RESULTS

Table (1) showed the work done to all the 30 leukemic patients including clinical examination, laboratory and radiological investigations and morphological classification (FAB). Age ranged from 1 to 15 years, 19 cases were males (63.3%), and 11 were female (36.7%), with definite male: female predominence, male: female ratio was 1.72:1.

Table (2) showed the distribution of clinical manifestation in whole cases (30 cases). We recorded fatigue (or general weakness) in 28 cases (93.3%). Weight loss was noticed in 21 cases (70%). Bone and joint pain was recorded in 24 cases (80%). Fever without infection was recorded in 17 cases (56.7%). As regard hemorrhagic manifestation, purpra was recorded in 15 out of the 30 cases (50%). Other manifestations of hemorrhage (as epistaxis, bleeding per rectum) were recorded in 7 cases (23.3%). Table (2) showed also the presence or absence of infection at time of initial diagnosis. We recorded 5 cases with infection (16.7%), and 25 cases were free from infection at time of diagnosis (83.3%).

As regard the hematological findings. Table (3) showed that patients presented with a hemoglobin less than 10gm% were 22 cases (73%) and those more than 10gm% were 8 cases (26%). The mean of hemoglobin was 9gm% with SD± 1.84 (range from 6-12.5).

Twenty seven cases were recorded to have platelet count less than 150×10^9 (90%0, and only three cases had platelet count more than 150×10^9 /L (10%). The median of platelet count was 98.5 (range from 45 - 170). Table (3).

Concerning total leukocytic count at diagnosis (TLC). We found that 11 cases had initial TLC less than 10×10^9 /L (36.7%0, 10 cases had initial TLC from $10 - 49 \times 10^9$ /L (33.3%), 5 cases had initial TLC ranging from 50- 99 \times 109/L (16.7%) and 4 cases had an initial TLC more than 100×10^9 /L (13.3%). The median of TLC was 28.5 (range from 6 - 134) Table (4).

Karyotypic study was done to all leukemic patients, successful karyotypic picture was obtained on 22 cases out of 30 studied leukemic cases.

We followed up these 22 cases to observe their prognosis (the response to treatment, whether relapse had occured or not, and their survival during the period of our study). We classified the 22 cases into two groups, group 1 (11 cases) the group of good prognosis (Table 5), and group 2(11 cases) the group of bad prognosis (Table 6).

Followed up of group 2 showed that case number 1 died in the first month after initiation of treatment, case number 2, relapse had occured in both central nervous system and bone marrow, case number 3, relapse also had occured in central nervous system and bone

marrow, case number 4, died after 43 days from induction of treatment, case number 5, died in the first 3 weeks after initiation of treatment, case number 6, relapse had occured in bone marrow three months after induction of treatment, case number 7, died two months after induction, case number 8, relapse had occured in bone marrow six months after starting of treatment followed by testicular relapse within three weeks, cases number 9, relapse had occured in bone marrow and testes after 3 months from induction of treatment, case number 10, died in the third months after initiation of treatment, case number 11, relapse had occured in bone marrow in the ninth month after initiation of treatment.

In group 2 five cases out of the eleven cases died (45.5%), two cases out of eleven cases had testicular relapse (18.2%). Also two cases had central nervous system relapse (18.2%). Bone marrow relapse had occured in six cases (54.5%). Combined hematological and non hematological relapse had occured in 4 cases from the six cases experienced relapse, and isolated hematological relapse had occured in the remmaining 2 cases.

Table (7) showed that 24 cases were presented with hepatomegaly at diagnosis (80%), 21 cases had splenomegaly (70%), and 22 cases were presented, at initial diagnosis, with lymphadenopathy (either generalised or localised) (73.3%).

Three cases only out of the whole cases were presented at diagnosis with mediastinal mass (9%). Involvement of central nervous

system was recorded in 2 cases out of the 30 cases (6%) (the involvement was evaluated at initial diagnosis).

Table (7) showed also that there was neither CNS involvement nor mediastinal mass in cases of group 1 at their diagnosis. Eight cases in group 1 (72.7%) had lymphadenopathy at their diagnosis, all of them were described as a localised lymphadenopathy. Eight cases were presented with hepatomegaly (72.7%) and seven cases were presented with splenomegaly (63.6%).

The same table showed that two cases in group 2 (18.2%) had central nervous system involvement at diagnosis and nine cases (81.8%) were free form central nervous system involvement. Three cases (27.3%) were presented with mediastinal mass at their initial diagnosis. All the eleven cases with 100% presented with lymphadenopathy and hepatosplenomegaly at their diagnosis.

Table (8) showed the comparison between the two groups. Concering male distribution, it was of significant difference as (P < .05). The difference between the two groups (for range of age ≤ 2 years and ≥ 10 years) was of significant difference (P < .05). There was no significant difference between the two groups in distribution of central nervous system involvement and presence of mediastinal mass at diagnosis (p > .05). As regard distribution of generalised lymphadenopathy, it was of significant difference (p < 0.05). Concerning hepatomegaly and splenomegaly there was no significant difference between the two groupes (as regard the presence or absence

of the organomegaly) (p > .05). Table (9) showed the prognostic values of hepatomegaly at different cut off level. The best cut off level to discriminate between group 1 and group 2 suggested was 3 cm, the accuracy was 91%. Table (10) showed the prognostic values of splenomegaly at different cut off level. The best cut off level to discriminate between group 1 and group 2 suggested was 3.5 cm, the accuracy was 86%.

Table (11) showed that the mean of age of cases of group 1 was 5.95 years ($SD\pm 1.63$), the mean of hemoglobin was 8.09gm% ($SD\pm 1.04$). The mean of platelet was 116 ($SD\pm 26.85$) and the mean of total leukocytic count was 13.36 ($SD\pm 9.57$).

In group 2 the mean of age was 6.77 years (SD \pm 5.48), the mean of hemoglobin was 10.73 (SD \pm 1.57), the mean of platelet count was 71(SD \pm 13.94) and for total leukocytic count the mean was 85.36 and (SD \pm 34.41), table (11).

Table (11) showed also the distribution of comparison between group 1 and group 2. Concering the age, the t was 0.47 and p > 0.05 with insignificant difference. The t value of hemoglobin distribution was 4.63 and p < 0.0001 with significant difference. The t value of platelet count distribution was 5.02 and p < 0.0001 with significant difference. The t value of TLC distribution was 6.68 and p < 0.0001 with significant difference.

Concerning FAB morphological classification of leukemic children, we found 4 cases presented with L1 morphology (13.3%), L2 was found in 24 cases (80%) and L3 morphology was found in 2 cases (6.7%).

Table (12) showed that in group 1 only 2 cases (18%) presented as L1 and 9 cases (82%) presented as L2. It is to be mentioned that 10 cases out of the eleven had a normal karyotypic picture, only one case showed abnormal karyotype. And all of them enter remission and remained disease free with no relapse (Table 5).

In group 2 only one case (9.1%) was presented with L3 and the remaining 10 cases (90.9%) were of L2 morphology (Table 12).

Table (13) showed the association between prognosis and karytoype. There was significant difference between normal and abnormal karyotype regarding the prognosis. Normal karyotype was associated with good prognosis, and abnormal karyotype was associated with bad prognosis.

In groyp 2, table (14) showed the association between bad prognosis and abnormal karyotype. There was significant difference between structural and numerical chromosomal abnormalities regarding association with death or relapse. Structural abnormality was evident to be more worse as 5 out of 7 cases died.

Table (15) showed the fate of the studied cases (cases of group 1 and 2) in relation to different prognostic factors. As regard age, 5 cases were ≥10years, 1 case of them (20%) survived and 4 cases with 80% died. 14 cases were > 2 to 9 years, all of them (100%) were survived. 3 cases were ≤ 2 years, 2 cases of them (66.7%) survived and 1 case (33.3%) died. Concerning sex, 14 cases were male, 9 cases of them (64.3%) survived, and 5 cases (35.7%) died. 8 cases were female, all of them survived.

As regard hemoglobin level, 14 cases were < 10 gm/dl, all of them survived. Hemoglobin level > 10 was recorded in 8 cases, 3 of them (37.5%) survived and 5 cases (62.5%) died. Concerning TLC, 8 cases were < 10×10^9 /L, all of them survived. 6 cases were recorded to have TLC from 10 to 49×10^9 /L, 5 caseh (83.3%) survived, and 1 case (16.7%) died. 4 cases were reported to have TLC from 50 - 99 × 10^9 /L, 2 of them (50%) survived, and the remaining 2 died. 4 cases with TLC > 100×10^9 /L were reported, 2 out of them (50%) survived. As regard platelet count, 2 cases had initial platelet count > 150×10^9 /L both of them (100%) survived. 20 cases were < 150×10^9 /L, 15 cases out of the 20 (75%) survived and 5 cases (25%) died.

Concerning morphological classification of ALL patients. 2 cases were L 1, both of them (100%) survived. 19 cases were L2, 14 cases out of the 19 (73.7%) survived, 5 cases (26.3%) died. Only 1 cases recorded to have L3 morphology, this case survived (100%) incidence.

Central nervous system involvement with leukemia was recorded in 2 cases, one of them survived (50%) incidence and the other died (50%). Mediastinal mass was reported in 3 cases, 1 case out of them (33.3%) survived and 2 cases died.

As regard lymphadenopathy, 6 cases were reported to have generalised lymphadenopathy, 3 cases out of 6 survived with an incidence 50%. 13 cases were found to have localised lypmph adenopthy, 11cases out of the 13 survived (84.6%), the remaining 2 cases died with an incidence of 15.4%. Only 3 cases were reported without lymphadenopathy, all of them survived.

Concerning hepatomegaly, 19 cases were found to present with hepatomegaly, 14 out of the 19 cases survived (73.7%), and the remaining 5 cases died (26.3%). Splenomegaly was found in 18 cases, 13 out of 18 cases survived (72.2%) and the remaining 5 cases died (27.8%).

As regard karyotypic picture, normal karyotype were reported in 10cases, all of them (100%) survived. 5 cases were reported to have numerical chromosomal abnormality, all of them survived (100%). Structural chromosomal abnormality were reported in 7 cases, 2 out of the 7 survived (28.6%), and the remaining 5 cases died (71.4%).

We selected five families of luekemic children to study their chromosomes. karyotypic pictures of all members of these families were normal. Figure (22) showed the pedigree of these families. Family number (1) was the family of case number (4). Family number (2) was the family of case number (10). Family number (3) was the family of case number (3). Family number (4) was the family of case number (17). Family number (5) was the family of case number (30) (Table 1).

Figures from 10 to 20 illustrate the karyotypic picture of leukemic case of group 2. Figure number 21 illustrate also the karyotypic picture of case number 9 in group 1. It is to be mentioned that the karyotypic structure of both group was illustrated in table (5 and 6).

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Z	וגי	Z	1	-n	Z	Z	Z	3	Z	ידו	X	K	ידן		MF	Sex
11	7	8.5	9	∞	11.5	12	9.5	9.5	9	9	9.5	10.5	7.5	,	gm.	НВ%
75	95	155	72	90	70	65	89	102	120	115	70	60	110			platlet
134	45	9	6	7	80	95	35	22	8	000	113	120	9		×109/L	пс
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1.6	1.5	1.3	5 7	1	11	5 5	1 2 2	2 5	1 1	. I	1.5	7 2	<u> </u>		-	FAB

TLC: Total leukocytic countHB: HemoglobinM: Male

M HB

: Female: generalised lymphadenopathy: Localised lymphadenopathy

Yr.: Year

Table (1)

30	29	28	27	26	25	24	ម	23	21	8	19	18	17	ō	ŀ	- 2	740.	X.	Case	
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82	110	170	160	124	60	120	90	45	8	120	115	140	108		110	65			platlet	
110	00	32	30	17	36	000	99	76	95	9	25	000		3 \	9	36		×109/L	TLC	
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continue (Table 1)

Distribution of clinical manifestation in whole cases.

n = 30		
- Constitutional manifestation		
Fatigue	28	93.3%
Weight loss	21	70%
Bone and joint pain	24	80%
Fever without infection	17	56.7%
- Hemmorrhagic manifestation Purpra Other hemorhage	15 7	50% 23.3%
- Infection Cases with infection Cases without infection	5 25	16.7% 83.3%

Table (2)

Distribution f Hemoglobin and platelet in whole cases. (number of patient) n = 30

	No.	%
- Hemoglobin		
< 10 gm	22	73%
> 10 gm	8	26%
Range	Ť	12.5
mean		9
SD	1 -	.84
Median	9)
- Platelet		
< 150 × 109	27	90%
> 150 × 109	3	10%
Range	45 - 170	
Median	98.5	

Table (3)

Distribution of total leukocytic count in whole cases.

	n = 30	
	No.	%
TLC	11	36.7%
10 - 49 × 10/L	10	33.3%
50 - 99 × 10/L	5	16.7%
>100 × 10 /L	4	13.3%
Median		28.5
Range		28.5

Table (4)

Group 1

* good						-	1	-	32	170	9.5	T]	7	3
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	+12, +13, +17						-	,	00	120	6	Z	7	9024*
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	!	į	Spieno	нершо	Lymph	Lyn	Mediastinal	CNS	TLC	platlet	НВ%	Sex	Age	Case
e P/agnosis	Chromsomal picture	FAR	Culomo											l

Table (5) Listed the good prognostic cases

* good prognosis: enter in remision remain disease free, no relapse for 1 year.

(•) Indicate the number of the patient in the original table (table 1).

Group 2

	11(13•)	10(30•)		9(22-)		% (21-)	¥(15•)			6(14*)	300				3(7•)		20•)	1(3*)		Po.	CRE
	3	15				رن	10	000000	<u> </u>	2.5	7		115		11		1.5	2			Age
	ידו	M		Ξ	:		1	<		Z	×	;	Z		X		Z	Z		Mr.	Sex
	7	11.5	+		:	12		12.5		=	15		12		9.5		9.5	0.5		377	HB%
	95	82		t	,	 6		 6;		75	à	70	65		89		70	00	6		platlet
	ょ	110	;		7,	- Y	2	36		134	90	×	95		35		113	120	130		TLC
	•	-			+			+		,		•	•		,		ı		•		CNS Leukemia
	t.		,		•						 	+	-		•		•		+		Mediastinal mass
-		-					+	+	+-				-	+		-	-	+	+	80	Lymph
	+		+		+				+		+	+	1							Lo	прһ
	+	-	+		+		+	+	- -		+	+		+		+	- <u>-</u> -, -	+	+		Hepato megaly
-		+	+		+		+	-	-		+	+		+		+		+	+		Spleno megaly
-	ţ	1.2	L2	<u> </u>		-	L2	1	1.5		L3	7.7	;	L2		7.7		に	1.2		FAB
		46 XX - 6, + 22	46 XY t (5, 8)		46 XY - 9p	(8,14)	48 XY + 21, +19, 1		18 XY t (16.22)		45 XY - 12	40 (11 (1), 22)	16 VV + (0 77)	46 XY t (8,14)		45 XY - 22		46 XY - 10, +19	47 XY + 11, + (1, 11)		Chromsomal picture
	+ B.M	Relapse	Died	+ B.M	Relapse Test	+ B.M	Kelapse resu	Dolonce Test	Died	+ B.M	Relapse CNS		Died	Died	+ B.M	Relapse CNS	+ B.M	Relapse CNS	Died		P/agnosis

Table (6) Listed the bad prognostic cases.

B.M.: Bone Marrow.

(•) Indicate the number of the patient in the original table, table (1)

CNS: Central Nervous System.

n = II $0%$ $72.7%$ 11 $100%$ $0%$ 2 $18.2%$ $12.7%$ 11 $100%$ 11 $110%$ $110%$	Group 2 $n = II$ % No. No. 11 100% 22 2 18.2% 2 3 27.3% 3 11 100% 24 11 100% 21
1 1	% No. 100% 22 18.2% 2 27.3% 3 100% 24 100% 21

-117Distribution of comparison between group 1 and group 2.

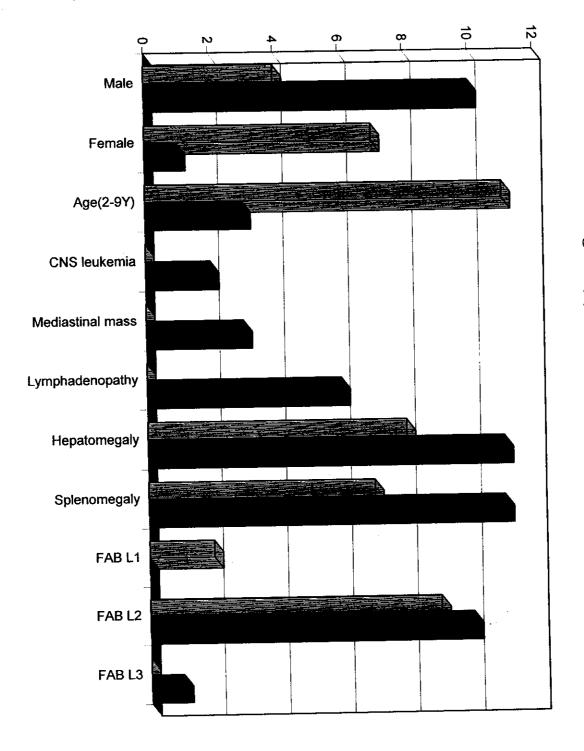
	group 1 n = 11	group 2 n = 11	p
	Number	oj cuses	
- Sex		10	
Male	4 cases	10 cases	p < .05
Female	7 cases	1	•
- Age			
Range from			
> 2 to 9	11	3	P > .05
others (≤ 2 and ≥ 10)	0	8	P < .05
- CNS leukemia	0	2	P>.05
 - Mediastinal mass	0	3	p > .05
- Lymphadenopathy	0	6	P < .005
(generalised)			
- Hepatomegaly	8	11	P > .05
- Splenomegaly	7	11	P > .05

Fisher exact probability test was used in this table.

Table (8)



Figure (1): Comparison between groups 1 & 2



■Group I

Prognostic values of hepatomegaly at different cut off level

Cut off value	2 cm	2.5 cm	3 cm
Diagnostic value			
sensitivity	82%	88%	82%
spicificity	73%	91%	100%
+ ve predictive	75%	90%	100%
- ve predictive value	80%	83%	85%
Accuracy (prevalence)	77%	86%	91%

Table (9)

The best cut off level to discriminate between bad and good groups suggested was 3 cm, the accuracy was 91%.

Prognostic values of splenomegaly at different cut of level

Cut off value	3ст	3.5cm
Diagnostic value		
sen sitivity	73%	73%
specificity	91%	100%
+ ve predictive	89%	100%
- ve predictive	77%	79%
Accuracy(prevalence)	82%	86%

Table (10)

The best cut off level to discriminate between bad and good groups suggested was 3.5cm, the accuracy was 86%.

Figure (2): Prognostic value of hepatomegaly at different cut off levels.

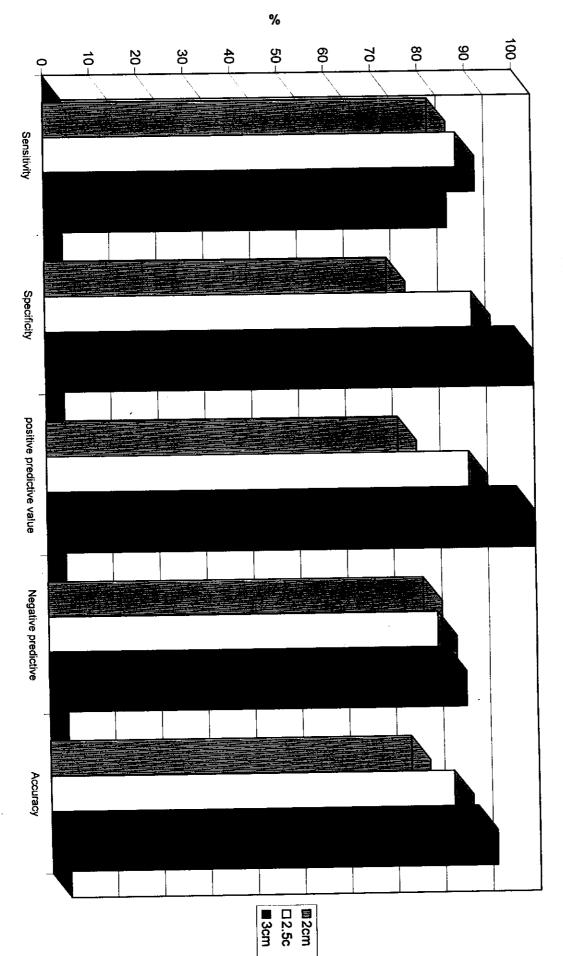
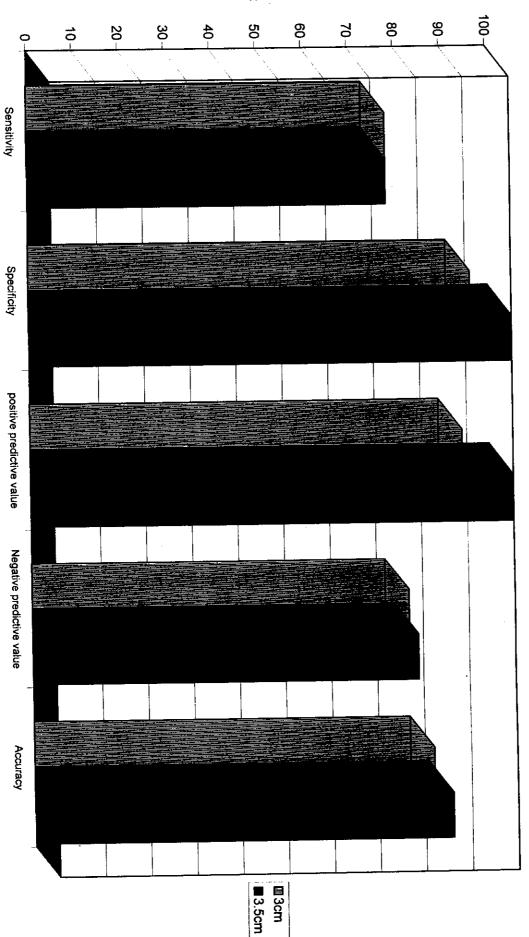




Figure (3): Prognostic value of splenomegaly at different cut off levels.



Comparison between group 1 and group 2

					 - -	
	Group I	p I	Group 2	lp 2		
	n = II	II	n = II	111	ť	p
	Mean	+ <i>SD</i>	Mean	±SD		
		1 63	6 77	+ 5.48	0.47	p > .05
Age	3.73	-				- 0 0001
HB	8.09	± 1.04	10.73	+ 1.57	4.63	p < 0.0001
		2/05	71	+ 13.94	5.02	p < 0.0001
Platelet	110	1- 20.00				0 0001
T1.C	13.36	± 9.57	85.36	± 34.41	6.68	p < 0.0001
ולל						

Table (11)

Figure (4): Age (in years) in groups 1 & 2

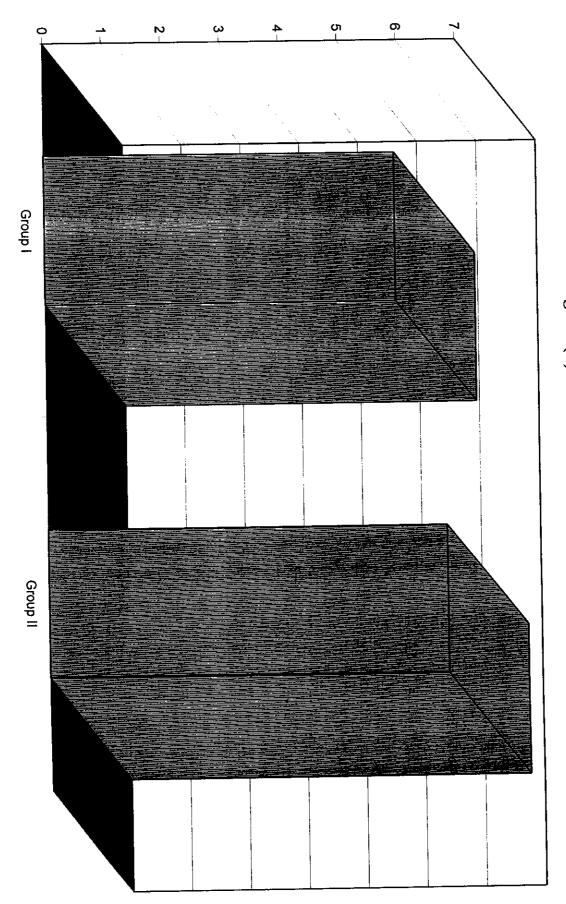


Figure (5): Hb level in groups 1 & 2

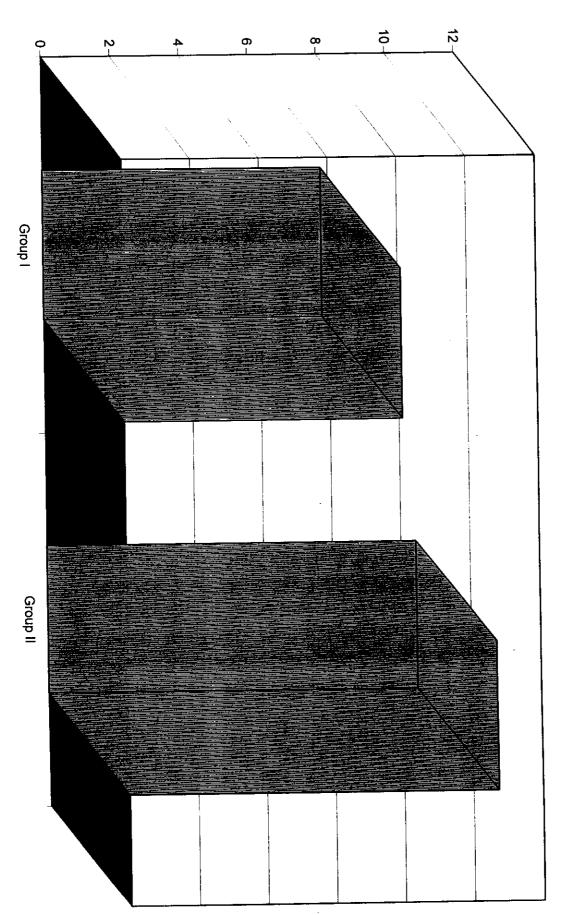


Figure (6): Platelet count in groups 1 & 2

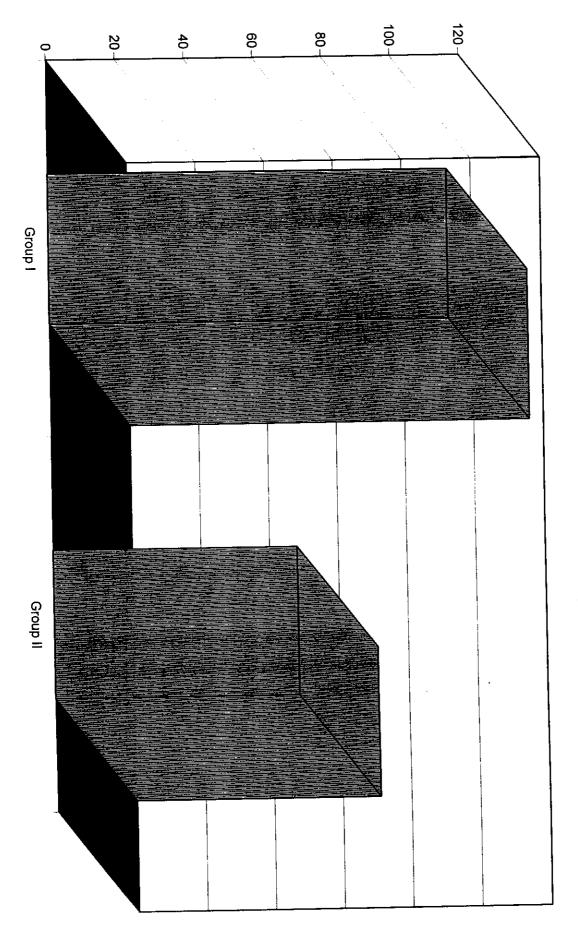
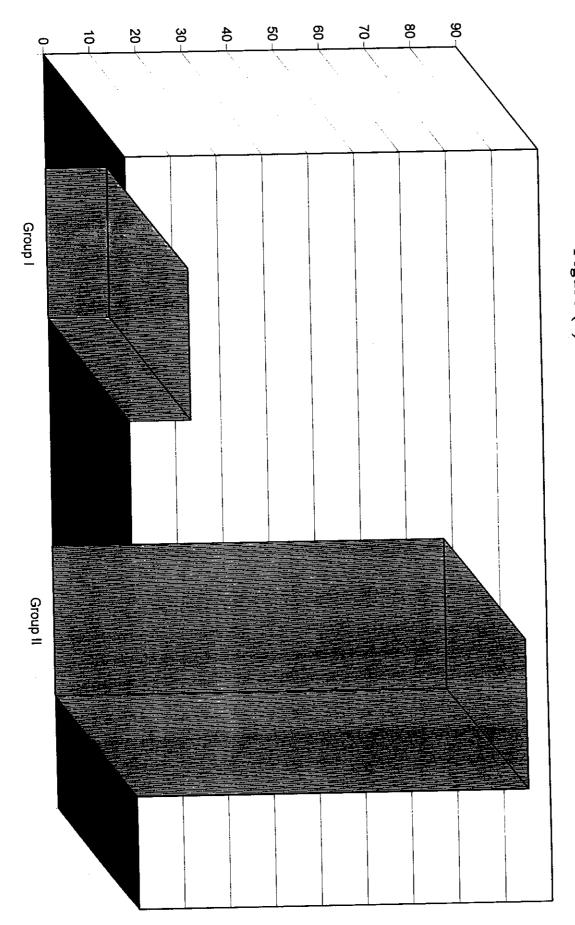


Figure (7): Total leucocytic count in groups 1 & 2



Distribution of FAB morphological classification.

	Group I	ıp I	Group 2	up 2	P
	n = 11	11	n = 11	111	
	Number	%	Number	%	
L1	2	18%	0%	0%	P > .05
L2	9	82%	10	90.9%	P > .05
ا ن	0%	0%	<u> </u>	9.1%	P > .05
L					

Table (12)

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Association between prognosis and karyotype.

	Good p	rognosis	Bad prognosis		
	No	%	<i>No</i>	%	
Normal karyotype	10	90.9%	0	0%	
Abnormal karyotype.	1	9.1%	11	100%	

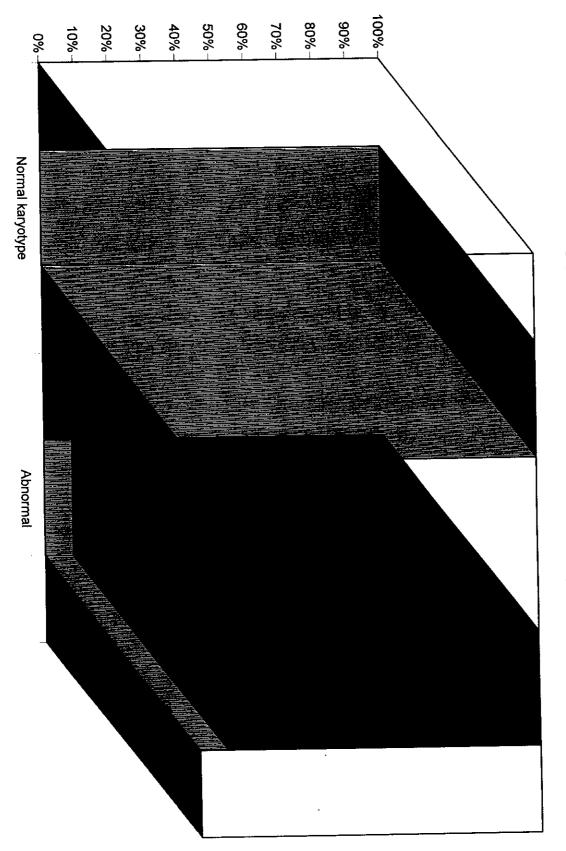
Table (13)

Association between bad prognosis and a bnormal karyotype

	Relapse n = 6		Death n =5	
	No.	%	No.	%
- Structural chromosomal abnormality	2	33.3%	5	100%
- Numerical chromosomal abnormalily	4	66.7%	0 .	0%
	P < 0.05			

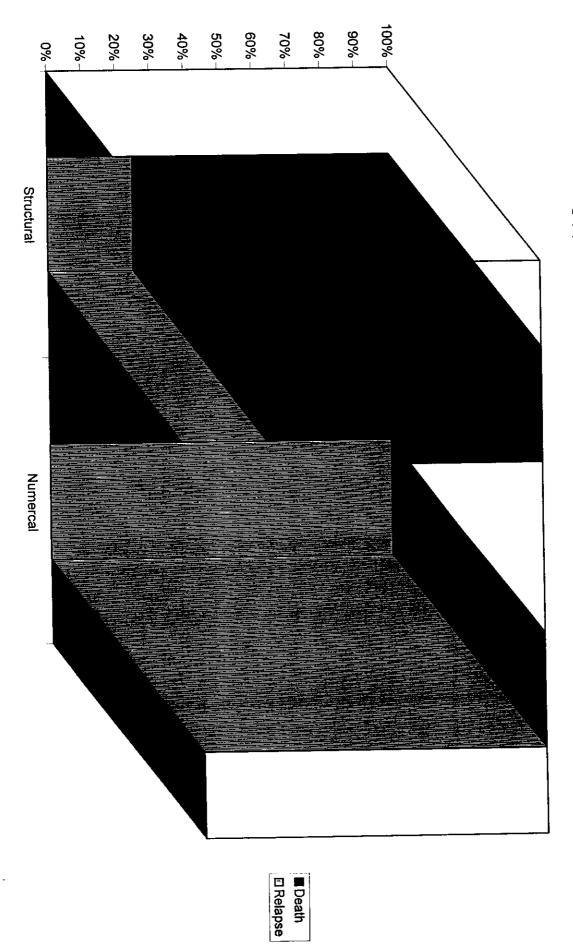
Table(14)

Fig.(8):Association between prognosis and karyotype.



■Bad ■Good

Fig.(9):Association between bad prognosis and abnormal karyotype.



Fate of cases in relation to different prognostic factors.

n = 22 (Two groups).

Patient's data	No. of case	No. of case Sur		Deaths	
_		No. of case	%	No. of case	%
Patient's data					ļ
-Age:					80%
\geq 10 years	5	1	20%	4	
> 2 to 9 years	14	14	100%	0	0%
\leq 2 years	3	2	66.7%	1	33.3
-Sex:				_	25.7
Male	14	9	64.3%	5	35.7
Female	8	8	100%	0	0%
- Hematological data			1		
-H b level gm / d L					
< 10	14	14	100%	0	0%
> 10	8	3	37.5 %	5	62.5%
-TLC × 10 ⁹ / L					
< 10	8	8	100%	0	0%
10- 49	6	5	83.3%	1	16.7%
50 - 99	4	2	50%	2	50%
> 100	4	. 2	50%	2	50%
-Platelet × 10 ⁹ /L					
> 150	2	2	100%	1 .	0%
< 150	20	15	75 %	5	25%
- Morphological			-		
classification					
L 1	2	2	100%	1.	0%
L 2	19	14	73.7%	6 5	26.3°
L 3	1	1	100%	6 0	0%

Patient's data	No. of case	Surv	Survival		Deaths	
Funch Same	9	No. of case	%	No. of case	%	
CNS leukemia	2	11	50%	11	50%	
Mediastinal mass	3	i	33.3%	2	66.7%	
Lymphadenopathy						
Generalised	6	3	50%	3	50%	
Localised	13	11	84.6%	2	15.4%	
No lymphadenopathy	3	3	100%	0	0%	
Hepatomegaly	19	14	73.7%	5	26.3%	
Splenomegaly	18	13	72.2%	5	27.8%	
Karyotype						
Normal	10	10	100%	0	0%	
Numirecal above could	5	5	100%	0	0%	
Structural	7	2	28.6%	5	71.4%	

Continue Table (15)

Figure (10): : Karyotypic picture of case No. 1, group 2, 47 XY + 11, t (1, 11)

Figure (11): Karyotypic picture of case No. 2, group 2, 46 XY - 10, + 19

Figure (12): Karyotypic picture of case No. 3, group 2, 45 XY - 22

Figure (13): Karyotypic picture of case No. 4, group 2, 46 XY t (8, 14)

Figure (14): Karyotypic picture of case No. 5, group 2, 46 XY t (9, 22)

Figure (15): Karyotypic picture of case No. 6, group 2, 45 XY - 12

Figure (16): Karyotypic picture of case No. 7, group 2, 46 XY t (16, 22)

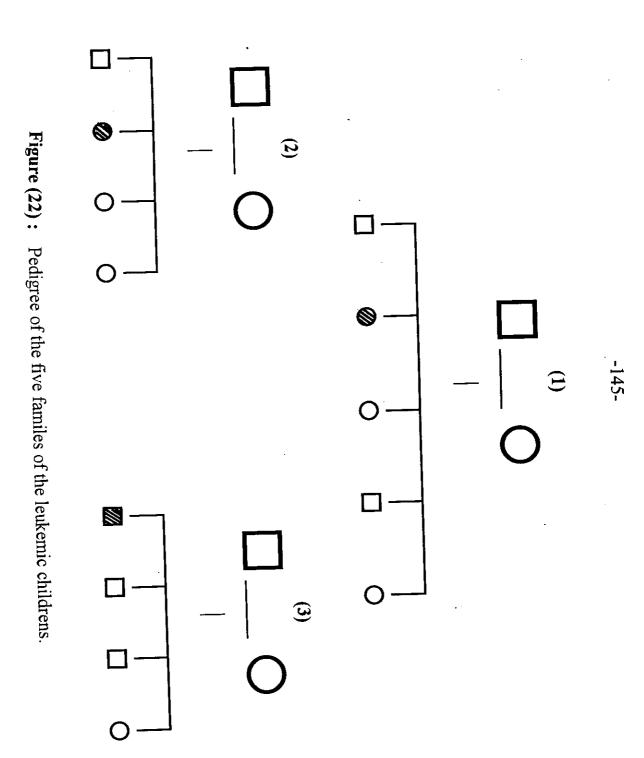
Figure (17): Karyotypic picture of case No. 8, group 2, 48 XY + 21, +19, t (8, 14)

Figure (18): Karyotypic picture of case No. 9, group 2, 46 XY - 9p

Figure (19): Karyotypic picture of case No. 10, group 2, 46 XY t (5, 8)

Figure (20): Karyotypic picture of case No. 11, group 2, 46 XX - 6, + 22

Figure (21): Karyotypic picture of case No. 9, group 1, 50 XY + 6, + 10, - 11, + 12, + 13, + 17



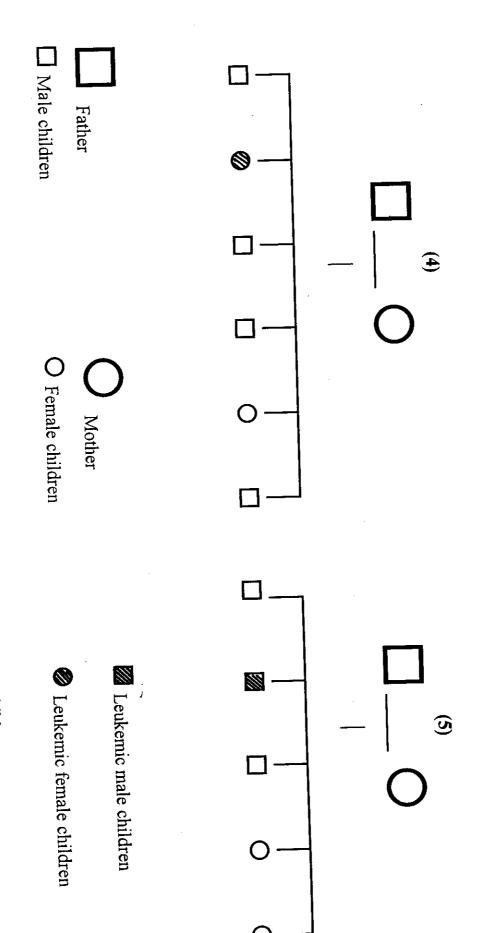


Figure (22): Pedigree of the five familes of the leukemic childrens.