## THE ROLE OF GLYCOSYLATED HEMOGLOBIN AND THE PRO-INFLAMMATORY MARKERS (INTERLEUKIN-6 & C-REACTIVE PROTEIN) IN ASSESSING THE SEVERITY OF DIABETIC RETINOPATHY IN PEDIATRICS

## By

#### Prof. Hanan Abd EL-Moniem Mohamed\* Dr. Mohammed Bahaa El-Ameer Hawary\* Dr. Ahmed Fathy Gabr\*\* Dr. Abdalla M. El-Ebidi\*\*\* Dr. Tereza Saad Abd El-shahed\*

\*Pediatrics \*\* Ophthalmology \*\*\*Biochemistry departments Aswan University

## ABSTRACT

**Background**: Diabetic retinopathy (DR) is one of the vascular complications of diabetes mellitus. It is caused by high blood sugar levels damaging the network of tiny blood vessels that supply blood to the retina. Circulating biomarkers of these pathways such as tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), interleukin–6 (IL-6), C-reactive protein (CRP) in correlation with HbA1C values which reflect the state of glycemic control. Inflammation may be associated with development of both type 1 and type 2 diabetes complications

**Objectives**: To assess the correlation between the level of pro-inflammatory markers (IL-6, CRP), HbA1c and the severity of the diabetic retinopathy

**Patients & Methods:** The study was cross sectional study. It conducted on eighty patients. The patients were selected from the Endocrine & Metabolism pediatric Unit at pediatrics Department in Aswan University Hospital during the period from March 2016 to October 2016.

**Results:** normal fundus examination was found in 74 cases (92.5 %) under 18 years old, while abnormal fundus was detected in 6 cases (7.5 %). IL-6 level in cases with normal fundus examination was ( $19.72 \pm 7.14$ ) and those with abnormal fundus examination was ( $32.85 \pm 9.84$ ) with P. value (0.011). CRP was ( $9.75 \pm 9.5$ ) mg/dl, with P value 0.503 which was insignificant. HbA1c in patients with normal fundus examination was ( $9.826 \pm 1.742$  while those of vitreous hemorrhage ( $9.41 \pm 0.1$ ) and those with macular edema ( $9.79 \pm 0.883$  %) with P value 0.923 which was insignificant.

**Conclusion:** There was significant correlation between IL-6 level in diabetic patient and abnormal fundus examination but there is no correlation with CRP level or HbA1c regarding abnormal fundus findings.

Vol. 20

*Key words; Glycosylated Hb (HbA1c) - tumor necrosis factor-* $\alpha$  (*TNF-* $\alpha$ ), *interleukin–6 (IL-6), C-reactive protein (CRP) - diabetic retinopathy.* 

#### INTRODUCTION

Diabetic retinopathy (DR) is one of the vascular complications of diabetes mellitus. It is caused by high blood sugar levels damaging the network of tiny blood vessels that supply blood to the retina (Mody. 2014).

In general, the progression of retinopathy is orderly, advancing from mild non-proliferative abnormalities, to severe proliferative diabetic retinopathy (PDR), characterized by the growth of new blood vessels on the retina and posterior surface of the vitreous (American diabetic Association. 2002).

Pathophysiological mechanisms leading to the vascular complications of diabetes include (1) endothelial dysfunction, (2) activation of the inflammation cascade, and (3) pro-coagulant imbalance (**Dorata et al., 2014**). Their circulating biomarkers may therefore provide opportunities for early diagnosis and targets for novel treatment of DR.

Circulating biomarkers of these pathways such as tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), interleukin–6 (IL-6), C-reactive protein (CRP) (inflammation), vascular cellular adhesion molecule-1, interstitial cellular adhesion molecule-1, Eselectin, von Willebrand factor (endothelial dysfunction), plasminogen activator inhibitor-1, fibrinogen, P-selectin (procoagulant state), and adiponectin (anti-inflammation) may be associated with development of both type 1 and type 2 diabetes complications (Raczynska et al., 2014).

No. 2

Diabetic retinopathy (DR) is a serious complication of diabetes mellitus and a major cause of visual loss globally. Prevalence of DR is rising (Taylor-Philips et al., 2014) Early detection and timely treatment of sightthreatening DR have reduced the and progression incidence of visual loss. Screening for DR ophthalmoscopy and (using fundus photography) is accurate, safe and cost-effective (Britta & Nicolas. 2016).

### AIM OF THE WORK

- To confirm the correlation between HbA1c, IL-6, CRP and the development of diabetic retinopathy.
- To assess the correlation between the level of proinflammatory markers and the

severity of the diabetic retinopathy.

## PATIENT AND METHODS

The study was cross sectional study. It conducted on eighty patients. The chosen patients were selected from the Endocrine & Metabolism pediatric Unit at pediatrics Department in Aswan University Hospital during the period from March 2016 to October 2016.

## **Ethical consideration:**

- **1.** Approval of research by the local ethical committee was obtained before conducting the study.
- 2. Verbal and/or written consent were obtained from all parents and controls after explanation of the whole procedures.
- **3.** All the data and the patients and results of the study are confidential and the patient has the right to keep it.

**Financial disclosure/ funding:** The authors received no financial support for the work or publication of this article.

Claim: no conflict of interest.

## **Inclusion criteria:**

1. Age above five years and to eighteen years.

- 2. An extended group age above eighteen years to twenty-five years.
- 3. Duration of disease  $\geq$  3 years

## **Exclusion criteria:**

- 1. Patient less than five years old and more than twenty- five years old
- 2. Duration of disease < 3 years
- 3. Patients had a diagnosis of other disease.
- 4. Patients with known eye disorder e.g. congenital or traumatic.

# All patients included in the study were subjected to:

## **Clinical assessment:**

- 1. Full medical history.
- 2. History of insulin intake (its type, and number of subcutaneous injection, history of regular check blood glucose at home.
- 3. Complete physical examination.
- 4. Assessment of: weight, Bl. pressure.

## Laboratory Tests:

- 1. Routine investigation; e.g. CBC, blood sugar. urine analysis.
- 2. Lipid profile (Total cholesterol, triglyceride, LDL, MDL)

- 3. Serum creatinine, blood urea.
- 4. CRP (Quantitative): (normal < 0.5mg/dl)
- 5. Assessment of IL6: (normal < 5 pg/dl)
- 6. HbA1c: (normal  $\leq$  5)

The entire laboratory tests were done by Awareness-technologiesstat-fax-2100-microplatereader.USA, 2012.

## Methodology of Lab Investigation:

Patient blood samples (5 cc) were taken from an antecubital vein. 2 cc of the sample is taken on EDETA vacutainer tube & the remaining 3 cc is taken on Serum separator tubes (SST). The EDETA sample is sent directly to the lab estimation for of HbA1c concentration. The serum sample is allowed to clot for 30 minutes at room temperature before centrifugation for 15 minutes at approximately 1000 x g. Serum was removed and stored at -20° C until assayed.

#### **Ophthalmological examination:**

All patients were subjected to full ophthalmological examination including:

- Visual acuity, using Snellen chart or crowded Kay pictures for children having difficulty in responding to it. - Refraction and recording best corrected visual acuity (BCVA)

No. 2

- Anterior segment examination using standard slit lamp for older patients and portable slit lamp young children.
- Goldmann applanation tonometry if possible.
- Fundus examination after dilatation of the pupils using cyclopentolate 0.5% - 1% eye drops.
- Fundus photography using (Topcon OCT/fundus Camera) and fundus photographs were examined by an experienced ophthalmologist.

Diabetic retinopathy (DR) when present is classified as either nonproliferative or proliferative and macular edema is reported also.

## Nonproliferative diabetic retinopathy (NPDR):

- 1. Mild non-proliferative diabetic retinopathy: where a few micro aneurysms are present.
- 2. Moderate NPDR: there are less than 20 microaneurysms, hard yellow exudates, cotton wool spots, and venous beading are present also in only one quadrant;
- 3. Severe NPDR: when any of following clinical features is

there; micro aneurysms in all 4 quadrants; venous beading in two or more quadrants; intraretinal microvascular abnormalities (IRMA) in one or more quadrants.

- 4. Very severe NPDR: This form includes two or more of the criteria for severe NPDR.
- 5. Proliferative diabetic retinopathy (PDR): There are neovessels growing at the optic nerve (NVD) and elsewhere in the retina except the optic disc

(NVE). Also, NV may cause preretinal and subhyaloid vitreous hemorrhages and can form membrane on the posterior hyaloid surface.

**Diabetic macular edema (DME):** Macular edema is defined as retinal thickening or the existence of hard exudates within 2 disk diameter of the macula. As DME occurs apart from the stage of diabetic retinopathy, so it evaluated independently (Sayin et al 2015).

	Range	Mean ± SD	
Age (years)	6 - 18	$15.5 \pm 4.4$	
Disease duration (years)	3 - 12	$7.9\pm4.3$	
Hb (g/dl)	11.5 - 15.9	$12.62 \pm 1.64$	
Creatinine (mg/dl)	0.1 - 1.3	$0.712\pm0.24$	
Cholesterol (mg/dl)	55 - 352	$169.04 \pm 48.02$	
TG (mg/dl)	26 - 724	$116.70\pm83.9$	
HDL (mg/dl)	27 - 117	$47.03 \pm 13.2$	
LDL (mg/dl)	30 - 400	$120.78\pm57.3$	
CRP (mg/dL) (N= < 0.5mg/dl)	5-24	6.11 ± 3.5	
HbA1c (%) (N= < 5)	5-12.8	$8.9 \pm 2.4$	
IL-6 (pg/dL) (N= < 5 pg/dl)	5.5 - 46.1	$20.2 \pm 7.7$	

### RESULTS

Table (1): Descriptive characteristics of the study cases.

Fundus examination	No. (80)	%
Normal fundus	74	92.5
Vitreous Hge/Neovascularization	2	2.5
Macular edema	4	5

Table (2): Distribution of the fundus findings in studied patients.



Figure (1): Distribution of the fundus findings in studied patients.

Table (2) & Fig. (1) show the results of the fundus in the studied cases. It revealed normal fundus examination in 92.5 %, those with vitreous hemorrhage were 2.5 % and those with macular edema were 5%.

 Table (3): Correlation between fundus examination and age of the studied group

Age	Age (years)		Devalues
Fundus ex	Mean	SD	r value
Normal fundus (n=74)	15.77	3.704	
Vitreous Hge/Neovasc. (n=2)	17.64		0.004
Macular edema (n=4)	15.02	0.712	



Figure (2): Correlation between fundus examination and age of the studied group.

Table (3) & fig.(2) show that; there was a positive correlation between duration of diabetes and fundus findings in the studied patients.

CRP	CRP (mg/dl)		Devolue
Parameter	Mean	SD	r value
Normal fundus (n=74)	6.18	3.36	
Vitreous Hge/Neovasc. (n=2)	24.00	0.0	0.24
Macular edema (n= 4)	5.00	0.0	

Table (4): Correlation between CRP level and fundus examination findings



Figure (3): Correlation between CRP level and fundus examination findings

Table (4) & Fig. (3) show that; CRP level between cases with normal fundus, examination and those with retinopathic changes as follow: CRP level was  $(5.96 \pm 3.02)$ in normal cases and those of vitreous hemorrhage was  $(24 \pm 0)$ , macular edema was  $(5 \pm 0)$ . P. value (0.24) which was insignificant.

IL-6	IL-6 (pg/dl )		Dyalwa
Parameter	Mean	SD	r value
Normal fundus(n=74)	21.99	5.79	
Vitreous Hge/Neovasc. (n=2)	46.100	3.01	0.05
Macular edema(n= 4)	28.43	5.31	

Table (5): Correlation between IL6 level and fundus examination findings



Figure (4): Correlation between IL6 level and fundus examination findings

Table (5) & Fig. (4) show that; there was a positive correlation between IL6 level and fundus findings in the studied patients.

HbA1c (%)	HbA1c (%)		Darahaa
Fundus ex	Mean	SD	P value
Normal fundus(n=74)	8.826	1.742	
Vitreous He/Neovasc. (n=2)	9.41		0.923
Macular edema(n= 4)	9.79	0.883	

 Table (6): Correlation between HbA1c percent and fundus examination findings.

Table (6) shows that; there was no correlation between HbA1c % and fundus examination findings. P value was 0.923 which is insignificant.

### DISCUSSION

The risk of micro vascular disease in diabetes is dependent on hyperglycemia and other risk factors such as hyperlipidemia, hypertension and obesity. From this point, delaying of micro vascular disease (diabetic retinopathy) could be done by control of hyperglycemia. (Wolfsdorf, 2014).

As regard to mean age in years for total number of **cases**: we found that cases with normal fundus examination was (15.05  $\pm$ 4.02) compared to cases with abnormal fundus examination was (25.18  $\pm$  0.66) with P value was 0.001 that is highly significant. And regarding to comparison of age in years between normal and abnormal cases: we found cases with normal fundus was  $(15.05\pm$ 4.02) and those with vitreous Hemorrhage  $(25.64 \pm 0)$  and those with macular edema  $(25.02 \pm$ 0.71) years, P. Value (0.004)which is highly significant.

While in comparison of age in between patient with vears normal fundus and those with abnormal fundus: we found that age in years in patient with normal fundus examination (15.77  $\pm$  3.7) and those with abnormal fundus examination were (25.18  $\pm$ 0.66) years with P value 0.001 which is highly significant. Regarding to comparison of age in years according to the abnormal

fundus finding: we found that **patients** with normal fundus examination were  $(15.77 \pm 3.704)$  years, those with vitreous hemorrhage were  $(25.64 \pm 0)$  years and those with macular edema were  $(25.02 \pm 0.712)$  with P value 0.004 which is highly significant.

Come in agreement to our study, Luis et al., 2016 who said that the rate of diabetic retinopathy is higher in patients who were older at diagnosis in type1 diabetes. In contrast to our study, the study by American Diabetic done Association, 2010 which said that patient at highest risk of development proliferative of retinopathy are those of age from 5 -14 years and almost all patient with type 1 diabetes after 20 years will show sign of retinopathy, also Britta & Nicolas, 2016 said that retinopathic diabetic changes begin in patient after 10 years of age and had diabetes not less than 3 years duration

Comparing the age groups between total number of **cases** with normal fundus examination, cases with vitreous Hemorrhage, revascularization and those with cases with macular edema: we found that cases with age group above 18 years we found 25 (86.2%) with normal fundus examination, 3 (10.3%) with macular oedema and 1 (3.4%) had vitreous hemorrhage P.

As regard to Comparison of age groups between **patients** with normal fundus examination, cases with vitreous Hemorrhage. revascularization and those with patients with macular edema. We found that patients less than 18 years were 55 person (100%) with no one had abnormal fundus examination while patients with age group above 18 years we found 21 (86 %) with normal fundus examination, 3 (12 %) with macular oedema and 1 (4%) had vitreous hemorrhage P. Value (0.001)which is highly significant.

Come in agreement to our results, the study done by kim, 2015 who said that we should start fundus examination later as he found that the youngest person reported to have sever diabetic retinopathy was between 15 - 19 years old. In contrast to our study, the study done by Mulugeta et al., **2016** in which they said that 4.7%of diabetic patients less than 18 years had diabetic retinopathic changes and also these patient were poorly controlled, and also according to Royal College of ophthalmology, 2013 they found that the prevalence of diabetic retinopathy in children from 10-13 years 1%, from 14-15 years

was 5.8% and from 16- 18 years was 17.7%

Regarding disease duration in patients: we found that patients with normal fundus examination. mean duration was  $(7.45 \pm 3.7)$ years) and those with abnormal fundus examination. mean duration was (15.5  $\pm$ 7.9) (P Value significant. 0.020) which is comparison Regarding to of disease duration between patients with normal fundus and those with retinopathic changes: we found that disease duration in patients with normal fundus mean of  $(7.45\pm 3.7)$ , vitreous hemorrhage mean  $(11\pm 0)$ , and macular edema mean  $(17 \pm 8.89)$  and P. Value (0.049) which is significant. And so there is relationship between disease duration and occurrence of DR

Come in agreement to our study, ADA, 2010 and Sobrin et al., 2015 who said that some patient with diabetes don't show any DR changes even after long time period, this may be due to genetic susceptibility (Luis et al., 2009). In contrast to our study, the study done by Nentwich, 2015 who said that about 1/3 of patients had sign of diabetic retinopathy at time of initial diagnosis and after 5 years, approximately 25 % of type 1DM will have retinopathy, after 10 years almost 60% have retinopathy. And Britta & Nicolas, 2016 who said that diabetic retinopathy changes begin after 3 years of disease

No. 2

As regard to comparison of CRP between total numbers of cases: we found mean for CRP in cases with normal fundus (5.96  $\pm$ those of vitreous 3.02). hemorrhage mean  $(24 \pm 0)$ , and macular edema (5  $\pm$  0) with P. Value (0.019) which is significant. While in Comparison of CRP between patients with normal and abnormal fundus: we found fundus patients with normal examination, CRP was (6.18 ± 3.36) mg/dl while those with vitreous hemorrhage CRP was (24  $\pm$  0) mg/dl, and those with macular edema CRP was  $(5 \pm 0)$ with P value 0.024 which is significant.

Come in agreement to our study (Karaman et al., 2012) and (Stong et al., 2015) who said that there is elevation of CRP with diabetic retinopathy. This result support that there is strong relation between level of CRP and severity of diabetic retinopathy

As regard to comparison of IL-6 between total number of **cases**: we found in **cases** with normal fundus examination IL-6 was (19.72  $\pm$  7.14) and those with abnormal fundus examination IL-6 was

 $(32.85 \pm 9.84)$  with P. Value (0.011) which is significant. While in Comparison of IL-6 between **patients**: we found that **patients** with normal fundus examination, **IL-6** was (21.99 ± 5.79) pg/dl while those with abnormal fundus examination **IL-6** was (32.85 ± 9.84) pg/dl, with P value 0.02 which is significant.

From the previous results in our study we found that there is correlation between level of IL-6 in the diabetic cases and those of non-diabetic cases (control), come in agreement to our study these Goldberg,2009 studies and symeinidis et al 2011. Also we found there is correlation between IL-6 in diabetic patients with normal fundus examination and that with diabetic retinopathic change. Come in agreement to our study these studies done bv American ophthalmology association, 2013.

As regard to HbA1c between total numbers of **cases**: we found HbA1c in cases with normal fundus examination was ( $8.88\% \pm$ 2.42), cases with vitreous Hemorrhage was ( $9.4\% \pm 0$ ), and those with macular edema ( $9.79 \pm$ 0.88). P value 0.899 which is insignificant. While regarding to comparison between HbA1c in patients: we found that patients with normal fundus examination HbA1c was (9.826 %  $\pm$  1.742), those of vitreous hemorrhage (9.41 %  $\pm$  0) and those with macular edema (9.79 %  $\pm$  0.883 %) and the P value 0.923 which is insignificant.

Come in agreement to our study, Luis et al., 2009 who said that HbA1c variability is an independent risk factor for diabetic retinopathy. In contrast to our study, Dohl- Jorgens et al., 2015, Kahler et al., 2015 and ADA, 2015 who stated that there is a strong relationship between the level of HbAIc and incidence chronic of micro vascular complications diabetic and retinopathy.

## CONCLUSION

- Our results show significant positive correlation between diabetic patients and their IL-6 in relation to diabetic retinopathy. There is no significant difference between diabetic patients with normal fundus examination and those with abnormal fundus examination regarding CRP level.
- There was a positive correlation between duration of diabetes and fundus findings in the studied patients.
- There is no significant difference between diabetic patients with normal fundus

examination and those with abnormal fundus examination in their HBAIc.

#### RECOMMENDATIONS

- Screening for diabetic retinopathy must be annually after 18 years of age and not before three years of illness duration this play a very important role in early detection and treatment.
- Further studies are recommended with more patients and long follow up to firmly establish the pathophysiological relationship between IL-6 and severity of DR.

#### REFERENCES

- **1. American Diabetic Association (2015):** Stages of diabetic retinopathy. Diabetes care, 38 (2): 308-315.
- American Diabetic Association. (2016): Standards of medical care in diabetes. Diabetes care, 15: 203-299.
- **3.** Britta M, Nicolas J (2016): Definition of Diabetes Mellitus, Nelson textbook of pediatrics, 20th edition, Philadelphia, Saunders Elsevier; Chapter 589, : 2760.
- 4. Dahl Jorgensenk, et al, (2015): Hemoglobin A1c as a control for diabetes (Middle east Afr J. Ophtallmol). 322 (2):151 – 156.
- 5. David M, Nathan R. (2014): the diabetes control and complication. Vol, 37 (1): 9-16.
- 6. Davis SM, Davis AB, Maddux, Guyt, Maahs (2016): Profound hypokalemia associated with DKA.

Pediatericdiabetes ;17: 61 – 65.

No. 2

- 7. Diabetic Retinopathy Study Research Group. (1981): A modification of the Airlie House classification of diabetic retinopathy.
- 8. Goldberg RB (2009): Cytokine and cytokine-like inflammation markers, endothelial dysfunction, and imbalanced coagulation in developdiabetes and of its ment complications. Journal of Clinical Endocrinology Metabolism. and 94(9):3171-3182
- 9. Gologorsky D, Thanos A, and Vavvas D, (2012): "Therapeutic interventions against inflammatory and angiogenic mediators in proliferative diabetic retinopathy," Mediators of Inflammation, vol10.
- 10. Kahler PE, Grevstad BE, Almdell D, Gluud C (2015): The relationship of glycemic Exposure to the risk of development and progression of retinopathy, Feb. 3 (2): 107 – 117.
- 11. K|araman K, Gverovic AA, Znaor L, Sapunar A. et al. (2012): IL-12 concentration in the aqueous tumour and serum of diabetic retinopathy patients, Graefe's Archive for Clinical and Experimental Ophthalmolgy, vol 250, (6): 815 – 821.
- 12. Kim JH, kang SW, His Ha and Kim JR. (2013): Vitrectomy combined with intravitrial triamcinolone acetonide injection and laser for diabetic macular edema, Korean Journal of ophthalmology, vol. 27, no.3 PP. 186 – 193.
- **13. Luis et al (2016);** Journal of diabetes research volume (2016) Article ID 9898309; Page 7
- 14. Mayer-Davis EJ, Busik JV,

**Esselman WJ and Gavin (2012):** Examining the role of lipid metabolism in diabetic retinopathy, Dec. 17 (6): 661 - 675

- **15. Mulugeta et al (2016):** Pediatric diabetic retionopathy experience of a tertiary hospital in Ethiopian; BMC Res Notes 9:116
- 16. Murata T, Ishibashi T, Khalil A, Hata Y, Yoshikawa H, Inomata H. (1995): Vascular endothelial growth factor plays a role in hyperpermeability of diabetic retinal vessels. Ophthalmic Res; 27: 48-52.
- 17. Murugesari P, Shukla D, Rajendran A, Kim R et al. (2008): proinflammatory cytokines and angiogenic and anti- angiogenic factors in vitreous of patients with proliferative diabetic retinopathy and eales' disease Retina, vol 28 (6):817– 824.
- **18.** Nentwish MM. (2015): Diabetic retionopathy ocular complication. Diabt sche retinopathy der diabeto-logy 6:491-502.
- 19. Raczyńska K, Zorena K, Myśliwska J, **Myśliwiec** M, Raczyńska-WoźniakD. **Balcerska** A. (2008): Analysis of the proangiogenic factor influencing the development of retinopathy in children with diabetes mellitus type 1. Polish Journal of Environmental Studies;17(1A):132-136.
- 20. Raczynska D, et al (2014) ; Current

trend in monitoring and treatment of diabetic retinopathy in adolescence 492926 ,Feb 13

- **21. Royal colleague of ophthalmology** (2013): Definition of diabetic retinopathy. Guidelines for the management of diabetic retinopathy., London.
- 22. Sayin N, Kara N, Pekel G. (2015): Ocular complications of diabetes mellitus. World J Diabetes; 6 (1): 92-108.
- **23. Sobrin L, Green T, et al (2015):** Candidate Gene association study for diabetic retinopathy. Ophthalmol. vissci 52:7593 – 602.
- **24.** Stong J et al. (2015): Relationship between CRP and diabetic retionopathy. journal plos org. Dec 4. 2015
- 25. Symeonidis C, Papakonstantinou E, Androudi S. et al. (2011): "Interleukin-6 and the matrix metalloproteinase response in the vitreous during proliferative vitreoretinopathy," Cytokine, vol. 54, no. 2, pp. 212–217.
- 26. Wolfsdorf JI, (2014): international society for pediatric and adolescents diabetes (ISPAD) guidelines for management of DKA: Do the guidelines need to be modified. Ped Diab. 15 (4): 277 86.



أ.د. حنان عبدالمنعم محمد \* - د. محمد بهاء الأمبر هوارى \* - د. أحمد فتحى جبر \* \* د. عبد الله محمد العبيدى \* \* \* - ط. تريزه سعد عبد الشهيد \*

أقسام \*طب الأطفال \*\* طب العيون \*\* \* الكيمياء الحيوية - جامعة أسوان

إن التهابات الشبكية في أطفال مرضى السكر تعد واحدة من أهم وأخطر مضاعفات مرض السكر

هناك مراحل كثيرة لالتهابات الشبكية بدءا من الارتشاح الشبكي وحتى حدوث نزيف في السائل الزجاجي مما يترتب عليه فقد الإبصار

ويصاحب هذه الالتهابات ارتفاع في مستوى معاملات الالتهابات بالدم وهي كالآتي: انترليوكين 6 – وبروتين س التفاعلي

أيضاً زيادة نسبة الهيموجلوبين التراكمي وخاصة في الأطفال الذين يعانون من سوء ضبط مستوى السكر

- وقد هدف هذا البحث إلى: • تاكيد العلاقة بين الهيموجلوبين التراكمي والانترليوكين 6 – وبروتين س التفاعلى وحدوث التهابات الشبكية • البحث عن وجود علاقة بين معدلات معاملات الالتهابات (الانترليوكين 6 –
  - البخت على وجود عرف بين معدرت معامرت الالتهابات (الالترنيودين ٥ -وبروتين س التفاعلي) بالدم وشدة خطورة التهابات الشبكية

وتتلخص خطوات العمل في التالي:

فحص ثمانين من مرضى السكر يتراوح أعمارهم ما بين 5 إلى 18 سنة فحصا
 إكلينيكياً.

قياس نسبة الانترايوكين 6 – وبروتين س التفاعلى وكذلك معدل الهيموجلوبين
 التراكمى
 فحص قاع العين لهؤ لاء الأطفال

وقد أوضحت نتيجة البحث أن هنالك علاقة بين حدوث مرض السكر وارتفاع معدل معامل الالتهاب (الانترليوكين 6) ولا يوجد علاقة واضحة بين حدوث مرض السكر ومستوى معامل الالتهاب (وبروتين س التفاعلى) كذلك وجود علاقة واضحة بين حدوث التهابات الشبكية وعمر المرضى حيث إن التهابات الشبكية تزداد بتقدم عمر المرضى وتقدم سنوات المرض.

وقد أوصت الدراسة ضرورة متابعة الأطفال المصابون بالسكر وعمل فحص لقاع العين بصفة دورية لاكتشاف أي تغيرات مبكراً.