hypertension include the drugs' interference with the energy dependent absorption mechanism by affecting cyclic adenosine monophosphate at the arachnoid granulations.[8] Although no clear genetic link exists to increased intracranial pressure, a genetic predisposition was proposed when dizygotic twin sisters developed the disorder. Both were taking tetracycline for acne. One was symptomatic with papilloedema, headache, and intracranial hypertension, while the other was found to have asymptomatic papilloedema after her twin had received her diagnosis.[10] Most of what we know about benign intracranial hypertension concerns the idiopathic form. More common than previously recognised, idiopathic intracranial hypertension occurs in 10-20/100 000 obese women. This means that idiopathic intracranial hypertension is as common among obese women as multiple sclerosis. The disorder affects women (7:1), who present with symptoms of intracranial hypertension (headache, diplopia, whooshing noises in the head) and signs of intracranial hypertension (papilloedema, palsy of the sixth cranial nerve). Ninety per cent of the patients are obese.[3]

In contrast, intracranial hypertension due to the tetracycline antibiotics (including doxycycline) occurs in both sexes, at almost any age, and without concomitant obesity. The symptoms and signs of intracranial hypertension, however, are the same. How quickly a person develops intracranial hypertension after ingesting doxycycline is unknown, but in the largest review of intracranial hypertension induced by minocycline, some participants had used the drug for up to a year before developing symptoms whereas others became symptomatic within two weeks.[8] Some dispute whether tetracyclines cause intracranial hypertension at all since so many individuals are treated with the drug every year without developing intracranial hypertension. However, individual cases have been reported where stopping the drug resolved symptoms and signs of intracranial hypertension, and restarting the drug brought recurrence of intracranial hypertension.[11]

No matter whether the disorder is idiopathic or secondary, it is known to be anything but benign. Corbett et al found that idiopathic intracranial hypertension often persists up to 41 years after the initial diagnosis, and that over 25% of patients have severe visual loss.[12] Patients with secondary forms of intracranial hypertension such as those using doxycycline are also not immune to visual loss. In 12 patients with minocycline induced intracranial hypertension 25% had notable visual field loss.[8] Therefore, patients who complain of headache after using doxycycline should be examined carefully, including their visual acuity, and formal testing of the visual fields. Funduscopy after dilating the pupils to look for papilloedema is mandatory.

Treatment of the primary and secondary forms of intracranial hypertension is similar—reduce intracranial hypertension. While there are no randomised controlled trials to guide the choice of treatment most practitioners recommend acetazolamide and weight loss to treat primary idiopathic hypertension. In the secondary forms, correcting the underlying mechanism, for example, treating the venous thrombosis, or stopping the causative medication is indicated. If visual loss progresses despite optimal medical therapy (usually acetazolamide, methazolamide, or furosemide (frusemide) in adequate doses), consideration of optic nerve sheath fenestration or lumbar peritoneal shunt is warranted to prevent further visual loss.[3] Risk factors for visual loss include a delay in diagnosis due to failure to diagnose the disorder, inadequate treatment, and delayed treatment.[3] The outcome of increased intracranial pressure due to doxycyclines is generally good if recognised early, before vision has been affected seriously.

Despite the many controversies, intracranial hypertension due to use of doxycycline does occur. Practitioners prescribing the tetracyclic antibiotics should be aware of the syndrome of increased intracranial pressure, and pay particular attention to the ocular fundus for papilloedema. Appropriate referral for visual testing including visual fields should be made, and treatment directed at stopping the drug, and instituting symptomatic treatment that lowers the intracranial pressure.

Kathleen B Digre *professor of neurology and ophthalmology*

University of Utah, Departments of Neurology and Ophthalmology, Salt Lake City, Utah 84132 USA (Kathleen.digre@hsc.utah.edu)

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**Correction**

*Tobacco, coffee, and Parkinson's disease*

An error occurred in the authors' names in this editorial (15 March, p 562). The name of the second author should have been Catharine Gale, not Chris Gale, and she should have been listed as the first author. We apologise for this editorial error.