BOTOX (XEOMIN®): CLOSTRIDIUM BOTULINIUM NEUROTOXIN TYPE A 150 KD INJECTION FOR BILATERAL ESSENTIAL BLEPHAROSPASM : PORT HARCOURT EXPERIENCE

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ABSTRACT

BACKGROUND: Blepharospasm is a focal dystonia manifested by repetitive spasms of eyelid muscles which results in involuntary eye closure and often accompanied by sustained contractions of other facial and neck muscle. It is usually progressive and causes significant disability. It is commoner in females and prevalence increases with advancing age. Though distressing, it is successfully treated with botulinium toxin injection

CASE PRESENTATION: We present two cases of blepharospasm. Both patients had prior history of eye conditions requiring surgery. One had cataract extraction while the other had corneal transplant surgery prior to onset of spasm. The two conditions were successfully treated with botulinium toxin injection although the second patient had his dose of injection repeated and doubled before he responded to treatment.

CONCLUSION: Although blepharospasm is distressing and leads to functional blindness, it still has a high curative rate in its response to botulinium toxin injection even in African subjects.

NigerJmed2019: 331- 333

INTRODUCTION

Blepharospasm is a focal dystonia of the orbicularis oculi muscles. It may occur in isolation, or be associated with other dystonias such as Parkinsonism, dystonic cerebral palsy, and tardive phenomena. It is usually bilateral and is characterized by involuntary closure of the eyelids .[1] It is usually progressive and could cause significant disability. Blepharospasm often begins as excessive blinking, usually accompanied by feelings of dryness or irritation of the eyes which can progress into clonic and finally sustained tonic eyelid closure that can interfere with daily activities.[2] Blepharospasm can be disabling to the point of rendering a person functionally blind if left untreated.[3]

The pathophysiology of blepharospasm is thought to involve a combination of loss of inhibition within the sensorimotor circuitry, disrupted sensorimotor integration, and maladaptive homeostatic plasticity. The interaction between cholinergic and dopaminergic transmission allows excessive unwanted movement resulting in the clinical manifestation of dystonia, abnormal plasticity, and sensory dysfunction.[3] Studies using transcranial magnetic stimulation (TMS) in dystonia show exaggerated cortical excitability,[4] which supports the role of abnormal plasticity.

Symptoms are more likely to spread beyond the orbicularis oculi in patients who are women, have an older age at onset, or have a history of significant head or facial trauma.[5]
Men with primary blepharospasm tend to develop symptoms at an earlier age.[3]
The risk of developing blepharospasm increases with age .[6] One age-specific estimate showed a prevalence of 26.6 per 100,000 in people aged 50–69, 31.9 per 100,000 in people aged 60–69, and 74 per 100,000 in people over age 69 .[7] Possible risk factors for the development of primary adult-onset dystonia include family history of dystonia, postural tremor and prior significant head or facial trauma, while hypertension and smoking cigarettes appear to be protective.[5] For blepharospasm specifically, prior history of eye disease is a possible risk factor, while drinking coffee may be protective.[8]

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Adequate treatment of blepharospasm is necessary to maintain quality of life, avoid functional blindness, and prevent complications such as corneal abrasions and dermatochalasis.[3]Botulinum toxin (BoNT) is the most commonly used therapy for blepharospasm. It is produced by the bacteria *Clostridium botulinum*. It causes flaccid paralysis by blocking the release of acetylcholine at the neuromuscular junction.[9,10] Intra muscularly , 2.5-10 iu is given every 3-6 months with some exceptions where it could last longer.The expected response rate to BoNT is very high. In a retrospective study, sustained benefit of about 2 years was seen in up to 92% of the patients .Usually patients that receive BoNT injections can expect long-term efficacy of 3-6 months. Loss of effectiveness is rare, .[11]

CASES 1. MS

A 65 year old male who presented with inability to open both eyes due to spastic contraction of the lids for more than 2 months. He reported that the condition followed bilateral cataract surgery. Any other history that we need to know? NO

Examination finding showed he had spastic lid closure which is consistent with bilateral blepharospasm .

5IU of Botox (Xeomin[R] Clostridium Botulinium neurotoxin type A 150 Kd) was injected into each lid and adjourning face at eight standard points following standard procedures. Two points each on the upper and lower lids, two points above the brow, a point each at the lateral canthus and the glabella.

The patient was reviewed 2 weeks later. He could now open the eyes freely with minimal residual spasm still observed. Three months post injection the patient was still doing well evidenced by ability to open the eyes, sustain it and with improved quality of life.

CASE 2. OG

75 years old man presented with inability to open both eyes as a result of sustained contraction of the lids for more than 2 years following bilateral corneal transplantation surgery.

A working diagnosis of bilateral essential blepharospasm was made and 5iu of Botox ((XeominR Clostridium Botulinium neurotoxin type A 150 Kd)) injections administered to eight marked out points on the lids and adjourning face using standard procedure These included Two points each on the upper and lower lids, two points above the brow, a point each at the lateral canthus and the glabella.A dose of 5iu at each point making a total of 40iu. The patient was reviewed two weeks and one month later with no improvement in the condition. A repeat injection was given one month from the first and patient was reviewed again 2 weeks later with condition remaining the same. The dose was doubled six months later to 10iu per point and patient reviewed a week later with dramatic response.

DISCUSSION

Blepharospasm is a focal dystonia of the orbicularis oculi muscles, .It is usually bilateral and is characterized by involuntary closure of the eyelids just as in our patients.[1] It is usually progressive and can cause significant disability as seen in the cases presented.

The risk factors for development of blepharospasm includes presence of eye diseases[8] and/ or head or face trauma. This could be the trigger in our patients who respectively had cataract and also corneal pathology that could have necessitated a corneal transplant surgery. Furthermore surgical trauma itself could be the predisposing factor to the spasm since it involved an organ in the face/head region.[5] Onset of symptoms were six month after the surgeries.

The risk of developing blepharospasm increases with age [6] which was the case in our patients. One age-specific estimate showed a prevalence 31.9 per 100,000 in people aged 60–69, and 74 per 100,00 in people over age 69.[7]This corresponds to the age group of our patients who were respectively 65 and 75 years.

The first patient recovered after an initial dose of botox. This is in keeping with finding in a study which showed a 92% success rate following botox injection.[10]The second patient recovered after the initial dose was doubled. There was no recorded side effects in both patient even though studies have documented associated side effects with botox.[12]The advanced age of patients is also a risk factor for hypertension which incidentally is protective for blepharospam.[5]This could be a positive factor in preventing recurrence although our patients were not hypertensive.

CONCLUSION

Eye diseases and/or surgery involving organ(s) in the head region could be risk factors for

blepharospasm. However despite being a distressing condition, it is well managed with botulinium toxin injection although more cases would have been needed to substantiate this claim.

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