Only 46 workers had died, and the statistical power to detect a significant increase for any specific cause of death was low (eg, 0.60 for a threefold increase in lung cancer). However, no overall increase in mortality was seen for all causes of death, all cancer deaths, or any specific cause of death. There were no cases of mesothelioma.

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GLAUCOMA THERAPY: IMPLICATIONS FOR THE INTERNIST

Although treating glaucoma is the province of the ophthalmologist, internists should be familiar with the glaucoma medications their patients may be taking, since even topical agents can cause systemic side effects. Conversely, oral medications given for other conditions can affect the intraocular pressure.

SIDE EFFECTS OF GLAUCOMA DRUGS

Topical agents

Topical beta blockers can cause all the side effects of oral beta blockers: exacerbation of asthma or chronic obstructive pulmonary disease, congestive heart failure, sinus bradycardia, heart block, depression, confusion, impotence, masking of symptoms of hypoglycemia, and exacerbation of myasthenia gravis. Conversely, oral beta blockers given for high blood pressure or angina can reduce intraocular pressure. The nonselective topical beta blockers (timolol, levobunolol, metipranolol, carteolol) tend to control intraocular pressure better than the selective beta blocker betaxolol, but the latter is safer for patients with asthma or obstructive lung disease.

The nonselective adrenergic agonists epinephrine and dipivefrin can cause hypertension, angina, tachycardia, palpitations, headaches, and tremor, in addition to the ocular complications of blurred vision (due to pupil dilation) and chronic red eye.

Topical cholinergic agonists (pilocarpine, carbachol, echothiophate, demecarium, physostigmine) are infrequently used, but they can increase gastrointestinal motility, salivation, and respiratory secretions. The anticholinergic agents (atropine, homatropine, scopolamine, cyclopentolate, tropicamide) can cause dry mouth, a flushed feeling, tachycardia, and atonic bowel.

The alpha-2 adrenergic agonist apraclonidine does not cause systemic complications, but it can cause lid elevation, conjunctival blanching, and pupil dilation.

Patient education

Systemic absorption of topical ophthalmologic medications can be minimized by instructing the patient to occlude the lacrimal sack when applying eye drops, to close the eye immediately afterward and to immediately blot up any excess fluid.

Oral agents

The oral carbonic anhydrase inhibitors (acetazolamide, methazolamide, dichlorphenamide) frequently cause potentially severe side effects: rash, fatigue, malaise, anorexia, weight loss, paresthesia, dysesthesia, depression, impotence, gastritis, renal calculi (with acetazolamide), and aplastic anemia. They increase excretion of bicarbonate and can cause metabolic acidosis, hypokalemia (especially when used with potassium-wasting diuretics), and hyperchloremia. These agents can aggravate gastritis or peptic ulcer disease. Methazolamide is metabolized in the liver and should be avoided in patients with hepatic failure.

The oral osmotic agents (glycerine, isosorbide) can cause nausea, vomiting, severe headache, dehydration, severe systemic fluid shifts, and subarachnoid hemorrhage; glycerine can cause severe glucose overload in diabetes mellitus. These agents and the intravenous osmotic diuretic mannitol are rarely used.

OTHER FACTORS AFFECTING INTRAOCULAR PRESSURE

Systemic corticosteroid therapy can substantially increase the intraocular pressure; caffeine mildly increases it, and nicotine has little effect. Antihistamines, decongestants, and anticholinergics usually have no effect. Excessive water imbibing can increase the intraocular pressure. Ethanol lowers it, as does exercise (except for gravity-inversion exercises).

DETECTING GLAUCOMA

Because glaucoma damages the eye insidiously before the patient becomes aware of it, young adults should visit an ophthalmologist every 3 or 4 years, and older adults every 1 to 2 years. Most primary glaucoma is hereditary; however, the hereditary patterns are poorly understood. The prevalence of glaucoma increases with age. There is a higher risk of glaucoma severity and blindness in blacks than in whites. Diabetes and myopia are mild risk factors.

Symptoms

Ocular pain is uncommon in glaucoma except with very high intraocular pressure. Headache, often unilateral over the brow, may represent referred pain. Blurred vision usually does not occur until late in the disease. At very high intraocular pressure, corneal edema may produce visual halos.

The patient loses vision in a distinctive pattern of peripheral or paracentral scotomas (areas of lost or depressed vision), a steplike defect in the nasal side of the visual field, and generalized depressed vision. However, other diseases can cause visual-field defects that mimic glaucoma. Visual acuity is often normal until late in the disease process.

Ophthalmologic findings

The hallmark of glaucoma is optic nerve damage and associated visual-field loss, which is asymptomatic and often asymmetric. The optic nerve shows progressive cupping (optic cup enlargement), a notch or other focal progressive thinning, a defect of the nerve fiber layer, or progressive optic-disc hemorrhaging.

TREATING GLAUCOMA

The patient should understand that treatment may prevent future damage only and will not restore lost vision.

The traditional American approach has been to start with medical therapy (topical, then oral) and then use laser therapy and ultimately surgery if ocular hypertension continues or visual fields show progressive defects. In contrast, the British approach is to resort to surgery early. Studies are underway to determine the best approach for various forms of glaucoma. An early laser iridectomy must be done for pupillary block and angle-closure glaucoma, and panretinal laser photocoagulation for neovascular glaucomas.

Some ophthalmologists question the value of glaucoma therapy for people with mild ocular hypertension without optic nerve damage, as there is no evidence that treating ocular hypertension prevents the onset of glaucoma. However, severe ocular hypertension always causes visual loss and other ocular damage and should be treated. A family history of glaucoma in a patient with ocular hypertension also would prompt the ophthalmologist to initiate treatment, even in the absence of damage.

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