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OXIDATIVE STRESS AND BIOCHEMICAL PARAMETERS IN PATIENTS WITH FACIAL BOTOX TREATMENT

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OXIDATIVE STRESS AND BIOCHEMICAL PARAMETERS IN PATIENTS WITH FACIAL BOTOX TREATMENT

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May 2022

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ABSTRACT

OXIDATIVE STRESS AND BIOCHEMICAL PARAMETERS IN PATIENTS WITH FACIAL BOTOX TREATMENT

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Master of Science in Chemistry

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The purpose of this study was to measure the concentration of total antioxidant capacity, malondialdehyde, cholinesterase, glutathione peroxidase, and peroxidase and the potential relationship between the total antioxidant capacity, malondialdehyde, peroxidase, glutathione peroxidase and choline esterase in men and women in individuals undergoing Botox injections with of some complications and study their impact. The results for men showed significant differences for the Serum Choline esterase test as well as for the Serum Total Antioxidant test and Malondiaaldehyde MDA test. Conversely, the Serum Catalase, Thyroid peroxidase (TPO) and Glutathione peroxidase GPX tests had no statistical significance. This suggests that some antioxidant tests are affected by the number and quality of Botox injections in men. As well as for women, the results indicated significant differences in the tests of serum cholinesterase, total antioxidant capacity and serum Malondiaaldehyde MDA, which indicate the effect of their levels when performing Botox injections for women. While there was no significance for the Serum Catalase, Thyroid peroxidase (TPO) and Glutathion peroxidase GPX tests at $P < 0.05$. This issue still needs further studies.

2022, 30 pages

Keywords: Oxidative stress, Total antioxidants capacity, Malodialdehyde, Choline esterase, Botox

ÖZET

YÜZ BOTOKS TEDAVİSİ OLAN HASTALARDA OKSİDATİF STRES VE BİYOKİMYASAL PARAMETRELER

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Bu çalışmanın amacı erkek ve kadınlarda toplam antioksidan kapasite, malondialdehit, kolinesteraz, glutatyon peroksidaz ve peroksidaz konsantrasyonunu ve toplam antioksidan kapasite, malondialdehit, peroksidaz, glutatyon peroksidaz ve kolin esteraz arasındaki potansiyel ilişkiyi ölçmektir. Botox enjeksiyonlarının bazı komplikasyonları ve etkilerinin incelenmesi. Erkekler için sonuçlar, Serum Kolin esteraz testinin yanı sıra Serum Toplam Antioksidan testi ve Malondiaaldehit MDA testi için önemli farklılıklar gösterdi. Tersine, Serum Katalaz, Tiroid peroksidaz (TPO) ve Glutatyon peroksidaz GPX testleri istatistiksel olarak anlamlı değildi. Bu, bazı antioksidan testlerinin erkeklerde Botox enjeksiyonlarının sayısından ve kalitesinden etkilendiğini düşündürmektedir. Kadınlar için olduğu kadar, sonuçlar, serum kolinesteraz, toplam antioksidan kapasite ve serum Malondiaaldehit MDA testlerinde de anlamlı farklılıklar gösterdi; bu, kadınlar için Botox enjeksiyonları yaparken seviyelerinin etkisini gösterir. $P < 0,05$'te Serum Katalaz, Tiroid peroksidaz (TPO) ve Glutatyon peroksidaz GPX testleri için bir anlam bulunmazken. Bu konunun hala daha fazla araştırmaya ihtiyacı var.

2022, 30 sayfa

Anahtar Kelimeler: Oksidatif stres, Toplam antioksidan kapasite, Malondialdehit, Kolin esteraz, Botox

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LIST OF SYMBOLS

%	Percent
**	Significant
/	Divide
+	Plus
<	Greater than
=	Equel
>	Less than
±	Plus-minus
µmol	Mole
g	Gram
IU	Milli-international units
kg	Kilogram
L	Liter
mL	Milliliter
mmol	Milli mole
ng	Nanogram

LIST OF ABBREVIATIONS

BTX	Botulinum toxin
CGRP	Calcitonin gene-related peptide
CM	Chronic migraines
COX	Cyclooxygenases
ETC	Electron transport chain
FRAP	Ferric reducing antioxidant power
LOX	Lipoxygenases
ROS	Reactive oxygen species
SNAP	Synaptosomal-associated protein 25
TMJ	Temporomandibular joint
VAMP	Vesicular-associated membrane protein

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1. INTRODUCTION

Botulinum toxin, sometimes known as the "poison that heals," is a neurotoxin that is released by bacteria belonging to the clostridium botulinum species, the toxin 150 kda is a polypeptide with two light and heavy chains linked by disulfide bonds with non-covalent forces and is sensitive to heat only two types are used for clinical purposes, namely type a and type b out of 7 other types. In 2009, the term botox was defined as a description of botulinum toxin by the us food and drug administration, it has widely uses, in ophthalmology, otolaryngology, dermatology, urology, gynecology and non-surgical reconstructive plastic medicine and treat some neurological problems, botox has also proven its therapeutic efficacy in treating chronic migraines and other headache disorders if new types of botulinum toxin are formed and produced that are more selective for the sensory and nerve cells that cause headache, its therapeutic efficacy will be better uses.

Botox type a is in a wide range of clinical applications, where it reversely inhibits acetylcholine secretions by presynaptic vesicles, as it works to inhibit muscle neurotransmitter release at synapses. Un botulinum toxin, such as overactive bladder and sweat glands, the fda-approved drug formulations for botox are una botulinum toxin, which is diluted 100 μt in 1 to 8 ml of saline and 300 μt in 0.6 to 2.5 μm of saline. Botox requires accuracy and caution because it may cause undesirable results, including eyelid drooping Botulinum toxin causes muscle or gland degeneration because it blocks the cholinergic nerve's release of acetylcholine (Ach) Muscle contraction and glandular secretion are both triggered by the neurotransmitter Ach.

The docking proteins are essential for docking acetylcholine-carrying vesicles to the presynaptic membrane. Because of its endopeptidase activity, Botulinum toxin A cuts the membrane-associated docking protein "synaptosomal-associated protein 25" (SNAP-25), a member of the N-ethylmaleimide-sensitive factor attachment receptor protein family (SNARE protein). This synaptobravin, found in the vesicular-associated membrane protein, is cleaved by the type B botulinum toxins (VAMP). Since docking proteins are disrupted by botulinum toxin, the release of Ach from cholinergic nerve endings in motor

neurons is hampered. As a result of the presynaptic block's functional denervation, muscles begin to atrophy and lose mass. An injection of this type causes a temporary loss of muscle tone, resulting in flaccid paralysis for approximately three to four months, after which a repeat injection is advised.

Regeneration of new axon terminals restores neuromuscular transmission. BTX A's therapeutic effects begin to manifest within the first 1-3 days, peak within the first 1-4 weeks, and diminish within the first 3-4 months of use. In order to counteract this decline, it is recommended to administer botox injections twice a year, with a minimum of three months between injections. Preventing antibody formation to protein prevents subsequent botox treatments from being possible due to this.

2. LITERATURE REVIEW

(Abbruzzese and Berardelli 2006), Botulinum toxin type A is a neurological mechanism of action. Works. By inhibiting the release of acetylcholine from the presynaptic muscle nerve endings, this leads to a temporary paralysis of muscle contraction, as this toxin changes the sensory receptors of the central nervous system, which in turn leads to secondary changes that result in long-acting clinical responses.

(Kroumpouzos et al. 2021), upper face rejuvenation with botulinum toxins has been practiced for quite some time. Using Botulinum toxin on the top face is most commonly used to treat wrinkles. Botulinum toxin side effects are possible in this area, however they are quite rare. The incidence of adverse events was determined to be just 2.6 percent of the time in a research that looked retrospectively at data from 845 patients who had upper face rejuvenation with 18-point Abobotulinum toxinA. The procedure was used to treat wrinkles and fine lines on the face. In this investigation, bruises on the lateral canthal region were shown to be the most common side effect. Blepharoptosis, post-touch-up eyebrow asymmetry, and headache were the additional side effects. Besides brow ptosis and diplopia, botulinum toxin can cause ectropion, lagophthalmos, and xerophthalmia (Amer *et al.* 2021).

Botulinum toxin is a safe treatment for horizontal and vertical neckbands, however side effects are prevalent. Injections of botulinum toxin may cause hoarseness, dysphagia, and neck weakness. Because the muscles beneath the neckbands are involved in phonation, deglutition, and neck flexion, deeper injections or higher doses of the toxin may induce dysphagia, dysarthria, neck paralysis, and xerostomia. Dysphagia and pain during swallowing were observed in 27% and 5% of patients, respectively, in a study of dystonia patients.

Patients over the age of 60 are more likely to experience these side effects, they need a bigger dosage of the poison, and they have less support from the soft tissues in their necks, making it easier for the toxin to reach the deeper neck muscles. Some individuals may have trouble lifting and maintaining their head in an upright position. These side effects

are quite infrequent, and recovery time ranges from 3-4 weeks. Because of the potential for sternocleidomastoid muscle effects from BoTNA, some people experience a floppy neck as a result of the drug.

When Botulinum toxin is used for the treatment of masseter hypertrophy, there is a possibility that it will have an effect on the temporomandibular joint (TMJ), which can result in problems opening the mouth. Reduced masticatory force after masseter injection can produce masticatory fatigue, reduced bite force, reduced masticatory force, impaired masticatory function, and trouble chewing hard meals, However, this often begins to improve anywhere from three to eight weeks after treatment begins and recovers to normal levels by twelve weeks.

(Adel et al. 2022), the purpose of this research was to investigate the effects of repeated injections of botulinum toxin and their long-term consequences in the treatment of excessive gingival display. A customized injection location and dosage were used during the course of this investigation. Twenty female participants were recruited for the research, each of them had hypermobility of the upper lip or gingival show measuring between 4 and 6 mm. These women had gummy smiles when they smiled. All of the patients got Botox injections at different places on their face, depending on the type of smile. We took digital measurements of how the gums looked when we smiled before and after surgery using standardized digital photographs and Adobe Photoshop software. At 14 days, 4 months, 8 months, and 12 months, the patients were checked on again. At the 14-day follow-up after surgery, measurements showed a big improvement and a big drop in the amount of gum showing. Relapse, on the other hand, happened after 4 months and after. Botox is a good treatment for gums that show too much. It works for 2–3 months and almost completely wears off after 4 months. Based on the results of our study, Following two rounds of Botox injections at follow-up intervals of four and eight months, we came to the realization that the effects of these injections may not be lasting, even though they were given twice.

2015 The rise in the production of free radicals, along with a deficiency of antioxidants, causes a condition known as oxidative stress, which may be quantified by determining

the levels of reactive oxygen species that are present, some diseases or abnormal indicators in the lifestyle of the living organism can be determined.

(Arora 2022), chemical denervation, also known as the inhibition of acetylcholine release into the synaptic cleft, is the primary means by which BT exerts its therapeutic effect. After being injected into the skin or the muscle, the heavy chain will attach to receptors that are found on cholinergic nerve terminals. The light chain of the molecule is coupled to the soluble N-ethylmaleimide-sensitive fusion (SNARE complex) protein receptors after it has been endocytosed after its separation from the heavy chain and endocytosis. Synaptobrevin (VAMP), syntaxin, and synaptosome associated protein (SNAP-25) are the three protein receptors that make up the SNARE. Synaptobrevin is cleaved by type B BT, while SNAP 25 is cleaved by type ABT. Because BT binds to these receptor proteins, acetylcholine vesicle attachment to nerve terminals and subsequent release into the synaptic cleft are both inhibited. The sections that follow will provide a description of the impact that BT has on a variety of glands, muscles, and pathways that lead to therapeutic action in dermatological disorders.

(Guida *et al.* 2018), Neurotransmitters such as acetylcholine glutamate,, norepinephrine, calcitonin gene-related peptide [CGRP], and substance P release at the presynaptic neuromuscular junction are the most commonly characterized mechanisms of action of BoNTs. Since acetylcholine is a neurotransmitter in the autonomic nervous system, BoNTs can affect both sympathetic and parasympathetic functions. BoNTs have been shown to affect a wide range of human cell types, including both non-neuronal and neuronal cells. Epidermal keratinocytes, mesenchymal stem cells derived from subcutaneous adipose tissue, neutrophils and macrophages, dermal fibroblast cells, and mast cells are all components of every single one of them. As a result, dermatology is seeing a rise in therapeutic applications.

(Satriyasa 2019), there are four essential steps in the process of Botox working; Presynaptic cell receptors on their surface are targeted by the (C-terminal) of the heavy chain, which then binds to the toxin. Over the course of around 30 minutes, this stage is completed. Next, the receptor-mediated endocytic process known as internalization

occurs. In this stage, the nerve cell plasma membrane invades the toxin-receptor complex to produce a vesicle carrying poison in the nerve terminal, the third step is relocating. Disulfide bonds are broken and the toxin light chain is released into nerve terminal cytoplasm after it has been endocytically split.

Blocking is the final phase in the process. The cytoplasmic protein known as SNAP-25, which is essential for the docking of acetylcholine receptors in nerve terminals on the inner side of the neuronal membrane, may be cleaved by anti-acetylcholine light chains A and E.

Following injection, acetylcholine excretion is aided by the toxin adhering to the presynaptic terminal of the neuromuscular junction. After this, the toxin attaches itself to a specific protein-membrane that is responsible for acetylcholine excretion. This is the last step in the process.

Relaxation of local muscles that is reversible as a consequence of the toxin's quick suppression of the release of acetylcholine at the neuromuscular junction leads to a reduction in the appearance of facial wrinkles and lines, some of which are generated by the constant contraction of facial muscles.

(Lewis *et al.* 2018), when it comes to treating the appearance of glabellar frown lines, injections of the muscle relaxant botulinum toxin (BTX) are a common procedure. This cosmetic procedure has been shown to reduce depression, according to studies. Reduced frowning is less likely to cause negative feedback in patients, which is in line with the theory that emotions are embodied. In the present research, these and three additional hypotheses about the effects of cosmetic (BTX) injections on embodied emotions were investigated to see whether they hold any water. Treatment of crow's feet was predicted to lower mood because patients' Duchenne smiles would be compromised as a result of the treatment. Face BTX treatments were thought to impair recognition of emotional expressions because of the reduction in one's capacity to act out different feelings. In addition, it was expected that following BTX therapy, sexual function would be impaired since the therapies suppress facial expressions that are related with sexual arousal.

Comparisons were made between twenty-four (BTX-treated) individuals and twelve matched participants (all of whom were females) both before and after treatment. According to the findings of the research, receiving (BTX) therapy for laughing lines was associated with an increased risk of developing depression. BTX treatment was also linked to decreased ability to recognize emotions and reduced sexual function. Adding to our understanding of the psychological effects of neurotoxin injections, these new findings expand our understanding of how emotions are manifested.

(Radhakrishnan *et al.* 2022), this study was conducted on mice, similar to our study similar to our study BoNT, a widely used therapeutic agent, prevents the neuromuscular junction from releasing too much acetylcholine. The testicles of experimental mice were shown to be negatively impacted when they were subjected to repeated intracremasteric injections of BoNT as well as a modest overdose of the substance. A modest dosage of BoNT may be used to treat a variety of conditions, including premature aging of the skin, neuromuscular deficiencies, issues with an overactive urine bladder, testicular discomfort, and sexual dysfunction. Even at a low dose and via a distal pathway, it is possible to achieve therapeutic benefits from BoNT on the testis. As a result, we studied the effects of BoNT on the testes, antioxidant levels, and sperm parameters in aging experimental mice using a minuscule dose injected into the vastus lateralis of the thigh muscle. It has been shown that BoNT has an effect on spermatogenesis, which in turn has an effect on the total sperm count and sperm motility in animals injected with 1 U/kg bodyweight of BoNT. This study shows that a low intramuscular dose of BoNT can be used to treat and prevent male infertility.

(Althawadi *et al.* 2022), a prior history of allergy to BT product constituents is the only absolute contraindication. If the skin is infected or has eczema or psoriasis, it should not be injected directly into it. Breastfeeding, contraception, or pregnancy are all examples of "relative contraindications" to using BT injections. Injections into the skin have very low systemic levels of botulinum toxin, and this suggests that it does not cross the placenta. Patients with neurological or neuromuscular disorders (e.g., myasthenia gravis) may experience severe muscle weakness as a result of BT injections. Because of their ability to block the neuromuscular junction, antibiotics such as gentamycin, amikacin,

tobramycin, and neomycin can increase the effects of botulinum toxin. Botulinum toxin antagonists, patients diagnosed with myasthenia gravis are sometimes given drugs, such as acetylcholinesterase inhibitors, which might lessen the effectiveness of other treatments (common medication includes pyridostigmine). A decrease in nerve excitation is caused by the antimalarial drug aminoquinoline, which has an adverse effect on acetylcholine. As a result, it has the potential to increase the impact of (BT) further. Cyclosporin: cyclosporin may enhance the effects of (BT) through an unknown mechanism. These are possible side effects of injection site keloid scar Ptosis occurs when a toxin solution diffuses into the levator muscles of the upper eyelid due to an injection being administered too close to the brow. The injection of the sternocleidomastoid muscle or the major salivary gland can cause dysphagia. This problem may arise as a result of toxin diffusion into nearby muscles. When treating hypersalivation with an injection into the submandibular gland, there is a risk that the toxin solution may seep out of the gland capsule and into the surrounding muscles that are involved in breathing4, causing respiratory embarrassment Toxin-induced facial paralysis - especially when injected into the masseter muscle - causes facial weakness, there is a risk of injury or infection as a result of these symptoms. Most BT injections in this area can be safely administered, But before the treatment can begin, the patient has to have a comprehensive evaluation and their medical history reviewed. The medical notes should include a clear explanation of the risks involved.

(Hamblin *et al.* 2016), when it comes to aesthetic medicine, Botox injections are one of the most common procedures. United Kingdom (UK) government agency, the (MHRA) Medicines and Healthcare Products Regulatory Agency, is responsible for ensuring that medicines are safe to use. Using data from the MHRA's government registry, we compared the reported incidence of facial cosmMigraine transformation's underlying pathophysiological mechanisms are still being debated. Plasma oxidative stress biomarker changes in chronic migraine have been documented. Treatment with onabotulinumtoxinA (BoNT/A), which has been given the green light for the prevention of chronic migraines, has the potential to lower pain neurotransmitters and products of oxidative stress. We intended to investigate whether or not there were variations in the plasmatic oxidative stress indicators advanced oxygenation protein products, ferric

reducing antioxidant power (FRAP), and thiolic groups (SH) between patients who suffer from chronic migraines (CM) and healthy controls (HC). After 6 months of treatment with (BoNT/A), we also examined possible changes in the CM group's clinical and biochemical parameters. The (CM) group started with higher (AOPP) (p 0.001) and lower (SH) (p 0.001) as well as (FRAP) (p = 0.005) values. A decrease in AOPP (p = 0.023) was found in the control group after six months, while increases in FRAP (p = 0.023) and SH (p=0.001) were discovered in the CM group after the same time period. Migraine symptoms were alleviated in the (CM) group after receiving (BoNT/A) treatment. Antioxidant mechanisms were found to be unbalanced in chronic migraineurs, and patients had lower antioxidant capacities than controls, as we had previously reported. An increase in plasma levels of oxidative stress biomarkers showed that BoNT/A is an effective preventative treatment for chronic myeloid leukemia (CM). The potential antioxidant properties of BoNT/A treatment should be examined in future studies.etic BoNT-A injection-related adverse events to that of published retrospective and prospective studies.

(Dini *et al.* 2019), migraine transformation's underlying pathophysiological mechanisms are still being debated. Plasma oxidative stress biomarker changes in chronic migraine have been documented. Treatment with onabotulinumtoxinA (BoNT/A), which has been given the green light for the prevention of chronic migraines, has the potential to lower pain neurotransmitters and products of oxidative stress. We intended to investigate whether or not there were variations in the plasmatic oxidative stress indicators advanced oxygenation protein products, ferric reducing anti-oxidant power (FRAP), and thiolic groups (SH) between patients who suffer from chronic migraines (CM) and healthy controls (HC). Following a course of therapy with BoNT/A lasting for six months, we additionally investigated any potential changes in the clinical and biochemical parameters of the CM group. The CM group started with higher AOPP (p 0.001) and lower SH (p 0.001) and FRAP (p = 0.005) values. A decrease in AOPP (p = 0.023) was found in the control group after six months, while increases in FRAP (p = 0.023) and SH (p=0.001) were discovered in the (CM) group after the same time period. Migraine symptoms were alleviated in the (CM) group after receiving (BoNT/A) treatment. Antioxidant mechanisms were found to be unbalanced in chronic migraineurs, and patients had lower

antioxidant capacities than controls, as we had previously reported. An increase in plasma levels of oxidative stress biomarkers showed that BoNT/A is an effective preventative treatment for chronic myeloid leukemia (CM). In further research, it will be important to investigate whether or not the BoNT/A therapy has any possible antioxidant effects.

(Jakubczyk *et al.* 2020), molecules with an oxygen atom and one or more unpaid red electrons are known as reactive oxygen specie. Free oxygen radicals, such as hydroxyl radicals, hydroperoxyl radicals, singlet oxygens, superoxide anion radicals, and free nitrogen radicals are all included in this category of free radicals. Cell processes such as aerobic respiration and inflammation generate small amounts of ROS, which are concentrated in the hepatocytes and macrophages of the digestive tract. Signaling molecules such as (ROS), reactive oxygen species are made up of ROS. They also cause cell differentiation and apoptosis, which aids in the aging process naturally. Muscle contractions, the regulation of vascular tone, and the determination of the activity of bactericidal and bacteriostatic enzymes are all part of their role. There are many factors that contribute to an increase in the production of free radicals in the body, including long-term stress, intense physical exercise, poor diet and stimulant use. Free radicals are produced and removed from the body in equilibrium under physiological conditions. Oxidative stress, free radical function, and free radical disease were the primary foci of this article's research. Search engines like PubMed and Google Scholar were used in the investigation. Oxygen radicals, oxidative stress, and diseases associated with free radicals were some of the search terms. Damage to the cellular and molecular level is caused by excessive production of free radicals, which contribute to oxidative stress. Damage to proteins (aggregation, denaturation), fats (peroxidation), carbohydrates and nucleotides can be caused by reactive oxygen species in vitro (changes in the DNA structure). Free radical-related illnesses are exacerbated as a result of this change, when the body is under excessive oxidative stress, the cardiovascular, respiratory, and nervous systems are particularly.

(Hajam *et al.* 2022), antioxidants, both enzymatic and non-enzymatic, generate (RONS) through a variety of exogenous processes and endogenous. The condition known as oxidative stress (OS) is caused when there is an unbalance between the generation of

oxidants and their ability to be neutralized, which in turn leads to a variety of diseases, disorders, and the aging process. The loss of function in organs and tissues is one of the hallmarks of aging. As we get older, our bodies produce more reactive oxygen species, which can lead to tissue damage and deformities. Chronic obstructive pulmonary disease, chronic kidney disease, cancer neurodegenerative diseases are just a few of the illnesses caused by OS. OS is prevented from damaging cells, tissues, and organs by different enzymatic and non-enzymatic antioxidants, which are induced by ROS. It is possible that long-term exposure to OS causes a decrease in cell antioxidant status, which can lead to various pathological conditions.

An example of a reactive oxygen species is the combination of superoxide radicals (O_2), hydrogen peroxide radicals (H_2O_2), and hydroxyl radicals (ROS), which is one of the most prevalent kinds of ROS (OH) A low amount of ROS generation and presence inside cells is required for several cellular processes, including protein phosphorylation, the activation of transcription factors, apoptosis, and immune function. For example, proteins, lipids, and nucleic acids are damaged when ROS production increases Many studies have shown that oxidative stress can play a role in the onset and/or progression of various diseases (such as metabolic disorders, cancer, atherosclerosis, and cardiovascular diseases, diabetes,). Under both normal and pathological situations, the mitochondria are the primary source of reactive oxygen species (ROS). This indicates that oxygen may be produced by cellular respiration, the activity of cyclooxygenases (COX) of lipoxygenases (LOX) during the metabolism of arachidonic acid, as well as by endothelial and inflammatory cells. These organelles not only have an innate ROS scavenging capability, but they also have the ability to, but they are also responsible for scavenging ROS that have been produced by mitochondria, but they also have a cellular need for clearing the ROS produced by mitochondria. ROS-induced cellular damage can be prevented by enzymatic components, such as SOD, Cat, and GPx, which protect cells from ROS-induced oxidative stress.

(Rinnerthaler *et al.* 2015), as we age, our skin is subjected to a significant amount of oxidative stress. Extrinsic aging is even more damaging than intrinsic aging. In spite of the fact that the outcomes differ greatly between the dermis and the epidermis, oxidative

stress brought on by UV irradiation is a major contributor to extrinsic aging. ROS are discussed in this review, along with their origins, such as the mitochondrial electron transport chain (ETC), peroxisomal and ER-localized proteins, the Fenton reaction, and enzymes such as cyclooxygenases, lipoxygenases, and xanthine oxidases. More specifically in this chapter, the various mechanisms that protect the body from oxidative stress, such as enzymes such as superoxide dismutases and catalases as well as organic compounds for example tocopherol, L-Ascorbate, and beta carotene as well as (CoQ10) and glutathione are described. Oxidative stress-induced protein, lipid, and DNA modifications are also discussed. We also take a look at the effects of aging on skin. An alteration in the structure of the cornified envelope occurs in old skin in addition to the disruption of the epidermal calcium gradient that goes along with it. As a result of this altered cornified envelope, the epidermis has a decreased ability to resist oxidative stress and a decreased barrier function.

3. MATERIALS AND METHODS

3.1 Materials

3.1.1 Study design

We conducted research in Baghdad/ Iraq for both genders, this study included 120 people and it was divided into four groups, 30 men before Botox injection and 30 after Botox injection, 30 women before Botox injection, and 30 after Botox injection.

3.1.2 Blood sample

Samples were collected from all people by drawing blood from a vein in the arm using a syringe, Disposable Syringe, the volume of blood withdrawn ranges from about (3 -5 mL) from men and women before and after Botox injections. Each drawn blood sample was placed in separating tubes with tight covers containing gel tubes, which isolates the serum as it acts as a separating layer between the serum and the blood. The separation was done using a centrifuge at 408 x g for 10 -5 minutes.

3.1.3 Instruments

Centrifuge (Beckmen Model Tj-6/ Germany), Freezer (Gibson/USA), pH meter (HANNA Instrument,China), Sensitive Balance, Spectrophotometer UV-VIS computer (Cecil, CE10N/ Germany), water bath (Memmert/ Germany) all instruments were used in this study.

3.2 Methods

3.2.1 Measurement of biochemical parameters

Choline esterase: Butyrylthiocholine is hydrolyzed to thiocholine and butyric acid by cholinesterase (SChE). The Equation (3.1) and Equation (3.2) show the interaction for the current study.

$$\text{Butyrylthiocholine} + H_2O \overset{\text{SChE}}{\Longleftrightarrow} \text{Thiocholine} + \text{Butric acid} \quad (3.1)$$

$$2\ \text{Thiocholine} + 2OH + 2\text{Hexacyanoferrate (lll)} \longrightarrow \text{Dithobis (choline)} + H_2O + 2\text{Hexacyanoferrate (ll)} \quad (3.2)$$

At 405 nm, the reduction in absorbance owing to the conversion of Hexacyanoferrate (III) to Hexacyanoferrate (II) is measured, and it is proportional to SChe activity in the material.

Manual procedure: Allow reagents and specimens to reach room temperature. Pipette into a temperature-controlled cuvette with a 1 cm route length:
Reagent; 15 mL; when the temperature reaches 37°C, then add Specimen (25 µL), Mix. Set the timer for 15 minutes. After ninety seconds, take a reading of the initial absorbance at 405 nm. Record the absorbance at intervals of 30 seconds for a total of 90 seconds. Determine the change in absorbance per minute (ΔAbs/min). Calculation: Calculate as the following: With theoretical factor as shown in Equations 3.3, 3.4.

$$\text{IU/L} = (\Delta\text{Abs} / \text{min}) \times 65804 \quad (3.3)$$

$$\mu\text{Kat/L} = (\Delta\text{Abs/min}) \times 1097 \quad (3.4)$$

With serum multicalibrator according to Equation 3.5:

$$\text{SChe Activity} = \frac{(\Delta\text{Abs/min})\text{Assay}}{(\Delta\text{Abs/min})\text{Calibrator}} \times \text{Calibrator Activity} \quad (3.5)$$

3.2.2 Total antioxidant capacity

With the use of the TAC Assay Kit, one may determine how much total antioxidant capacity a sample has. Within a 96-well microtiter plate configuration, samples are compared to a known uric acid standard concentration. After the samples and standards have been diluted with a reaction reagent, the reaction continues for a few minutes after it has begun. A typical 96-well spectrophotometric microplate reader is used to read the reaction at 490 nm.

Procedure: Each specimen and uric acid standard should be tested in triplicate. Each time the experiment is done, a newly constructed standard curve must be utilized.

1. Fill the 96-well microtiter plate with 20 liters of diluted Uric Acid Standards or samples.
2. Pour 180 l of the 1X Reaction Buffer into each well using a multichannel pipette or a plate reader liquid handling tool. Blend well.
3. At 490 nm, read the plate to get an initial absorbance.
4. Pour 50 liters of 1X Copper Ion Reagent into each well to start the reaction. On an orbital shaker, incubate for 5 minutes.
5. To finish the reaction, pour 50 mL of 1X Stop Solution into each well.
6. At 490 nm, read the plate once again.

3.2.3 Malondialdehyde

The concentration of malondialdehyde in serum is measured using a spectrophotometer. It is done by measuring the amount of malondialdehyde, which is one of the main products of lipid peroxidation, and the method relies on the interaction between lipid peroxides, primarily malondialdehyde, and thiobarbituric acid (TBA), which took place in an acidic medium and resulted in a colored product whose intensity of absorption was measured at 532 nm.

Procedure:

1. In a test tube (glass), 1 mL of 17.5 percent, trichloroacetic acid solution and 1 mL of 0.6 percent. Thiobarbituric acid solution are added to 150 mL of serum and mixed with a vortex.
2. In a 15-minute incubation period, place the mixture in boiling water.
3. The mixture was left to cool and 1 mL of, trichloroacetic acid solution of 70% concentration was added to it.
4. Allow for a 20-minute resting period at room temperature.
5. Place the mixture in the centrifuge at 300 rpm for 15 min.
6. At a wavelength of 532 nm, the absorbance was measured with a spectrophotometer.

Calculation: The concentration of Malondialdehyde was estimated based on the Equation 3.6.

$$\text{Malondialdehyde } (\mu\text{mol/L}) \quad \frac{\text{Absorbance of sample}}{\text{E}\circ\text{xL}} \text{X D} \qquad (3.6)$$

Where:

Eo = Extinction coefficient 1.56 x 105 M-1 cm-1

L = light path cm.

D = dilution factor 6.7 x 106

3.2.4 Glutathione peroxidase

The activity of Gpx enzyme was estimated using the colorimetric method and based on the Green and Hill method.

Procedure:

Phenol + 4-Amino antipyrine	1.4 mL
Hydrogen peroxide (0.0017 M)	1.5 mL

The above mixture was prepared at a temperature of (25 °C) for 3 to 4 minutes, the reaction was started by adding (0.1 mol) of serum and then the absorbance was recorded from the time of addition directly at a wavelength of 510 nm and the absorbance was recorded after 5 minutes, the calculation of Gpx enzyme are shown in Equations 3.7, 3.8.

$$GPX(U/L) = \frac{\Delta A/min}{E} \times \frac{Vt}{Vs} \times 100000 \quad (3.7)$$

Where Vt = Total volume, Vs = serum volume, E = 50000

$$\Delta A/min = \frac{\text{Absorbance in 5 min - Absorbance in 0 min}}{5} \quad (3.8)$$

3.2.5 Catalase

The catalase enzyme's activity is calculated using a technique that is based on the catalase enzyme's ability to fractionate the hydrogen peroxide molecule H_2O_2 into H_2O water and O_2 gas, causing the spectrophotometer's optical absorbance to drop, as well as the time the reaction takes place. The blood serum is diluted by taking 0.050 cm^3 of serum, and diluting it with 5 cm^3 of buffer solution, after that we take 2 cm^3 of diluted serum and add 1 cm^3 of hydrogen peroxide solution to it, then we read the absorbance of it with a spectrophotometer after 15 seconds (A1) at the wavelength is 240 nm and using quartz cells, then the second reading (A2) is recorded after 30 seconds.

Calculation: The activity of the catalase enzyme is calculated based on the following Equation 3.9.

$$\text{Catalase k/mL} = 13.8 \log A1/A2 \quad (3.9)$$

3.2.6 Thyroid peroxidase

The human thyroid peroxidase solid-phase sandwich ELISA (enzyme-linked immunosorbent test) is used to determine how much of a target is bound between two

matching antibodies. In the wells of the supplied microplate, a target-specific antibody has been pre-coated. After that, samples, standards, or controls are put to these wells, where they attach to the immobilized (capture) antibody. The second (detector) antibody is added to the sandwich, and then a substrate solution is added, which interacts with the enzyme-antibody-target complex to create a quantifiable signal. The strength of this signal is related to the target concentration in the original material.

3.3 Statistical Analysis

The data is expressed using the mean, standard deviation, minimum, and maximum values. An ANOVA test was used to compare groups. Bonferroni corrections were used in the posthoc analysis. In order to compare three different sets of data that did not follow a normal distribution, the Mann–Whitney test with the Bonferroni adjustment was used. To analyze the differences between groups that had a normal mean, a test called the T-test for normally distributed data was used. Data were analyzed using SPSS. Diagrams will be plotted. $P<0.05$ was considered normal, <P0.01 was seen as very significant, $P<0.001$ was regarded as extremely significant, while $P > 0.05$ was regarded as inconsequential.

4. RESULTS

Before and after receiving Botox injections, we subjected women and men to a battery of chemical testing, with the intention of determining how the treatment affects antioxidants in the body. The study was divided into four groups, women and men, and each group included a before and after. And we will explain the interpretation of the results in this chapter and the men will be before and after the Latox injection, and then the results of the women, as shown in Table 4.1 and Figure 4.1.

4.1 The Results in Men Before and After Botox

Table 4.1 The mean age and others parameters in men patients with botox

GROUP STATISTICS						
	Groups	N	Mean	Std. Deviation	Std. Error Mean	P-value
Age	Grup A: MEN befor botox	30	26.47	5.03699	1.59283	0.057
	Grup B:Men after botox	30	25.63	4.50900	1.42587	
SCE	Grup A: MEN befor botox	30	2.507	1.867392	0.590521	0.019
	Grup B:Men after botox	30	3.595	2.301643	0.727843	
STAC	Grup A: MEN befor botox	30	14.945	5.946259	1.880372	0.021
	Grup B:Men after botox	30	25.091	16.275824	5.146867	
MDA	Grup A: MEN befor botox	30	4.439	1.64953	0.52163	0.681
	Grup B:Men after botox	30	2.970	2.02662	0.64087	
Catalase	Grup A: MEN befor botox	30	95.424	11.581665	3.662444	0.637
	Grup B:Men after botox	30	98.709	6.893350	2.179869	
TPO	Grup A: MEN befor botox	30	7.524	3.574437	1.130336	0.748
	Grup B:Men after botox	30	8.892	3.596478	1.137306	
GP	Grup A: MEN befor botox	30	145.22	31.794011	10.054149	0.039
	Grup B:Men after botox	30	122.40	34.694944	10.971504	

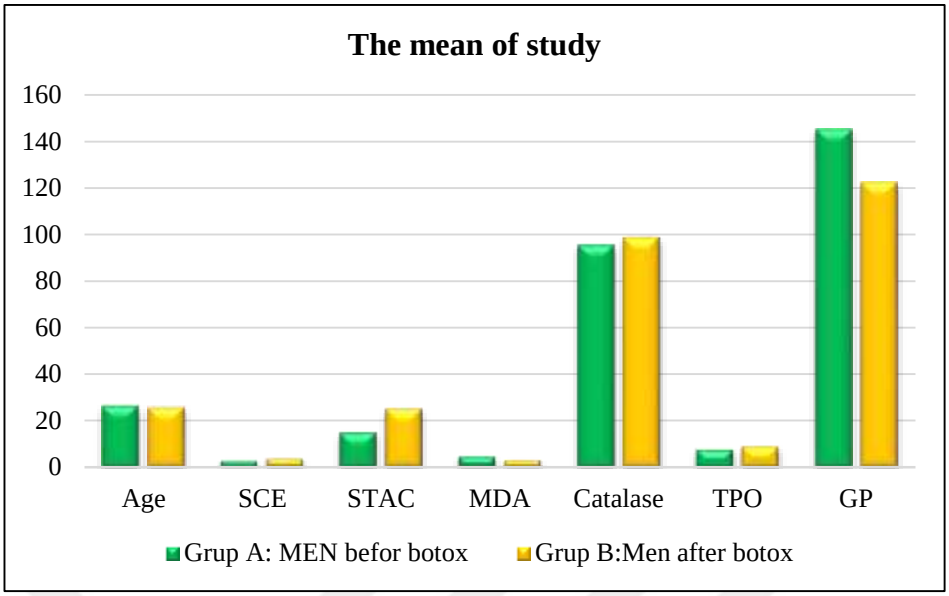

Figure 4.1 The mean age and other parameters in men patients with botox

4.1.1 The age

The indication of results in men before and after Botox injections for age (26.4700 ± 5.03699 and 25.6320 ± 4.50900 years, respectively) at P = 0.057, which indicates that there are minor differences due to the duration of the study. But the results confirm that men who do Botox injections are in their twenties, as shown in Table 4.1 and Figure 4.1.

4.1.2 Serum choline esterase

In men before and after botox injections, the results were (2.50763 ± 1.867392 and 3.59527 ± 2.301643 U/mL, respectively) at P = 0.019, which indicates that there are statistically significant differences that indicate the effect of the serum choline esterase levels when performing botox injections, as shown in Table 4.1 and Figure 4.1.

4.1.3 The serum total antioxidant capacity

Before and after Botox injections in men, the results were (14.94545 ± 5.946259 and 25.09129 ± 16.275824 mmol/1, respectively) at P = 0.021, which indicates that there are

statistically significant differences that indicate the effect of The serum Total antioxidant capacity levels when performing Botox injections, as shown in Table 4.1 and Figure 4.1.

4.1.4 The serum malondiaaldehyde

Also, the results of serum malondiaaldehyde (MDA) in men before and after Botox injections for (4.4393 ± 1.64953 and 2.9704 ± 2.02662 μmol/L, respectively) at P = 0.681, which indicates that there are non-statistically significant differences that indicate the effect of the Serum Malondiaaldehyde capacity levels donot change when performing Botox injections, as shown in Table 4.1 and Figure 4.1.

4.1.5 Glutathion peroxidase

In men before and after Botox injections, the results Glutathion peroxidase were (145.2269 ± 31.794011 and 122.4036 ± 34.694944 U/L, respectively) at P = 0.039, which indicates that there are statistically significant differences that indicate the effect of The Serum Glutathion peroxidase levels when performing Botox injections, as shown in Table 4.1 and Figure 4.1.

4.1.6 Catalase and thyroid peroxidase

Also, the results of Catalase and Thyroid peroxidase in men before and after Botox injections for (95.42474 ± 11.581665, 98.70952 ± 6.893350 mU/L and 7.52460 ± 3.574437, 8.89216 ± 3.596478 IUM/L, respectively) at P = 0.637 and 0.748, which indicates that there are non-statistically significant differences that indicate the catalase and thyroid peroxidase levels don't change when performing botox injections, as shown in Table 4.1 and Figure 4.1.

4.1.7 The Results in Women Before Botox

The results of the effect of Botox injections on antioxidants and some chemical tests in women before and after Botox injections and the Latox injection are shown clearly in Table 4.2 and Figure 4.2.

Table 4.2 The mean age and others parameters in patients women with botox

GROUP STATISTICS						
	Groups	N	Mean	Std. Deviation	Std. Error Mean	P-value
Age	Grup C: Wemon befor botox	30	30.842	4.99216	1.57866	0.051
	Grup D: Wemon after botox	30	31.470	5.03699	1.59283	
SCE	Grup C: Wemon befor botox	30	6.0637	3.168614	1.002004	0.037
	Grup D: Wemon after botox	30	2.5076	1.867392	0.590521	
STAC	Grup C: Wemon befor botox	30	27.928	15.184962	4.801907	0.041
	Grup D: Wemon after botox	30	14.945	5.946259	1.880372	
MDA	Grup C: Wemon befor botox	30	2.5695	1.50611	0.47627	0.026
	Grup D: Wemon after botox	30	4.4393	1.64953	0.52163	
Catalase	Grup C: Wemon befor botox	30	90.358	10.979380	3.471985	0.793
	Grup D: Wemon after botox	30	95.424	11.581665	3.662444	
TPO	Grup C: Wemon befor botox	30	8.3927	3.367141	1.064783	0.682
	Grup D: Wemon after botox	30	7.5246	3.574437	1.130336	
GP	Grup C: Wemon befor botox	30	145.61	34.115761	10.788351	0.851
	Grup D: Wemon after botox	30	143.22	31.794011	10.054149	

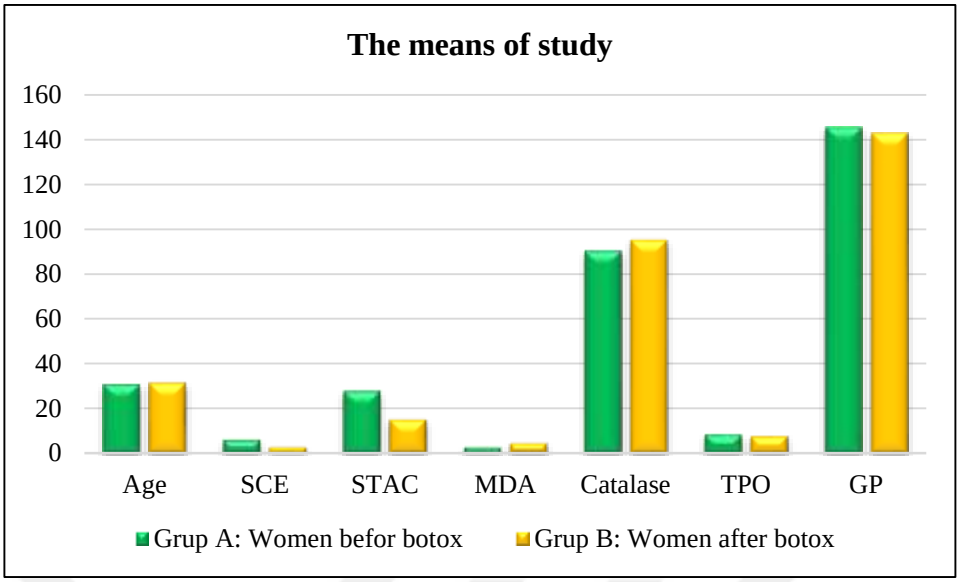

Figure 4.2 The mean age and other parameters in women patients with botox

4.1.8 The age

The results in women before and after Botox injections for age (30.8420 ± 4.99216 and 31.4700 ± 5.03699 years, respectively) at P = 0.051, which indicates that there are minor differences due to the duration of the study. But the results confirm that women who do Botox injections are in their thirties, these results and comparison for the age for women studied groups are shown clearly in Table 4.2 and Figure 4.2.

4.1.9 Serum choline esterase

In women before and after botox injections, the results were (6.06376 ± 3.168614 and 2.50763 ± 1.867392 U/mL, respectively) at P = 0.037, This suggests that there are variations between the groups that are statistically significant and point to the influence of, the serum choline esterase levels when performing Botox injections, these results and comparison for the Botox injections for women studied groups are shown clearly in (Table 4.2) and (Figure 4.2).

4.1.10 The serum total antioxidant capacity

Before and after Botox injections in women, the results were (27.92831 ± 15.184962 and 14.94545 ± 5.946259 mmol/L, respectively) at P = 0.041, It suggests that there are variations between patients that are statistically significant and point to the influence of the serum total antioxidant capacity levels when botox injections are being performed, these results and comparison are shown clearly in Table 4.2 and Figure 4.2.

4.1.11 The serum malondiaaldehyde

The results of serum malondiaaldehyde (MDA) in women before and after Botox injections for (2.5695 ± 1.50611 and 4.4393 ± 1.64953 μmol/L, respectively) at P = 0.026, It implies that there are changes that are statistically significant and that reflect the influence of the serum malondialdehyde capacity levels while doing Botox injections, this also suggests that the differences, these results and comparison are shown clearly in Table 4.2 and Figure 4.2.

4.1.12 Glutathion peroxidase

In women before and after Botox injections, the results Glutathion peroxidase were (145.6130 ± 34.115761 and 143.2269 ± 31.794011 U/L, respectively) at P = 0.581, which indicates that there are statistically non-significant differences that indicate the donot found any change in Glutathion peroxidase levels when performing Botox injections, these results and comparison for the glutathion peroxidase for the women are shown clearly in Table 4.2 and Figure 4.2.

4.1.13 Catalase and thyroid peroxidase

Also, the results of Catalase and Thyroid peroxidase in women before and after Botox injections for (90.35874 ± 10.979380, 95.42474 ± 11.581665 mU/L and 8.39272 ± 3.367141, 7.52460 ± 3.574437 IU/mL, respectively) at P = 0.793 and 0.682, which

indicates that there are non-statistically significant differences that indicate the Catalase and Thyroid peroxidase levels donot change when performing Botox injections, these results and comparison for the Catalase and Thyroid peroxidase in women before and after Botox injections are shown clearly in Table 4.2 and Figure 4.2.

4.2 The Correlation between Catalase and Studied Parametres

When Pearson's test was conducted to find out the correlations between catalase and the result of the parameters, there was a correlation between catalase with age and MDA at P = 0.001 and P = 0.025. While there was no correlation with the rest of the tests, the correlation between catalase and all studied parameters are shown clearly below in (Table 4.3).

Table 4.3 The correlation between catalase and all studied parameters

CORRELATIONS			**RESULTS**
Catalase	Age	Pearson Correlation	0.504**
		Sig. (2-tailed)	0.001
		N	40
	SCE	Pearson Correlation	-0.720
		Sig. (2-tailed)	0.048
		N	40
	STAC	Pearson Correlation	-0.274
		Sig. (2-tailed)	0.088
		N	40
	MDA	Pearson Correlation	0.880*
		Sig. (2-tailed)	0.025
		N	40
	TPO	Pearson Correlation	-0.162
		Sig. (2-tailed)	0.317
		N	40
	GP	Pearson Correlation	0.093
		Sig. (2-tailed)	0.568
		N	40

5. DISCUSSION AND CONCLUSION

The aim of the study was to evaluate some antioxidants before and after Botox injections in men and women. And the results for men indicated that there were statistically significant differences for test Serum Choline estrase, as well as for test Serum Total antioxidant capacity and Serum Malondiaaldehyde MDA. On the contrary, tests Serum Catalase, Thyroid peroxidase (TPO) and Glutathion peroxidase GPX had no statistical significance. This indicates that some antioxidant tests are affected by the number and quality of botox injections in men. As well as for women, According to the findings, there were differences between the tests that were statistically significant in the cases of serum choline estrase. Serum Total antioxidant capacity and Serum Malondiaaldehyde MDA, which indicate the effect of their levels when Botox injections were made for women. While there was no significance for tests Serum Catalase, Thyroid peroxidase (TPO) and Glutathion peroxidase GPX at a P less than 0.05.

A comparison of the TAC's pre- and post-exposures to the magnetic field revealed a statistically significant increase in the TAC's post-exposures ($p < 0.05$). Both (OSI) and (TOS) demonstrated a statistically significant reduction in post-exposures as compared to pre-exposures to a magnetic field of (1.5 T) (for each of the two, $p < 0.01$). The 1.5 T static magnetic field that was employed in the (MRI) machine did not generate a detrimental impact; on the contrary, it had the good effect of lowering oxidative stress in males after just a brief period of exposure to the field (Sirmatel *et al.* 2007). In the blood of patients with non-metastatic disease, changes in the levels of total antioxidant capacity (TAC), uric acid, nitric oxide, cup-per, malon-dialdehyde, and iron were determined. In comparison to the control group, a significant decrease in (TAC) (32.7 percent), uric acid (28.1 percent -49.2 percent), MDA (20.7 percent -25.2 percent), and nitric oxide (50.4 percent -61.9 percent) was found in both groups of patients. The control group had a decrease in all four of these factors as well (Omar *et al.* 2011).

Thyroid peroxidase, often known as TPO, is the enzyme that plays a role in the production of thyroid hormone. In vitro studies have shown that arsenic trioxide, sometimes known as AS_2O_3, may suppress TPO activity. It is assumed that this inhibition takes place when

AS_2O_3 binds to the free sulfhydryl groups found in TPO. In vitro studies have shown that reduced glutathione, often known as GSH, may block TPO activity. This suppression might be the result of GSH acting as a competitive substrate for hydrogen peroxide, or it could be the result of GSH reducing the oxidized form of iodide, both of which are necessary for TPO activity. (Palazzolo and Ely 2015).

REFERENCE

Abbruzzese, G. and Berardelli, A. 2006. Neurophysiological effects of botulinum toxin type A. Neurotoxicity Research., 9(2): 109-114.

Adel, N. 2022. A Standardized Technique for Gummy Smile Treatment Using Repeated Botulinum Toxins: A 1-year Follow-up Study. Plastic and Reconstructive Surgery Global Open., 10(4).

Althawadi, N., Ujam, A. and Visavadia, B. 2022. Botox hidden dangers. British Dental Journal., 232(4): 192-193.

Amer, A., Amer, M. and Nofal, H. 2021. Botulinum Toxin for the Face. In Cosmetic Surgery. IntechOpen.

Arora, G. 2022. Botulinum toxin beyond aesthetics in dermatology. Cosmoderma., 2.

Dini, E., Mazzucchi, S., De Luca, C., Cafalli, M., Chico, L., Lo Gerfo, A. and Gori, S. 2019. Plasma levels of oxidative stress markers, before and after BoNT/A Treatment, in chronic migraine. Toxins., 11(10): 608.

Guida, S., Farnetani, F., Nisticò, S. P., Mariarosaria, C. G., Babino, G., Pellacani, G. and Fulgione, E. 2018. New trends in botulinum toxin use in dermatology. Dermatology Practical & Conceptual., 8(4): 277.

Hajam, Y. A., Rani, R., Ganie, S. Y., Sheikh, T. A., Javaid, D., Qadri, S. S. and Reshi, M. S. 2022. Oxidative Stress in Human Pathology and Aging: Molecular Mechanisms and Perspectives. Cells., 11(3): 552.

Hamblin, M. R. 2016. Shining light on the head: photobiomodulation for brain disorders. BBA Clinical., 6: 113-124.

Jakubczyk, K., Dec, K., Kałduńska, J., Kawczuga, D., Kochman, J. and Janda, K. 2020. Reactive oxygen species-sources, functions, oxidative damage. Polski merkuriusz lekarski: Organ Polskiego Towarzystwa Lekarskiego., 48(284): 124-127.

Kroumpouzos, G., Kassir, M., Gupta, M., Patil, A. and Goldust, M. 2021. Complications of botulinum toxin A: an update review. Journal of Cosmetic Dermatology., 20(6): 1585-1590.

Lewis, M. B. 2018. The interactions between botulinum-toxin-based facial treatments and embodied emotions. Scientific reports., 8(1), 1-10.

Omar, M. E., A. S., Eman, R, Y. and Hafez F, H. 2011. The antioxidant status of the plasma in patients with breast cancer undergoing chemotherapy. Open Journal of Molecular and Integrative Physiology., 1(3): 29-35

Palazzolo, D. L. and Ely, E. A. 2015. Arsenic trioxide and reduced glutathione act synergistically to augment inhibition of thyroid peroxidase activity in vitro. Biological Trace Element Research., 165(1): 110-117.

Radhakrishnan, R. K., Ravichandran, S., Sukesh, A., Kadalmani, B., & Kandasamy, M. 2022. Single injection of very mild dose botulinum toxin in the vastus lateralis improves testicular spermatogenesis and sperm motility in ageing experimental mice. Laboratory Animal Research., 38(1), 1-11.

Rinnerthaler, M., Bischof, J., Streubel, M. K., Trost, A. and Richter, K. 2015. Oxidative stress in aging human skin. Biomolecules., 5(2): 545-589.

Satriyasa, B. K. 2019. Botulinum toxin (Botox) A for reducing the appearance of facial wrinkles: a literature review of clinical use and pharmacological aspect. Clinical, Cosmetic and İnvestigational Dermatology., 12: 223.

Sirmatel, Ö., Sert, C., Sirmatel, F., Selek, S. and Yokus, B. 2007. Total antioxidant capacity, total oxidant status and oxidative stress index in the men exposed to 1.5 T static magnetic field. General Physiology and Biophysics., 26(2): 86.

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