Clinical Nutrition ESPEN 10 (2015) e66–e70

Contents lists available at ScienceDirect

Clinical Nutrition ESPEN

journal homepage: http://www.clinicalnutritionespen.com

Original article

Can we rely on predicted basal metabolic rate in chronic pancreatitis outpatients?☆

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ARTICLE INFO

Article history:
Received 15 September 2014
Accepted 22 December 2014

Keywords:
Chronic pancreatitis
Basal metabolic rate
Nutritional support
Malnutrition

SUMMARY

Background/objectives: Malnutrition is a common complication to chronic pancreatitis (CP) and many patients need nutritional support. An accurate estimation of the basal metabolic rate (BMR) is essential when appropriate nutritional support is to be initiated, but in the clinical settings BMR is cumbersome to measure. We therefore investigated whether BMR can be reliable predicted from a standard formula (the Harris–Benedict equation) in CP outpatients.

Methods: Twenty-eight patients with clinical stable CP and no current alcohol abuse were enrolled. Patients were stratified according to nutritional risk using the Nutrition Risk Screening 2002 system. Body composition was estimated using bioelectrical impedance. BMR was measured using indirect calorimetry and predicted using the Harris–Benedict equation based on anthropometric data.

Results: The average predicted BMR was 1371 ± 216 kcal/day compared to an average measured BMR of 1399 ± 231 kcal/day (P = 0.4). The corresponding limits of agreement were −347 to 290 kcal/day. Twenty-two patients (79%) had a measured BMR between 85 and 115% of the predicted BMR. When analysing patients stratified according to nutritional risk profiles, no differences between predicted and measured BMR were evident for any of the risk profile subgroups (all P > 0.2). The BMR was correlated to fat free mass determined by bioelectrical impedance (rho = 0.55; P = 0.003), while no effect modification was seen from nutritional risk stratification in a linear regression analysis (P = 0.4).

Conclusion: The Harris–Benedict equation reliable predicts the measured BMR in four out of five clinical stable CP outpatients with no current alcohol abuse.

1. Introduction

Chronic pancreatitis (CP) is an inflammatory disease of the pancreas that causes irreversible damage to the pancreatic tissue. As the disease evolves, significant impairment of exocrine and endocrine pancreatic functions becomes evident and affects digestion and absorption of nutrients [1]. However, due to a large reserve capacity of exocrine pancreatic function, exocrine pancreatic insufficiency (EPI) is often a late complication of CP, and its evolution depends on the severity and aetiology of the underlying disease [2,3]. Many patients develop malnutrition early in their disease course and prior to evolution of EPI. Hence, other factors contribute to malnutrition in CP and its pathogenesis is probably multifactorial in most patents [4]. The most frequently reported causes, in addition to EPI, is depletion of nutritional intake due to anorexia secondary to pain, duodenal obstruction or alcoholism, metabolic disorders secondary to diabetes or associated liver diseases and increased metabolic activity due to activity in the underlying disease [5,6]. There is, however, a paucity of data on this important area and more research is needed to understand the complex nature of malnutrition in CP.

As a consequence of malnutrition many patients will require nutritional support during their disease course. It has been

☆ Supported by: Danish Council for Strategic Research, The Danish Agency for Science, Technology and Innovation.

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http://dx.doi.org/10.1016/j.clnesp.2014.12.003

estimated that 10%–15% need oral nutritional supplements, 5% need enteral tube feeding and around 1% will require parenteral nutrition [4,7]. Institution of nutritional support necessitates accurate determination of the individual patient's energy requirements. The basal metabolic rate (BMR) is the rate of energy expenditure at rest and accurate estimation of the BMR is essential when appropriate nutritional support is to be initiated. In clinical practice the BMR is typically predicted from calculations based on the patient's demographic and anthropometric characteristics. The most cited and used predictive formula is the Harris–Benedict equation, which includes age, stature, and body weight to estimate BMR [8]. However, the accuracy of predicted BMR has been questioned in patients with acute and chronic pancreatitis. Accordingly, an increased BMR was previously reported in patients with acute pancreatitis and associated with septic complications, fever and abscesses [9,10]. Furthermore, increased BMR was reported in hospitalised CP patients and in stable outpatient patients with alcoholic CP and a low body mass index (BMI) [11,12]. The underlying cause(s) of increased BMR was, however, not explored in these studies and they did not control for ongoing alcohol abuse, which may significantly increase BMR and thus bias the findings [13].

This study was designed to characterise BMR in clinical stable CP patients with no current alcohol abuse and to investigate the influence of nutritional risk profile on BMR. We hypothesized that the BMR can be reliably predicted from the Harris–Benedict equation. The aims of the study were: 1) To compare measured and predicted BMR in a population of CP patients with clinical stable disease and no current alcohol abuse; and 2) to investigate the influence of nutritional risk profile on BMR.

2. Methods

This was a cross-sectional study conducted at Department of Gastroenterology and Hepatology, Aalborg University Hospital, Denmark. The local ethic committee approved the protocol (N-20120001) and all patients provided written informed consent prior to investigations.

The diagnosis of CP was based on the Mayo Clinic diagnostic criteria – Table 1 [2]. Only patients with clinical stable CP and no signs of acute pancreatitis (i.e. an amylase level below 3 times the normal upper level) or other intercurrent diseases were included [14]. Patients with alcohol consumption above the safe limits recommended by the Danish Health and Medicines Authorities (i.e. >7 units of alcohol per week for women and >14 units of alcohol per week for men) were excluded. Information on patient demographics, aetiology of CP, EPI, diabetes, the use of pain medications, alcohol consumption and smoking habits were obtained from clinical interviews in the outpatient clinic of our institution and through review of the individual patient records.

The Nutrition Risk Screening 2002 system (NRS 2002) was used to determine patients individual nutritional risk profiles [15]. This instrument is a nutritional risk screening system based on four basic questions: recent weight loss, recent food intake, BMI and disease severity. Each question is assigned a score depending on severity of impairment (0 = none; 1 = mild; 2 = moderate; 3 = severe). An additional score of 1 point is added for age above 70 years. A score equal to or greater than 3 indicates a patient at nutritional risk [15]. The NRS 2002 has been validated for nutritional risk screening in various clinical settings and is recommended by the European Society for Clinical Nutrition and Metabolism (ESPEN) guidelines for nutritional risk screening [16].

To ensure best possible accuracy, the same operator (MK) conducted all anthropometric, bio-impedance and BMR measurements. Standing height was measured without shoes to the nearest 0.1 cm using a wall-mounted stadiometer (Seca 222, SE 0123, Seca, Birmingham, United Kingdom). Body weight was measured wearing light indoor clothing without shoes to the nearest 0.1 kg using a digital electronic weight (Seca 701, CE0109, Seca, Birmingham, United Kingdom). BMI was calculated using the actual body weight divided by the square of the height (kg/m^2).

Bioelectrical impedance was measured using a multi-frequency analyzer (BioScan 920-II, Maltron, Essex, United Kingdom) to estimate body composition. Adhesive electrodes were placed in a tetra polar arrangement, on the dorsal surface of the hand, wrist, foot and ankle at the patient's right side and resistance against an alternating electric current was recorded. To ensure accuracy of the measurement, the patient was told to fast for at least 4 h (water was allowed until 2 h before the test), to retain from physical activity within 8 h prior to assessment, to urinate and to lay down in a supine position at a non leading examination couch for 10 min before measurement. The patient was positioned with approximately 45° between the legs and approximately 30° between each arm and the torso. Body composition was reported as fat free mass (FFM) (kg) and fat mass (FM) (kg). To account for inter-individual differences in anthropometry a height adjusted index was calculated dividing the FFM and FM by the squared height (i.e. the FFMi and FMi, respectively).

Basal metabolic rate was measured as indirect calorimetry based on the canopy method (Jaeger Oxycon Pro, CareFusion, San Diego, California, USA). Gas samples were continuously obtained and analysed from a mixing chamber to determine oxygen consumption (VO_2) and carbon dioxide production (VCO_2). BMR for each subject was calculated based on the Weir equation with a pre-set value for urine nitrogen (UN) of 15 g/day [17]: $BMR = [(VO_2 \times 3941) + (VCO_2 \times 1106)] \times 1{,}44 - (UN \times 217)$.

Immediately before each measurement the equipment was calibrated with a gas containing 15.0% oxygen, 5.85% carbon dioxide and 5.85% nitrogen to ensure accuracy and reliability of the results. As the measurement of respiratory gases is only accurate when conducted under standard conditions, the patients was told to fast for at least 8 h, to retain from physical activity within 24 h and to lay down in a supine position for 30 min before measurement. The measurement was carried out in quiet and termoneutral surroundings for approximately 30 min to ensure at least 20 min of stable readings. The patient had to be awake and relaxed before and during measurement. Only patients without fever (i.e. core body temperature <37.5 °C) or other conditions contributing to a disturbance of steady-state BMR were measured.

The predicted BMR was estimated by the revised Harris–Benedict formulation, based on the actual weight and height at the time of BMR measurement [8]:

$$\text{BMR (Men)} = 66.5 + (13.8 \times \text{weight in kg}) + (5.0 \times \text{height in cm}) - (6.8 \times \text{age in years})$$

$$\text{BMR (Women)} = 655.1 + (9.6 \times \text{weight in kg}) + (1.8 \times \text{height in cm}) - (4.7 \times \text{age in years})$$

Table 1
Diagnostic criteria for chronic pancreatitis according to the Mayo Clinic diagnostic criteria [2].

Pancreatic calcifications	4
Histology	4
Characteristic ductal pathology (MRCP or ERCP)	3
Pancreatic exocrine insufficiency	2
Diabetes	1
Clinical symptoms (Recurrent acute pancreatitis, chronic abdominal pain)	2

4 Points ≥ Chronic pancreatitis, MRCP, magnetic resonance cholangiopancreatography, ERCP, endoscopic retrograde cholangiopancreatography.