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Some of Hematclogical and Biochemical Parameters of Children Infected with Anemia in Samawa City

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Abstract

Anemia spreads a widely in many countries in the world particularly the poor and developing countries . Iraq is one of these countries and many of children suffer from bad environmental health , and economic conditions . Samples of blood collected from patients infected with anemia in the hospital of children and delivery in samawah city . Hematological and biochemical parameters were determined , hematological parameters included packed cell volume , hemoglobin concentration , total white blood cells count while biochemical parameters included glucose , cholesterol , billrubin and urea concentrations were measured .

Results showed High significant increase (P<0.01) in the number of children infected with iron deficiency anemia in comparison with other types of anemia , so number of children infected with sickle cell anemia and hemolytic anemia . Also there was high significant decrease (p<0.01) in hemoglobin concentration and packed cell volume in all types of anemia in comparison with a control group Glucose and bilirubin increased significantly (p<0.01). Total white blood count increased significantly (p<0.01) in patients with thallasemia . sickle cell anemia and hemolytic anemia compared with patients with iron deficiency anemia and a control group . Total serum cholesterol high significant decreased (p<0.01) in all types of anemia compared with a control group . Results showed there were non significant differences in urea levels in all types of anemia compared with a control group .

Introduction

Children infected with anemia in all parts of word , and this disease caused by genetic acquired factors . Iron deficiency anemia infects large number of children than other types of anemia in poor and development countries . There are about three quarters of children over five years infected with iron anemia in eastern Africa . Iron deficiency anemia has been associated with impaired growth , decreased appetite , reduced activity and increased incidence of some infections. (Demayer and Adils , 1995) .The factors caused anemia not only the deficiency of some enzymes or elements but involved genetic factors produced . Thalassemia is an hematological disorder characterized by the gentic defect of globin chain synthesis resulting in unproductive erythropoiesis and enormous expansion of the reticuloendethial system (Barone *et al* . , 1998) .

Thalassemia is highest is countries bordering on Mediterranean sea , much in meddle east , Africa and southeast Asia . (Kazazian , 1988) . Population migration during the past decades has led to increasing numbers of patients with thalassemia being encountered in all parts of the world , including in the united states . (Cuningham *et al* . , 2004) .Regular blood transfusion programs and chelation treatment have considerably improved survival of patiens with sthalaseima . However disease and treatment related complications in these patient progress over time , causing severe morbidity and shortened life expectancy (Gaziev *et al* . , 2005) .

Sickle cell anemia is one of the variants of disorders of hemoglobin synthesis inherited from both parents in an autosomal recessive fashion . It is characterized by chronic anemia which exacerbated during period of rapid red blood cell destruction , failure of the erythroid cell line in the bone marrow (Juwah *et al* . , 2004) . Erythrocyles have tendency to become grossly abnormal in shape (sickle shaped) under condition of oxygen tension , hemoglobin characterized that glutamic acid replaced by valine in the sixth amino acid position of globin chain (Saraswathly and Aggarwal , 2005) . Patients with sickle cell anemia have very high levels of HbF and low levels of HbA2. in comparison with health peoples (Lielnjo *et al* . , 1986) . The etiologies of hemolytic anemia often are categorized as acquired or hereditary , comon acquird causes are antierythrocyte drugs , transfusion reaction , damage of erythrocyle membrane toxins and infections agents while the disordes of erythrocyte membrane , enzyme and hemoglobin cause hereditary hemolytic anemia (Gurpreet *et al* . , 2004) . Sickle cell anemia and thalassemia are examples for the disorder in hemoglobin syhthesis and in the current study both considerd as two types because their importance and prevalence.

Jacobasch and Rapoport (1996) indicated that glucose - 6- phosphater dehydrogenase deficiency is the most common deficiency and is prevalent among Africans , Black Amricans and population of Mediterranean countries and east Asia . The current study aimed at determine the extent of infection of children with most types of anemia in the city of Samwa /Iraq in addition to study of some hematological and bioehemical parameters some in these types.

Materials and Methods

This study carried out in the delivery and children hospital is Samawa city/Iraq during the period between January to April in 2007. The study included 152 children aged between (2-7)

years , 130 children were infected with some types of anemia while 22 children were healthy and considered as a control group samples of blood were taken from vein , to measures some hematological and biochemical parameter :

1-The hematological parameters :-

a- Packed cell volume (PCV) : Capillaries contained anticoagulant were used and put in microcenterfuge . PCV was measured by hematocrit reader (Schalm et al . ,1975)

b- Hemoglobin conecnteration : Hemoglobin meter and Drabkn solution were used according to (Sood , 1992) .

c- Total white blood cells count (TWBCs) : according to the method used by Dacie and Lewis (1984) Hemocytometer and Drabkun solution were used in this method .

2-The biochemical parameters :

a- Total serum cholesterol : Enzymtic method was used for determine the total serum cholesrol , absorbability of sample read at 500nm.. (Burtis Ashwoo, 1999) .
Serum glucose : according method by (Tietz , 1982)

b- Urea concenteration : Diacetyl manoxime method was used for measure urea concenteration (Al Khyat , 1992) .

c- Bilirubin concenteration : bilirubin was measured by using indirect method (Health ministry , 1995) .

Results

In this study results showed that there was high significant increase ($p<0.01$) in number of children infected with iron deficiency anemia compared with other types of anemia and control groups . Also the children infected with thalassemia increased signlficantly ($p<0.05$) compared with sickle cell anemia and hemolytic anemia and control group. (Figure. 1)

Table (1) demonstrate high significant decrease ($p<0.01$) in hemoglobin concenteration and packed cell volume in all types of anemia compared with a control group . and this decrease was high significant ($p<0.01$) in children with thalassemia in comparison with other types of anemia . Total white blood cells count increased significantly ($p<0.01$) in children with thalassemia , sickle cell anemia and nomolytic anemia compared with iron deficiency anemia and control group. Total serum cholesterol was decreased significantly ($p<0.01$) in all types of anemia compared with a control group as well as it was high significant decreased ($p<0.01$) in children with iron deficiency anemia in comparison with other types of anemia. Glucose was increased significantly ($p<0.01$) in children with thalasemia , sickle cell anemia and hemolytic anemia compared with iron deficiency anemia and a control group . Also there was high significant increase ($p<0.01$) in children with thalassemia in comparison with children with thatlassemia in comparison with children with sickle cell anemia , iron deficiency anemia and a control group . There was no significant difference between iron deficiency and control group, in glucose concenteration .Results showed that bilirubin was high significant increased ($p<0.01$) in thalassemia sickle cell anemia and hemolytic anemia compared with iron deficiency anemia and a control group . There were non significant differences in urea levels in all types of anemia compared with a control group .

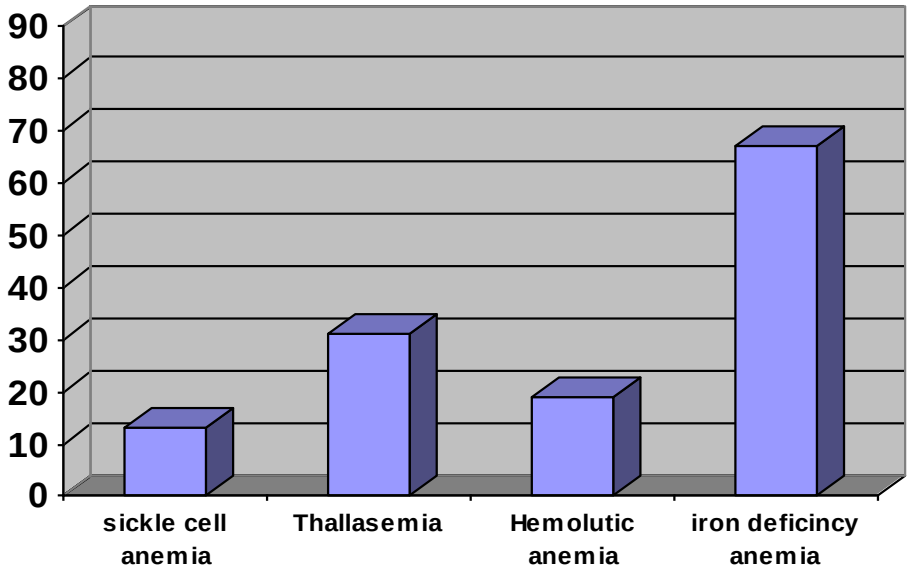

Figure (1) the number of children aged (2-7) years whom infected with some types of anemia in Samawa city

Table (1) : Some hematological parameters of children infected with different types of anemia in Samawa city

Types of anemia	Hb (g/dl)	PCV (%)	TWBC ($x10^3$)/mm2
Tallasemia	6±0.23 a	17.4±0.72 a	22.4±0.059 b
Sickle cell anemia	7.5±0.29 b	22.95±0.76 b	21.5±0.105 b
Hemolytic anemia	7.8±0.19 b	24.2±0.60 b	24±0.050 b
Iron deficiency anemia	7.6±0.30 b	23.6±1.03 b	12.5±0.063 a
Control	11.2±0.38 c	37.3±1.5 c	104±0.309 c

Numbers=Means + SD
Different litters mean significant differences among the groups

Table (2) : Some biochemical parameters of children infected with different types of anemia in Samawa city

Types of anemia	Glucose conceteration (mg/dl)	Total serum cholesterol (mg/dl)	Billrubin concentration (mg/dl)	Urea concentration (mg/dl)
Tallasemia	132.09±3.91 a	122.35±3.71 b	5.06±0.47 a	30.45±0.72 a
Sickle cell anemia	115.5±9.26 cb	124.5±9.8 b	55.5±0.53 a	27.56±1.84 a
Hemolytic anemia	130.6±5.69 ab	113.8±4.47 b	5.37±0.49 a	29±0.91 a
Iron deficiency anemia	101±5.11 cd	90±862 a	1.47±0.20 b	26.8±2.06 a
Control	91.25±3.37 d	143.75±3.09 c	0.68±0.05 b	27.87±0.54 a

Numbers=Means ± SD
Different litters mean significant differences among the groups

Discussion

The world Health organization WHO and unicef demousterated that iron deficiency anemia involve the half number of anemia cases in world (WHO , 1991). These reports which it agree with with the results of this study .

The importance factor , that cause iron deficiency anemia is iron deficiency in food which necessary for hemoglobin synthesis . Iron deficiency increased in children period because accelerated growth reqiurements in this period , also chronic blood loss from gastrointestinal tract and infection with parasites are considered caused factors for iron deficiency anemia . (Hoffbrand *et al* , 2004).The prevalence of other types of anemia resulted from genetic disorder is nearly limited but the migration from region to other regions and gene help in the prevalence of these types (Al-Madany ,2001) genetic disorder in hemoglobin synthesis such as sickle cell anemia and thalasemia caused chronic hemolysis .

In sickle cell anemia memberane abnormalities from sickling and oxidative damage lead to splenic trapping removed of cells and intravascular hemolysis occur , while in thalassemia the deficiency in one globin chain lead to decrease in hemoglobin and the intracellular precipitation of excess chain which damages the membrane and lead to hemolysis . (Maedel, 1993).

Hemolysis and ineffective erythropoiesis cause the anemia that occurs in thalassemia (Pootrakul *et al* . 2000)

Erythrocytes that are exposed to phosphatisylserires may also contribute directly to the vascular damage obsorved in thalassemia .(Muntori et al.,1992)
Erythrocytes and platelets in thalassemia contain higher levels of reactive oxygen species and lower levels of intracellular glutathione and this may be attributed to continious exposure to oxidative insults (Amer and Fibach , 2004). Hemolysis involves the destruction of erythrocytes before their normal life span, also hemolysis most often presents as anemia when erythrocytes can't match the pace of erythrocyte destruction . (Gurpreet *et al* , 2004) .

The most common enzymopathy causing hemolysis is G6PD deficiency . Most patient have no clinical evidence of ongoing hemolysis unit infection , drug reaction or ingestion of fava beans-causes oxidative damage to hemoglobin (Beulter , and Luzzatto , 1999)
There are many canlitions associated with high lemolysis of erythrocytes in circulation include incompatible transfusion , G6PD deficiency , severe burns and certain infections (Tabbara , 1992) .

Hemolysis which occurs in patents with thalasemia , sickle cell anemia and hemolytic anemia causes decease in erythrocytes count and hemoglobin concenteration and this explain the high significant decrease in packed cell volume and hemoglobine cenceteration in current study The children infected with lron deficiency anemia increased in Diwanyah city because nutrient deficiency and economic conditions (Al-Salman *et al* , 1999) .
Iron deficiency anemia caused by iron deficiency food , some materials which decrease iron absorption from intestine such as phytic acid , phosphate and oxalate , and dysfunction in gastric sections which convert iron to ferrous form which absorbed in intestine easly. (Ganong 2003) Iron deficiency affect hemoglobin sythesis then affect hemoglobin concenteration and packed cell volume

The elevation in glucose level may be resulted from the toxic effect of iron overlaod and ferritin increase . Low (1997) indicated that glucose increase occur in persons whom have high levels of ferritin which causes dysfunction in pancreas . patients with Beta-thalassemia infected by mclitus diabetis and this resulted from iron overload in Beta-cells and then decrease of insulin secretion (Soliman *et al* , 1996 , Khalifa *et al* , 2004)

In study carried out by (Suvarn *et al* , 2006) showed that insulin resistance was higher in patients not on chelation theraby with those on chelation theraby , chelation is removal of iron from body. Insulin resistance releated with the increase of ferritin levels , decrease the tissues seusitivity to insulin and increase insulin levels in blood. (Tsapas , *et al* , 2007) .

Hemolysis caused thalassemia , sickle cell anemia and hemolytic result in iron overload , iron overlaod affect in cholosterl levels , (Piatti *et al* 1999) demonstrated that iron accumulation eventually occurs and this cause dysfunction in target organs such heart liver and pancreas and this affect cholostrol synthesis in the liver espcially . Patient infected with G6PD deficiency have low levels of Total cholesterol and low density lipoprotein in comparison with normal persons. (Muntoni *et al* 1992) In hemolytic anemia caused by G6PD deficiency , cholostrol synthesis and LDL related mRNA is very low (Batella *et al* , 2002) .
Thus the dysfunction in liver lead to decrease in total serum cholestrol which

occurd , in other hand iron deficiency causes cholesterol decease . Lewis *et al* . , (2001) indicated that cholesterol level was low in mice fed little amount of iron compared with a control group .

Erythrocytes in sickle cell anemia thalassemia and G6PD deficiency anemia are more sensitive to osmotic shock , oxidative stress and energy depletion leading to enhance the hemolysis of those cells (Karl lang *et al* , 2002)

Hemolysis considered as main factor causes the increase in an conjugated bilirubin.(Murray *et al*.,2003)

Ataly *et al* (2006) indicated that anemia resulted from G6PD deficiency is common enzymopathy which caused evelation of bilirubin in children .

Also most children infected with sickle cell anemia suffer from jaundice caused by high significant increase in bilirubin in blood. (Aljuwah *et al* , 2004)

References

. Al-Juwah , E U Niemadim and W Kaine(2004) .Types of anaemic crises in paediatric patients with sickle cell anaemia seen in Enugu , Nigeria.ADC ONLINE.89; 572-576

. Al-Khyat , M.H.(1992) .Method for evalution blood urea by diacetyl manoxime.Health Ministry .Baghdad .Iraq.

. Al-Salman , A. K. ; Obead , A. H. ; Hussein , H. S. ; Karwan H.C. (1999) . Evalusion the effect of embargo on primary children as reflected by percentage of Hemoglobin in Al Qadisiya province.Al Qadisya J.4(1):22-33.

. Al-Madany , T. A.(2001) incidence of thalassemia trait among group of adolescents in Najaf. Al-Qadisiah . Med. J. (1) : 26-30.

. Amer .J and Fibach , E. (2004) : Oxidative status of platelets in normal and thalassemia blood .Thromb . Haemost 92:1052-1059.

. Atay E , Bozaykut A and Ipek IO (2006)Glucose-6-phosphate dehydrognase deficiency in neonatal indirect hyperbilirubinemia .J Trop pediatr;52(1) : 56-58.

. Barone , R. Romeo , M.A. ; Schiliro , G. and pavone , L. (1998) . plasma chitotriodase activity in patient with B.thalasemia B.C.M.D. (1) :1-8 .

. Batetta B , Bonatesta RR , Sanna F , Putzolu M ,Mulas MF , Collu M and Dessi S. (2002) Cellgrowth and cholesterol metabolism in human glucose-6-phosphate dehy drogenase deficient 1 ymphomononuclear cells.Cell Prolif ; 35(3):143-154.

. Beulter E and Luzzatto , L. (1999) Hemolytic anemia , Semin Hematol. 36(4) : 38-47.

. Beulter E. (1994) G6PD Deficiency Blood 84:3613-3636.

. Burtis , C. A. and Ashwood E. R(1999) Teitz Textbook Clinical Chemistry . 3rd ed. , W. B. Saunders Company . London , pp. 826-835.

. Cunningham , M.J. ; Macklin , E. A. ; Neufeld , E.J. and choen , A. R. (2004) complications of B.thalassemia major in north America Blood , 104:34-39.

. DeMayer , E. and Adiels , T.M. (1985) The prevalence of anemia in the world . World Health State . Q. 38:302-316.

. Dhaliwal , G. ; cornett , A. C. and Tierney , J. R. (2004) Hemolytic anemia , American family physician 69 (11).

. Dacie , J. V. and Lewis , S. M. (1984) . Partial hematology 6th . ed. Edinburgh , Churchill.

. Ganong, W.F. (2003) Review of Medical physiology . 21 ed . Lang Medical Publication . California.

. Gaziev , J ; Sodani , P. , polchi , p ; Andreani , M and Lucarelli , G. (2005) . Bone marrow transplantation in adults with thalassemia : treatment and long-term follow-up . Ann N.Y. Acod Sci. 1054:196-250.

. Gurpreet Dhaliwal , M.D. , Patricia A. Cornett , M.D. , and Lawrence M .Tierney , J R . , M.D.(2004) Hemolytic Anemia. American Family Physician.VOL.69.NO.11.

. Health ministry (1992) . Laboratory Method . Baghdad . Iraq.

. Hoffbrand , A. N ; Pettit , J. E. and Moss , P. A. H. (2001) Essential Haematology . 4th ed. Blackwell publishing company.

. Jacobasch , G. and Rapoprt , S. M. (1996) . Hemolytic anemias to due erythrocyte enzyme deficiencies . Mol. Aspects. Med. 17(2):143-170.

. Juwah , A. I. ; Nlemadim , E. U. and Kaine , W. (2003) types of anaemic crises in paediatric patients with sickle cell anemia seen in Enugu , Nigeria . Arch . Dis. Child. 89: 572-576.

. Karl Lang , Benjamin Roll ,Svetlana Myssina , Markus Schittenhelm , Hans-Gerhard scgeel-walter , Lothar Kanz ,Jasmin Fritz , Florian Lang ,Stephan Huber and Thomas wieder.(2002)Enhanced erythrocyte apoptosis in sickle cell anemia, Thalassemia and glucose-6-phosphate dehydrogenase deficiency .International Journal of Experimental and clinical cellular physiology , biochemistry and pharmacology .Vol.12,NO.5-6.

. Kazazian H . Boehm C.D.(1988) Moleculaer basis and prenatal diagnosis of B-thalassaemia blood .72,1107.

. Khalifa As , Salem M , Mounir E ,EL-Tawil MM , EL-Sawy M and Abd AL-Aziz MM.(2004) Abnormal glucose to lerance in Egyptian beta-thalassemic patients : possible association with genotyping. Pediatr Diabetes ;5(3):123-132.

. Lewis RM , James LA , Zhang J , Byrne CD , Hales CN (2001)Effects of maternal iron restriction in the rat on hypoxia-induced gene expression and fetal metabolite levels. Br J Nutr ; 85(2):193-201.

. Lie-injo , L.E. Hassan , K ; Joishy , S. K. and lim , M L. (1986) sickle cell anemia associated with alph-thalassemia in Malaysian Indians . Am. J. Hematol . 22(3) : 265-274.

. Low LC (1997) Gr0wth puberty and endocrine function in beta-thalassaemia major. J pediaty Endocrinol Metab ;10(2):175-184.

. Maedel , S. S. (1993) Morpgological changes in Erythrocytes . American society of clinical pathology press

. Mandori , A. B. ; Brabino G.A. ; Lubin , Blt Kuypers , FA. (2000) Adherence of phosphatidylserine exposing erythrocytes to endothelial matrix thrombospondin blood 95: 1293-1300.

. Muntoni S, Batetta B, Dessi S, Muntoni S and Pani P.(1992)Serum lipoprotein profile in the Mediterranean variant of glucose-6-phosphate dehydrogenase deficiency.Eur J Epidemiol.1:48-53.

. Murry , K. Robert , Granner , K. Daryl , Mayes , A. Peter and Rodwell , W. Victor (2003) Harpers Illustrated Biochemistry. 26th ed , McGraw-Hill Companies .

. Piatti G ,Allegra L ,Ambrosetti U , Cappellini MD ,Turatif and Fiorelli G .(1999) Thalassemia and pulmonary Function.Haematologic ;84:804-808.

. Pootrakul , P. , Sirankapracha , P. and Hemosorach , S. (2000) Acorrelation of erythrokinetics ineffective erythropoiesis and erythroid apoptosis in Thai patients with , thalassemia . Blood . 96:2606-2612.

. Saraswathy , K.N. and Aggawal , S. (2005) Study of glucose -6- phosphate dehydrogehase deficiency and sickle cell anemia among the Tamil Brahmins of Delhi. Anthropologist . 7(1) : 69-70.

. Schalm , O.W. ; Jain , N.c. and Carrol , E.J(1975)Veterinary haematology.3rd.ed.Philadeliphia.

. Sood, R .(1992)Technigue of medical lubratory methods and explains .Translated by ,Haidr ,S.K.Jabar , A. and Aubeas, B. Baghdad.Iraq.

. Soliman AT , el Banna N , alsalmi I and Asfour M .(1996) Insulin and glucagons responses to provocation with glucose and arginine in prepubertal children with thalassemia major befour and after long-term blood transfusion . J Trop pediatr ; 42(5):291-296.

. Suvarna J , Ingle H and Deshmukh CT (2006) Insulin resistance and beta cell function in chronically transfused patients of thalassemia major .Indian pediater ;43(5):393-400.

. Tabbara , I . A. , (1992) Hemolytic anemias , Dignosis and management . Med . Clin. North . Am. 76(3): 649-668.

. Tietz , N.W(1982) Fundamentals of Clinical Chemistry . 2nd ed , . W.B Saunders company Philadelphia .

. Tsapas A , Vlachaki E , Christoforidis A , Sarigianni M , Bekiari E ,Perifanis V , Tsapas V, Paletas K , and Athanassiou-Metaxa M .(2007) Insulin sensitivity assessment with euglycemic insulin clamp in adult beta-thalassaemia major patients.Eur J Haematol;79(6):526-530.

. World Health organization.World statistics annual.Geneva:World Health organization.

بعض المعايير الدمويه والكيموحيويه للاطفال المصابين بفقر الدم في مدينة السماوه

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الخلاصـــة

ينتشر مرض فقر الدم بشكل واسع في كثير من دول العالم بشكل خاص الدول الفقيرة والنامية ويعد العراق احد هذه الدول لظروف صحية وبيئية واقتصادية . اجريت الدراسة الحالية لمعرفة مدى اصابة الاطفال ببعض انواع فقر الدم في مدينة السماوة اضافة الى دراسة بعض المعايير الدموية والكيموحيوية . تم اخذ عينات من الدم لقياس تركيز الهيموكلوبين و حجم الخلايا المرصوص والعدد الكلي لخلايا الدم البيض في حين تم عزل المصل لقياس الكلوكوزوالكوليسترول الكلي والبايلوربين واليوريا . لقد اثبتت النتائج وجود زيادة عالية المعنوية (P<0.01) في اعداد الاطفال المصابين بفقر دم نقص الحديد مقارنة مع بقية الانواع كما كانت هذه الزيادة معنوية في حالة الاطفال المصابين بالثالاسيميا مقارنة مع فقر الدم المنجلي وفقر الدم التحللي . كما اشارت النتائج الى وجود انخفاض عالي المعنوية (P<0.01) في كمية الهيموكلوبين وحجم الخلايا المرصوص في جميع انواع فقر الدم مقارنة مع مجموعة السيطرة الى حصول انخفاض عالي المعنوية في العدد الكلي لخلايا الدم البيض في فقر الدم المنجلي وفقر الدم التحللي والثلاسيميا مقارنة مع فقر دم نقص الحديد ومجموعة السيطرة . اما بالنسبة للمعايير الكيموحيوية فقد حصل ارتفاع عالي المعنوية (p<0.01) في مستويات الكلوكوز والبايلروبين في الاطفال المصابين بالثالاسيميا وفقر الدم التحللي وفقر الدم المنجلي مقارنة مع مجموعة السيطرة في حين انخفض مستوى الكوليسترول معنوياً (P<0.01) في جميع انواع فقر الدم مقارنة مع مجموعة السيطرة . في هذه الدراسة اظهرت النتائج عدم وجود اختلافات معنوية في مستوى اليوريا في الانواع المختلفة لفقر الدم مقارنة مع مجموعة السيطرة .