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Ocular Toxicity of Antidepressants, Pathophysiology and Prevention: About Two Cases

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Abstract | **Case Report**

Often unknown, the ocular side effects induced by antidepressants are rare but can however be severe with risk of compromising the functional prognosis of the eye. We report in this article, the case of two patients who presented an ocular toxicity induced by antidepressants, then we will support the pathophysiological mechanisms of occurrence of these ophthalmological effects as well as the importance of the multidisciplinary therapeutic component of induced ocular toxicities. At the end of this article, and aimed at prevention, we will discuss the interest of taking into consideration the risk of occurrence of ophthalmological damage when prescribing antidepressants.

Keywords : Ocular toxicity, secondary ocular involvement, antidepressant, clinical cases.

INTRODUCTION

Antidepressants are psychotropic drugs prescribed mainly to treat depressive states, anxiety disorders, certain chronic pain pathologies, eating disorders and sleep disorders [1].

Their therapeutic action is linked to their effects on brain neurotransmitters, which modifies mood and behavior. However, increased levels of these neurotransmitters can impact the eye as well as other systems in the body [2].

Induced eye damage, sometimes unknown, includes accommodation disorders, dry eye, mydriasis, acute glaucoma or retinal alterations.

We present, through this article, the case of two patients followed for a depressive disorder, each under a different class of antidepressant, and in whom ocular toxicity was induced by these antidepressants.

The aim of this article is to warn the psychiatrist and the ophthalmologist about these ocular risks induced by antidepressants and to understand their pathophysiological mechanisms, and finally to update on the recommendations of prevention and management in order to minimize the risk of functional impairment and thus preserve optimal visual function.

Clinical vignettes

Clinical case number 1:

This is a 60-year-old patient known for chronic narrow-angle glaucoma stabilized under well-conducted medical treatment (prostaglandin analogs), followed for six months for depressive disorder (diagnosed according to DSM-V criteria) [3] on Amitriptyline 50 mg. The patient experienced severe eye pain associated with blurred vision and a rainbow-colored halo around lights, headaches and vomiting.

An ophthalmological consultation is then requested in order to carry out diagnostic examinations but also to determine whether the prescribed antidepressant would be incriminating to induce these ocular symptoms and then discuss its discontinuation.

Clinical and paraclinical ophthalmological examinations were in favor of acute angle closure glaucoma requiring discontinuation of the antidepressant.

The clinical course was marked by improvement of ophthalmological symptoms after change of antidepressant venlafaxine 75mg / day

Clinical case number 2:

Patient of 35 years followed for a somatoform disorder sertraline 100 mg / day for two months. For several days now, she has presented with progressive worsening ocular symptoms suggestive of induced dry eye: Burning and conjunctival itching, foreign body sensation, redness and sensitivity to light and his eyes became extremely watery. The iatrogenic effect of the antidepressant was retained after eliminating any other etiology.

DISCUSSION

However, all structures of the ophthalmologic organ can be affected by toxic adverse effects of antidepressants, affecting mainly the anterior part of the eye. Often unknown, because of their usual benign nature, they can be severe with risk of compromising the functional prognosis of the eye [4].

These adverse effects are due to the neuro-biological action of antidepressants, to varying degrees depending on their classes, on neurotransmitters and on their receptors.

There are different classes of antidepressant drugs; tricyclic antidepressants (TCAs), selective serotonin reuptake inhibitors (SSRIs), serotonin and norepinephrine reuptake inhibitors (SNRIs), norepinephrine and dopamine reuptake inhibitors (NDRIs), monoamine oxidase inhibitors (MAOIs) [5].

For tricyclic antidepressants (Amitriptyline, Imipramine) cause mydriasis, paralysis of accommodation, blurred vision and dry eye. This is due to the antagonistic action of muscarinic receptors. Risk of increased intra-accular pressure (IOP) in patients with angle-closure glaucoma.

Selective serotonin reuptake inhibitors (fluoxetine, sertraline, paroxetine): by their anti-cholinergic action (weaker than ATC) cause moderate mydriasis, dry eye. Increased risk of angle closure in ocular anatomical abnormalities [7].

Serotonin and norepinephrine reuptake inhibitors (venlafaxine, dulàoxetine) have a sympathomimetic action caused indirectly by an inhibition of the effect of acetylcholine, would cause mydriasis via the action on the dilator muscle of the pupil which would exert a mechanical blockage of the resorption of the aqueous humor. The increase in the production of aqueous humor and the dilation of the pupil causes an increase in intraocular pressure and the occurrence of acute glaucoma by closure of the angle especially in people with a physiologically narrow drainage angle that is encountered mainly in hyperopic patients [6].

Other risk factors have been reported such as a family history of glaucoma, enlargement of the lens (e.g. senile cataract) and ciliary muscle hyperplasia [6].

By comparison, SSRIs and SNRIs have a lower anticholinergic effect compared to ATCs, so they affect the tear film less. Some studies have shown that SNRIs, in particular, have an even lower impact than SSRIs [6].

Simple, conscientious and planned monitoring in a large majority of cases reduces the risk of eye damage and preserves optimal visual function. Treating physicians should be aware that an IRS may raise eye pressure in predisposed patients (elderly, hyperopic, previously glaucoma, familial glaucoma).

It is therefore important, before any prescription of antidepressants, to conduct an ophthalmological examination and carefully evaluate the risk factors (narrow-angle glaucoma, ophthalmological history, myopia / hyperopia) and potential drug interactions. In case of doubt, it will be a question of seeking the opinion of the ophthalmologist and the clinical pharmacologist.

In all patients on antidepressants, ophthalmological advice is required to perform complete and regular ophthalmological examinations (at least one fundus examination, IOP control, ocular surface assessment if symptom).

In case of symptoms or complications, adapt or change the antidepressant in consultation psychiatrist and ophthalmologist, in parallel with symptomatic treatment of dryness and urgency in case of glaucoma.

Finally, favor, in patients at ophthalmological risk, antidepressants with a more favorable eye profile (IRSN according to some studies) [8].

CONCLUSION

The ocular side effects of antidepressants, due to their action on neurotransmitters, are numerous and their recall is thus of obvious importance.

The potential risks of ocular toxicity should not be ignored and should lead the psychiatrist to inform the patient and systematically seek them during the controls or refer him to a specialized ophthalmological opinion in case of symptoms or presence of risk factors.

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