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Allopurinol-Induced Toxic Optic Neuropathy: An Unusual Cause

I.ER-RACHIQ, I.Elboukhani, S.Houmaid ,A.Mchachi , L.Benhmidoune ,A.Chakib , R.Rachid , M.Elbelhadji

Department of Adult ophthalmology , Faculty of Medicine and Pharmacy , Hassan II University-Casablanca , Hospital 20 August 1953 , IBN ROCHD UHC , Morocco

Abstract

Purpose .*To report a rare toxic optic neuropathy due to allopurinol*

Case report.*A 47 year old women with history of hyperuricemia treated with allopurinole for one month, thyroidectomized 10 months ago for a papillary carcinoma having received an iratherapy and who is currently on replacement therapy. She presented with complaints of severely decreased vision in both eyes. Her symptoms started 24 hours earlier and had gradually worsened. Entering acuities measured 3/10 in both eyes. There was no improvement in acuities with pinhole or manifest refraction in either eye. Her pupils were round,equal , and reactive to direct and consensual light. There was no afferent pupillary defect in either eye. Slit lamp examination revealed a disc edema stage 1 in the both eyes. Neurological examination revealed no signs of intracranial hypertension, meningeal syndrome or cranial pair involvement. Cerebral scan with cerebral angio-MRI turned out to be normal. Lumbar puncture with monomeric study and CSF analysis proved normal. The infective and inflammatory results also turned out to be normal. The visual field objectified an absolute deficit at the level of paracentral and peripheral temporal visual field in the both eyes. The evolution was marked by the occurrence of a generalized macular rash associated with facial edema in a context of deterioration of the general state. The diagnosis of drug toxicities was retained at the dermatological emergencies where they indicat to stop taking allupurinol, and we retained the diagnosis of toxic optic neuropathy to allopurinole with maintenance of the stopping of the allopurinole.*

Conclusion. *If the suspected diagnosis is drug-related toxic optic neuropathy, and the withdrawal of one medication does not lead to visual recovery or there is a further deterioration of vision, the possibility of toxicity because of other medications should be considered.*

Keywords*: Allopurinol, toxic optic neuropathy, visual field, ocular side-effects*

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I. Introduction :

Opticneuropathyrefers to injury to the opticnerve.Commonmechanisms of opticneuropathyinclud compressive, inflammatory and ischémicinsults to the optic nerve [1].Toxicopticneuropathyis a sideeffect of somemedication. Patient education about possible sideeffectsis crucial in prompt diagnosis of drugrelatedtoxicoptic neuropathies and discontinuation of the causative agent to increase the chance of vision recovery[2].

We report whatis to ourknowledge the first case of allopurinol toxicopticneuropathyoccuring in a patient treated for hyperuricemia.

II. Case Report :

A 47 yearoldwomenwithhistory of hyperuricemiatreatedwithallopurinole for one month, thyroidectomized 10 monthsago for a papillarycarcinomahavingreceived an iratherapy and whoiscurrently on replacement therapy. She presented to the ophthalmological emergencies with complaints of severelydecreased vision in botheyes. Hersymptomsstarted 24 hoursearlier and hadgraduallyworsened. Shedeniedanyheadache,recent trauma or pain.

Enteringacuitiesmeasuredwas about 3/10 in bothbothseyes. There was no improvement in acuities with pinhole or manifest refraction in either eye. Herpupilswereround,equal , and reactive to direct and consensual light. There was no afferentpupillarydefect in eithereye.Extraocular muscles demonstrated full motility in both eyes , with no pain or diplopia on eye movement.

Slitlampexaminationrevealedmildinferiorpunctuateepithelialerosions on bothcorneas ,cappedmeibomian glands in botheyes , brown and flat irides ans nuclearcataracts in the both eyes.Intraoccular pressures by goldmann tonometry measured 14mmhg OD and OS.Thevitrouswasclear and quiet.Retinalvasculaturewas normal. The fovea was normal but in the optic disc we noted a cup to disc ratio

equal to 0,4 in OD and OS and a disc edema stage 1 in the both eyes. Neurological examination revealed no signs of intracranial hypertension, meningeal syndrome or cranial pair involvement. The general examination revealed a deterioration in the general condition classified OMS3, hemodinamycic and respiratorystability and body mass index estimated at 35kg / m2. We did a cerebral scan with cerebral angio-MRI which turned out to be normal. Lumbar puncture with monomeric study and CSF analysis proved normal. The infective and inflammatory results also turned out to be normal.
The retinalangiography made confirmed the bilateralpapillaryedema and the visualfieldobjectified an absolutedeficit at the level of paracentral and peripheral temporal visualfield in the botheyes.

The evolution was marked by the occurrence of a generalized macular rash associated with facial edema in a context of deterioration of the general state.

The diagnosis of drugtoxicosiswasretained at the dermatological emergencies wheretheyindicat to stop takingallupurinol, and weretained the diagnosis of toxicopticneuropathy to allopurinolewith maintenance of the stopping of the allopurinole.

The evolution was marked after 4 weeks by a spontaneous recovery of a visual acuity estimated at 10/10 in the both eyes with regression of the papillary edema but a persistence of a slight pallor.However, thevisualfieldsobjectified a persistence of the campimetricdeficit.

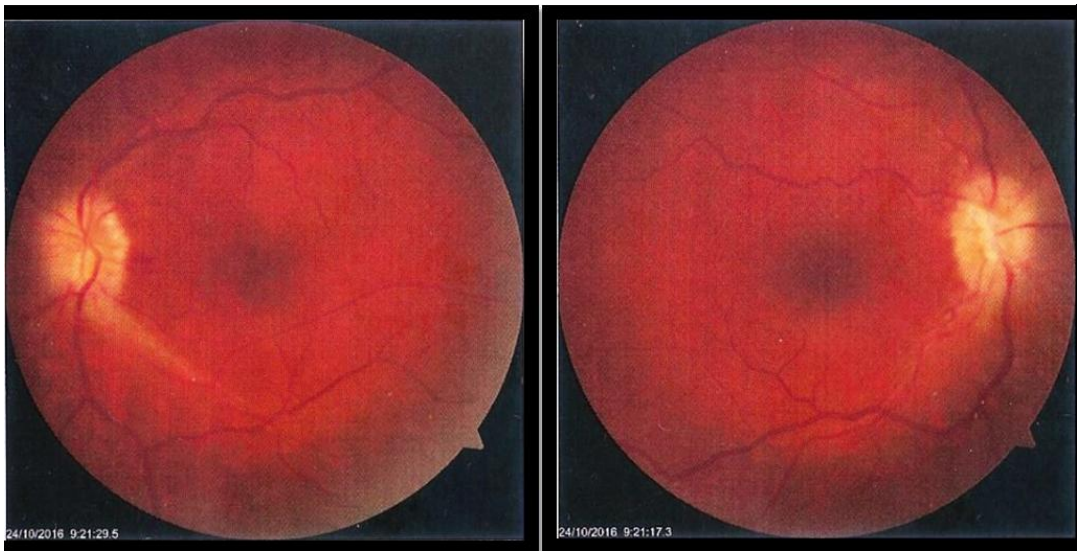

Fig 1 : Stereo photos demonsrtrating a disc edema in the OS and OD

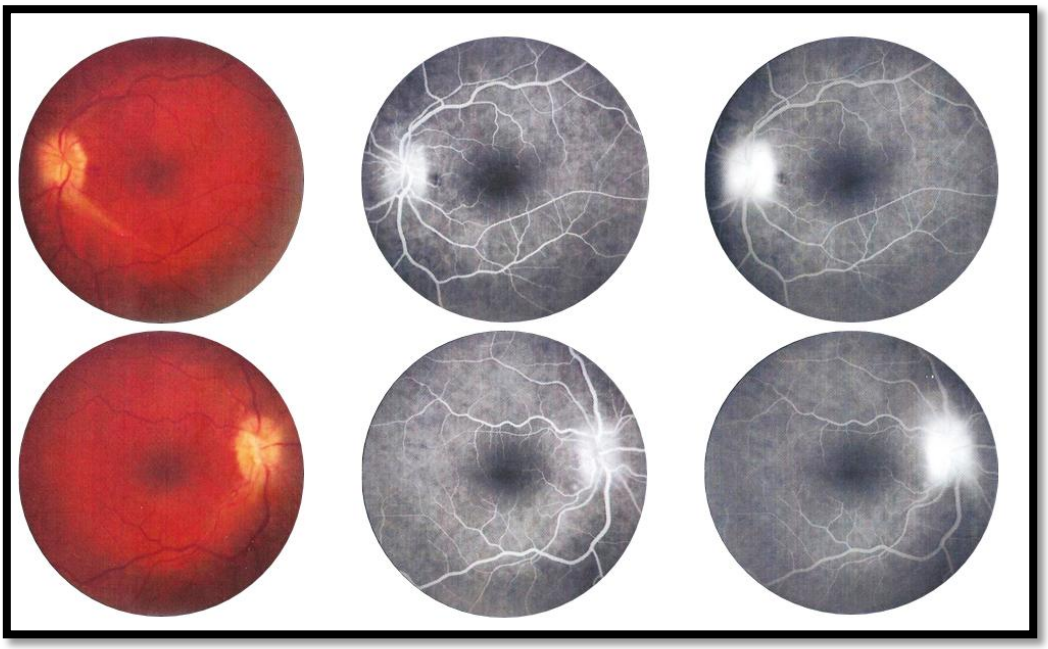

Fig2 : Retinalangiography made confirmed the bilateralpapillaryedema

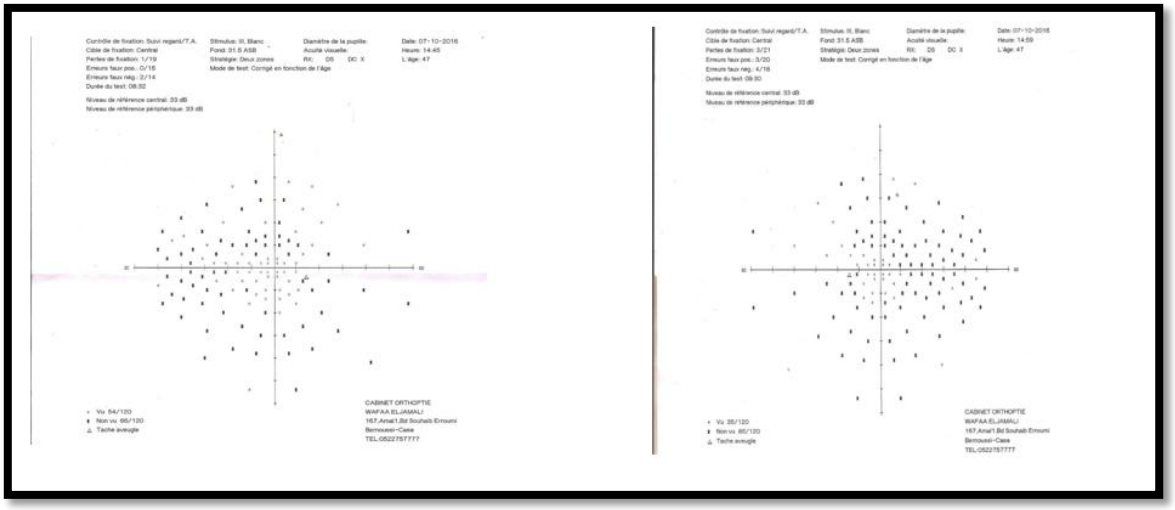

Fig3 : Absolutedeficit at the level of paracentral and peripheral temporal visualfield in OD and OS

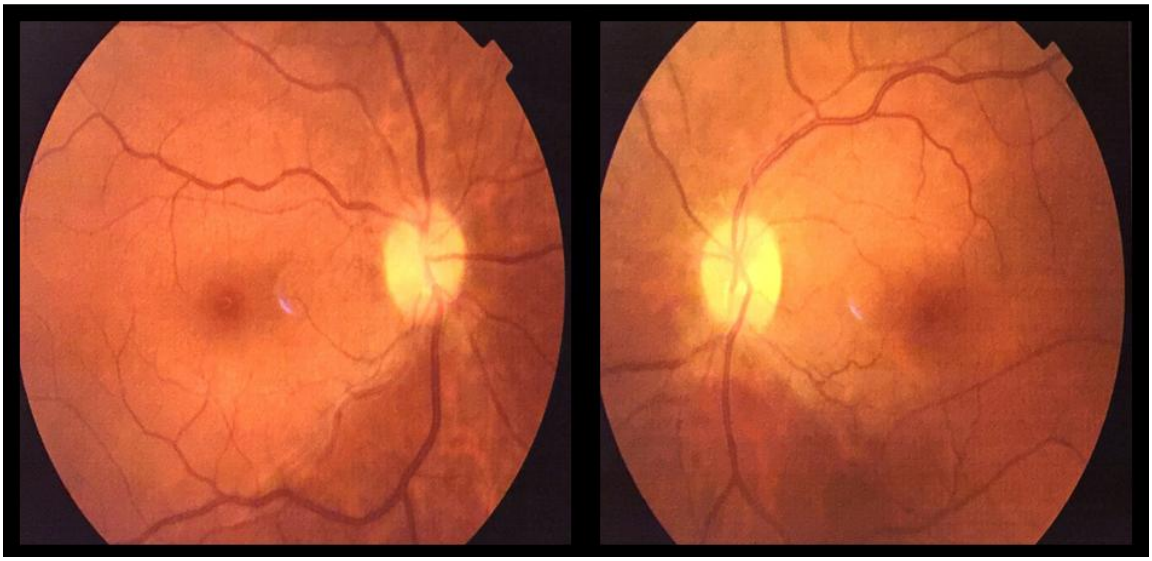

Fig 5 : Stereo photos demonsrtrating the regression of the papillaryedema

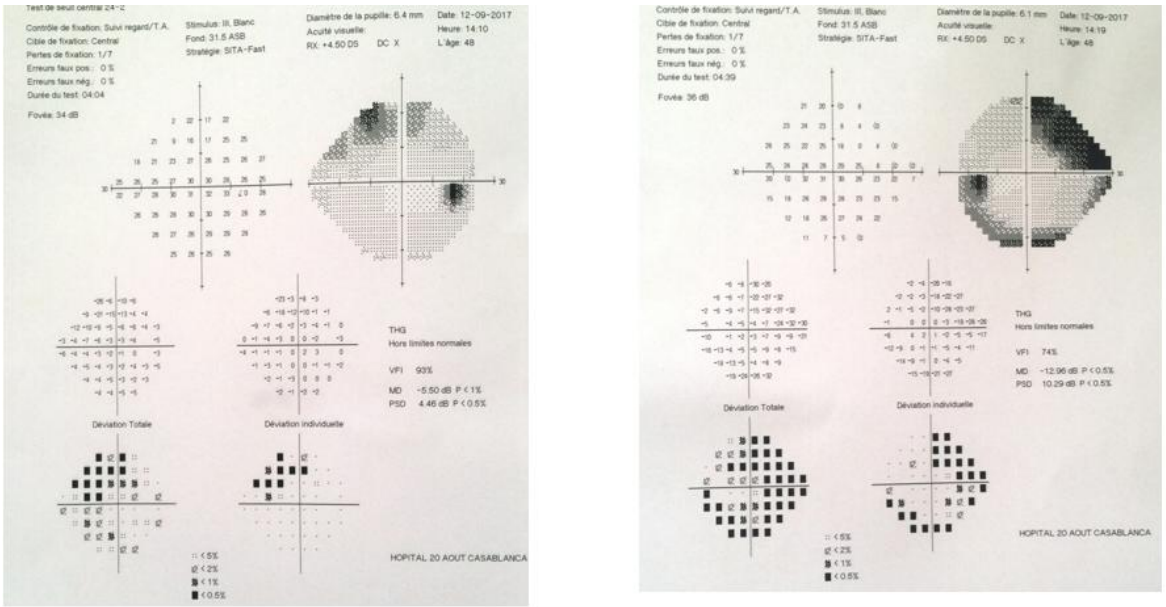

Fig 6 : Visual fielddemonstrating a persistence of the campimetricdeficit

III. Discussion

Toxicopticneuropathyis the result of damage to the optic nerve caused by varioustoxins [3]. The medicationsthatmay cause toxicopticneuropathy are antituberculosis (ethambutolisoniasid) , antimicrobial(linezolid and ciprofloxacine) , antiepileptic (vigabarrin),phosphodiesterase type 5 inhibitors (sildenafil , tadalafil ,vardenafil) , anti-tumornecrosis factor alpha agents , amiodarone and tamoxifen[2]. No case of allopurinol toxicopticneuropathy has been described in the literature .

Differentialdiagnosis for toxicopticneuropathyincludenutritional and tobacco/alcohol ,ischémic , compressive , demyelinating , and hereditaryoptic neuropathies [2].

Because the patient followed a healthydiet , showed normal serum B12 and redbloodcell folate levels , nutritionalopticneuropathycouldberuledout.Our patient was not tabagic or alcoholic . Ischemicopticneuropathywasunlikelybecause the patient did not have vasculopathydisease and his vision losswasbilateral. Dymyelinating and compressive optic neuropathie couldberuled out because of the magneticresonanceimaging of the brain and orbits. The hereditaryopticneuropathycouldalsoberuled out becauseit'stypicallypresents at a youngerage[4]. This case providesthat allopurinol can cause bilateralopticneuropathy. Causality for optic neuropathies fromdrugtoxicitygenerallyrequires a biologicmechanism,adequate duration of treatment , a dose-responsecurverelated to the medication , recoveryduringtreatment cessation, and involvement of bothoptic nerves at presentation or with progression [5]. The biologicmechanismsrelated to allopurinol opticneuropathy are not known at present. Clinciansneed to beaware of this rare but important toxicity.Immediatetermination of therapyis the only effective management thatcan stop the progression and allowrecovery of vision [3].

IV. Conclusions

In conclusion , if the suspecteddiagnosisisdrug-relatedtoxicopticneuropathy , and the withdrawal of one medicationdoes not lead to visualrecovery or thereis a furtherdeterioration of vision , the possibility of toxicitybecause of othermedicationsshouldbeconsidered.Werecommendconsideringbaselinevisualassessment if patients experiencevisualsymptomsafter commencement of allopurinol therapy and assessingvisualacuity , color vision, and visualfields to detectearlyevidence of toxicopticneuropathy.

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