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IMBALANCE BETWEEN FREE RADICAL PROPAGATION AND SOME ANTIOXIDANTS IN PATIENTS WITH CATARACT

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Abstract

The relation of oxidative stress to the occurrence of cataract remains to be undetermined and must be clarified. So, the aim of this work was to study the effect of 02-free radical and some antioxidants in the pathogenesis of senile and diabetic cataract. This work was carried on 30 patients and 10 healthy subjects as control. They were 23 males and 17 females. Their ages ranged from 50 to 64 years. Patients were classified into senile cataract, diabetic without cataract and diabetic cataract groups. Each group included 10 patients.

The results of this work showed that, in patients with senile cataract, there were non-significant increase of fasting serum glucose (FSG) and serum total bilirubin (serum T. bilirubin) compared with the control group. Serum lipid peroxide (S.LP) and serum ceruloplasmin (S. Cp) were signficantly increased ($p<0.05$) while plasma superoxide dismutase (SOD) and serum uric acid were significantly decreased ($p<0.05$) compared with the control group. Moreover, diabetic patients with and without cataract showed a significant increase of FSG, S. total bilirubin, S. LP, S. Cp, and S. uric acid ($p<0.05$) while plasma SOD was significantly decreased ($p<0.05$) compared with the control group.

Comparative study of the diabetic cataract versus senile cataract and diabetic without cataract, our results showed a significant increase of FSG, S.LP, S. total bilirubin and S. Cp, while there was significant decrease of plasma SOD in diabetic cataract compared with both senile cataract and diabetic without cataract ($p_1<0.05$ & $p_2<0.05$), respectively.

S. uric acid was significantly increased in diabetic cataract compared with senile cataract group ($p_1<0.05$) while it was non-significantly increased compared with diabetic without cataract group.

Also, aqueous humor study of diabetic cataract group versus senile cataract group showed that LP and uric acid were significantly increased ($p<0.05$) while SOD was significantly decreased ($p<0.05$).

Correlation study revealed that, age was significantly and positively correlated with LP, but negatively correlated with SOD both in serum and aqueous in all patient groups. FSG was significantly and directly corrleated with serum and aqueous LP, uric acid, S. total bilirubin, and S. Cp in diabetics with and without cataract. Moreover, serum and aqueous LP was significantly and inversely correlated with serum and aqueous SOD in all patient groups, while, it was positively correlated with S. total bilirubin, and S. Cp in diabetics with and without cataract.

We could conclude that the imbalance between generation of 02-free radical and plasma SOD may have an etiological implication in the occurrence of cataract.

Introduction

The paradox of aerobic life or the oxygen paradox is that higher eukaryotic aerobic organisms cannot exist without oxygen, yet oxygen is inherently dangerous to their existence. Thus, dark side of oxygen relates directly to the fact that each oxygen atom has one unpaired electron in its outer valence shell, and molecular oxygen has two unpaired electrons thus atomic oxygen is a free radical (FR) and molecular oxygen is a free bi-radical. Concerted tetravalent reduction of oxygen by the mitochondrial electron transport chain to produce water is considered to be a relatively safe process, however, the univalent reduction of oxygen generates reactive intermediates, which are responsible for oxygen toxicity. The reductive environment of the cellular milieu provides ample opportunities for oxygen to undergo unscheduled univalent reduction (Davies, 1995).

The antioxidant activity decreases with aging, so the human is exposed more to oxidative stress

and their various diseases such as atherosclerosis, cancer and various types of eye diseases such as cataract (Davies, 1995).

Cataract is one of the major causes of blindness worldwide. Although several mechanisms have been suggested to explain the etiopathogenesis of cataract, none of them is conclusive (Balisubramanian et al., 1990).

It is generally accepted that opacification of the lens is the last step of a complex process in which oxidation is a predominant initiating factor (Siezen, et al., 1989). The oxidative mechanisms seem even to have a great role in the onset of cataract in diabetic patients. Oxidation of the lens proteins is more common in older than younger lenses (Spector 1985).

Free radicals (FRs) can damage the lens by peroxidizing membrane lipids which result in the formation of malondialdehyde (MDA), which in turn can cross-links between membrane lipids and proteins, introducing damage to bases of DNA and polymerizing and cross-linking proteins (Lerman, 1992).

The lens defense mechanisms against oxidative insult include many antioxidants, e.g. the superoxide dismutase (SOD) which catalyzed dismutation of the toxic superoxide anion to hydrogen peroxide, which is detoxified by other antioxidant enzymes (Costarides et al., 1991).

Ceruloplasmin has a powerful antioxidant activity. It is suggested that it acts as a scavenger of superoxide anion radicals (Ali et al., 1995). Total bilirubin is also considered as antioxidant by scavenging the peroxyl radical. Moreover, uric acid acts as a powerful antioxidant against many FRs (Halliwell et al., 1994).

Aim of the Work

The aim of this work is to study the effect of 02-free radical and some antioxidants in the pathogenesis of senile and diabetic cataract.

Subjects and Methods

This study included 40 subjects (23 males and 17 females).

Their ages ranged from 50-64 years. They were selected from those attending the Ophthalmology Clinic at Benha University Hospitals. The selected subjects were categorized into the following groups :

Group I : Ten normal healthy subjects (7 males and 3 females). Their ages ranged from 51 to 64 with a mean value of 56.6 ± 1.249 years, as control group.

Group II : Ten subjects (7 males and 3 females) with senile cataract. Their ages ranged from 51 to 64 years, with a mean value of 56.4 ± 1.536 years.

Group III : Ten diabetic subjects with type II D.M. (6 males and 4 females) without cataract. Their ages ranged from 51 to 64 years, with a mean value 57.8 ± 1.4 years. The duration of D.M. ranged from 3 to 5 years with a mean value 3.9 ± 0.28 years. They were treated with oral hypoglycemic drugs and dietary regimen, but they were uncontrolled.

Group IV : Ten diabetic subjects with type II D.M. (6 males and 4 females), having diabetic cataract. Their ages ranged from 50 to 62 years, with a mean value 56.4 ± 1.314 years. The duration of D.M. ranged from 5 to 9, with a mean value 6.8 ± 1.23 years. They were treated with oral hypoglycemic drugs and dietary regimen, but they were uncontrolled.

All subjects included in the present study were subjected to the followings :

- Full history to exclude the presence or absence of diabetes mellitus (DM), liver or kidney diseases and endocrinal disorders.

- Full ophthalmologic examination including visual acuity testing, measurement of intraocular pressure, slit lamp biomicroscopy, determination of cataract status and fundus examination.

Three samples were taken :

1- A venous blood sample (5 cc) was taken during fasting and divided into 2 parts :

One. The first part was taken on ethylene diamine tet- ra-acetic acid (EDTA) powder then

centrifuged and the separated plas- ma was kept frozen at - 80°C until assay of SOD (Nebot et al., 1993).

Two. The second part was left to be clotted, then centri-fuged and serum separat- ed was used for determi- nation of :

- S. glucose (Trinder et al., 1969).
- SGOT (Reitman & Frankel, 1957).
- SGPT (Reitman & Frankel, 1957).
- S. Creatinine (Husdan and Rapoport, 1968).
- S. Uric acid (Fossati et al., 1980).
- S. Total bilirubin (Jendrassik, 1938).

The remaining part of the serum was kept frozen at -80°C until assay of :

- S. Lipid peroxide (Draper & Hadley, 1990).
- S. ceruloplasmin (Cunningham et al., 1995).

2- Another venous blood sample (2.0 cc) was taken 2 hours postprandial, then centrifuged and the separated serum was used for determination of glucose (Trinder et al., 1969) to assure the diabetic condition.

3- An aqueous humor sample (bout 150 µl) was taken from patients with senile and diabetic cataract during surgery by puncturing the anterior champer of the eye for estimation of uric acid (Fossati et al., 1980). The remaining part was kept frozen at-80°C for determination of :

- Lipid peroxide (Draper & Hadley, 1990).
- SOD (Nebot et al., 1993).

Exclusion Criteria :

* Smoking, obesity and patients with liver, kidney or endocrinal diseases, glaucoma and diabetic retinopathy.

Results and Discussion

Table (1) : The mean ± SE and p values of FSG, S.LP, plasma SOD, S. uric acid, T. bilirubin and Cp in the studied groups compared with each other.

Biochemical Parameters / Groups	FSG (mg/dl)	S.LP (nmol/ml)	Plasma SOD (U/ml)	S. Uric acid (mg/dl)	S.T. bilirubin (mg/dl)	S.Cp (mg/dl)
Control	80.8 ± 1.794	11.39 ± 0.754	41.6 ± 4.52	5.53 ± 0.342	0.579 ± 0.05	14.456 ± 0.554
Senile cataract	88.6 ±1.714 NS	15.3 ± 0.861 p<0.05	29.02 ±2.318 p<0.05	4.19 ± 0.28 p<0.05	0.587 ± 0.027 NS	18.466 ± 0.623 p<0.05
DM without Cataract	121.5 ± 2.35 p<0.05	16.76 ± 0.943 p<0.05	19.4 ±1.916 p<0.05	6.68 ± 0.261 p<0.05	0.695 ± 0.028 p<0.05	27.473 ± 1.614 p<0.05
Diabetic cataract	144.8 ± 1.298 p<0.05 p_1<0.05 p_2<0.05	21.19 ± 1.19 p<0.05 p1<0.05 p2<0.05	14.7 ± 1.49 p<0.05 p_1<0.05 p_2<0.05	7.13 ± 0.38 p<0.05 p_1<0.05 NS	0.783 ± 0.04 p<0.05 p_1<0.05 p_2<0.05	48.532 ± 2.756 p<0.05 p_1<0.05 p_2<0.05

p : Probability versus control

p_1 : Probability versus senile cataract

p_2 : Probability versus diabetics without cataract

NS = Non significant

Table (2) : The mean ± SE and p values of aqueous LP, SOD, uric acid levels in diabetic cataract compared to senile cataract

Biochemical Parameters / Groups	Aqueous		
	LP (nmol/ml)	SOD (U/ml)	Uric Acid (mg/dl)
Senile cataract group	6.51 ± 0.433	13.85 ± 0.773	0.205 ± 0.028
Diabetic cataract group	9.86 ± 0.554 p<0.05	10.33 ± 0.463 p<0.05	0.367 ± 0.066 p<0.05

Table (3):The correlation coefficient (r) between age and various biochemical parameters (LP, SOD, uric acid, T. bilirubin and Cp) in serum and aqueous of all patient groups.

Biochemical Parameters / Groups		LP		SOD		Uric Acid		T.bilirubin	CP
		S.	Aq.	S.	Aq.	S.	Aq.	S.	S.
Senile cataract group	"r"	0.757	0.651	-0.861	-0.786	0.491	0.049	0.563	0.478
	P	<0.05	<0.05	<0.05	<0.05	NS	NS	NS	NS
D.M. without Cataract group	"r"	0.820	-	-0.723	-	0.462	-	0.505	0.476
	P	<0.05	-	<0.05	-	NS	-	NS	NS
Diabetic Cataract group	"r"	0.683	0.659	-0.665	-0.706	0.170	0.39	0.449	0.445
	P	<0.05	<0.05	<0.05	<0.05	NS	NS	NS	NS

Table (4):The correlation coefficient (r) between FSG and various biochemical parameters (LP,SOD,uric acid,T.bilirubin and Cp)in serum and aqueous of diabetic patients,with and without cataract

Biochemical Parameters / Groups		LP		SOD		Uric Acid		T.bilirubin	CP
		S.	Aq.	S.	Aq.	S.	Aq.	S.	S.
D.M. without Cataract group	"r"	0.684	-	-0.537	-	0.796	-	0.664	0.649
	P	<0.05	-	<0.05	-	<0.05	-	<0.05	<0.05
Diabetic Cataract group	"r"	0.650	0.662	-0.446	-0.419	0.777	0.695	0.843	0.784
	P	<0.05	<0.05	NS	NS	<0.05	<0.05	<0.05	<0.05

Table (5):The correlation coefficient (r) between serum and aqueous LP and other biochemical parameters (LP,SOD,uric acid,T.bilirubin and Cp)in serum and aqueous of all patient groups.

Biochemical Parameters / Groups		SOD		Uric Acid		S.total bilirubin	Serum Cp
		S.	Aqueous	S.	Aqueous		
Senile cataract group	"r"	-0.719	-0.673	0.280	-0.213	0.534	0.550
	P	<0.05	<0.05	NS	NS	NS	NS
D.M. without Cataract group	"r"	-0.616	-	0.402	-	0.649	0.688
	P	<0.05	-	<0.05	-	<0.05	<0.05
Diabetic Cataract group	"r"	-0.865	-0.636	0.401	0.408	0.776	0.675
	P	<0.05	<0.05	NS	NS	<0.05	<0.05

S = Serum Aq = Aqueous NS = non-significant

Oxidative stress is implicated in several age-related ocular diseases including : senile cataract, glaucoma and senile macular degeneration. Also, the oxidative mechanisms seem even to have a great role in the onset of cataract in diabetic patients (Spector, 1985).

Serum free radicals and antioxidants may affect the lens through the aqueous, so measuring the levels of LP and some antioxidants, e.g. SOD and Cp in serum and aqueous correlates with their lens levels (Obara et al., 1995). The extent of tissue damage is a result of the imbalance between the generation of free radicals and their elimination by the defense mechanisms (Reddy and Bhat ,1999)

Our results showed a significant increase of S. LP (Table 1) in all studied groups compared with the control group ($P<0.05$). Moreover, there was a significant increase of S. LP in the diabetic cataract patients compared with both senile cataract patients ($P_1<0.05$) and diabetics without cataract ($P_2<0.05$). On the other hand, aqueous LP was significantly increased in diabetic cataract (Table 2) compared with the senile cataract patients ($P<0.05$).

The role of LP in the development of senile cataract could be explained by damage to the lens membrane proteins as receptors and enzymes, e.g. Na^+/k^+ ATPase leading to increase leaking of ions resulting in lens hydration and opacification (Lucas et al., 1986). Also, since MDA is diffusible (Esterbauer et al., 1991), it causes lens protein modification and aggregation and thus leads to opacification (patmore & Duncan, 1981). Moreover, increased lipid peroxidation leads to glutathione consumption (Babizhayev et al., 1992), which is important in the prevention of lens opacification (Reddy, 1990).

The increase of LP in senile cataract could be explained by Varma et al., (1997) who found that with aging, antioxidant enzymes activities were decreased due to non-enzymatic glycation of these enzymes. However, the same mechanism of non-enzymatic glycation of antioxidant enzymes

occurs both in diabetics with and without cataract (Obara, 1995).

In addition to increased glycation of antioxidant enzymes in diabetics, increased glycation of lens collagen and plasma proteins occurs which leads to stimulation of lipid peroxidation that in turn enhances lipid and protein damage that continuously supply free radicals leading to a vicious circle of damage (Baynes, 1991).

To our mind, there were no previous reports about S. LP assay in senile and diabetic cataract in comparison with the control group, and in diabetic cataract compared with diabetics without cataract, but there were many reports about LP assay in lens homogenates like Lian et al., (1993) and Takoyama et al., (1999).

In accordance to our results Griesmacher et al., (1995) and Telci et al., (2000) found a significant increase of S. LP in diabetics without cataract compared with normal control. Also, Obara, (1995) reported a significant increase of serum and aqueous LP in diabet - ic cataract compared with senile cataract.

The present study showed a positive significant correlation ($p<005$) between age and LP level (Table 3) both in serum and aqueous in senile, ($r=0.757$ & 0.651, respectively) and diabetic cataract ($r=0.683$ & 0.694, respectively) patients. This may be explained by the decrease of the antioxidant defenses with aging (Lian et al., 1993). Esmat & Shaker, (1998) reported significant increase of MDA level with increasing age both in lens and erythrocytes of diabetic and senile cataract patients signifies the importance of the oxidative stress and lipid peroxidation in the aging and senile cataract formation.

Moreover, a positive significant correlation ($P<0.05$), (Table 4) was reported between FSG and LP level in both serum ($r=0.650$) and aqueous ($r=0.651$) of patients with diabetic cataract. This may be explained by more enhanced protein glycation with increased FSG (Varma, 1997).

Our data showed a significant decrease of plasma SOD activity

(Table 1) in all studied groups compared with the control group ($p<0.05$). Moreover, there was a significant decrease of plasma SOD in the diabetic cataract group compared with both senile cataract ($p_1<0.05$) and diabetics without cataract groups ($p_2<0.05$). On the other hand, aqueous SOD activity was significantly decreased in diabetic cataract (Table 2) compared with senile cataract group ($p<0.05$).

The decreased plasma SOD activity in both senile cataract and diabetics without cataract may be attributed to aging and abnormal glycation (Varma, 1997). The more decrease in plasma SOD activity in diabetic cataract patients may be explained by more enhanced glycation of SOD in diabetic patients (Özmen et al., 1997).

There wee no previous reports about plasma SOD activity in both senile and diabetic cataract in comparison with the control group, but there were some reports about SOD activity in lens homogenates like, Fecondo and Augusteyn, (1983), Lian et al., (1993) and Varma, (1997).

In agreement to our results Rema et al., (1995) and Fukui et al., (2000) found a significant decrease of plasma SOD activity in type II diabetics without cataract compared with control.

Also, Obara, (1995) reported a significant decrease of plasma and aqueous SOD activity in diabetic cataract compared with senile cataract. However, on contrary to our results, Kernell et al., (1992) reported non-significant difference between aqueous SOD in senile and diabetic cataract patients.

The current study revealed a negative significant correlation ($p<0.05$) between age and plasma and aqueous SOD levels (Table 3) in both senile ($r=-0.861$ & -0.723), respectively and diabetic cataract patients ($r=-0.784$ & -0.66), respectively. Picot et al., (1992) found decreased SOD activity with age and they suggested that SOD may be irreversibly inactivated by its product (H_2O_2) in a concentration dependent manner or by an increase in the glycation of SOD as a function of age.

Relative to out findings but in

Lens homogenate Scharaf et al., (1987) found reduced lens SOD activity with increased age in cataract of both diabetics and normal subjects. Also, Esmat & Shaker, (1998) found a significant decrease of erythrocyte and lens SOD with increasing age in senile and diabetic cataract.

Also, the current study showed a negative non-significant correlation between FSG and SOD levels in serum of diabetics with and without cataract (r=-0.446, & -0.537), respectively and in aqueous of diabetic cataract (r=-0.419), (Table 4).

The results of this work showed a significant decrease of S. uric acid (Table 1) in senile cataract patients, but significantly increased in both diabetics with and without cataract compared with the control group ($p<0.05$). Also, serum and aqueous uric acid (Table 1 & 2) was significantly increased in diabetic cataract patients compared with senile cataract ($p_1<0.05$), while S. uric acid was non-significantly increased compared with diabetics without cataract ($p_2>0.05$).

There are many explanations for the hyperuricemia associated with D.M. First, in diabetic patients, there was an increased tissues protein catabolism (Collier et al., 1990), which may lead to hyperuricemia. Second, in patients with D.M., oxaloacetate was decreased and Kreb's cycle is inhibited. So, pyruvic acid was accumulated with subsequent increase in lactic acid. However, hyperlactacidemia leads to increase renal threshold level for uric acid clearance (Yokogoshi & Saito, 1996). Third, Madianov et al., (2000) reported that hyperuricemia in diabetic patients may be due to renal dysfunction and/or hyperproduction of uric acid. This later factor may be attributed to the higher activity of xanthine oxidase recorded in D.M.

Although the occurrence of hyperuricemia in diabetic patients as a counteracting mechanism against oxidative damage (Nieto et al., 2000) it has a lens capsular insult which is a possible pathophysiological explanation for cataract formation (Beiran et al., 1994).

The significant decrease of S. uric acid level in senile cataract may be due to age-related changes of xanthine oxidoreductase. This complex enzyme is formed of xanthine dehydrogenase and xanthine oxidase. So, uric acid level decreased when the ratio between xanthine dehydrogenase and xanthine oxidase increase and vice versa (Chung et al., 1999).

Uric acid mostly acts by promoting the binding of iron and copper, thus removing the free iron and copper from the plasma leading to prevention of Fe^{2+} and Cu^{2+} catalyzed lipid peroxidation and Fenton reaction (Marino, 1998). This suggests the role of uric acid as a strong antioxidant against the development of senile cataract (Kaluzny et al., 1996).

Kaluzny et al., (1996) reported a significant decrease of S. uric acid in senile cataract while Madianov et al., (2000) reported a significant increase of S. uric acid in diabetic cataract compared with the control group. The previous two reports were compatible with our result. On the other hand, Cerrellio et al., (1997) reported a significant decrease of S. uric acid in diabetics compared with the control group, which is contradictory to our results.

The significant increase of aqueous uric acid in diabetic cataract compared to the senile cataract group confirms the previous explanations about the role of diabetes as an intervening factor in the occurrence of cataract (Metelitsyna, 1998 and Nieto et al., 2000). Thus, the compensatory increase of aqueous uric acid may be a protective mechanism against oxidant stress to delay cataract formation.

There were no previous reports about aqueous uric acid in diabetic cataract compared to the senile cataract, but only one report of Kalunzy et al., (1996) who compared aqueous uric acid in senile with pre-senile cataract was recorded.

The obtained data showed non-significant correlation between age and serum and aqueous uric acid (Table 3) in all patient groups.

The present study revealed a

Positive significant correlation between FSG and serum, uric acid ($r=0.796$, $P<0.05$) in diabetic without cataract. Also, there was positive significant correlation between FSG and serum and aqueous uric acid levels in diabetic cataract patients, ($r=0.777$ & 0.695 $p<0.05$, $P<0.05$), respectively (Table 4). This may be attributed to the increased renal dysfunction and production of uric acid with increased severity of diabetes manifested by increased FSG (Madianov, et al., 2000). This result coincided with that obtained by Wakabayashi, (1998) who reported a positive significant correlation between FSG and serum uric acid in age groups between 40 to 59 years.

Our work showed a non-significant increase of S. total bilirubin (Table 1) in senile cataract group, while it was significantly increased in diabetics with and without cataract compared with the controls ($p<0.05$). Moreover, S. total bilirubin was significantly increased in diabetic cataract compared with both senile cataract ($p_1<0.05$) and diabetics without cataract ($p_2<0.05$).

The significant increase of S. total bilirubin in diabetics with and without cataract may be attributed to intrahepatic cholestasis associated with D.M. There are many factors that may lead to intrahepatic cholestasis, like, autonomic neuropathy, which decrease biliary tract motility, fatty liver that leads to canalicular compression and cholangitis (Clark, 1996).

Our results were in accordance with Tu~non et al., (1991) and in disagreement with the results of Chorne et al., (1994) and Ko et al., (1996).

Although our finding showed non-significant increase in S. total bilirubin in patients with senile cataract, Donnelly et al., (1995) had reported a significant increase of S. total bilirubin associated with senile cataract. They explained this finding by mild intrahepatic cholestasis. However, the cause remains unclear.

The present study showed a positive non-significant ($p>0.05$) correlation between age and serum bilirubin (Table 3) in senile

cataract (r=0.563), diabetic without cataract (r=0.505) and diabetic cataract (r=0.449) groups.

Also, there was a positive but significant correlation ($P<0.05$) between FSG (Table 4) and serum T. bilirubin in diabetic with (r=0.843) and without cataract patients (r=0.664). This may be explained by increased liver affection by increased severity of diabetes mellitus (Foster, 1988).

Our findings showed a significant increase of S. Cp (Table 1) in all studied groups compared with the control group ($p<0.05$). Moreover, there was a significant increase of S. Cp in diabetic cataract compared with both senile cataract and diabetics without cataract groups ($p_1<0.05$ and $p_2<0.05$). Ceruloplasmin was not detected in aqueous humor of our patients by radial immunodiffusion method.

Previous reports were implicated about the role of oxidative stress in the development of senile cataract (Lucas et al., 1986) and diabetics without cataract (Androne et al., 2000) and diabetic cataract (Esmat & shaker, 1998). Ceruloplasmin has been found to be an acute phase reactant protein that has a strong antioxidant activity, which increased in response to oxidative stress (Goldstein et al., 1979) and (Ali et al., 1995).

Also, oxidative stress has been reported to be more effective in the genesis of diabetic cataract than senile cataract (Obara, 1995), which explains why S. Cp was significantly higher in diabetic cataract than senile cataract.

Our results were in agreement with the results of Androne et al., (2000) and Metelitsyna, (1998) who found a significant increase of S. Cp in senile cataract and diabetics without cataract compared with the control group. To our mind, there were no previous reports about S. Cp assay in diabetic cataract compared with the control group for comparison contrary to our results Collier et al., (1990) and Telci et al., (2000) reported a non-significant difference in S. Cp concentration between diabetics without cataract and control group.

The obtained data showed a non-significant correlation ($p< 0.05$) between age and serum Cp in senile ($r=0.478$) and diabetic with ($r=0.445$) and without ($r=0.476$) cataract patients (Table 3). This agreed with Metelitsyna, (1998) who explained this by decreased antioxidant defenses with aging.

The present study revealed a positive significant correlation ($p<0.05$) between FSG and serum Cp, (Table 4) in diabetic without cataract ($r=0.649$) and diabetic cataract patients ($r=0.784$). This result may be attributed to the fact that Cp being an acute phase reactant protein that has strong antioxidant activity and increases more with the increase of the oxidative stress associated with increased severity of diabetes mellitus (Metelitsyna, 1998).

Finally, correlation study of serum and aqueous LP, in senile cataract group (Table 5), Showed an inverse significant correlation with serum and aqueous SOD, ($r=-0.719$ & -0.673), respectively while, was correlated non-significantly with serum and aqueous uric acid, S. total bilirubin and S. Cp. In addition, serum LP in diabetics without cataract showed an inverse significant correlation with plasma SOD, ($r=-0.616$) while it was significantly and positively correlated with S. total bilirubin ($r=0.649$) and S. Cp ($r=0.688$). However, S. LP showed a non-significant positive correlation with S. uric acid. Furthermore, serum and aqueous LP in diabetic cataract group showed a significant and inversely correlation with plasma and aqueous SOD,($r=-0.865$&-0.636), respectively, while directly with S. total bilirubin ($r=0.776$) and S. Cp ($r=0.675$). However, it was directly and non-significantly correlated with serum and aqueous uric acid, ($r=0.401$ & 0.408), respectively.

There were no similar reports about the correlation between serum and aqueous LP with plasma and aqueous SOD, serum and aqueous uric acid, S. total bilirubin or S. Cp, but Lian, (1993) reported no significant correlation between MDA content and SOD activity in cataractous lenses and RBCs, but there was a negative correlation between SOD and

MDA in normal human lenses.

Out the results of the current study, it can be concluded that the increased oxidative stress is somewhat responsible for the increase in the risk of occurrence of senile and diabetic cataract. However, O_2 free radicals are more important in the pathogenesis of diabetic cataract as well as senile cataract. The oxidative stress produced by aging and diabetes lead to damage of the genetic material, lipid peroxidation of cell membrane and inactivation of membrane bound enzymes. Accumulation of such oxidants in the lens probably would accelerate the process of cross-linking and polymerization of lens proteins with subsequent cataract formation. So, the imbalance between generation of O_2-free radical and some antioxidants may have an etiological implication in the occurrence of cataract.

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الملخص العربى

عدم الإتزان بين انتشار جذور الأوكسجين الحرة وبعض مضادات الأكسدة فى مرضى المياه البيضاء

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شملت هذه الدراسة على ثلاثين مريضا وعشرة أشخاص أصحاء كمجموعة ضابطة منهم 23 من الذكور و 17 من الأناث ممن تتراوح اعمارهم من 50 الى64 عاما.وقد تم تقسيم المرضى إلى ثلاث مجموعات كل مجموعة عبارة عن عشرة من المرضى.إحتوت المجموعة الأولى على المرضى كبار السن الذين يعانون من مرض المياه البيضاء والمجموعة الثانية على مرضى البوال السكرى ولا يعانون من مرض المياه البيضاء أما المجموعة الثالثة فهم مرضى البوال السكرى والذين يعانون من المياه البيضاء.

وكانت نتيجة البحث وجود زيادة ذات دلالة إحصائية فى مستوى بيروكسيدات الدهون والسيريلوبلازمين بمصل الدم وان هناك نقص ذات دلالة إحصائية فى مستوى أنزيم السوبر أكسيد ديسمبتاز بالبلازما وحمض البوليك بمصل الدم بينما وجد أن هناك زيادة بدون قيمة إحصائية فى مستوى الجلوكوز والصفراء الكلية بمصل الدم وذلك فى مجموعة مرضى كبار السن المصاحب للمياه البيضاء. أما مجموعة مرضى البوال السكرى سواء الذين الذين يعانون أو لايعانون من المياه البيضاء وجد أن هناك زيادة ذات قيمة إحصائية فى مستوى الجلوكوز وبيروكسدات الدهون وحمض البوليك والصفراء الكلية والسيريلوبلازمين بمصل الدم بينما يوجد نقص ذات دلالة إحصائية فى مستوى إنزيم السوبر أكسيد ديسميتاز بالبلازما. وعلى الجانب الآخر فعند قياس مستوى بيروكسيدات الدهون وحمض البوليك فى سائل الغرفة الأمامية فقد وجد أن هناك زيادة فى كليهما بينما يوجد نقص فى انزيم السوبر اكسيد ديسميتاز وذات دلالة إحصائية وذلك فى مرضى البوال السكرى الذين يعانون من المياه البيضاء عند مقارنتهم بالمرضى كبار السن والذين يعانون من المياه البيضاء.

نستخلص من هذا البحث أن زيادة جذور الأكسجين الحرة ونقص بعض مضادات الأكسدة يمكن أن يكون لها دور هام في حدوث المياه البيضاء المرتبطة بالسكر أو وجود مرض البوال السكرى .