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Anorexia and megestrol acetate: treatment versus placebo controlled study

Nefrologia 2014;34(3):416-7
doi:10.3265/Nefrologia.pre2014.Mar.12391

To the Editor,

Loss of appetite is a frequent symptom in a dialysis patient, which causes anxiety and affects perceived quality of life. Deficient food intake as a result of anorexia is the main cause of type 1 uraemic malnutrition, which is not related to intercurrent inflammatory processes[1]. Megestrol acetate increases the appetite and improves the nutritional parameters in patients treated with periodic haemodialysis who suffer from anorexia[2]. Given the subjective nature of anorexia, there is doubt surrounding the extent to which the effect of megestrol acetate can be attributed to a placebo effect.[3] In order to clarify this, we studied anorexia's response to megestrol acetate in a randomised controlled study.

In 2011 and 2012, 122 patients were treated in our haemodialysis unit. 19 of the patients suffered from anorexia, without a known trigger factor. In order to diagnose anorexia, we used the appetite questionnaire from the HEMO and DOPPS studies[4,5] in which the patient has to indicate how he/she regards his/her current appetite on a Likert scale with five possibilities: very good, good, normal, bad or very bad. The patient is then asked whether his/her appetite had improved, stayed the same or diminished in the last four weeks. Anorexia is diagnosed when a patient reports that his/her current appetite is normal, bad or very bad and that it has not changed or has diminished in the last four weeks. The 19 patients were treated randomly with megestrol acetate, 160mg/day (10 patients; 4 males and 6 females), or placebo (9 patients; 4 males and 5 females). We analysed anorexia evolution and evolution of nutrition-related clinical parameters over three months.

Initially, there were no differences between the treated group and the control group with respect to age (73±9 vs. 69±18 years, *P*=.544), time on dialysis (40±46 vs. 47±41 months, P=.731), dry weight (58.1±10.7 vs. 61.9±7.2kg, P=.377), weight loss in the last two months (0.6±1 vs. 0.6±0.5kg, *P*=.903), albumin concentration (3.25±0.62 vs. 3.33±0.57 g/dl, *P*=.781) or dialysis dose (Daurgidas spKt/V 1.70±0.28 vs. 1.79±0.22, *P*=.456). All the patients received dialysis three times a week, with high-flux biocompatible membrane and ultrapure dialysate.

After three months of treatment, 9 out of the 10 patients treated with megestrol acetate and 4 out of the 9 patients treated with placebo reported an improvement in appetite (*P*=.046, Fisher test). Evolution of weight and other nutritional parameters are shown in Table 1. An increase in weight and in concentrations of albumin, creatinine and urea, without modification to dialysis dose, were only observed in the group of patients treated with megestrol acetate. None of these parameters varied significantly in the group of patients treated with placebo.

The feeling of loss of appetite is partly subjective, which can be seen to be influenced by placebo administration. However the stimulating effect on appetite induced by megestrol acetate cannot be exclusively attributed to a placebo effect. The proportion of patients who reported an improvement in appetite following megestrol acetate treatment is higher than that reported by the placebo-treated control group.

Table 1. Evolution of nutritional parameters.

	Megestrol Acetate Group			Placebo Group		
	Baseline	3 months		Baseline	3 months	
Weight	58.1 ± 10.7	59.8 ± 9.9	P = 0.003	61.9 ± 7.2	61.1 ± 7	P = 0.100
Albumin (g/dl)	3.25 ± 0.62	3.49 ± 0.68	P = 0.009	3.33 ± 0.57	3.31 ± 0.46	P = 0.608
Cr (mg/dl)	8.5 ± 2.7	9.7 ± 2.5	P < 0.001	9.2 ± 2	8.7 ± 2.3	P = 0.189
Urea (mg/dl)	136 ± 40	161 ± 52	P = 0.067	141 ± 40	139 ± 50	P = 0.853
Kt/V	1.70 ± 0.28	1.67 ± 0.32	P = 0.587	1.79 ± 0.22	1.83 ± 0.22	P = 0.431

Most significantly, an improvement in the parameters related to nutrition status was only observed in the group of patients treated with megestrol acetate.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Hyperkalaemia in hospitalised patients. How to avoid it?

Nefrologia 2014;34(3):417-9

doi:10.3265/Nefrologia.pre2014.Feb.12322

To the Editor,

Hyperkalaemia is a serious electrolyte disorder whose incidence is increasing, especially among the elderly, in which renin release is reduced and to whom drugs favouring hyperkalaemia, such as renin-angiotensin-aldosterone system inhibitors (RSAA) or non-steroidal anti-inflammatory drugs, are regularly administered[1,2,3]. It is not uncommon for several of these drugs to be used simultaneously in a patient, increasing thus the risk of hyperkalaemia[4].

We decided to study the epidemiology of hyperkalaemia in patients admitted to a hospital centre, carrying out a cross-sectional observational prevalence study over a year (01/06/2009-31/05/2010), which included adults with principal or secondary hyperkalaemia diagnosis in the discharge report.

Demographic variables, the origin of hyperkalaemia and trigger factors, among others, were studied.

Hyperkalaemia was considered of domiciliary origin if the patient presented hyperkalaemia on admission and of hospital origin if the potassium readings were within the normal range on admission and hyperkalaemia developed during admission.

Hyperkalaemia was defined as mild if potassium was below 5.9mmol/l, as moderate if it was between 6-6.9mmol/l and as serious if it was above 7mmol/l.

The possible causal factors for hyperkalaemia were reviewed, with a maximum of two from the following: acute renal failure (ARF), administration of potentially hyperkalemic-inducing drugs, potassium supplements, heart failure (HF) or the escape of potassium from cells.

Associated history and predisposing factors for hyperkalaemia included arterial hypertension, chronic HF, chronic kidney disease (CKD), diabetes mellitus, chronic liver disease and volume depletion.

Out of the 11 856 adult patients admitted in the study period, hyperkalaemia diagnosis figured in the discharge report of 96 patients (0.8%). Detection of hyperkalaemia by the laboratory was higher: 26% (3098/11 856) of the patients presented potassium levels above the range (5.1-12.8mmol/l). Hyperkalaemia was mild in most cases (n = 2715 [87.6%]), moderate in 303 (9.8%) and serious in 80 (2.6%).

Of the 96 patients with hyperkalaemia identified in the discharge report, the disease evolved during hospital stay in 32 of the cases (33.3 %). Among the precipitating factors, drugs and/or ARF were responsible for hyperkalaemia in 80.2% of the patients. Mean age was 74 (14.3) [19-97] years and 59.4% were female.

We found an accumulated hyperkalaemia incidence of 0.81% of the admissions in a year. It is an underestimated incidence, given that detection was made based on diagnoses at discharge and many cases of hyperkalemia were not included in the diagnoses in the discharge report. Only 15.4% (59/383) of moderate and serious hyperkalaemia detected by the laboratory (K≥6mmol/l) were included in the discharge reports. This indicates a possibility for improvement, since the discharge report, the mean of communication between various activity levels, must be as complete as possible.

Hyperkalaemia was more frequent in older patients, in diabetics and in patients with CKD, and was frequently of multifactorial origin, combining comorbidity factors and drugs.

76 % of patients with hyperkalaemia were on treatment with a potentially hyperkalaemic-inducing drug, and of these, 54.8 % were taking two or