Dietary Intake, Resting Energy Expenditure, Weight Loss and Survival in Cancer Patients

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ABSTRACT Anorexia, hypermetabolism and weight loss are common in advanced cancer. The progressive wasting may be due to diminished dietary intake as well as to increased energy expenditure mediated by metabolic alterations caused by the tumor. We studied dietary intake, resting energy expenditure and weight loss in 297 patients with generalized malignant disease and their relation to survival. Patients were examined cross-sectionally at entry into an outpatient palliative care program that included anti-inflammatory treatment and nutritional counseling. Survivors were studied longitudinally after 4 mo during palliative care. We found at entry that the patients’ mean dietary intake was low. Weight loss of >10% was present in 43% of the patients, and hypermetabolism was present in 48%. Dietary intake did not differ between normometabolic and hypermetabolic patients, nor was tumor type or gender related to energy and protein intake. Weight loss could not be accounted for by diminished dietary intake alone. Increased resting energy expenditure was not compensated for by an increase in spontaneous food intake. These findings indicate that feedback regulation of dietary intake in relation to energy expenditure is frequently lost in patients with cancer. Hypermetabolism and weight loss were significant predictors of decreased survival. Mean survival time was about 8 mo; 189 patients survived 4 mo or more, and 153 could be reexamined. At the 4-mo follow-up during palliative care, group mean weight was nearly maintained, with large individual variations. Weight loss during follow-up predicted decreased survival. Energy intake increased slightly, also with great variation, and an increased energy intake predicted longer survival.


KEY WORDS: cancer cachexia, dietary intake, energy expenditure, weight loss, survival

Weight loss, anorexia and increased resting energy expenditure (REE) are frequent findings in advanced cancer. The mechanisms of cancer cachexia have been extensively studied but not fully clarified (1). In terms of energy balance, progressive wasting can be attributed to changes in dietary intake, energy expenditure or both, mediated by metabolic alterations. Several aspects of the energy balance equation in advanced cancer are not well known. Although anorexia is very common in progressive cancer disease, we have not found many quantitative measurements of dietary intake in clinical cancer reported in the literature. Increased REE is frequently found, and a large span in REE from hypometabolism to hypermetabolism has been reported in malnourished patients with cancer. Sustained hypermetabolism over a long period of disease progression can make a large contribution to negative energy balance and wasting if not compensated for by an increase in energy intake. Hypermetabolism and diminished energy intake due to anorexia may thus constitute a vicious cycle in the development of cancer cachexia.

We studied dietary intake, REE and weight loss in unselected patients with generalized malignant disease of a solid tumor type, mainly gastrointestinal tumors. Inclusion criteria were generalized malignant disease with a solid tumor type, no other efficient or established tumor treatment available to the patient and expected survival of ≥6 mo. Patients were examined at entry to an outpatient palliative care program that included anti-inflammatory treatment with indomethacin (2), treatment of anemia with erythropoietin (3), dietary advice and nutritional support.

REE by indirect calorimetry, height, weight and weight loss were recorded for 297 patients with cancer. REE was determined in the morning after an overnight fast by indirect calorimetry (Deltatrac; Datex, Helsinki, Finland). Predicted basal metabolic rate was calculated using the Harris-Benedict equation. Patients were classified as hypermetabolic if mea-
that entry, weight loss of >10% was present in 43% of the patients and elevated REE (>110% of predicted) was present in 48%. Mean dietary intake was low: 2.6 ± 10 kcal/(kg · d). Dietary intake did not differ between normometabolic and hypermetabolic patients, nor was tumor type or gender related to energy and protein intake. The proportions of macronutrients were not different from those of a healthy population living in the same area.

Weight loss could not be accounted for by diminished dietary intake because energy intake in absolute amounts was not different and intake per kilogram body weight was higher in weight-losing patients than in weight-stable patients. Thus dietary intake of energy was low. Weight loss and hypermetabolism were frequent and not compensated for by an increase in spontaneous food intake. These findings indicate that feedback regulation of dietary intake in relation to energy expenditure is frequently lost in patients with cancer.

At follow-up, mean survival time was 8 mo. Hypermetabolism and weight loss were associated with decreased survival; 189 patients survived ≥4 mo, of whom 153 could be reexamined. Reasons for nonparticipation at follow-up were mainly unwillingness to participate in further investigations and terminal illness.

At the 4-mo follow-up during palliative care, group mean weight was nearly maintained, with large individual variations (mean difference, –0.3 ± 4.4 kg). Weight loss during follow-up predicted decreased survival. Energy intake increased slightly, also with large variation (mean difference, 146 ± 661 kcal/d). Increased energy intake predicted increased survival. Group mean REE was unchanged (mean difference, 15 ± 162 kcal/d). Hypermetabolism at follow-up remained associated with decreased survival. Thus hypermetabolism and weight loss were associated with decreased survival, whereas an increase in energy intake during follow-up was associated with increased survival.

The metabolic alterations in advanced cancer have many parallels with a chronic systemic inflammatory response and differ considerably from the metabolic changes in starvation (1). Artificial nutrition alone does not appear to affect overall survival in advanced cancer. Appetite stimulants such as high-dose progestins can improve anorexia and, to a lesser extent, promote weight gain, but timing, duration and dosage for optimal therapeutic effect are still unclear (5). Therapeutic strategies aimed at modulating the mediators of the catabolic response, such as cytokines and eicosanoids (6), or metabolic regulation, such as anabolic and anticyclic agents, may offer more promise in the future. Also, early detection and intervention may be more effective. Thus strategies to counteract hypermetabolism and anorexia may be important for the survival of patients with cancer and should be further explored in interventional studies.

LITERATURE CITED