

Assay White Blood Cell Differential Count in Group of Iraqi Patients' Child with Type 1 Diabetes Mellitus

La.Huda Farhan

Department of Laboratory Technic in Collage of Health and Medical Technic in Baghdad Iraq

Abstract

This study was included (76) subjects, (46) child with diabetic patients, and (30) of them were healthy child considered as a control group. The average age of those children were (3- 13) years. All the samples were collected from the Yarmok hospital in Baghdad. The period between August 2014 to December 2015. All samples were estimated by Complete Blood Picture (CBP) by manual procedure and used whole blood, and Fasting Serum Glucose (FSG) estimated by enzymatic method.

The study showed that the age group (9-11) is the most among the group of children with diabetes type I. Serum sugar level increased was highly significant ($p < 0.01$) in comparison between control and patient. And the results showed there is significance ($p < 0.05$) between the child patients and control groups in no. of WBC.c. In compared between the neutrophil cell count the results showed there is significance ($p < 0.05$) between the studies group, while showed highly significant ($p < 0.05$) from children and healthy children in lymphocytes cell count. And showed there is non-significant ($p < 0.05$) in monocytes, eosinophil and basophile cells count.

The aim of this study is to assess the role of the white blood cells of children with type I diabetes patients and the aged between (3-13) years.

Keywords: White Blood Cells, Type 1 diabetic.

الخلاصة

شملت هذه الدراسة (76) عينه، (46) طفلاً مصاباً بمرض السكري ، و (30) منهم أطفال أصحاء يعتبرون مجموعة سيطرة. كان متوسط عمر هؤلاء الأطفال (3- 13) سنة. جمعت جميع العينات من مستشفى اليرموك في بغداد. الفترة ما بين اب 2014 كانون الثاني 2015. تم حساب الخلايا بواسطة الصورة الكاملة للدم عن طريق الإجراء اليدوي واستخدمه الدم الكامل لذلك، واستخدمه المصل لقياس الكلوكوز بالطريقة الأنزيمية .

وأظهرت الدراسة أن الفئة العمرية (9-11) هي الأكبر بين مجموعة الأطفال المصابين بنوع السكر (1) كان مستوى السكر في المصل مرتفعاً للغاية مقارنة بين السيطرة والمرضى وبمستوى معنوية عالية ($P < 0.01$) ، وأظهرت النتائج وجود أهمية معنوية ($p < 0.05$) بين الأطفال المرضى ومجموعات المراقبة في العدد الكلي لخلايا الدم البيض. بالمقارنة بين عدد خلايا العدة أظهرت النتائج وجود أهمية معنوية ($P < 0.05$) بين مجموعة الدراسات ، في حين أظهرت معنوية كبيرة ($P < 0.05$) من الأطفال والأطفال الأصحاء في عدد الخلايا للمفاوية. وأظهرت أن هناك أهمية غير معنوية ($p < 0.05$) في عدد الخلايا أحادية الخلية ، الحامضية و القاعدية.

الهدف من هذه الدراسة هو تقييم دور خلايا الدم البيضاء للأطفال الذين يعانون من مرض السكري من النوع الأول والذين تتراوح أعمارهم بين (3-13) سنة.

Introduction:

Type 1 diabetes mellitus is a chronic metabolic syndrome defined by an inability to produce insulin, a hormone which lowers blood sugar. This leads to inappropriate hyperglycaemia (increased blood sugar levels) and deranged metabolism of carbohydrates, fats and proteins. Insulin is normally produced in the β -cell of pancreas, a glandular organ involved in the production of digestive enzymes and hormones such as insulin and glucagon. These functions are carried out in the exocrine and endocrine (Islets of Langerhans) pancreas respectively¹. It's usually first develops in children or young adults. With type 1 diabetes the illness usually develops quite quickly, over days or weeks, as the pancreas stops making insulin². Certain gene variants that carry instructions for making proteins called Human Leukocyte Antigens (HLAs) on white blood cells are linked to the risk of developing type 1 diabetes. The proteins produced by HLA genes help determine whether the immune system recognizes a cell as part of the body or as foreign material. Some combinations of HLA gene variants predict that a person will be at higher risk for type 1 diabetes, while other combinations are protective or have no effect on risk³. The researches show that type 1 diabetes modulated the nerve supply and immune function of bone marrow. This altered communication head to increase the level of monocyte being produced by bone marrow and show these cell infiltrate the brain and cause an increase in the inflammatory signal in its sympathetic center⁴. The cause of type 1 diabetes is unknown. A number of explanatory theories have been put forward, and the cause may be one or more of the following: genetic susceptibility, a diabetogenic trigger, and/or exposure to an antigen⁵.

The aim:

The aim of this study is to assess the W.B.C. differential count between Type I diabetic children and normal.

Subjects and methods:

Subjects:

This study included 30 sample of blood from the diabetic children from 3 to 13 year old, and 30 blood sample of non- diabetic children were collected as a control. All studied samples were conducted from the laboratory of Yarmouk Teaching Hospital in Baghdad city from the period of August 2014 till February 2015.

Specimen collection and Methods:

Specimen collection;

2ml of blood was collected from all children by a sterilized syringe and transfer to EDTA tube and tested immediately.

Methods:

1- Manual W.B.C Count

2. Differential leukocyte count

Results and discussion

Table (4.1): distribution of age group.

years		No. and % of groups		Total	p.value		
		patients	control		X ²	sig	
Age group	(3-5)	2	2	4	1.020	Non sig. p <0.05	
		2.6%	2.6%	5.3%			
	(6-8)	16	12	28			
		21.1%	15.8%	36.8%			
	(9-11)	18	12	30			
		23.7%	15.8%	39.5%			
	(12-13)	10	4	14			
		13.2%	5.3%	18.4%			
	Total		46	30			76
			60.5%	39.5%			100 %

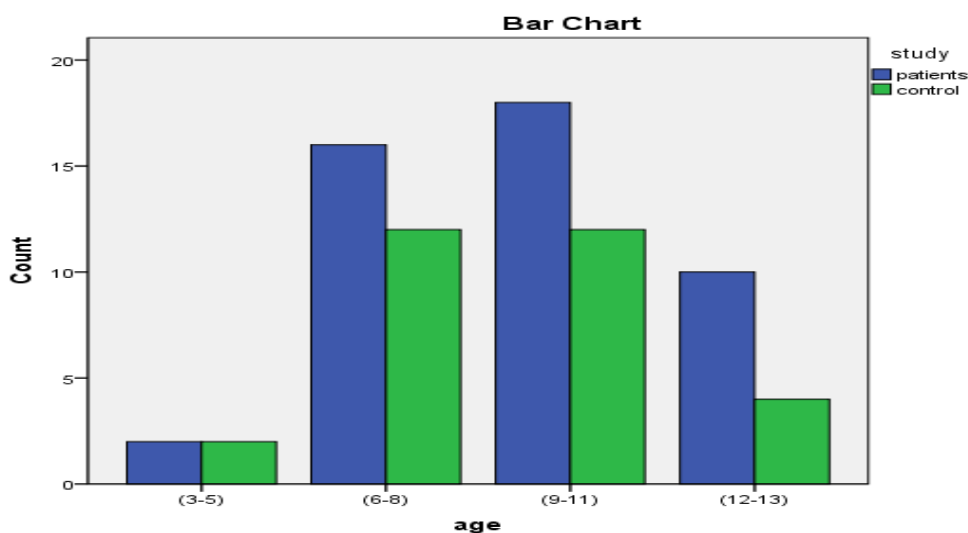


Figure (4.1) distribution of age group

Table and figure (4.1) showed that the age group between (9-11) years is more affected than the other age group in patients with type 1 diabetes mellitus. Data showed no significant difference

between the numbers of patients in different age group. For many reasons, postulated to involve population hygiene, sun exposure, and other environmental factors, its incidence has increased

Figure (4.2): distribution of serum sugar level

Dramatically over the last two decades, especially in children less than five years old. Those under the age of 18 are most often afflicted⁶ .

Table (4.2): statistical analysis of serum glucose level

		study		Total	p.value	
		patients	control		X2	sig
Sugar level	(65-110)normal	0	28	28	.000	Highly sig. P<0.01
		.0%	36.8%	36.8%		
	(<65->110)abnormal	46	2	48		
		60.5%	2.6%	63.2%		
Total Count		46	30	76		
of Total %		60.5%	39.5%	100.0%		

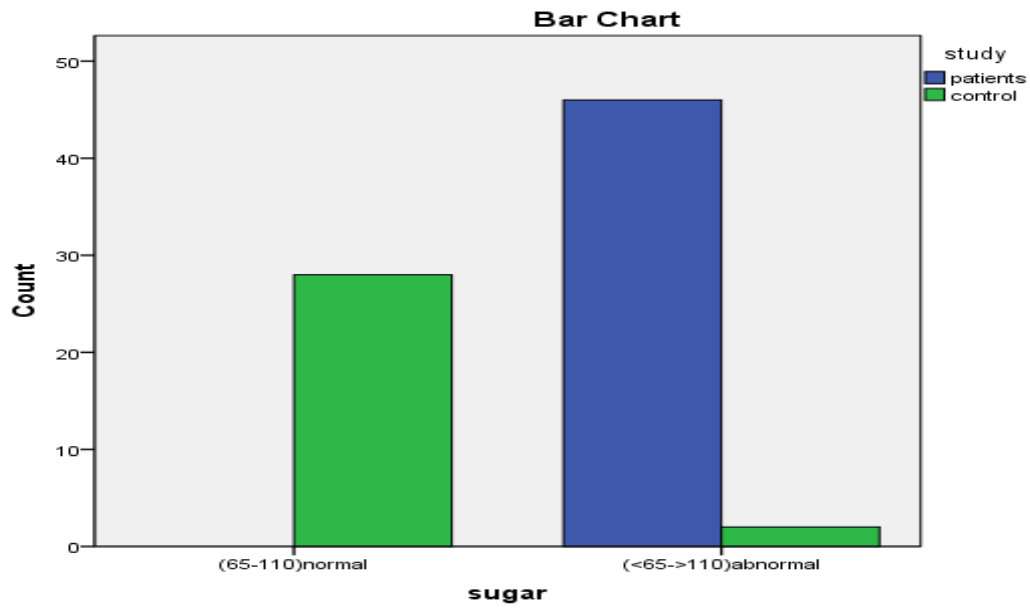


Figure (4.2): distribution of serum sugar level

Table and figure (4-2) showed that serum sugar level increased was highly significant in comparison between control and patient ($p < 0.01$) because all the patients suffered from diabetes mellitus type 1.

Table (4.3) distribution analysis of W.B.C count

		study			p.value	
		patients	control	Total	X ²	Sig.
WBC. Count	(4000-10000)normal	6 7.9%	14 18.4%	20 26.3%	.09	Sig.p<0.05
	higher than normal	40 52.6%	16 21.1 %	56 73.7%		
Total	Count	46	30	76		
	% of Total	60.5%	39.5%	100.0%		

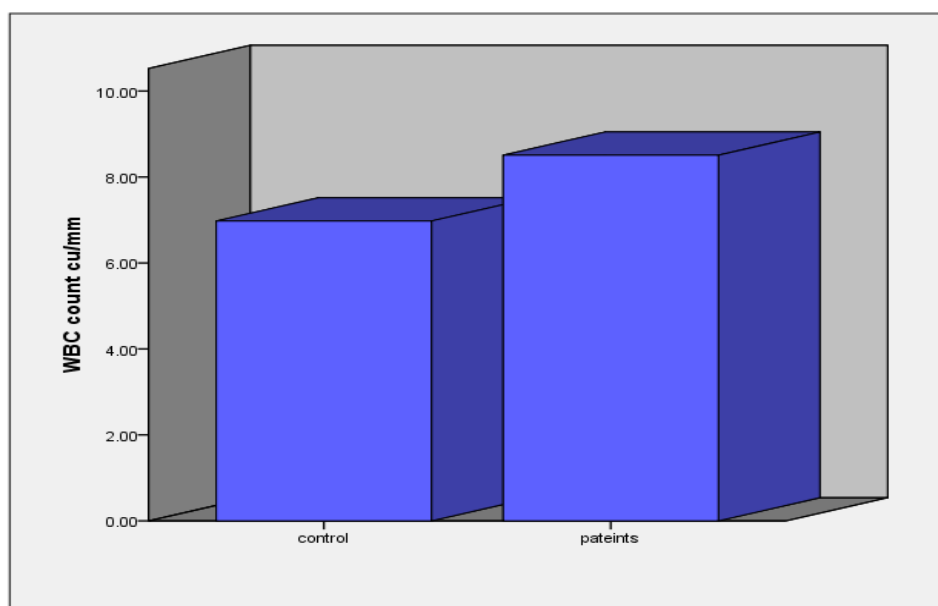


Figure (4.3) distribution of W.B.C count

Table and figure (4.3) showed there is significance ($p < 0.05$) and this value cannot be considered in diagnosis Type 1 diabetes, because the increasable among the types of white blood cells.

Table (4.4): distribution of neutrophil count group.

		study			p.value	
		patients	control	Total	χ^2	sig.
neutrophil count	<63)cell normal	13 28.3%	16 52.0%	29 38.2%	0.58	sig. p<0.05
	higher than normal	33 71.7%	14 48.0%	47 61.8%		
total	Count	46	30	76		
	of Total	60.5%	39.5%	100.0%		

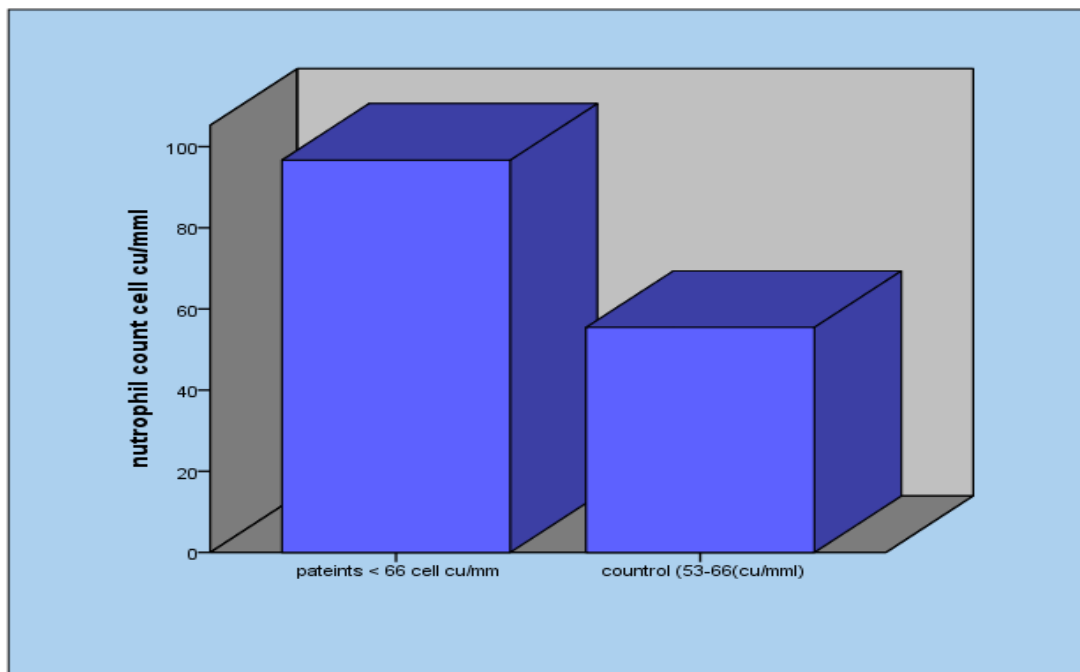


Figure (4.4): distribution of neutrophil count

Table and figure (4.4) showed there is significance ($p < 0.05$) between the studies group.

Table (4.5): distribution of lymphocyte count among the studies groups

		study			p. value	
		patients	control	Total	X ²	Sig.
Lymphocyte count	(28-33)cell normal	10 13.2%	10 13.2%	20 26.3%	0.000	Highly sig.p<0.005
	higher than normal	34 44.7%	20 26.3%	54 71.1%		
	less than normal	2 2.6%	0 .0%	2 2.6%		
Total	Count	46	30	76		
	% of Total	60.5%	39.5%	100.0%		

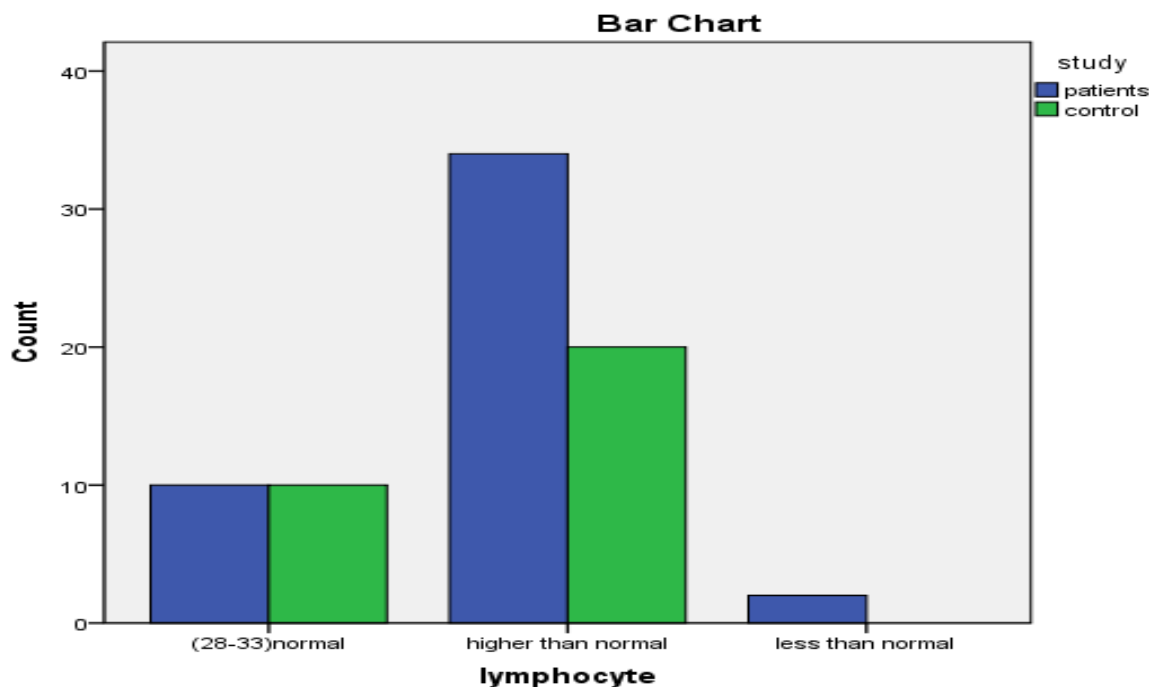


Figure (4.5): distribution of lymphocyte count/year among the studies groups.

Table and figure (4.5) showed significant ($p < 0.05$) Because the samples from children and healthy children who are considered the control group may also suffer from other diseases caused increases in cells histological evaluation of pancreas from patients with type 1 diabetes patients reveals insulinitis - immune cell infiltrates around and in the islets – and cellular immune reactivity is considered to be the direct cause of beta-cell destruction, mainly mediated by T-lymphocytes / T-cells. The consensus has been that autoreactive T cells are responsible for the b-cell destruction resulting in overt T1D, whereas the autoantibodies are innocent bystanders useful as predictive markers of future disease. The observation in patients with recent-onset T1D that treatment with the anti-CD20 monoclonal antibody targeting B cells results in a retarded loss of endogenous insulin secretion of the same magnitude as that seen with therapies affecting T-cell function⁷.

Table (4.6): the distribution of monocyte count groups.

		study			p.value	
		patients	control	Total	X ²	Sig.
Monocyte count	(2-10) cell normal	3 3.9%	0 0%	3 3.9%	.069	No sig.p>0.005
	higher than normal	43 56.6%	26 34.2%	69 90.8%		
	less than normal	4 5.3%	0 0%	4 5.3%		
Total		Count	46	30	76	
		% of Total	60.5%	39.5%	100.0%	

Table (4.6): the distribution of monocyte count groups.

		study			p.value	
		patients	control	Total	X ²	Sig.
Monocyte count	(2-10) cell normal	3 3.9%	0 0%	3 3.9%	.069	No sig. p>0.005
	higher than normal	43 56.6%	26 34.2%	69 90.8%		
	less than normal	4 5.3%	0 0%	4 5.3%		

Total	Count	46	30	76		
	of Total	60.5%	39.5%	100.0%		

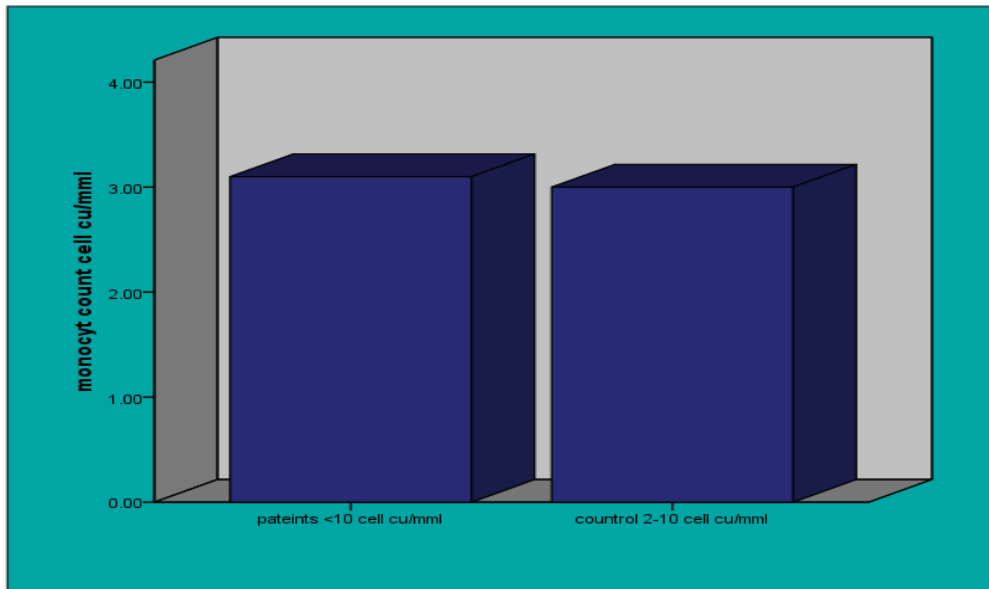


Figure (4.6): distribution of monocyte count

Table and figure (4.6) showed there is non-significant ($p < 0.05$) because the monocyte cells do not have an active role in patients with type 1 diabetes mellitus. In type 1 diabetes, WBC counts were not increased but featured a general activation of adaptive immunity, the number of monocytes correlates negatively with FBG.⁸

Table (4.7): the distribution of eosinophil count

		study			p. value	
		patients	control	Total	X ²	Sig.
Eosinophil count	6)normal	22 28.9%	36 47.4%	58 76.3%	.553	No sig. p>0.005
	her than normal	3 3.9%	0 0%	3 3.9%		
	s than normal	10 13.2%	5 6.6%	15 19.7%		
total		Count	46	30	76	
		% of Total	60.5%	39.5%	100.0%	

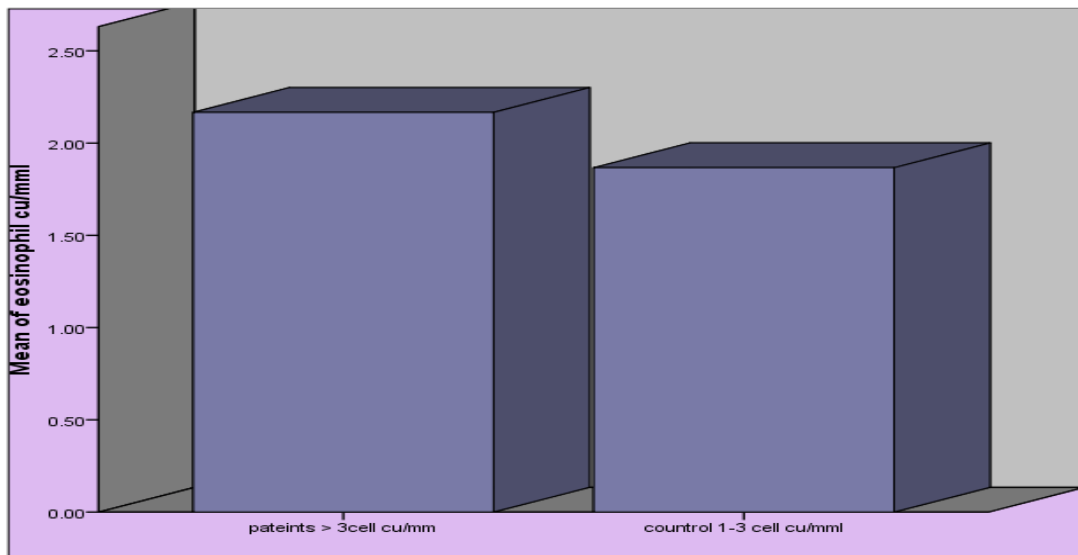


Figure (4.7): distribution of eosinophil count

Table and figure (4.7) show there is non-significant ($p < 0.05$) between patient and control .This cell is not affected with Type 1 diabetes.

Table (4.8): distribution of basophil count

		study			p. value		
		patients	control	Total	X ²	Sig.	
Basophil	1 cell normal	Count	18	10	28	2	No sig. p>0.005
		of Total	23.7%	13.2%	16.8%		
	Abnormal	Count	28	20	48		
		% of Total	66.8%	26.3%	33.2%		
Total		Count	46	30	76		
		of Total	60.5%	39.5%	100.0%		

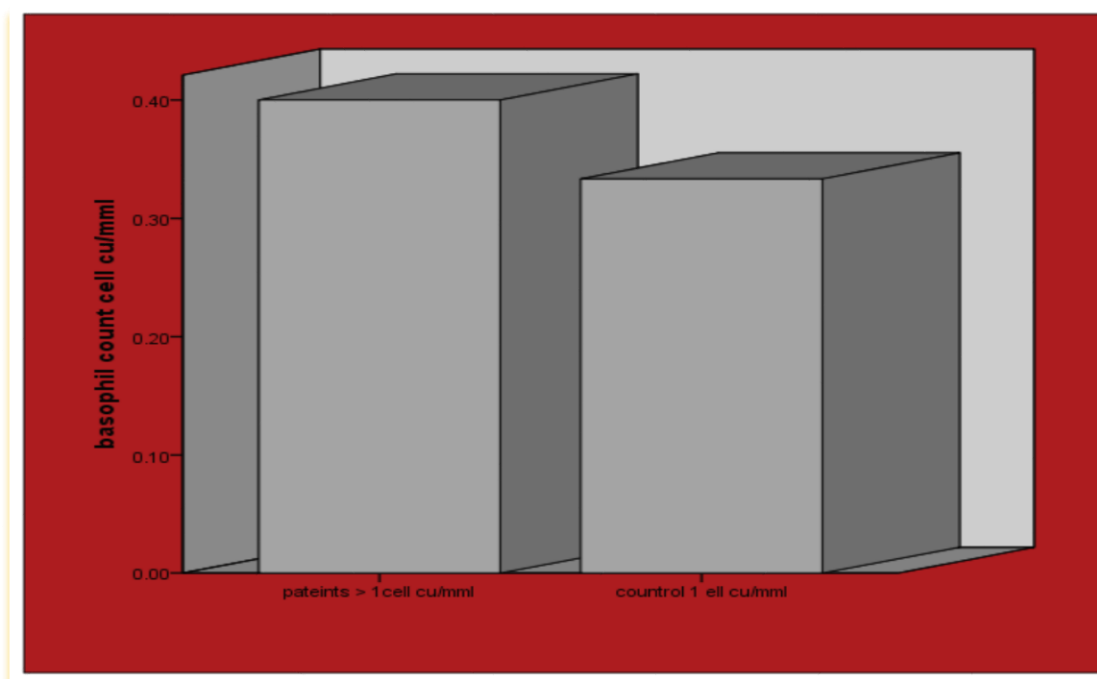


Figure (4.8): distribution of basophil count

Table and figure (4.8) showed there is no significance ($p < 0.05$) in patient with Type 1 diabetes has increased level of basophil than the normal individual.

Conclusions and recommendations

Conclusions

- 1- There are no any notice able increase in other types of white blood cells numbers
- 2- A significant increase in numbers of lymphocyte, this is due to the nature of the disease, which is classified as an autoimmune disease
- 3- The study showed that there increase numbers of neutrophil cell.
- 4- There is no increase in the number of other types of white blood cells.

Recommendations

- 1- Increase no. of studies groups
- 2- Genetically study.
- 3- Detection of the real causes of lymphocytes to damage the Langerhans cells.

References

- [1] Carotenuto P, Gazda LS, Pratt PF. (1987) Circulating lymphocyte populations and autoantibodies in non-obese diabetic (NOD) mice : a longitudinal study.:84-93.
- [2] Patients S. (2002) Anti-cd3 monoclonal antibody in new-onset type 1 diabetes mellitus.;346(22):1692-1698.
- [3] Factors R, Examination C. (2006) Diabetes mellitus type 1 (insulin dependent , juvenile onset) What is Diabetes Mellitus Type 1 (insulin dependent , juvenile onset)?;1:1-7.
- [4] Cooke DW, Plotnick L, Cooke DW. (2012) Type 1 Diabetes Mellitus in.. doi:10.1542/pir.29-11-374.
- [5] Knip M, Simell O. (2012) Environmental Triggers of Type 1 Diabetes.:1-15.
- [6] Bluestone JA, Herold K, Eisenbarth G. (2010) interventions in type 1 diabetes. *Nature*.;464(7293):1293-1300. doi:10.1038/nature08933
- [7] Becker DJ, Gitelman SE, Goland R, et al. (2009). Rituximab, B-Lymphocyte Depletion, and Preservation of Beta-Cell Function.

[8] Menart-houtermans B, Ruth R, Nowotny B, et al. (2014) Leukocyte Profiles Differ Between Type 1 and Type 2 Diabetes and Are Associated With Metabolic Phenotypes : Results From the German Diabetes Study (GDS).;37(August):2326-2333. doi:10.2337/dc14-0316.