as found in the recurrent lesion of our patient, has been described only once as an orbital primary tumour[3] and has not been previously reported as a recurrent orbital liposarcoma.

A recent classification from The Armed Forces Institute of Pathology[8] groups together the WDL, atypical lipoma, and pleomorphic lipoma into one category of 'atypical lipomatous tumor' located between the benign and malignant groups, as it contains cells with irregular, hyperchromatic nuclei but does not metastasize. It was also argued that the differentiation between lipoma-like, sclerosing, and inflammatory subgroups have no significance and may occur in the same tumour. Our finding of the same chromosomal imbalances in the primary lipoma-like tumour and in the inflammatory relapsing tumour also suggests that these subtypes should be grouped together.

In summary, liposarcoma with metaplastic bone and recurrent liposarcoma with inflammatory component should be added to the differential diagnosis of rare orbital lesions.

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Sir,

Simultaneous bilateral acute angle closure glaucoma following venlafaxine treatment

A 49-year-old woman presented to eye casualty with blurred vision and pain in both eyes. She had been diagnosed with irritable bowel syndrome (IBS) 4 years earlier and her gastroenterologist had prescribed venlafaxine earlier that day. At 4 h after taking the first oral dose, she developed symptoms of pain and blurred vision simultaneously in both eyes. She also reported nausea and vomiting.

On examination, visual acuities were 6/18 in the right eye and 6/12 in the left. Both pupils were fixed and mid-dilated. There was bilateral corneal oedema and shallow anterior chambers were evident. Gonioscopy revealed angle closure in both eyes. Intraocular pressures (IOPs) were 49 mmHg in the right eye and 54 mmHg in the left. A diagnosis of bilateral acute angle closure glaucoma was made.

Immediate therapy was commenced with a standard acute angle closure glaucoma treatment regimen. IOPs were normalised by the next day and YAG laser peripheral iridotomies were performed. She made a full visual recovery to 6/6 in each eye. The venlafaxine was discontinued.

Discussion

Venlafaxine is a new class of SSRI, which is a potent inhibitor of serotonin and, to a lesser extent, of noradrenaline reuptake. It is widely used in the treatment of depression and has become widely accepted as a treatment for IBS by modulating central and peripheral sensory mechanisms and by reducing depression, which may be an aetiological factor.[1]

There has been a previous report of increase in pressure with chronic narrow angle glaucoma and venlafaxine.[2] We also note a report of nonsimultaneous bilateral acute angle closure glaucoma with venlafaxine treatment,[3] although the causative link is weak as the patient developed the closure many days after treatment began and there was intervening orbital trauma. In addition, the patient was commenced with other drugs, including chlorpromazine, which is known to cause angle closure by anticholinergic effects.[4]

Although SSRIs have been implicated in long-term IOP rise,[5] the very dramatic bilateral pressure rise so soon after treatment with venlafaxine is likely to implicate a mydriatic aetiology in this event. Animal studies have shown that SSRIs induce mydriasis by acting through central mechanisms.[6,7]

Acute angle closure glaucoma has been reported with older SSRIs such as paroxetine occurring 2 weeks[8] and 1 day[9] after commencing therapy. Although a serotoninergic mechanism for increase in pressure over the longer term has been postulated,[10] it was thought that cases of acute glaucoma were due to the weak anticholinergic effects of paroxetine.[11]

Venlafaxine does not have anticholinergic effects and as such may strengthen the premise of a seratoninergic aetiology for acute angle closure reported with older SSRIs.[8,9] Alternatively, the weak adrenergic effect of this medication may well be responsible.

While the exact mechanism of precipitation of acute angle closure remains unclear, awareness among ophthalmologists and physicians prescribing these drugs must be raised.

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Sir,

Abnormalities on the multifocal electroretingram may precede clinical signs of hydroxychloroquine retinotoxicity

Hydroxychloroquine is a widely used and effective antirheumatic drug since its production in 1940s, especially for systemic lupus erythematosus, rheumatoid arthritis, and Sjögren's syndrome. Retinotoxicity as a side effect is well known despite concerted efforts by physicians and ophthalmologists in monitoring these patients.[1–6] The estimated risk of macular toxicity in patients on chronic hydroxychloroquine has been estimated to be less than 0.5%,[7] although Bernstein reports that incidence can be as high as 3–4% in