinoside, daunorubicine) [9], TAD-HAM regimen (TAD + high dose cytosine arabinoside, mitoxantrone) [21]. VDPC regimen (vincristine, doxoru-

bicine, prednisolone, cytosine arabinoside) [42], BFM-78 protocol (prednisone, vincristine, adriamycine, cytosine arabinoside, 6-thioguanine, cyclophosphamide, CNS prophylaxis) [11] and the BFM-83 protocol (VP-16, daunorubicine, cytosine arabinoside + BFM-78 regimen) [35]. Complete remission (CR) was defined according to the CALGB criteria [12].

For statistical analysis a two-sided chi-square test was used [38]. Survival curves were established by the method of Kaplan and Meier [24]. Statistical differences between the life-table curves were calculated by the log-rank test [32].

Chromosome analysis

Chromosome analysis was performed according to standard techniques for all patients prior to any specific therapy. Bone marrow cells were cultured in medium RPMI with 15% calf serum without cell synchronization for 2, 24 or 48 h. Unstimulated peripheral blood cells were cultured for 24 or 48 h. G-banding was performed in all cases according to Seabright's technique [37]. In most cases 15–25 mitoses were counted and photographed. With rare exceptions, at least 5 mitoses were karyotyped. Chromosomes were classified according to the ISCN criteria [23]. A clonal anomaly was defined according to the recommendations of the First International Workshop on Chromosomes in Leukemia [13]. The karyotype was further classified as NN (only normal mitoses), AN (mixture of normal and abnormal mitoses) and AA (only abnormal mitoses).

Results

The patients' clinical data are summarized in Table 1. The only essential difference between the treated and the untreated group was the advanced age in the untreated group. Cytogenetic data are summarized in Table 2. Overall incidence of chromosomal anomalies in all patients was 53%. It was neither significantly influenced by sex (male patients: 38/71 = 54% anomalies, female patients: 35/68 = 51% anomalies), age (children < 16 years: 11/17 = 65%, adult patients: 62/122 = 51%, adult patients 16–60 years: 35/70 = 50%, adult patients > 60 years: 27/52 = 52% anomalies) nor the FAB-classification (M_1: 16/30 = 53%, M_2: 20/38 = 53%, M_4: 16/36 = 44%, M_5: 14/24 = 58% anomalies).

Table 1. Comparing clinical data of 112 patients treated by combination chemotherapy and 27 patients receiving only symptomatic treatment

	$n = 112$	$n = 27$	Total
Adults (no.)	97	25	122
Children (< 16 years) (no.)	15	2	17
Sex (no.): males	57	14	71
females	55	13	68
Mean age (years)			
– all patients	43	63	47
– adults only	49	67	52
– children only	7	7	7
FAB classification (no. of patients)			
M_1	22	8	30
M_2	32	6	38
M_3	1	1	2
M_4	30	6	36
M_5	19	5	24
M_6	4	—	4
M_7	4	1	5
Treatment protocols (no. of patients)			
TAD	79		
TAD/HAM	8		
VDPC	7		
BFM-78	14		
BFM-83	4		

Table 2. Comparing summarized cytogenetic data of 112 patients treated by combination chemotherapy and 27 patients receiving symptomatic treatment only

	$n = 112$	$n = 27$	Total
Overall incidence of chromosomal anomalies:	59/112 = 53 %	14/27 = 52 %	73/139 = 53 %
NN	53/112 = 47 %	13/27 = 48 %	66/139 = 47 %
AN	34/112 = 31 %	11/27 = 41 %	45/139 = 32 %
AA	25/112 = 22 %	3/27 = 11 %	28/139 = 21 %
Chromosomal aberrations most often found (no. of patients):			
Complex[a]	9	4	13
t (8;21)	10	—	10
+8	8	1	9
−7	5	1	6
−5	3	1	6
5q−	1	3	4
+11	3	1	4
12p−	3	1	4
+6	3	—	3
11q−	3	—	3
inv (16)	2	1	3

[a] Four or more anomalies

Table 3. Clinical and cytogenetic findings in 59 patients with abnormal karyotype treated by combination chemotherapy

Patient	Sex / age (years)	FAB classification	Treatment protocol	Treatment result	Duration of CR (months)	Survival (months)[a]	Material studied	Number of abnormal cells / total cells	Main clone(s)	AN/AA
1 BT	F/67	M_2	TAD	NR	—	3	BM 24[h]	12/23	45, X, + del (1) (p13), del (3) (p14), del (3) (p12), − 5, dup (12) (q13 → q24)	AN
2 HA	F/65	M_5	TAD	NR	—	2	BM 2, 24[h]	14/16	48, XX, − 2, − 21, + m_1, + m_2, + m_3, + m_4	AN
3 HP	M/63	M_4	TAD	+	—	1	BM 48[h]	15/15	43, XY, del (3) (p23), − 5, − 9, t (9; 17) (q11; p13), + 12, − 16,	AA
4 WI	F/61	M_2	TAD	CR	11+	13+	BM 24[h]	22/22	17p+, − 18, − 22, + m, fm 45, XX, − 3, − 6, − 7, + der (3), + m	AA
5 BM	F/51	M_1	TAD	CR	6	12	BM 24[h]	12/20	47, XX, + 4	AN
6 RK	M/68	M_4	TAD	PR	—	10	BM 48[h]	28/28	46, XY, 4p+, del (11) (q23)	AA
7 KI	F/50	M_2	TAD	NR	—	1	BM 48[h]	8/ 8	42, X, − 4, − 5, − 15, − 17, + m	AA
8 HH	F/74	M_2	VDPC	NR	—	1	BM 2, 24[h] Bl 48[h]	18/54	a) 49, XX, + 4, + 13, + 19 b) 48, XX, + 13, + 19	AN
9 GW	M/75	M_6	TAD	NR	—	2+			44, XY, del (5) (q12; q33), − 7, − 16	AA
10 HB	F/24	M_5	TAD	CR	3	4	Bl 24[h]	58/58	47, XX, + 6	AA
11 HT	M/26	M_5	TAD	CR	5	6	BM 2, 24[h] Bl 48[h]	29/31	48, XY, + 6, + 8	AN
12 QF	M/ 2	M^7	BFM-83	CR	7+	9+	BM 2[h]	15/15	48, XY, + 6, + 21	AA
13 SR	F/47	M_6	TAD	CR	3	11	BM 2[h]	7/ 8	47, XX, t (6; 11) (q23; p15), + der (3)	AN
14 FE	F/77	M_5	TAD	NR	—	1	BM 2[h] Bl 24[h]	14/20	46, XX, t (6; 21) (q13; q22)	AN
15 BH	F/40	M_2	TAD	CR	4	5	BM 24[h] Bl 24[h]	8/12	45, XX, − 7	AN
16 BK	M/20	M_7	TAD	CR	5	11	Bl 24[h]	9/18	45, XY, − 7	AN
17 PH	M/44	M_1	TAD	CR	12	24+	Bl 24[h]	12/15	46, XY, del (7) (q31)	AN
18 FH	M/64	M_1	TAD	PR	—	4	BM 48[h]	9/17	a) 45, XY, − 7, − 9, + m b) near tetraploid (n = 91)	AN
19 SA	M/ 4	M_4	BFM-83	CR	12+	14+	BM 24[h] Bl 24[h]	14/14	46, XY, t (8; 21) (q22; q22)	AA
20 OA	M/20	M_2	TAD	CR	2+	4+	BM 24[h] Bl 24[h]	13/13	a) 46, XY, t (8; 21) (q22; q22) b) 45, X, − Y, t (8; 21) (q22; q22)	AA
21 BU	M/44	M_2	TAD	CR	30	42	BM 24[h] Bl 24[h]	18/18	a) 46, XY, t (8; 21) (q22; q22) b) 45, X, − Y, t (8; 21) (q22; q22)	AA
22 LH	M/42	M_2	VDPC	CR	42+	44+	BM 24[h] Bl 24[h]	36/36	45, X, − Y, t (8; 21) (q22; q22)	AA
23 MH	M/51	M_2	TAD	CR	9+	10+	BM 24[h]	10/10	45, X, − Y, t (8; 21) (q22; q22)	AA
24 SG	M/39	M_2	TAD	CR	11+	13+	Bl 48[h]	7/ 7	45, X, − Y, t (8; 21) (q22; q22)	AA
25 MI	F/22	M_2	TAD	CR	7	23	BM 24[h] Bl 24[h]	12/12	45, X, − Y, t (8; 21) (q22; q22), del (9) (q13; q22)	AA
26 RS	F/20	M_1	TAD	CR	14+	18+	BM 24[h]	15/18	47, XX, + 8, t (8; 21) (q22; q22)	AN
27 MU	M/25	M_1	TAD	CR	15+	17+	BM 24[h] Bl 24[h]	22/23	46, XY, 7q+, t (8; 21) (q22; q22)	AN
28 MP	M/24	M_2	TAD/ HAM	CR	8+	10+	Bl 24[h]	15/15	46, XY, 7q+, t (8; 21) (q22; q22), del (9) (q22)	AA
29 MR	F/ 9	M_7	BFM-78	PR	—	22	Bl 24[h]	27/27	47, XX, + 8	AA
30 UH	M/42	M_4	TAD	CR	39+	41+	BM 24[h] Bl 24[h]	24/29	47, XY, + 8	AN
31 LA	F/74	M_2	VDPC	PR	—	6	Bl 24[h]	30/30	47, XY, + 8	AA
32 RD	M/43	M_5	TAD	CR	13+	15+	BM 24[h]	11/15	47, XY, + 8	AN

[a] From diagnosis

Table 3. Continued

Patient	Sex / age (years)	FAB classification	Treatment protocol	Treatment result	Duration of CR (months)	Survival (months)[a]	Material studied	Number of abnormal cells / total cells	Main clone(s)	AN/AA
33 KH	M/12	M_7	BFM-78	PR	—	10	Bl 24[h]	4/ 4	47, XY, +8, 18p+	AA
34 HK	M/61	M_4	TAD	PR	—	11	BM 2, 24[h]	5/27	46, XY, −8, +m	AN
35 KH	M/49	M_4	TAD	CR	1+	3+	BM 24[h]	5/10	46, XY, t (8; 10) (q21; q26)	AN
36 SE	F/48	M_5	TAD	NR	—	2	BM 24[h] Bl 48[h]	20/34	a) 47, XX, +8, t (9; 11) (q21; q23) b) 46, XX, t (9; 11) (p21; q23)	AN
37 KS	M/ 1	M_5	BFM-78	CR	38+	40+	Bl 24[h]	20/20	46, XY, −C (9?), +m	AA
38 WP	M/ 1	M_5	BFM-78	CR	7	10	BM 24[h]	9/14	46, XY, t (9; 11) (q12; q11)	AN
39 HI	F/57	M_2	TAD	CR	9	12	BM 24[h]	15/25	46, XX, t (9; 22) (q34; q11)	AN
40 CJ	FM/43	M_2	TAD	CR	3	5	BM 2[h] Bl 24[h]	12/16	46, XX, t (9; 22) (q34; q11)	AN
41 HI	F/64	M_4	TAD	CR	1+	3+	BM 24[h]	11/11	46, XX, del (9) (q22)	AA
42 HI	F/64	M_4	TAD	CR	1+	3+	BM 24[h]	11/11	46, XX, del (9) (q22)	AA
42 TH	M/45	M_4	TAD	CR	39	50+	BM 2, 24[h]	11/23	47, XY, +11	AN
43 MH	F/59	M_1	TAD	NR	—	2	BM 24[h]	10/10	47, XX, +11	AA
44 PG	F/61	M_1	TAD	CR	9+	11+	BM 24[h]	7/ 8	47, XX, +11	AN
45 VH	M/12	M_4	BFM-78	CR	35+	37+	Bl 24[h]	4/17	46, XY, del (11) (q23)	AN
46 KJ	M/22	M_5	TAD	NR	—	2	BM 2, 24[h] Bl 24[h]	41/41	46, XY, del (11) (q23)	AA
47 DW	F/44	M_4	TAD	CR	6	11	BM 24[h] Bl 24[h]	4/16	46, XX, del (12) (p12)	AN
48 TS	F/ 4	M_4	BFM-78	PR	—	5	BM 24[h] Bl 24[h]	8/17	46, XX, del (12) (p11)	AN
49 KM	F/ 1	M_5	BFM-83	CR	10	12+	Bl 24[h]	33/33	48, XX, del (12) (p12), +19, +19	AA
50 DH	F/31	M_4	TAD	PR	—	13	BM 24[h] Bl 24[h]	10/50	46, XX, 12p+	AN
51 DJ	M/51	M_4	TAD	CR	13	24	Bl 24[h]	7/17	46, XY, inv (16) (p13; q22)	AN
52 ME	F/44	M_4	TAD	CR	3+	4+	BM 2, 48[h]	6/20	46, XX, inv (16) (p13; q22)	AN
53 AF	M/ 1	M_1	BFM-78	CR	30+	32+	BM 24[h] Bl 24[h]	20/23	47, XY, +19	AN
54 HK	F/61	M_1	TAD/ HAM	CR	4	6+	Bl 48[h]	8/14	48, XX, 20q+, +m	AN
55 BJ	M/17	M_2	BFM-78	CR	4+	6+	BM 24[h]	3/ 3	45, X, −Y	AA
56 SG	F/65	M_1	TAD/ HAM	CR	4	15	BM 2[h]	2/ 4	46, XX, del (X) (q24)	AN
57 HE	M/65	M_1	TAD	PR	—	11	BM 24[h]	6/ 7	hyperdiploid (n = 53)	AN
58 TA	M/60	M_1	TAD	NR	—	2	BM 2, 24[h]	50/50	hyperdiploid − hypotetraploid	AA
59 JH	M/36	M_5	TAD	CR	4	17	BM 2, 24[h] Bl 24[h]	48/85	hypotetraploid	AN

[a] From diagnosis

The chromosomal aberrations most often found were (Tables 2–4): complex aberrations, t(8; 21), trisomy 8 (in four patients as sole anomaly), monosomy 5 or 7, a 5q-chromosome (always combined with other anomalies), trisomy 11 (in three patients as sole anomaly), a 12p-chromosome, trisomy 6, 11q- and inv [16].

The prognostic significance of the karyotype was evaluated only in the 112 patients treated by combination chemotherapy (Table 5). The proportion of patients with or without maintenance therapy was nearly equal in the groups NN, AN and AA. Overall CR rate was 67%, and this was not different between children (10/15 = 67%) and adults (65/97

Table 4. Clinical and cytogenetic findings in 14 patients with abnormal karyotype receiving only symptomatic treatment

Patient	Sex / age (years)	FAB classification	Material studied	Number of abnormal cells / total cells	Main clone(s)	AN/AA
1 KA	F/70	M_1	BM 2, 24h	3/18	47, XX, +1	AN
2 DE	F/71	M_1	BM 24h	31/31	46, XX, 1p+, del (5) (q12; q33)	AA
3 KE	F/62	M_4	BM 24h	29/41	49, XX, + del (1) (p13), + del (1) (p13), +7	AN
4 RH	M/65	M_1	Bl 48h	9/ 9	48, XY, der (1), +C, +16(?)	AA
5 WM	F/77	M_2	BM 2h	6/ 7	49, XX, +1, del (5) (q12; q33), + del (7) (q32), +11, −16, +22	AN
6 BM	M/70	M_5	BM 24h	2/ 6	46, XY, inv (2) (q22; q13)	AN
7 SA	M/76	M_1	BM 24h Bl 24h	24/27	42, XY, −4, del (5) (q12; q33), −7, 11p+, del (12) (p11), 17p+, −18, −20, +r(4?), dm	AN
8 MR	F/65	M_4	BM 2h	33/33	a) 47, XX, −5, +11, t (16; 17) (p11; p13), +22 b) hypertetraploid	AA
9 AM	F/59	M_3	BM 24h	4/24	46, XX, t (15; 17) (q22; q21)	AN
10 BG	M/52	M_4	BM 24h Bl 24h	15/23	47, XY, +8, inv (16) (p13; q22)	AN
11 MZ	F/14	M_5	Bl 24h	13/15	46, XX, del (17) (q22)	AN
12 SH	M/73	M_2	BM 2, 24h	29/36	45, X, −Y	AN
13 DA	M/75	M_5	BM 24h	20/34	47, XY, +m	AN
14 RM	F/76	M_2	BM 24h	10/17	multiple, complex anomalies	AN

Table 5. Prognostic significance of karyotype in 112 patients treated by combination chemotherpay according to the chromosomal status NN, AN, AA

	Complete remission rate (CR)	Median duration of CR (months)	Mean duration of CR (months)	Median survival time from the date of diagnosis (months)	Mean survival time from the date of diagnosis (months)
NN	36/53 = 68 %	7	13,6	14	24,0
AN	24/34 = 71 %	9	17,8	17	26,6
AA	15/25 = 60 %	30	29,3	42	35,6
	$p = 0.68$	$p = 0.02$		$p = 0.13$	

= 67%). The chromosomal status NN, AN and AA did not significantly influence the CR rate. On the other hand, when considering the types of chromosomal aberrations, remarkable differences were seen: all 10 patients with t (8; 21) achieved CR, whereas only two out of nine patients with complex anomalies did so ($p = 0.001$).

Duration of CR was significantly longer in those with an AA chromosomal pattern than in those with a NN or AN karyotype (Fig. 1). On the other hand, survival time was not significantly influenced by the chromosomal status NN, AN, AA (Fig. 2). When comparing the median duration of CR of the t (8; 21) patients with all NN patients, a highly significant advantage for the t (8; 21) patients is seen (NN: 7 months, t (8; 21): 30 months, $p = 0.003$) (Fig. 3). This good prognosis associated with t (8; 21) also explains the longer remission duration of the AA patients in comparison with the NN and AN group because in 8 of the 15 AA patients the translocation 8; 21 was found.

Discussion

The chromosomal aberration rate of 53% and the types of anomalies found in our patients correspond to the findings of other authors who used the metaphase technique [13–15, 25, 29, 31, 33] but surprisingly we did not find any of the well-known anomalies, such as t (6; 9), t (16; 16) and inv [3].

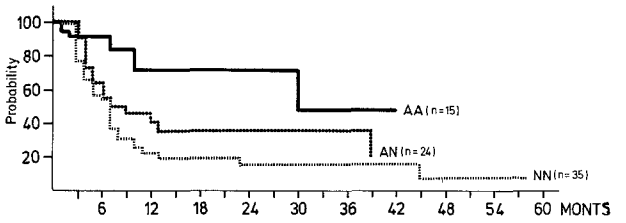

Fig. 1. Probability of complete remission duration according to the chromosomal status NN, AN, AA

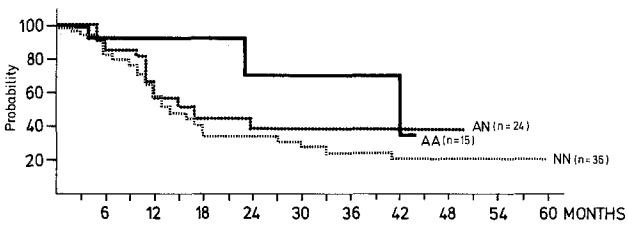

Fig. 2. Probability of survival according to the chromosomal status: NN, AN, AA

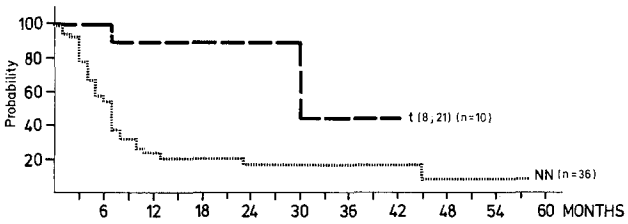

Fig. 3. Probability of complete remission duration of patients with t(8; 21) compared with NN patients

In the majority of cytogenetic studies [2, 5, 7, 13, 15, 25, 26, 31] in AML the karyotype was considered to be of prognostic value. Patients with an AA karyotype had a lower remission rate and/or shorter survival times than patients with a NN or AN chromosomal pattern. Sakurai and Sandberg [36] explained these findings by the hypothesis that AA patients had no more normal stem cells to repopulate the bone marrow after chemotherapy induced bone marrow aplasia. But there are also reports in which the chromosomal pattern NN, AN, AA was of no prognostic significance [8, 18, 19, 28, 29, 39], at least in patients older than 59 years [6]. In our 112 patients with de novo AML treated by combination chemotherapy the chromosomal classification NN, AN and AA was of no predictive value with regard to CR rate or survival after achievement of CR. Besides technical reasons there are several clinical explanations for the discrepancy between our results and those cited above: (1) in contrast to other reports [13, 14, 25], in our study all patients with a known preceding myelodysplastic syndrome were excluded; (2) the proportion of patients not treated by aggressive chemotherapy for clinical reasons is relatively high; (3) supportive treatment has undoubtedly improved over the last years, which led to better results, particularly in high-risk patients. For all these reasons our study and the favorable treatment results may be not completely comparable with other studies, particularly those conducted between 1973 and 1981 [2, 6, 26, 28, 39].

By using a prophase chromosomal technique much more prognostic value has been attributed to *the types* of aberrations rather than to the classification NN, AN, AA. For example, complex anomalies were associated with a very poor prognosis [30, 43], which is confirmed by our results with only 2 out of 9 patients achieving a complete remission. In older studies also, using the metaphase technique, the poor prognostic significance of complex aberrations as well as of anomalies of chromosomes number 5 and 7 was known [13, 17, 19, 29].

On the other hand, translocation 8; 21 seems to be associated with a favourable prognosis. Our high remission rate of 100% corresponds well with the experience of others [4, 7, 16, 17, 25, 29, 30, 34, 39–41]. In contrast the significantly longer duration of CR in comparison with NN patients is seldomly found [25, 30, 34]. Most authors found the duration of CR and survival of patients with t(8; 21) to be not different from other patients with AML [16, 17, 29, 40].

The prognostic significance of other anomalies, such as trisomy 8, inv (16), 11q-, t(9; 11) and t(15; 17) cannot be evaluated here because the number of patients studied is too small. Inv (16) is known to be associated with a favourable course of the disease [22, 27], while the aberrations + 8, 11q-, t(9; 11) and t(15; 17) seem to be of no or not yet evaluable prognostic value [1, 5, 17, 20, 30].

Are further large cytogenetic studies in AML justified and needed to define more exactly the prognostic significance of the karyotype? Therapeutically, combination chemotherapy stagnates more or less, but treatment of AML has been enriched over the last years by bone marrow transplantation and experimental therapeutic approaches such as treatment by low dose ARA-C [10].

If the worse prognosis of patients with complex aberrations is confirmed in further studies, then these patients could be candidates for experimental treatments and bone marrow transplantation in the first complete remission. On the other hand, provided that the good prognosis of inv (16) and t(8; 21)

is confirmed, some or even many of these patients may be cured by combination chemotherapy alone, and bone marrow transplantation would be indicated only in second complete remission. For these reasons the chromosomal status in AML could be of major clinical importance.

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