We report the case of a young man who developed permanent retinal changes leading to blindness as a result of chlorpromazine. It is particularly unusual as he received only very low doses of chlorpromazine and was poorly compliant.

Case report

SW is a 33-year-old male with a chronic schizophrenic illness. He first presented at the age of 19 with psychotic symptoms thought to be hallucinogen induced which settled quickly on very low dose chlorpromazine. Later that year he was readmitted and attracted a diagnosis of schizophrenia. Over the next nine years he received only very low doses of chlorpromazine (max 200 mg daily) and a short course of fluphenazine decanoate (25 mg four-weekly), but was very poorly compliant.

In 1992 he complained of poor vision, so was referred to an ophthalmologist where he was found to have greatly reduced visual acuity, grossly limited perimetry, bilateral proptosis, and fluorescein angiography revealed bilateral macular oedema. Extensive investigations performed were negative and chlorpromazine-induced retinopathy was diagnosed. Despite discontinuing chlorpromazine permanent retinal pigment epithelial damage was present at both foveas. He remained functionally impaired and was registered blind, and is seeking damages.

It would therefore seem prudent that clinicians are aware that chlorpromazine may cause severe ocular damage even at low dose. There may also be a case for screening for pigmentary retinopathy in patients who are exposed to chlorpromazine.

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Buspirone augmentation of sertraline

SIR: Refractory obsessive–compulsive symptoms are vexatious for both patient and clinician (Jenike & Rauch, 1994). In the considerable number of cases which fail to respond to SSRIs, augmentation strategies with lithium or buspirone have been disappointing (Dominguez & Mestre, 1994). After observing incomplete responses to sertraline, I report two cases of dramatic symptomatic improvement with the addition of buspirone.

Case 1 is a bus driver in his early twenties, with a 5–6-year history of obsessive–compulsive disorder. When unwell, he is unable to work, as he becomes preoccupied with checking his cash float and his schedules to the extent that his route runs progressively later and later. Having been suicidal, his OCD was originally mistaken for depression. Treatment with dothiepin and desipramine had been unhelpful, diazepam relieved his anxiety but not the rituals. After his diagnosis two years previously, he responded partially to fluoxetine (20–40 mg/day), but discontinued this due to persistent insomnia, headache and lethargy. Sertraline was somewhat better tolerated, but nausea became a problem each time the dose was pushed above 100 mg/day. At this dose his improvement was partial (Y-BOCS reduced from 28 to 20; Zung from 60 to 50); he was able to work (barely) but was tense and exhausted from struggling against his rituals. Addition of buspirone 10 mg/day was dramatically effective within days (Y-BOCS 8; Zung 27), without any return of his nausea. Attempts to withdraw sertraline or buspirone have been equally unsuccessful, due to the rapid return of his anxiety and rituals. He has remained well on this combination for 15 months.

Case 2 is a 36-year-old woman with a 20-year history of recurrent anorexia nervosa, OCD, migraine, and marked premenstrual irritability. She had responded poorly to various tricyclic antidepressants, and was unable to tolerate more than 75 mg of clomipramine. Her OCD was particularly debilitating; apart from rarely finishing her rituals before 2 am, she felt driven to arrange coloured clothes pegs 'properly' in order to protect her children from harm. Phenelzine 60 mg/day was effective in prophylaxis of her migraine and premenstrual mood change, but did not affect her OCD and was finally rejected because of its stimulation of appetite and weight gain. Fluoxetine was effective in partially suppressing her OCD, but worsened her headaches and was abandoned. Sertraline was less likely to aggravate her migrane, but was incompletely effective up to 200 mg/day with marked symptoms persisting (Y-BOCS 25; Ham-D 31).

Addition of buspirone up to 60 mg/day produced a dramatic remission of her compulsions and premenstrual dysphoria, her obsessions remaining largely unchanged (Y-BOCS 17; Ham-D 12). Her headaches, worsened by this combination, were relieved by the addition of atenolol. Attempts to withdraw sertraline or buspirone were quickly abandoned due to recrudescence of compulsive

rituals. Her improvement has maintained over two years.

These cases indicate a potentially useful energy between sertraline and buspirone in OCD, similar to their combined efficacy in refractory depression (unpublished observations). The cases reported here appear in contrast to controlled trials finding little effect of buspirone augmentation of other SSRIs (Dominguez & Mestre, 1994). Both cases also displayed significant depressive symptomatology, which I suggest may predict buspirone augmentation of SSRI non-response in OCD. This possibility, yet to be examined in a controlled study, suggests a biological difference between OCD with and without depression, and accords with the finding that tryptophan depletion worsens depression, not OCD, in patients responding to SSRIs (Barr *et al*, 1994).

A pharmacokinetic interaction between sertraline and buspirone could account for the augmentation, but seems unlikely, given the dramatic rapidity and reversibility of the effect, and the fact that Case 1 was clearly tolerant of higher doses of sertraline and did not suffer recurrence of side-effects after addition of buspirone. A final possibility is that sertraline may differ in some important way from other SSRIs, perhaps by enhancing NMDA receptor activation (Bergeron *et al*, 1993).

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Osteogenesis imperfecta and non-accidental injury

SIR: Bebbington *et al* (1994) report a strong link between ethnicity and diagnosis. We report a case in which failure to consider the cultural background led to misdiagnosis with far-reaching consequences.

Case report

Ms. A is a 30-year-old Nigerian woman who arrived in the UK in the early nineties, pregnant with her second child B and of undisclosed immigration status. There was no family or personal history of mental or physical illness.

When B was 3 weeks old, Ms. A brought her to casualty with painful swellings of both lower limbs. B had bluish sclera and a flat back to her head, but it was stated that these features "are common in African babies". X-rays showed bilateral transverse fractures of the femurs and multiple older rib fractures. The paediatrician commented that Ms. A gave "the usual story" to account for B's injuries.

Infants in Ms. A's community are given regular massage. She had noticed cracking noises from the legs the previous day while massaging B. The paediatrician thought this explanation unlikely. An Emergency Protection Order was made placing B with foster parents.

Five months later Ms. A took a pair of shoes from the children's ward where B had been an in-patient. She was detained under Section 2 of the Mental Health Act (1983). She was virtually mute, and ate and drank little for three days. She behaved in a vague and distracted way with ritualistic movements, but afterwards had no memory of these events. To a Nigerian social worker she denied psychotic phenomena. Further bizarre behaviour in public led to more hospital admissions. Each time, Ms. A's mental state returned to normal rapidly without medication. Culture-bound adjustment reaction was diagnosed.

In foster care, B failed to gain weight and had another fracture. Review of the original X-rays and collagen banding indicated severe osteogenesis imperfecta (type 111). Ms. A was admitted to a Mother and Baby Unit and was rehabilitated as sole carer of B. Her mental state was normal. She felt angry with professionals, believing she would have been listened to had she been white. On discharge she was caring fully for B and cooperating with supporting agencies.

When B was born, Ms. A had been in the UK for less than three months. For a non-Caucasian immigrant there is a time of adjustment and probably racism, overt and covert, to face (Littlewood & Lipsedge, 1989). Ms. A was fearful of deportation and losing B. Her guarded behaviour raised suspicions in professionals. Diagnosis of child abuse is more likely if the explanation is insufficient or inconsistent (Ablin *et al*, 1990).

Non-accidental injury is a common differential diagnosis for milder forms of osteogenesis imperfecta, but is rare in Type 111 (Paterson & McAllion,