Patients’ self administration of hydrocortisone

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Properly managed, Addison’s disease carries a normal life expectancy.1 1 The only related risk of death is inadequate glucocorticoid replacement treatment at times of illness or other stresses; this can lead to an adrenal crisis. All patients should know how to adjust their steroid dose when they are ill, and they should know which illnesses require such adjustment. We believe that all patients should have a supply of injectable hydrocortisone at home and be able to give themselves an injection if vomiting persists or if there is delay in obtaining medical attention. As it is the policy of our unit to instruct all patients in these matters we audited our patients to determine their knowledge.

Patients, methods, and results

Twenty six patients with Addison’s disease were identified from unit records, 22 women and four men with a mean age of 49 (range 18-79) years and mean duration of disease of 15 (1-35) years. Patients were sent a multiple choice questionnaire of 33 questions: six questions inquired about background information and current steroid replacement treatment and nine referred to self treatment practice with oral steroids during illness and 18 to self treatment with parental steroids. Twenty five (96%) patients replied.

Fifteen patients had never changed their oral steroid dosage. Nine of these had had Addison’s disease for over 16 years and three for over 30 years. Seven patients were wholly correct about which illnesses required adjustment of dosage, 15 were partially correct, and three were totally wrong.

Ten respondents (40%) claimed never to have been instructed in injecting hydrocortisone. Although the case notes recorded instruction in only three of these patients, all should have been instructed in accordance with unit policy. None of the 10 patients had a home supply of injectable hydrocortisone. Fifteen respondents recalled instruction, and written confirmation of this appeared in six of their case notes. Only six had a home supply of injectable hydrocortisone. One of these six had no needles or syringes, and the expiry date of one patient’s supply had passed. A further two patients thought they would not be able to inject themselves, thus only two patients among the cohort of 25 responders could self administer parenteral hydrocortisone, but one of them did not take the parenteral steroids on holiday.

Seventeen of the 25 patients had received parenteral steroids in the past. For 11 patients this had been an imminent or established adrenal crisis requiring inpatient treatment with intravenous hydrocortisone; the other six had received parenteral steroids at the time of elective surgery. None of these patients had had intramuscular hydrocortisone before admission. Only two of these patients currently had injectable hydrocortisone at home, and both were unable to self-administer it. Twenty patients (80%) always carried either a steroid card or Medicalert bracelet indicating that they were on maintenance steroids for Addison’s disease.

Comment

Nearly half of these patients with Addison’s disease had had an adrenal crisis, yet the group as a whole remained ignorant. Only a quarter had injectable hydrocortisone at home, and only two could self-administer it. In a population well served by general practitioners this may not jeopardise the patient, but in rural areas, inner city environments, or transient accommodation patients may be in danger of an adrenal crisis if medical advice is delayed. Rectal hydrocortisone is well absorbed and provides adequate cortisol for up to eight hours and may be an effective alternative to parenteral hydrocortisone.2

All our patients have now been sent a follow up instruction sheet and will be issued with injectable hydrocortisone and needles. Further instruction in using intramuscular hydrocortisone will be arranged.

We suggest that, as part of the routine care of patients with Addison’s disease, the knowledge of self treatment should be reinforced verbally and with an instruction sheet so that patients can self administer intramuscular steroids if necessary.

We thank Dr M Hartog, reader in medicine, Bristol Royal Infirmary, for permission to study his patients and Mrs Marion Mitchard for secretarial assistance. GDB is funded by Fidia.


Hypoalbuminaemia after prolonged treatment with recombinant granulocyte macrophage colony stimulating factor

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Treatment with recombinant granulocyte macrophage colony stimulating factor is being evaluated in various clinical circumstances including after chemotherapy, and bone marrow transplantation, and in aplastic anaemia, the myelodysplastic syndrome, and AIDS. Most trials have used intermittent dose regimens (for example, two weeks of treatment followed by two weeks without treatment), and dosages up to 32 µg/kg/day have been well tolerated with only minor side effects. At very high dosages serious side effects such as fluid retention, pericarditis, and thromboembolism have occurred.1,2

We are evaluating the clinical value of recombinant granulocyte macrophage colony stimulating factor in the myelodysplastic syndrome and aplastic anaemia. The factor is given daily subcutaneously for 12 weeks, the initial dosage of 3 µg/kg/day being increased to 10 µg/kg/day after four weeks if there has been no response. Patients who respond (neutrophil count >1·5 x 10⁹/l) subsequently receive maintenance treatment for two weeks in every four. Patients are required to have normal renal and liver function at entry. We have treated nine patients, four of whom developed