Letters to the Editor

Radioiodine therapy in thyrotoxicosis

Sir,
I read with interest the excellent article by Sweatman & Chambers. However, they did not mention radioiodine therapy which is the agent of choice, especially in the patient described (a 71 year old woman with recurrent thyrotoxicosis). Radioiodine is the only reliable therapy in recurrent thyrotoxicosis and hyperthyroid heart disease.1,2 In a recent review3 it is noted that in Grave’s disease and toxic multinodular goitre the remission rate is only 40–50% after antithyroid drugs, these drugs have little effect in toxic adenoma, and after radioiodine therapy, the risk of malignant disease and genetic abnormalities is not significantly greater in any age group. In our institute, radioiodine therapy has been administered since 1981, 160 patients have been followed up, no malignancy or birth-defect has been found, and only two patients have become hypothyroid.

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References

This letter has been shown to Dr Sweatman who replies:

Sir,
We were very pleased to note Dr Taher’s interest in this article and agree wholeheartedly with his views on the excellence of radioiodine therapy in the treatment of thyrotoxicosis, particularly in the age group referred to in our paper. In fact we followed a reasonably conventional course with this lady in that she had oral anti-thyroid therapy in the first instance and subsequently radioiodine was administered once her thyrotoxicosis was controlled. We were unable to mention this in the case report owing to shortage of space and the need to report concisely the essential features in our patient.

M.C.M. Sweatman
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Hypoglycaemia related to falciparum malaria – a correctable cause of death

Sir,
The paper entitled ‘Hypoglycaemia and cerebral malaria’ by Kiire4 published in a recent issue was read with interest. The pathogenesis of this correctable and preventable metabolic disorder in the course of falciparum malaria brought out by the author is very relevant and informative, more so to clinicians from South East Asia and Africa where quinine is being increasingly used in the light of chloroquine resistance. In an earlier Indian Armed Forces study in north-eastern India covering 560 patients with falciparum or mixed (falciparum and vivax) infections, hypoglycaemia was not observed, presumably following the routine use of glucose infusions in all patients with pellaginous manifestations.2

The recent report of a patient with cerebral malaria who manifested with recurrent neurological disturbance while on quinine sulphate and complete recovery after supportive glucose infusion indicate the need for awareness of a treatable but life threatening complication in the process of management.3

It is hence recommended, as has already been highlighted by Kiire4, that clinicians involved in the management of pellaginous malaria should routinely monitor parenteral glucose infusion prophylactically to prevent morbidity and mortality related to hypoglycaemia.

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References
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