Drug-Induced Glaucoma: Causes and Prevention

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60 million Americans are at risk for developing glaucoma. Glaucoma is characterized by optic nerve damage and visual field defects, for which elevated intraocular pressure (IOP) is the major risk factor. An elevated intraocular pressure is caused by an obstruction in the circulatory pathway of aqueous humour in the interior chamber of the eye. The higher the level of IOP, the greater the likelihood of optic nerve damage and visual field loss.

Drug-induced glaucoma can be considered as a form of secondary glaucoma because it is brought about by specific systemic, topical, or ophthalmic medications, which include both prescription and over-the-counter drugs. The drug may cause the elevation of IOP via an open-angle mechanism or a closed-angle mechanism. Patients taking potentially glaucoma-inducing medications can remain undiagnosed with an elevated IOP, which can result in permanent optic nerve damage and lose of visual field.

Every patient is susceptible to drug-induced glaucoma. The risk is even greater in glaucoma suspects, or those already diagnosed with glaucoma, as these medications can exacerbate a more pronounced attack of elevated IOP. Although some severe acute attacks may result in immediately recognizable symptoms, most drug-induced elevation of IOP is gradual and symptomless, allowing it to go unaddressed until there is complaints of significant lose of the visual field.

Patients taking drugs that have the potential to increase IOP should have tests to detect this condition and to allow early diagnosis. If glaucoma inducing drugs must be used, IOP should be monitored closely. A favorable outlook exists if IOPs are controlled. If untreated, glaucoma can lead to permanent visual damage and blindness.

Product package inserts may mention glaucoma or the risk of increased intraocular pressure as a contraindication or as an adverse effect. Clinicians should be mindful of the possibility of drug-induced glaucoma, whether or not it is listed as a contraindication. With certain basic safeguards, virtually all of the medications are acceptably safe to prescribe.

Not all clinicians are aware of this issue. Thus far, these drugs have often been prescribed in glaucoma patients, while some other clinicians avoid these medications altogether in higher risk patients, while others pay little attention to glaucoma. Routine screening can allow patients to safely benefit from such medications.

At the present time, guidelines for glaucoma screening by the primary care practitioner have not been firmly established. Improvement in the diagnostic skills for the early detection of glaucoma in the primary care setting, coupled with clear guidelines for referral to an ophthalmologist, could have significant economic and health implications.
IOP measurement by tonometry can easily be used as a routine part of the periodic health examination performed by the primary care physician for the purpose of detecting glaucoma. Non invasive tonometry has gained general acceptance, mostly among eye care professionals as a screening tool for glaucoma. It is not widely used by primary medical care physicians who may have within their practices many patients with undiagnosed glaucoma.

Tonometry is useful for primary medical care physicians to evaluate adverse effects of drugs and prevent unrevealed manifestations of glaucoma. General practitioners should be cognizant of the risk factors for glaucoma before and after prescribing a drug that has the potential to cause, precipitate or exacerbate glaucomatous disease of the eye.

Prevention relies on screening and early detection, especially in those cases where a drug-inducing medication must be taken. Many of these medications are prescribed in the primary care setting. Therefore, an increased awareness and improvement in early detection of elevated IOP by primary care could significantly decrease the prevalence of drug-induced glaucoma.

**Glaucoma inducing medications:**

This review is based in part on papers published in peer-reviewed, ophthalmic and non-ophthalmic scientific journals, drug product inserts, and the 2007 and 2008 Physicians Desk Reference. Searches included various combinations of terms such a Glaucoma, Elevation of IOP, Increase Intraocular Pressure, Intraocular Hypertension within fields such as Contraindications, Side Effects and/or Warnings. Thus far, over three hundred prescription and over-the-counter medication are listed.

Several different drugs have the potential to cause the elevation of intraocular pressure, which can occur via an open-angle mechanism or a closed-angle mechanism. These medications can be subdivided into steroidal or nonsteroidal medications. Steroid induced glaucoma is a form of open-angle glaucoma. Medications prescribed for a variety of systemic conditions can produce pupillary dilation and therefore angle-closure glaucoma. Children are as susceptible to drug-induced glaucoma as adults.

In addition, many over-the-counter medications have within their package inserts warnings regarding presence and/or history of glaucoma. However, patients are unaware of any presence or history of glaucoma if they have never been screened, and are therefore continuing to take those medications, not knowing of the risk.

**List of High Risk Over-The-Counter:**

Actifed, Advil, Benadryl, BioLean, Cleareyes Drops, Comtrex, Dimetap, Dramamine, Excedrin, PediaCare, Primatene Mist, Robitussin, Simply Sleep Caplet, Sudafed PE, Theraflu, Triaminic, Tylenol, Vicks, VIVA Redness Relief Eye Drops, Zicam
List of high risk prescription therapeutic drug classes:

Acetylcholinesterase Inhibitors
Alpha-Adrenergic agonist
Aminoketones
Analgesics: Opioid Analgesic / Central Acting Analgesic / Agonist-Antagonist / Narcotic
Anticholinergic Agents
Antihistamines
Antiarrhythmic (Class I)
Antitussives (Non-Opioid) / Opioid Antitussives
Barbiturates
Benzodiazepines
Benzothiazole
Beta₂ Agonists
BOTOX Purified Neurotoxin Complex
Bronchodilators
Calcium channel blocker / HMG-CoA reductase inhibitor
Carboxamides / Carboxamide Anticonvulsants
Chelating agent
Corticosteroids
COX-2 inhibitor
Decongestants
Dibenzoapine Derivatives
Diuretic
Dopa-Decarboxylase Inhibitor/ Dopamine Precursor/ COMT Inhibitor / Dopamine Agonist / Non-Ergoline
Dopamine Agonist / Dopamine Reuptake Inhibitor
Expectorants
Fluoroquinolone
GABA analog
Glucocorticoids
H₁ Antagonists
HMG-CoA reductase inhibitor
Imidazopyridine Hypnotics
Immunomodulatory agent
Monoamine oxidase inhibitor / MAOI (type B)
Muscarinic Receptor Antagonists
NMDA receptor antagonist
NSAID
Phenothiazine / Phenothiazine Derivatives
Phosphodiesterase Type 5 inhibitor
Prostaglandin E₂
Protein-tyrosine kinase inhibitor
Proton Pump Inhibitors
Pyrazolopyrimidine (Non-Benzodiazepine)
Selective Serotonin and/or Norepinephrine and/or Dopamine Reuptake Inhibitors
Serotonin/histamine antagonist
Skeletal muscle Relaxants (Central-Acting)
Smooth muscle antispasmodic
Sulfamate-substituted monosaccharide antiepileptic
Sulfonamide anticonvulsant
Sympathomimetic Amines / Anorectic Sympathomimetic Amines
Synthetic octapeptide analogue of somatostatin
Thienobenzodiazepines
Tricyclic Antidepressants
Works Cited


