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RESEARCH ARTICLE

CASE REPORT OF DIETHYL CARBAMAZINE INDUCED ANTERIOR UVEITIS WITH RAISED INTRAOCULAR PRESSURE IN KNOWN CASE OF LYMPHATIC FILARIASIS

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Abstract

Drug induced uveitis (DIU) is relatively rare entity .Diagnosis of DIU is a challenge as no diagnostic test will help in diagnosis and is not necessary for the drug to cause uveitis in all patients who receive it .Time between medication used and occurrence of symptoms varies ranging from few days to months .Drug may be administered through any route including systemic ,topical and intravitreal .Cause of drug induced inflammation is not well known. Diethyl carbamazine(DEC)therapy for filariasis frequently results in systemic and ocular complications but pathogenesis of such complications is unclear.A case diagnosed with filariasis is presented here . complains of Redness, ocular pain and blurred vision LE , appeared after DEC treatment and patient visited the ophthalmology OPD ,where she was diagnosed with acute anterior uveitis and raised intraocular pressures .Patient was treated with topical antibiotic , topical corticosteroids , cycloplegics , antiglaucoma medication and DEC was discontinued, which immediately improved patient presenting complains. Therefore where ocular inflammatory disease exists drug induced uveitis must be ruled out as possible cause.

Introduction:-

Diethyl carbamazine is powerful microfilaricide drug used as second line of treatment for filarial infections it was widely used prior to introduction of ivermectin.DEC causes death of microfilaria including those that are present within the eye leading to an intense inflammatory reaction known as mazzotti reaction[1-2]. ocular complications require timely diagnosis and early treatment

Case Report :

A 54 year old female diagnosed with Lymphatic Filariasis LE. Treatment with Diethyl carbamazine was initiated .After two days of DEC treatment patient began to experience redness ,ocular pain and blurred vision for which she visited ophthalmology OPD ,where she was diagnosed with acute anterior uveitis(AAU) and raised intraocular pressure (IOP) .She had best corrected visual acuity of 20/20 in RE and 20/200 in left eye and left eye showed keratic precipitates ,microcystic edema and Tyndall ++,IOP with Goldman applanation tonometry Right eye -18mm Hg and Left eye 44 mm Hg with no hypopyon .posterior segment findings was within normal limits on fundoscopy.

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Treatment with prednisolone eyedrops ,cylopentolate , timolol eye drops were started. Ophthalmologist expert in uveitis considered DEC to be the primary cause of ocular inflammation since the mechanism of action of the drug can induce ocular inflammation and there are similar cases reported in literature. They discontinued the treatment with DEC and was replaced with Ivermectin . after this Examination & treatment plan was set up which consisted of reducing the topical steroids until their elimination .Patient was reassessed after 2 days with decreased intraocular pressure left eye to 18 mm Hg and follow up at one week with following outcomes 20/20 visual acuity both eyes ,no tyndall and single keratic precipitate were seen and there was tremendous symptomatic improvement in left eye .Patient was advised repeated checkups every month with ophthalmologist .Moreover after stoppage of DEC, no similar episodes have been observed in this patient.

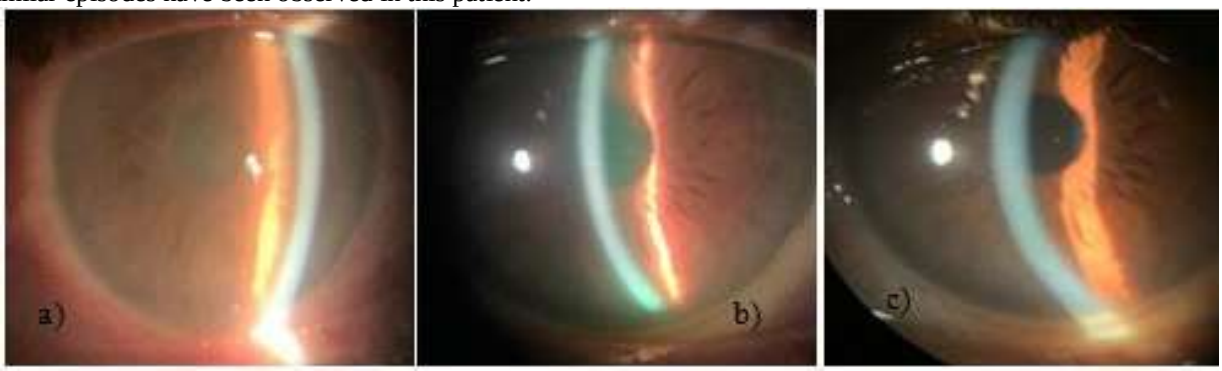

Figure 1:- a) Left eye acute anterior uveitis and raised intraocular pressures with hazy cornea with microcystic edema and fine pigmented Keratic precipitates (presentation- 2 days after initiation of DEC therapy)
b) decreased intraocular pressures and corneal edema subsided (2 days after stoppage of DEC and ocular treatment)
c)Nokeratic precipitate Seen , clear cornea and normal intraocular pressures seen (1 week after ocular management.)

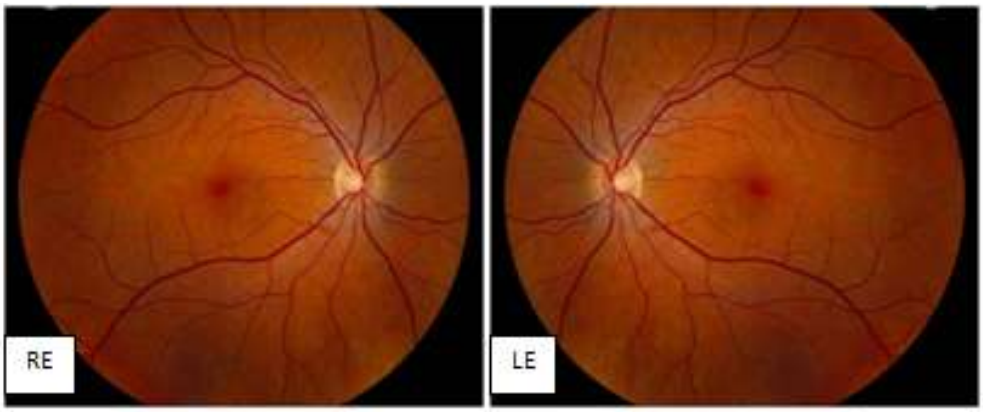

Figure 2:- Both eyes fundus within normal limits.

Discussion:-

Drug-induced uveitis is claimed to account for less than 0.5% of cases in a tertiary referral uveitis clinic, despite the fact that autoimmune disorders and infections still account for the majority of occurrences of uveitis[3]. Proteinuria, severe pruritus, night blindness, punctate keratitis, visual field constriction, anterior uveitis, transitory retinal pigment epithelial lesions, chorioretinitis, and optic nerve inflammation have all been reported as serious systemic and ocular problems[4-6]. Although both toxic and inflammatory responses have been proposed as potential contributors, the mechanism(s) behind drug-induced uveitis are mostly unknown[7-9]. There is no clear explanation for how drugs can produce inflammation. Mechanisms that are postulated both directly and indirectly. Usually with topical or intracamerally implanted medicines, direct mechanisms are observed shortly after pharmaceutical usage. A change in melanin's capacity to scavenge free radicals is one example of an indirect method. Other examples include immune complex deposition in uveal tissues, immunological responses to antigens released after antibiotic-induced microorganism death, or immune reactions to antigens[7] Taylor and .et.al demonstrated that ivermectin and DEC have comparable efficacy at reducing intraocular microfilariae in a double-masked, placebo-controlled

prospective experiment, with ivermectin causing none of the ocular inflammatory side effects[10]. Therefore, the uveitis linked to DEC cannot be explained by the Mazzotti reaction alone. DEC may be a potential contributor to drug-induced uveitis due to the lack of data on dosage or rechallenge.

In the late 1980s, Naranjo et al [11] proposed a set of criteria to establish a causality of adverse drug effects-(Noted that not all these criteria have to be fulfilled.)

1. The reaction is frequently described and documented.
2. Recovery of symptoms occurs when the drug is tapered or discontinued.
3. Other causes for symptoms have been excluded.
4. Symptoms worsen when the dose of the drug is increased.
5. The adverse event is documented by objective evidence.
6. Similar effects occur in a patient with similar drugs.
7. Symptoms recur with re-challenge of the suspected drug

Using an algorithm originally proposed by Naranjo[11] and et.al., it was quantitatively described that there is probable (Naranjo scores of 5 to 8) association of Diethyl carbamazine to uveitis. Drug-induced uveitis typically resolves within weeks with discontinuation of the offending agent and treatment of the ocular inflammation. In our case, the withdrawal of Diethylcarbamazine was definitive and treatment with topical steroids , cycloplegics and antiglaucoma medication was necessary to completely solve the problem.

Conclusions:-

A wide range of topical, systemic, and intraocular drugs have been shown to cause drug-induced uveitis. Therefore, all patients with uveitis that is otherwise unexplained should have a thorough drug history obtained. Drugs suspected of producing uveitis may need to be reduced or stopped when the inflammation is either severe or prolonged, even though drug-induced anterior uveitis normally responds to extensive topical corticosteroid and cycloplegic/ mydriatic therapy. With the help of this case, we are able to emphasize how crucial it is to eliminate therapy as a potential reason when an ocular inflammatory illness manifests.

Declaration Of Patient Consent

The authors attest that they have all necessary patient consent forms. The patient(s) has(have) indicated in the form that he/she/they are/are willing for his/her/their clinical imaging and other information to be reported in the journal. The patients are aware that while every attempt will be made to keep their identities hidden and their names and initials kept confidential, anonymity cannot be guaranteed.

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Conflicts Of Interest –

Nil.

There are no conflicts of interest.

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