Completing surgical management of morbid obesity

Sir,

We read with interest the review article *Surgical management of morbid obesity* by Aggarwal et al (vol 69(2), 2008, p. 95) and agree that the exponential rise in obesity fuels the demand for bariatric surgery. A recent audit (January 2008) by the British Obesity Surgery Patient Association of English primary care trust criteria for funding bariatric surgery revealed fifteen primary care trusts required a patient to have a body mass index (BMI) of 50 kg/m^2 or more and four primary care trusts required a BMI of at least 60 kg/m^2. So for some primary care trusts their patients will have to be considerably larger than the National Institute for Health and Clinical Excellence (2006) clinical guidelines of BMI >40 kg/m^2.

Undoubtedly the improvement in patient co-morbidities following bariatric surgery is becoming increasingly recognized although the consequences of such massive weight loss resulting in excessive lax skin on the legs, arms, torso and neck can present the patient with new physical and psychological challenges, which often he/she had not considered before bariatric surgery.

The potential need for plastic surgery to manage these skin excesses should be brought to the patient's attention and also to the attention of those budgeting for the overall care of bariatric surgery patients.

The criteria set by some primary care trusts will only select those patients who will have the greatest skin excess when deflated after bariatric surgery. Functionally these skin folds are problematic for patients, causing difficulties with hygiene and the development of recurrent superficial skin infections. Many primary care trusts will fund bariatric surgery but will not sanction skin tailoring procedures which they incorrectly perceive as cosmetic, rather than the final phase of functionally and psychologically reconstructing the post-bariatric surgery patient.

We believe that the inclusion of a plastic surgeon in the multidisciplinary team and their involvement during the ongoing care of these patients should be regarded as essential for the surgical management of morbid obesity.

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Neuroleptic malignant syndrome induced by quetiapine overdose

Sir,

Quetiapine is a new class of antipsychotic which is frequently used in the treatment of schizophrenia and atypical depression. It is a dibenzothiazepine derivative, which has high affinity for D1 and D2 dopamine receptors. In comparison to other antipsychotics it has less alpha-1 antagonist and antimuscarinic activities (Whalley et al, 1999; Kobayashi et al, 2006). The data available on quetiapine overdose in the literature are sparse with a study in 2000 (Pollak and Zbuk, 2000) showing no fatalities and no correlation of high serum concentrations and toxicity of quetiapine.

Neuroleptic malignant syndrome is a rare, life-threatening condition characterized by fever, hyperthermia, muscular rigidity, change in mental status, tachycardia, hypertension or hypotension, metabolic acidosis and autonomic dysfunction. Although neuroleptic malignant syndrome is a well-recognized side effect of conventional antipsychotics, only four case reports have linked this condition with atypical antipsychotics such as quetiapine. In two of these reports, quetiapine was co-prescribed with sulpride (al-Waneen, 2000) and fluvoxamine (Stanley and Hunter, 2000).

Here the authors describe a female patient who developed neuroleptic malignant syndrome following an overdose of quetiapine. A 61-year-old woman presented with a reduced level of consciousness (Glasgow Coma Scale 9), tachycardia, hypotension and metabolic acidosis with a pH 7.32. She had taken an overdose of 60 tablets of quetiapine 150 mg. She was on no other medication apart from quetiapine. She was managed conservatively with intravenous fluids in the first 24 hours and her Glasgow Coma Scale improved to 14. She remained quite drowsy for another 24 hours following which she returned to her normal mental state. She started spiking temperatures up to 39.1°C. Blood cultures, mid-stream urine and chest X-ray were unremarkable and there was no obvious source of infection. Her creatine kinase was 4631 U/litre (normal range 0–200 U/litre). She was continued on intravenous fluids and treated with paracetamol and lorazepam as required. She recovered after a further 48 hours with her creatine kinase falling to 157 U/litre.

Neuroleptic malignant syndrome can result from an overdose of quetiapine. It can be treated with supportive measures as it settles with time. This report adds to the limited literature available to date on the effects of quetiapine overdose.

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