TJPS

TIKRIT JOURNAL OF PURE SCIENCE

Journal Homepage: http://main.tu-jo.com/ojs/index.php/TJPS/index



Cytogenetic study of Acute myelogenous Leukemia in Erbil City-Iraqi Kurdistan Region

Kazhall Muhammad Sulaeman

Biology Department, Education College, Salahadin University, Erbil, Kurdistan Region, Iraq

ARTICLE INFO.

Article history:

-Received: 22 / 6 / 2017 -Accepted: 29 / 10 / 2017

-Available online: / / 2018

Keywords:Acutemyelogenousleukemia,chromosomeaberrations,Blooddiseases,diseases, Smoking.Genetic

Corresponding Author: Name: Kazhall Muhammad Sulaeman E-mail: Kazhalbio@yahoo.com Tel:

Affiliation:

Abstract

 ${
m A}$ cute myelogenous leukemia is a blood disorder. It is tenth most frequent cancer, and the third leading cause of cancer death through the world. Acute myeloid leukemia is clinically heterogeneous disorder characterized by multitude of chromosomal changes. The objective of the present study is to study frequency of chromosomal aberrations (CA) in peripheral blood lymphocytes of Acute myelogenous leukemia patients in Erbil City. Blood samples were collected from 50 patients and 20 controls from both sex at different age groups. Because Acute myeloid leukemia is a heterogeneous disorder; genetic factors, smoking habit, patients occupation, blood diseases, genetic diseases and various environmental factors contribute to its development. Other factors was also studied included patient gender and age. The results showed that a significant increases in the chromosomal abnormalities in the patients when compare to control group and that highest value of chromosomal aberrations was (Dicentric chromosome) which occurred in males at fourth age group (60-69), also it showed that most patients are males who are at age group(60-69)year, Also the results showed that most of patients have smoking habit especially males, and because environmental and occupational exposures to chemicals play an important role, So the most patients have mylodysplasia blood disease.

1-Introduction

Acute myelogenous leukemia is cancer of myeloid part of blood cells, characterized by rapid growth of abnormal white blood cells which accumulate in bone marrow. Acute myeloid leukemia is the common type of acute leukemia that affecting older people [1,2].

The Commonest four Cancers death registered in Erbil City-Kurdistan Region, represent 100 case during 2011-2015[3]. The annual incidence rate for leukemia based on gender and age .The common type of leukemia in Sulaymaniyah Province for the period 2009–2013 was calculated from data collected at Hiwa Hospital. The statistics indicated that there was higher incidence rate of leukemia among male population (4.1) compared to females (3.6) .Acute myeloid leukemia represent 17% of all type of leukemia. This is in agreement with the latest international leukemia statistics [4].

Acute myeloid leukemia (AML) incidence is very strongly related to age, with the highest incidence rates being in older males and females [5]. The risk factors for adult acute myeloid leukemia include high dose of ionizing radiation, benzene and some types of chemotherapy, these factors in combination responsible for only a small percentage of cases. Smoking was first hypothesized as a risk factor [6,7]. Smoking is associated with nearly about 50% of increases in leukemia risk [8, 9].

Many risk factors for developing of acute myelogenous leukemia have been founded which including (blood disease, exposure to chemical substances and genetic disorder). Myelodysplastic syndrome can also evolve into acute meylogenous leukemia [10]. Exposure to aromatic organic solvents and benzene is a strong cause of acute myeloid leukemia[11]. A hereditary risk for acute myeloid leukemia appears to exist many congenital conditions that increase the risk of leukemia, the most common is Down syndrome [12,13].

At present, cytogenetic parameters are used successfully in clinics for risk stratification of leukemia [14]. Chromosome abnormalities are common and many patients have cytogenetic changes that are associated with poor risk features[15]. Diagnosis of patients who were suffering from acute myelogenous leukemia are influenced by genetic changes in leukemic cells, which include chromosomal aberrations, gene mutations and changes in gene [16].

The present study was conducted to determine the chromosomal aberration in acute myeloid leukemia (AML) patients and to determine the main risk factors for developing this diseases in Errbil City- Iraqi Kurdistan Region.

2- Materials and methods

The present study was carried out at Research center in Salahaddin University. This study was performed on 70 individuals, 50 patients diagnosed with acute myeloid leukemia (AML) in Erbil city from Nanakali hospital and 20 control healthy individuals, between February 2016 to July 2016, Each patient was provided with a special questionnaire form, which includes some fields of information to be filled about the patient that are (Name, Address, Gender, Age, smoking habit, blood diseases, genetic diseases and their occupation). Furthermore, the questionnaire forms were filled in through direct interviews with patients and their relatives as well as using medical records available in the hospital with the help of doctors and staff. Cytogenetic analysis was performed on metaphases from peripheral blood lymphocyte with the use of standard procedures.

2- Blood Sampling and culture

About two ml of blood were collected from each patients 6-7 drops of heparinized blood were incubated in 5 ml of culture media(Rosswell Park Memorial Institute 1640) (RPMI- 1640 with 0.5 ml fetal Bovin serum, then 0.3 ml of phytohemagglutinin(PHA) was added. Cell cultures were incubated for 72 h at 37°C After 71 h we added 0.2 ml Colcemid to each tube and then after 1 h incubation both cultures were harvested using a classic technique that include centrifugation, treatment with hypotonic solution of KCL for 20 min followed by repeated fixation with methanol/acetic acid(3/1) [17]. After centrifugation and adding of fixative take 4 drops of cell suspension and dropped on slide. The slide was stained with gimsa stain for 2-4 minutes. Then the slid was washed and left to dry at room temperature Microscopic examination was performed then at least 20 metaphases were analyzed to identify structural chromosomal aberrations. Cytogenetic analysis was performed in patients by using the GTG(Giemsa)banding technique according to the protocol of Iraqi center for cancer and medical genetic research (ICCMGR) [17].

Statistical Analysis of data was performed, using SPSS software application, the statistical test which is used called factorial experimental design to study the chromosomal aberrations in acute myelogenous Leukemia patients in different age and sex groups. Using a prepared program of SPSS version 18, the results were analyzed.

3- Results and Discussion

Figure (1) showed that most patients who were suffering from acute myeloid leukemia are males represent(68%) this because of their occupation, while female represent (32 %). Also it shows that most patients are at age (60-69) years, represent (34%). There are differences in cancer incidence between males and females [18]. These results were similar to finding reported by [19] who concluded from a study on the prevalence of acute leukemia among a group of Iraqi patients that acute myelogenous leukemia is more common in men than in females. [20] Concluded from a study of cancer mortality in the European union that in Acute myelogenous leukemia the mortality rate for males is higher than that for females. Also similar results were obtained by[21] who concluded that acute myelogenous leukemia accounts for 25% of all leukemia diagnosed in adults.

Figure (2) shows that most patients having smoking habit represent(50%) who are males The mechanism by which tobacco products may influence leukemia risk is unknown [22]. This results agree with results obtained by [23] who concluded that smoking is a main risk factor to cause leukemia in elderly people[24]. suggested that 58% of smoking that induced mortality among acute myeloid leukemia cases could be due to benzene which present in cigarette smoke.

Figure (3) shows that Acute myelogenous Leukemia were more commonly in house and cars dyer or painters represent(37%) who are males. The results were similar to finding reported by [25] who concluded that acute myeloid leukemia development has been correlated with exposure to many types of environmental agents, most of them due to links between the exposure history and cytogenetic changes. Exposure to radiation, alcohol consumption, inhalation of benzene, smoking, dyes and exposure to pesticide and herbicide have all been implicated as potential risk factors for development of acute myeloid leukemia. Also the results of the present study agree with results obtained by [26] who concluded from study of risk of developing acute leukemia after employment as a painter.

Figure (4) shows that AML patients having different blood diseases included Myelodysplasia (30%), Myelofibrosis (20%) Polycythaemia (20%) all represent (70%), while those without any blood diseases represent (30%) while figure (5) shows that most patients having different genetic diseases included Fanconi anemia (30%), Down syndrome (20%) all represent(50%), while those without any blood diseases represent (50%) .The myelodysplastic syndromes also known as MDS or myelodysplasia, are hematological (i.e., blood-related) medical conditions with ineffective of all blood cells [27]; [28] concluded that children with Down syndrom are known to have an increased high risk of acute leukemia, including AML, while[29] concluded that

Tikrit Journal of Pure Science 23 (1) 2018

Fanconi anemia FA patients have 500-fold higher risk of acute myelogenous leukemia than the general

population.







Figure 2: Distribution of patients with Acute myelogenous Leukemia according to smoking habit



Figure 3: Distribution of patients with Acute myelogenous Leukemia according to their occupations







Figure 5: Distribution of acute myelogenous leukemia patients according to the incidence of enetic diseases

The Study of chromosomal aberrations (CAs) in acute myelogenous leukemia patients from (50) samples of blood (5) samples show normal chromosomes. While (45) samples show different types of chromosomal aberrations. The value present in table(1) represent mean, while (C1 and C2) represent control and patients respectively. Age groups included (A1, A2,A3and A4) which represent age group (30-39), (40-49), (50-59), (60-69) respectively. Study of both sex, males and females represent (S1 and S2) respectively.

Table, (1) show highly significant effect at (P < (0.01))of both (control and patients) on different chromosomal aberrations including (Dicentric and chromatid interchange: chromosome quadriradialy), as shown in figures (7a, b and c) and all the differences between mean values were clear as shown in table (2,),were the value of L.S.D at both levels(P<0.05) and P<0.01) as follows (0.223,0.321), (0.264, 0.380) respectively. All age groups shown highly significant effect on all forms of chromosomal aberrations, The values of L.S.D. were (0.315,0.457) (0.374,0.540) respectively .Also sex shown highly significant effect, the value of L.S.D. (0.223,0.321), (0.264, 0.380) respectively in both males and females. Table (2) shown that higher value of chromosomal aberrations (Dicentric chromosome) (6.166 ±0.966) (0.223,0.321) which occurred in acute myelogenous Leukemia patients. While in age groups shown that the higher value of aberrations was (deicentric chromosome) (6.00±1.854) which occurred at fourth age group (60-69). From study of sex the results shown that the higher value of chromosomal aberrations was (dicentric chromosome) (4.375 ± 1.116) in males. The interaction between states, ages and sex, the results shown that higher value of aberrations was (Dicentric chromosome) (15.333±0.332), which occurred in Acute myelogenous leukemia patients who are males at fourth age group.

Acute myelogenous leukemia is classified as heterogenous disease leading to chromosomal changes, gene mutations and changes in expression of many genes and micro RNAs. Chromosomal aberrations can be founded in 50% to 60% of acute myeloid leukemia patients[30]. [31] concluded that many types of chromosomal aberrations like monosomy, deletion of chromosomes5 or 7 and trisomy 8 were common in acute myelogenous patients.[32] concluded that chromosome aberrations may also include 11q, balanced type of translocations between chromosome 15 and 17 t(15:17); chromosome 8 and 21 (t98;21)); and others like (q22;q22),(q31;q22) and (9;11).[33] noted an increase of chromosomal deletion with age mostly in chromosomes 5 and 7 [34]concluded that increases in the incidence of acute myelogenous leukemia with complexes chromosomal aberrations with increases age.

Chromosomal aberrations have been founded more frequently in acute myeloid leukemia patients who were smokers compare to non smokers including aberrations, The reasons for increased risk of acute meylogenous leukemia in smokers specially older individuals was decreased ability to repair DNA damage. [35,36] concluded that adult acute myeloid leukemia patients have chromosomal abnormalities present in all dividing bone marrow cells at diagnosis period which included translocation (t(1:8), (p32.1;q24.2), and inversions (19:9)(p13.3;q21q34) that may possibly have originated transcripts with leukemogentic potential.

both seats at unrefent age groups)				
		Mean square(MS)		
Source of variation	d.f	Dicentric chromosome	Chromatid interchange (Triradial)	
Cases(Control,C1: AML Patients,C2)	1	385.333**	280.333**	
Age(A)	3	56.944**	29.639 **	
Sex(S1,S2)	1	52.083**	12.000**	
Case/ Age(C/A)	3	66.833**	24.556 **	
Case/ Sex(C/S)	1	44.083**	18.750**	
Age/ Sex(A/S)	3	9.917**	1.556**	
Case/ Age/Sex (C/A/S)	3	5.806**	2.528**	
Error	32	0.208	0.292	
Total	48			

 Table 1 : Analysis of Variance to study the chromosomal aberrations in acute myelogenous patients (in both sexes at different age groups)

* P<0.05

* * P< 0.01

(Statistical Analysis of data was performed, using SPSS software version 18 Application) and the statistical test which is used called factorial experimental design.

	Chromosomal aberrations			
Factors	Dicentric	chromatid interchang		
	chromosome	(quadriradial)		
Control(C1)	0.500±0.104	0.458±0.103		
AML Patients(C2)	6.166 ±0.966	5.291±0.615		
L.S.D(0.05)	0.223	0.264		
(0.01)	0.321	0.380		
A1(30-39)	0.916±0.148	1.00 ± 0.248		
A2(40-49)	2.416±0.570	2.166±0.637		
A3(50-59)	4.000±1.337	4.500±1.161		
A4(60-69)	6.000±1.854	4.333±1.123		
L.S.D	0.315	0.374		
	0.457	0.540		
S1(Males)	4.375±1.116	3.375±0.779		
S2(Females)	2.291±0.549	2.375±0.517		
L.S.D	0.223	0.264		
	0.321	0.380		
C1A1	0.666±0.210	0.666 ± 0.210		
C1A2	0.833±0.166	0.333±0.210		
C1A3	0.000±0.000	0.500±0.223		
C1A4	0.500±0.223	0.666±0.210		
C2A1	1.166±0.166	1.166±0.210		
C2A2	4.000±0.632	4.000±0.632		
C2A3	8.000±1.211	7.500±0.991		
C2A4	11.5000±1.727	8.000±0.365		
L.S.D	0.446	0.529		
	0.646	1.255		
C1S1	0.750±0.130	0.333±0.142		
C1S2	0.250±0.130	0.583 ± 0.142		
C2S1	2.583±0.528	2.833±0.474		
C2S2	9.750±1.135	7.750±0509		
L.S.D	0.315	0.374		
	0.457	0.540		
A1S1	1.000±0.258	1.166±0.401		
A1S2	0.833±0.166	0.833±0.307		
A2S1	3.00±1.064	2.666±1.210		
A2S2	1.833±0.401	1.666±0.494		
A3S1	5.333±2.389	5.000±0.365		
A3S2	2.666±1.201	3.000±1.064		
A4S1	8.166±2.208	4.666±1.801		
A4S2	3.833±1.720	4.833±1.505		
L.S.D.	0.446	0.529		
	0.646	1.255		
C1A1S1	0.666±0.333	0.333±0.333		
C1A1S2	0.666±0.333	0.333±0.333		
C1A2S1	0.666±0.333	0.000 ± 0.000		
C1A2S2	1.000±0.000	0.666±0.333		
C1A3S1	0.000 ± 0.000	0.333±0.333		
C1A3S2	0.000±0.000	0.666±0.333		
C1A4S1	1.000±0.000	0.666±0.333		
C1A4S2	0.000±0.000	0.666±0.333		
C2A1S1	1.333±0.333	2.000±0.000		
C2A1S2	1.000±0.000	1.000±0.000		
C2A2S1	5.333±0.333	5.333±0.333		
C2A2S2	2.666±0.333	2.333±0.333		
C2A3S1	10.666±0.333	9.333±0.333		
C2A3S2	5.333±0.333	5.333±0.333		
C2A4S1	15.333±0.333	8.333±0.333		
C2A4S2	7.666±0.633	7.333±0.333		
L.S.D	0.631	0.748		
	0.914	1.083		
Statistical Analysis of data v	tatistical Analysis of data was performed, using SPSS software version 18 application			
and the statistical tes	and the statistical test which is used called factorial experimental design.			

Table 2 : Mean ± S.E to study chromosomal aberrations in acute meylogenous patients



Figure 6: Normal human chromosomes, male sample (1000 X, Giemsa stain)



Figure 7 a: Human chromosomes, male sample (1000 X, Giemsa stain)



Figure 7 b: Human chromosomes, male sample (1000 X, Giemsa stain)



Figure 7 c: Human chromosomes, female sample (1000 X, Giemsa stain) Figure (7a,b,c,): Chromosome aberrations in lymphocytes of patients who were suffering from Acute myelogenous leukemia in Erbil City in both sexes at different age groups. (1000 X, Giemsa stain)

4- Conclusions

From the results of the present study the following conclusions could be considered:

1- Chromosome aberrations was observed in patients who were suffering from Acute myelogenous leukemia in Erbil City included (dicentric chromosome and Chromatid interchange quadriradial). The highest value of chromosomal aberration was (Dicentric chromosome) which occurred in males at age (60-69) year.

References

1. Jemal A., Thomas A., Murray T., and Thun M. (2002). Cancer statistics, Cancer J Clin 52 (1): 23–47. 2. Xie Y., Davies S.M., Xiang Y., Robison L.L. and Ross J.A.(2003). Trends in leukemia incidence and survival in the United States (1973–1998). Cancer. 97: 2229–2235.

3. General directorate of health –Hawler-2012-2016.

4. Karim Zh., Khidhir K., Ahmed R., Hassan H.G. and Karim D.O. (2016). Leukemia Study in Sulaymaniyah Province, Kurdistan, Iraq, Chin Med J(Engl). 20; 129(2): 244–245.

5. Parkin D.M., Ferlay J., Raymond L. and Young J.(2000). Cancer Incidence in Five Continents. Volume VII. IARC Scientific Pub. No. 143. Lyon, France: IARC Scientific Publications

6. Pogoda J.M., Martin S.P., Nichols P.W. and Ross R.K. (2002). Smoking and Risk of Acute Myeloid Leukemia: Results from a Los Angeles County Case-Control Study. Am. J. Epidemiol.155 (6): 546-553.

7. Kinlen L.J. and Rogot E.(1988).Leukemia and smoking habits among United States veterans. Br Med J; 297: 657–659.

8. Garfinkel L. and Boffetta P. (2003). Association between smoking and leukemia in two American Cancer Society prospective studies, Cancer, 65: 2356–2360.

9. Chelghoum Y., Danaïla C., Belhabri1 B., Charrin C., Le O., Fiere1D. and Thomas S.(2002). Influence of cigarette smoking on the presentation and course of acute myeloid leukemia, Ann Oncol. 13 (10): 1621-1627.

10. Sanz GF., Sanz MA., Vallespí T., Cañizo MC., Torrabadella M., García S., Irriguible D.and San Miguel JF. (1989). Two regression models and a scoring system for predicting survival and planning treatment in myelodysplastic syndromes: a multivariate analysis of prognostic factors in 370 patients. Blood. 74 (1): 395–408.

11. Austin H., Delzell E. and Cole, P. (1988). Benzene and leukemia. A review of the literature and a risk assessment, Am J Epidemiol 127 (3): 419–39.

12. Taylor G.M. and Birch J.M. (2006). The hereditary basis of human leukemia, Leukemia (6th ed. p. 210.

13. Horrwitz M., Goode E.L. and Jarvik G.P. (1996). Anticipation in familial leukemia, Am. J. Hum. Genet. 59(5):990-8.

14. Haj F.B., Markoya L.D., Klaman F.D., Bohmer D. and Neel D.J.(2003). Regulation of receptor

2- Most patients who were suffering from Acute myelogenous leukemia are males at age 60-69).

3- Most of them have smoking habit specially males and there are painters.

4-Most of patients have mylodysplasia blood disease, while most of them were without any genetic diseases.

tyrosine kinase signaling by protein tyrosine phosphatase, J.BIOL. Chem. 278:739-744.

15. Heaney M.L and Soriano G. (2013). Acute myeloid leukemia following a myeloproliferative neoplasm: clinical characteristics, genetic features and effects of therapy, Curr Hematol Malig Rep.8(2):116-22.

16. Mrozek K. and Bloomfied C.D. (2006). Chromosome aberrations, gene mutations and expression changes, and prognosis in adult acute myeloid leukemia, Hematology Am Soc Hematol Educ Program.:169-77.

17. Yaseen N.Y. ,Humadi A.A. ,Tawfiq M.S. and Estivan A.G. (1998).Cytogenetic studies on patients with chronic Myelocytic leukemia, Med. J. Tikrit Univ.,4:5-9.

18. Drak M.T and Karpuzoglu E.(2012). Gender differences in cancer susceptibility: an inadequately addressed issue, Frontiers in Genetics.3(2).110-134.

19. Mohammad T.Kh., Mahmood A.H., Elew G.F. and Al-Khalidi S.J. (2009). A study of the prevalence of acute leukemia among a group of Iraqi patients, Journal of Al-Nahrain University.12 (2) ,pp.107-112.

20. Levi F., Lucchini F., Negri E. and La Vecchia, C. (2002).Cancer mortality in the European Union, 1988–1997: the fall may approach 80,000 deaths a year, Int J Cancer. 98: 636–637.

21. Jemal A., Siegel R., Xu J. and Ward E.(2010). Cancer statistics, Cancer J Clin.60.277-300.

22. Sandler D. P. and Collman G.W.(1987). Cytogenetic and environmental factors in the etiology of the acute leukemias in adults, Am J Epidemiol .126: 1017–1032.

23. Sandler D.P., Shore, D.L. and Anderson J.(2013). Cigarette smoking and risk of acute leukemia: associations with morphology and cytogenetic abnormalities in bone marrow. J Natl Cancer Inst; 85(4).

24. Korte J.E., Hertz-Piccioto I. and Schulz M.R. (2000). The contribution of benzene to smoking induced leukemia, Environ Health Perspect. 108:333-9.

25. Crane M.M., Strom S.S. and Halabi S. (1996). Correlation between selected environmental exposures and karyotype in acute myelocytic leukemia, Cancer Epidemiol Biomarkers Prev. 5:639-44.

26. Lindquist R., Nilsson BS., Eklund G., Odont D.and Gahrton G.(1987) Increased risk of developing

acute leukemia after employment as a painter. Cancer Journal. 60(6).P 1378–1384

27. Kasper D., Braunwald L. and Eugene. F. (2005). Harrison's Principles of Internal Medicine (16th ed.). New York: McGraw-Hill. p. 625.

28. Rosner F. and Lee S. (1972) .Down syndrome and acute leukemia: Myeloblastic or lymphoblastic: Report of forty-three cases and review of literature. Am J Med 53:203.

29. Shimamura A. and Alter B.P. (2010). Pathophysiology and management of inherited bone marrow failure syndromes, Blood Rev .24:101–122.

30. Martens J .H. and Stunnenberg H.G. (2010).The molecular signature of oncofusion proteins in acute myeloid leukemia, FEBS Lett.584:2662.

31. Byrd J.C., Mrozek K. and Dodge R.K.(2002). Pretreatment cytogenetic abnormalities are predictive of induction success, cumulative incidence of relapse, and overall survival in adult patients with de novo acute myeloid leukemia: results from Cancer and Leukemia Group B(CALGB 8461), Blood. 100:4325-36.

32. Mrozek K., Radmacher M.D., Bloomfield C.D .and Marcucci G. (2009). Molecular signatures in acute myeloid leukemia. Curr Opin Hematol. 16:64-9.

33. Moorman A.V., Roman E., Willett E.V., Dovey G.J., Cartwright R.A. and Morgan G.J. (2001) Karyotype and age in acute myeloid leukemia. Are they linked? Cancer Genet Cytogenet. 126:155-161. 34. Schoch C., Kern W. and Krawitz P.(2001). Dependence of age-specific incidence of acute mveloid leukemia on karyotype ,Blood J.; 98:3500. 35. Sandler D.P., Shore D. and Anderson JR.(1993). Cigarette smoking and risk of acute leukemia: associations with morphology and cytogenetic abnormalities in bone marrow. J Natl Cancer Inst;85: 36. Agnes C., Fett C., Roseli V., Cristina B., Vendrame G., Andréa B., Carvalho S., Octávio R. and Marileila V. (2007). Atypical chromosome abnormalities in acute myeloid leukemia type M4, Genet. Mol. Biol. 3 (1).

دراسة وراثة خلوية لسرطان الدم النخاعي الحاد في مدينة اربيل/ اقليم كردستان العراق كة ذال محمد سليمان

قسم علو م الحياة ، كلية التربية ، جامعة صلاح الدين ، اربيل ، العراق

الملخص

مرض سرطان الدم النخاعي هو احدى امراض الدم و واحدة من عشرة انواع السرطانات المالوفة والمسبب الثالث للموت في المصابين بالسرطان في العالم، ان سرطان الدم النخاعي تعرف على انه مرض متغاير سريريا وتتميز بكثرة التشوهات الكروموسومية. ان الهدف من الدراسة الحالية هي معرفة التشوهات الكروموسومية. ان الهدف من الدراسة الحالية هي معرفة التشوهات الكروموسومية في الدم المحيطي لمرضى سرطان الدم النخاعي في مدينة اربيل. لقد تم دراسة (50 مريض) مصابين بسرطان الدم النخاعي في مدينة اربيل. لقد تم دراسة (50 مريض) مصابين بسرطان الدم النخاعي في محافظة اربيل و (20 شخص من اصحاء) كمجموعة سيطرة في كلا الجنسين وفي مجاميع عمرية مختلفة. بسبب كون مرض سرطان الدم النخاعي في مدينة اربيل. لقد تم دراسة (50 مريض) مصابين بسرطان الدم النخاعي في محافظة اربيل و (20 شخص من اصحاء) كمجموعة سيطرة في كلا الجنسين وفي مجاميع عمرية مختلفة. بسبب كون مرض سرطان الدم النخاعي في موان الدم النخاعي في مدون متغاير لهذا تلعب عدة عوامل تساهم في تطوير المرض منها عوامل وراثية، التدخين، مهنة المريض، امراض الدم، امراض وراثية و عوامل بيئية مختلفة, لقد تمت دراسة عوامل اخرى ايضا درست تضمنت جنس المريض وعمره. من الدراسة الحالية تمت الاستنتاج المراض وراثية و عوامل بيئية مختلفة, لقد تمت دراسة عوامل اخرى ايضا درست تضمنت جنس المريض وعمره. من الدراسة الحالية تمت الاستنتاج بينه توجد زيادة معنوية في التشوهات الكروموسومية في المرضى مقارنة بمجموعة السيطرة، وان اعلى قيمة للتشوهات الكروموسومية هي التشوه البنه وركر وي الموسوم ثنائي السنترومير) التي وجدت في الذكور في المجموعة العمرية الرابعة (60-69) سنة، وايضا وجد بان معظم المرضى هم ذكور في عمر (60-69) سنة ومعظمهم كانوا مدخنين. لانه العوامل الوراثية والتعرض المهني للمواد الكيمياوية تلعب دورا مهما لهذا النتائج تبين بان معظم المرضى هم من خال هم من خال الموني المون وي منهم من يشرهم من يشرو موسوم ثنائي السنترومير ألتي مدخنين. لانه العوامل الوراثية والتعرض المهني للمواد الكيمياوية تلعب دورا مهما لهذا النتائج تبين بعظم المرضى هم من يشتعلون في مهنة الصباغة ولديهم مرض خلل التنسج المغي المواد الكيمياوية تلعب دورا مهما لهذا المرضى هم من يشانه المرضى هم من يشاني والم خال المن والمومي وي المومي مولم اللمما الموائي والم المومي المما مول المواه المومي والمما وم