Original article

Comparison of equations of resting and total energy expenditure in peritoneal dialysis patients using body composition measurements determined by multi-frequency bioimpedance

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SUMMARY

Background & aims: Waste products of metabolism accumulate in patients with kidney failure and it has been proposed that the amount of dialysis treatment patients require be adjusted for energy expenditure. This requires validation of methods to estimate energy expenditure in dialysis patients.

Methods: We compared values of resting energy expenditure (REE) estimated in peritoneal dialysis (PD) patients using a selection of available equations with estimates derived using a novel equation recently validated in chronic kidney disease patients (CKD equation). We also determined the relationship of these estimates of REE and of total energy expenditure (TEE) – which is REE plus physical activity associated energy expenditure (PAEE) estimated using the Recent Physical Activity Questionnaire – to bioimpedance-derived parameters of body composition.

Results: We studied 118 adult PD patients; 75 male (63.6%), 33 diabetic (28.5%), Caucasoid (42.4%), mean age 59.3 ± 18.2 years and weight 73.1 ± 16.6 kg. REE with the CKD equation was 1532 ± 237 kcal/day, which was more than that for Mifflin–St. Jeor 1425 ± 248 kcal/day, but less than Cunningham 1648 ± 248 kcal/day, Bland Altman mean bias ranged from –107 to 111 kcal/day. TEE was 1924 (1700–2262) kcal/day, and on multi-variate analysis was associated with appendicular muscle mass and nitrogen appearance rate (β 34.3, p < 0.001 and β 5.6, p = 0.002, respectively).

Conclusion: With reference to the CKD equation, the majority of standard equations underestimate REE in PD patients. Whereas the Cunningham equation overestimates TEE. REE was associated with appendicular muscle mass and estimated dietary protein intake.

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1. Introduction

The number of patients with chronic kidney disease (CKD) continues to increase worldwide, with more than 300,000 patients currently treated by peritoneal dialysis (PD). Urea is generated from protein metabolism and the amount of dialysis given to patients is assessed by measuring urea clearance, with clinical guidelines advising on minimal weekly urea clearance targets (Kt/Vurea) [1]. However, a number of both observational and randomised multicentre prospective studies have consistently failed to demonstrate that increasing the amount of dialysis, by increasing Kt/Vurea is associated with greater PD patient or technique survival [2–5]. As the products of cellular metabolism accumulate in patients with CKD, an alternative hypothesis has been that the amount of dialysis a PD patient requires should be proportionate to their metabolic activity [6]. To be able to do this we need to develop simple methods of estimating energy expenditure which are valid in patients with advanced CKD.

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Metabolic activity includes both resting metabolic rate and that secondary to physical activity. We have developed an equation to estimate REE in CKD patients and based on this a method to estimate total energy expenditure (TTE — comprising REE plus physical activity energy expenditure (PAEE)) which has been validated in doubly labelled water studies in this setting [7,8]. We wished to compare this chronic kidney disease (CKD) equation in PD patients with other equations estimating resting energy expenditure (REE), which have been developed from other patient populations. In addition, we wished to determine whether there was an association between TEE and REE and bioimpedance measurements of body composition.

2. Patients and methods

Adult PD patients with chronic kidney disease under the care of the University College London partner hospitals were enrolled when they attended for routine outpatient assessments of peritoneal membrane function. Patients with limb amputations, previous strokes, wheelchair bound or those clinically volume overloaded were excluded from study. Paired samples of spent dialysate effluent and serum were analysed by standard methods, and the weekly dialysis dose calculated as Kt/Vurea. Dietary protein intake was estimated by calculating the nitrogen protein accumulate rate (PNA g/day) using the Bergström equation [9], and adjusted for body weight (nPNA) g/kg.

Patient demographics were obtained from computerised hospital records and co-morbidity determined using a self-administered co-morbidity grading [10], based on medical conditions and complications, including diabetes mellitus (as defined by WHO criteria), cardiac disease, respiratory disease, liver disease, arthritis, depression and malignancy [10].

We compared estimates of REE using our CKD equation [7,8], with the modified Harris– Benedict equation [11,12], the Mifflin–St. Joer [13], Katch– Mc Ardle [14] and Cunningham equations [15]. We used the validated Recent Physical Activity Questionnaire (RPQ) to determine physical activity [7]. The RPQ collates information about activities performed during the preceding 4 weeks; including those at home, work and also during leisure time, and also the amount of time spent on each activity. Physical activity data was determined by each reported activity being assigned a Metabolic Equivalent of Task (MET) value according to the Compendium of Physical Activities [16].

Measurements of body composition were made using bioimpedance (InBody 720, InBody, Seoul, South Korea; Body Composition Monitor (BCM), Fresnius, Bad Homburg, Germany). Measurements were performed according to a standard protocol [17,18]. Bioimpedance measurements made by the BCM and InBody were standardised using previously derived equations [19]. Skeletal muscle mass (SMM) was derived from measurement of limb muscle mass (appendicular muscle mass) [20]. Body surface area was calculated using the Gehan and George equation as recommended by the European Best Clinical Practice guidelines [21].

Ethical approval was granted by the UK National Research Ethics Committee — North Wales Essex and all patients signed consent forms in accordance with the e principles of the declaration of Helsinki. Study registration was with the UK Clinical Research Network (portfolio number 12023).

3. Statistical analysis

Statistical analysis was by paired analysis, students’ t tests, or Mann–Whitney U test, ANNOVA or Kruskal–Wallis analysis, with post-hoc adjustment for multiple testing were appropriate, univariate correlation (GraphPad Prism version 6.0, San Diego, USA), and Bland Altman comparison (Analyse-It version 3.0, Leeds, UK). Variables associated with TEE, p < 0.1 were entered into a multi-variable analysis and then eliminated in a step back manner if variables were not significant, unless they improved model fit (SPSS 22.0, SPSS University Chicago, USA). Variables were checked for collinearity. Data are presented as mean ± standard deviation, median (inter quartile range), or mean and 95% confidence intervals (CI), or as a percentage.

4. Results

We enrolled 118 adult PD outpatients; 75 male (63.6%), 33 diabetic (28.5%), with a mean age of 59.3 ± 18.2 years, and a median treatment with PD of 9.4 (3.8–25.5) months. 42.4% were Caucasoid, 29.7% South Asian, 21.1% African/Afro-Caribbean, and 6.9% East Asian. Mean patient weight was 73.1 ± 16.6 kg with a body mass index of 26.0 ± 4.9 kg/m², and a median self-reported co-morbidity score of 2 (0–4). 19.7% had a history of heart disease and 9.4% previous myocardial infarction. 83.9% of the PD patients used automated peritoneal dialysis cycler, 24 h urine volume was 940 (448–1408) ml/day, with a weekly urea clearance (Kt/Vurea) of 2.1 (1.7–2.6), with an estimated nPNA of 0.89 ± 0.25 g/kg/day. Haemoglobin 110.5 ± 4.5 g/l, serum albumin 36.6 ± 5.6 g/l, C reactive protein 5.0 (2–15) mg/l, urea 18.5 ± 5.9 mmol/l, serum creatinine 696 (525–900) mmol/l, and glucose 5.7 (4.9–8.1) mmol/l.

The mean REE using the CKD equation was 1532 ± 237 kcal/day. The REE determined by the CKD equation was significantly greater than that for the Mifflin– St. Joer, and less than that for Cunningham equations (Fig. 1). On Bland Altman analysis, there was bias with the modified Harris–Benedict equation with increasing REE compared to the CKD equation for men with greater REE, and conversely reducing REE for women at greater REE values (Fig. 1). The Cunningham equation significantly over estimated REE compared to all the other equations.

Male patients had greater REE with all equations (Table 1). Apart from the Cunningham equation, all other equations gave lower REE values than the CKD equation (Fig. 1, Table 1), although the difference was only significant with the Mifflin–St. Joer equation for women.

We compared REE derived from the equations and body composition as measured by bioimpedance, and PNA (Table 2). The Katch–Mc Ardle and Cunningham equations use lean body mass (LBW) so accounting for the close association with LBW. All equations were associated with LBW, and SMM, and dietary protein intake as estimated from peritoneal and urinary urea. There was a weaker association with percentage body fat, which was lost when study numbers were reduced by subdividing according to gender.

We then calculated TEE using the RPQ and the CKD equation for REE, and TEE was associated with REE, body size, and muscle mass but not fat mass (Table 3). The association was weaker for body mass index compared to body surface area and muscle mass. On multivariable analysis, excluding REE for collinearity, then only SMM and PNA remained statistically significant (Table 4).

5. Discussion

Products of cell metabolism accumulate in patients with end stage kidney disease. Traditionally urea clearance has been used to assess dialysis adequacy. However, studies increasing urea clearance failed to demonstrate a survival advantage [2–4]. It has therefore been suggested that the amount of dialysis clearance delivered should be adjusted for metabolic rate [6].

A number of equations estimating REE have been developed from various populations, which have generally included healthy subjects of varying body sizes but predominantly of North