ORIGINAL ARTICLE

Postprandial secretion of ghrelin in hemodialysis patients with and without diabetic nephropathy

Piotr Firczyk[1], Witold Ignacy[2], Marcin Adamczak[2], Jerzy Chudek[2], Andrzej Więcek[2]

1 Department of Nephrology and Dialysis Unit, Silesian Hospital, Cieszyn, Poland
2 Department of Nephrology, Endocrinology and Metabolic Diseases, Medical University of Silesia, Katowice, Poland

KEY WORDS

diabetic nephropathy, gastroparesis, ghrelin, hemodialysis

ABSTRACT

INTRODUCTION Ghrelin is a hormone produced mainly by the stomach, which enhances gastric emptying. The disturbances of ghrelin secretion and/or biodegradation may play an important role in the pathogenesis of gastroparesis in hemodialysis patients, especially those with diabetic nephropathy.

OBJECTIVES The aim of the study was to assess postprandial changes in plasma ghrelin in hemodialysis patients with diabetic or non-diabetic nephropathy.

PATIENTS AND METHODS Thirty-two hemodialysis patients (16 with diabetic nephropathy [HD-DM], 16 with non-diabetic nephropathies [HD]) and 15 healthy subjects (C) were enrolled into the study. Plasma ghrelin concentrations were assessed in the fasting state and at 30, 60 and 120 min after ingestion of a test meal. Gastric emptying was assessed based on changes in serum paracetamol concentrations ingested with the meal.

RESULTS In both HD groups fasting plasma ghrelin levels were significantly higher than in C. After ingestion of a test meal plasma ghrelin levels declined mostly at 60 min. In contrast to C, decreased ghrelinemia persisted over 120 min after ingestion of a test meal in both hemodialysis groups. There was no correlation between postprandial reduction in plasma ghrelin at 60 and 120 min and serum paracetamol concentration in HD and HD-DM, while in C decrease in postprandial plasma ghrelin at 60 min correlated with serum paracetamol concentrations.

CONCLUSIONS In hemodialysis patients plasma ghrelin levels are significantly elevated compared to healthy subjects. Prolonged postprandial suppression of ghrelin secretion may contribute to the pathogenesis of gastric emptying disorders in hemodialysis patients. Abnormal postprandial ghrelinemia was observed especially in patients with diabetic nephropathy.

Correspondence to:
Prof. Andrzej Więcek, MD, PhD, FRCP (Edin), Klinika Nefrologii, Endokrynologii i Chorób Przmiany Materii, Śląski Uniwesytet Medyczny, ul. Francuska 20/24, 40-027 Katowice, Poland, phone: +48-32-255-26-95, fax: +48-32-255-37-26, e-mail: awiecek@spskm.katowice.pl
Received: March 1, 2009.
Revision accepted: May 5, 2009.
Conflict of interest: none declared.
Pol Arch Med Wewn. 2009; 119 (7-8): 447-452

INTRODUCTION Ghrelin is a polypeptide hormone with anabolic activity, which is secreted mainly by the stomach.[1] This 28-amino acid peptide increases food intake, simultaneously decreases energy expenditure and stimulates growth hormone release.[2] Ghrelin is an endogenous ligand of growth hormone secretagogue receptor type 1a, distributed in the hypothalamus, pituitary and other segments of the gastrointestinal tract.[3,4] Ghrelin is involved in various functions of the gastrointestinal tract. It increases gastrin and acid secretion in the stomach and modulates gastric motility by increasing the frequency of migrating motor complex. As a result, it accelerates gastric emptying.[5] Plasma ghrelin concentrations are the highest in the fasting state and decrease after food intake.[6]

When kidney function deteriorates, plasma ghrelin levels increase, reaching 2- to 3-fold higher values in patients with stage 5 of chronic kidney disease than in subjects with normal kidney function. This suggests that the kidneys play an important role in the biodegradation of ghrelin.[7,8,9]

Diabetic nephropathy is the most common cause of end-stage kidney disease.[10] Delayed gastric emptying occurs in up to 50% of patients with diabetes.[11] Diabetic gastroparesis may contribute to the development of malnutrition and is

associated with increased mortality in this group of patients in comparison to the general population.[12] The pathogenesis of diabetic gastroparesis has not been fully established. There is a view that impaired gastric emptying, among others, is a consequence of sympathetic nervous system dysfunction (diabetic neuropathy), ischemic degeneration of the smooth muscle cells of the gastrointestinal tract, and hormonal changes (increased serum motilin and cholecystokinin).[13] Similarly to diabetic patients, delayed gastric emptying is a common finding in hemodialysis patients (uremic gastroparesis).[14] To date, the role of ghrelin in the pathogenesis of uremic gastroparesis has not been investigated. Recently, Murray et al. have shown that parenteral administration of exogenous ghrelin enhances gastric emptying in patients with diabetic gastroparesis.[15] Ghrelin analogues may have clinical implications and in the future become a new gastric prokinetic drug. Moreover, Asai et al. demonstrated that plasma ghrelin levels are lower in diabetic patients with gastroparesis than in those without impairment of gastric emptying.[16] In addition, no significant decrease of plasma ghrelin after oral glucose loading during 180 min has been shown in patients with diabetic gastroparesis.[16]

So far, no study has been designed to assess postprandial secretion of plasma ghrelin in hemodialysis patients with chronic renal failure with or without diabetic nephropathy.

PATIENTS AND METHODS Thirty-two hemodialysis patients (16 with a long history of type 2 diabetes and diabetic nephropathy [HD-DM], 16 with other than diabetic nephropathies [HD]) and 15 apparently healthy subjects (C) were enrolled into the study. The underlying kidney diseases in HD patients were: chronic glomerulonephritis (40%), chronic interstitial nephritis (23%), polycystic kidney disease (8%), hypertensive nephropathy (8%), and those of unknown origin (21%). All HD-DM patients were on insulin therapy. Study protocol was approved by Bioethics Committee of the Medical University of Silesia in Katowice. Characteristics of study groups are given in TABLE 1. All hemodialysed subjects were recruited from the Dialysis Unit of Silesian Hospital in Cieszyn. Exclusion criteria were as follows: a past or present history of organic gastric and/or duodenal disorders (diagnosed during panendoscopy), liver and bile duct disease, a history of abdominal surgery (except appendectomy), poor glycemic control, acute infections, pregnancy or lactation. All subjects were asked to discontinue prokinetic drugs or proton pump inhibitors at least 2 weeks prior to the study.

In all subjects blood samples were drawn while fasting and at 30, 60 and 120 min after ingestion of a test meal. The standard test breakfast consisted of: 40 g corn flakes, 250 ml milk, 100 ml fresh grapefruit juice and 5 g sugar (14.4% protein, 12.0% fat, 73.6% carbohydrate; caloric value of 370 kcal [1545 kJ]), and additionally contained 1 g of paracetamol. In all hemodialysis patients study procedures were performed before hemodialysis sessions. Plasma ghrelin, growth hormone, insulin, and glucose were measured in blood samples. Gastric emptying was assessed indirectly based on changes in serum paracetamol concentrations taken with the test meal.[17] Paracetamol absorption takes place in the small intestine but not in

TABLE 1 Clinical and biochemical characteristics of the study groups

	C (n = 15)	HD (n = 16)	HD-DM (n = 16)	HD vs. HD-DM
gender (M/F)	7/8	11/5	7/9	0.29
age (years)	41.3 (34.2–48.3)	60.6[c] (54.2–65.8)	64.7[c] (60.3–69.0)	0.21
duration of hemodialysis (months)	–	28 (9–51)	25 (6–41)	0.41
body mass index (kg/m^2)	25.9 (23.4–28.4)	26.6 (24.5–28.7)	28.9 (26.5–31.4)	0.18
total fat mass (kg)	24.5 (17.9–32.2)	19.7 (15.2–24.1)	24.8 (19.4–30.2)	0.62
serum glucose (mg/dl)	78 (74–83)	81 (77–86)	104[c] (88–119)	0.002
serum insulin (μU/m)	6.6 (4.2–9.0)	8.1 (5.7–10.6)	12.6 (3.3–21.9)	0.81
plasma ghrelin (ng/ml)	4.33 (3.44–5.23)	6.47[b] (5.45–7.48)	5.78[a] (4.64–6.92)	0.17
plasma growth hormone (μIU/ml)	1.16 (0.39–1.93)	3.56[a] (1.83–5.28)	1.64 (0.59–2.70)	0.08

Statistical significance vs. healthy subjects: a $p < 0.05$, b $p < 0.01$, c $p < 0.001$

Abbreviations: C – control, HD – hemodialysis patients with non-diabetic nephropathies, HD-DM – hemodialysis patients with diabetic nephropathy

TABLE 2 Plasma ghrelin, serum glucose, insulin concentrations at 0, 30, 60, 120 min and paracetamol concentration at 15, 30, 60, and 120 min after ingestion of a test meal

		C (n = 15)	HD (n = 16)	HD-DM (n = 16)
plasma ghrelin [ng/ml]	0 min	4.33 ±1.62	6.47 ±1.90	5.78 ±2.14
	30 min	4.04 ±1.24[a]	6.31 ±2.12	5.49 ±2.21[c]
	60 min	3.89 ±1.21[b]	6.20 ±2.14[a]	5.34 ±1.82[b]
	120 min	4.19 ±1.32	6.26 ±2.14	5.38 ±2.07[c]
serum glucose [mg/dl]	0 min	78 ±8	81 ±8	104 ±29
	30 min	110 ±29[c]	139 ±19[c]	181 ±51[c]
	60 min	83 ±21	138 ±23[c]	209 ±59[c]
	120 min	82 ±12	104 ±23[b]	210 ±61[c]
serum insulin [μU/ml]	0 min	6.6 ±4.0	8.1 ±4.5	12.6 ±17.5
	30 min	73.4 ±40.2[c]	39.2 ±28.9[c]	28.6 ±26.3[c]
	60 min	47.5 ±32.5[c]	45.4 ±34.0[c]	33.9 ±28.7[c]
	120 min	21.7 ±18.5[c]	26.6 ±15.8[c]	32.7 ±26.7[c]
serum paracetamol [μg/ml]	15 min	3.53 ±4.57	2.92 ±4.72	4.08 ±6.55
	30 min	7.95 ±4.76[b]	6.17 ±4.21[c]	4.99 ±4.19
	60 min	10.00 ±4.26[b]	8.81 ±2.67[c]	6.26 ±4.27
	120 min	9.12 ±4.49[b]	7.99 ±2.33[b]	6.94 ±2.92

Statistical significance to 0 or 15 min: **a** $p<0.05$, **b** $p<0.01$, **c** $p<0.001$

Abbreviations: see TABLE 1

the stomach, therefore measurement of its serum concentration allows to assess gastric emptying. Serum paracetamol concentrations were measured in the blood samples withdrawn at the previously mentioned time points (at 30, 60 and 120 min after ingestion of a test meal), and additionally 15 min after intake of the test breakfast.

In thawed plasma samples total ghrelin (Ghrelin Ria Kit, Linco Research Inc., USA) and growth hormone levels (BioSource hGH-IRMA, BioSource Europe S.A., Belgium) were measured using radioimmunoassay. Serum insulin (Elecsys, Roche Diagnostics GmbH, Germany) and serum paracetamol levels (AxSYM Paracetamol, Abbott Laboratories Diagnostic Division, USA) were measured using an enzyme-linked immunosorbent assay. Serum glucose in venous blood was determined directly after drawing by a routine enzymatic method in the hospital laboratory.

In all subjects total fat mass was assessed by dual-energy X-ray absorptiometry (DEXA) using a Lunar DPX-L (Lunar Radiation Corporation, USA).

Statistical analysis was performed using Statistica 6.0 software showing results as mean values and 95% confidence interval. The intergroup differences were calculated using the Mann-Whitney U test. The Spearman test was used to assess correlations between variables. A $p<0.05$ was considered statistically significant.

RESULTS Both groups of hemodialysis patients were about 20 years older than C (TABLE 1). There was an overrepresentation of males in HD group and overrepresentation of females in HD-DM group; however, these differences were not statistically significant. Moreover, the HD-DM group had the highest body mass index (BMI) (TABLE 1). In both groups of hemodialysis patients (HD and HD-DM patients) plasma ghrelin concentrations were similar, 49% and 33% higher than in C, respectively (TABLE 1). The adjustment for age did not markedly change ghrelin values (HD 6.3 ±1.8; HD-DM 5.7 ±2.0; C 4.3 ±1.6 ng/ml). As expected, mean serum glucose levels were higher in hemodialiysis patients with diabetes (significantly compared to C alone). Fasting plasma levels of growth hormone were higher in hemodialysis patients (significantly in the HD group alone) when compared to C.

Intake of the test meal (equivalent of standard breakfast) was followed by a decline in plasma ghrelin levels, most evident at 60 min, 7.3, 5.4 and 6.7% in C, HD and HD-DM patients, respectively (TABLE 2, FIGURE). In all groups a reduction in plasma ghrelin levels 60 min after ingestion in comparison to the initial values was observed (significant in HD-DM and C). Suppression of ghrelin secretion persisted longer (over 120 min after ingestion) in both groups of hemodialysis patients, significant only in HD-DM. After 120 min ghrelinemia reached the baseline values only in C.

A postprandial increase of insulin secretion at 30 min was significantly higher in C when compared to both groups of hemodialysis patients (TABLE 2, FIGURE). The highest postprandial insulinemia in hemodialysis patients was delayed and was observed 60 min after ingestion of the test meal. A postprandial increase in insulin secretion lasted even 120 min after meal ingestion. Only in C

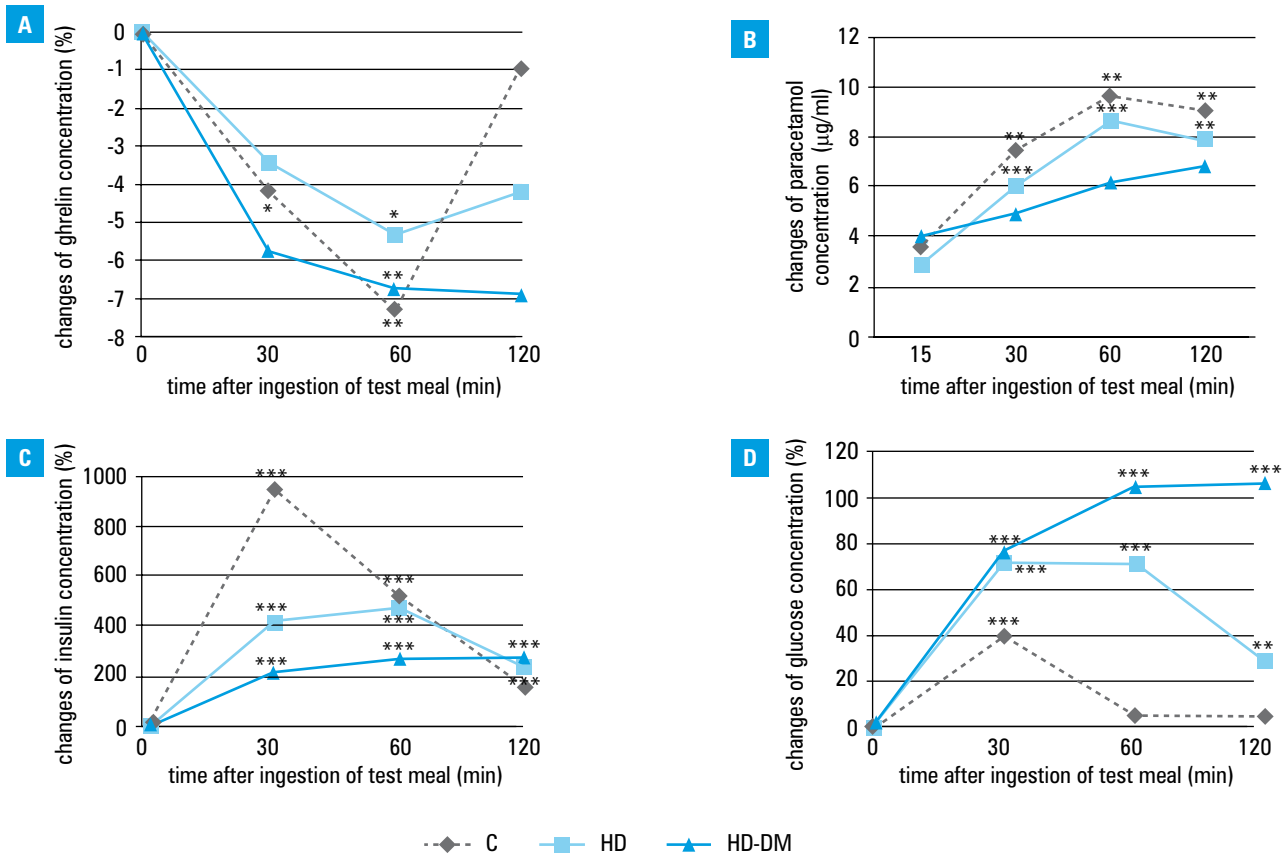

FIGURE Changes in plasma ghrelin (A), serum paracetamol (B), serum insulin (C) and serum glucose (D) in hemodialysis patients with diabetic nephropathy (HD-DM), hemodialysis patients with non-diabetic nephropathies (HD) and healthy subjects (C) at 0, 30, 60 and 120 min after ingestion of a test meal; statistical significance vs. 0 min
* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

the peak of insulin secretion preceded maximum decrease in plasma ghrelin levels.

Plasma growth hormone levels in both groups of hemodialysis patients were higher than in C, significantly only in the HD group. In all study groups postprandial decrease of plasma growth hormone levels was detected at 30 and 60 min, the most pronounced and statistically significant at 60 min in the HD group.

At 15 min after ingestion serum paracetamol concentrations were comparable in all groups. An increase in mean serum paracetamol at subsequent time points was observed until 60 min in C and HD groups, and until 120 min in the HD-DM group (TABLE 2, FIGURE). In individual profile analysis of serum paracetamol concentrations, 50% of HD-DM and only 31% of HD patients achieved the peak concentration at 120 min. In HD-DM patients with delayed paracetamol absorption, there was a more pronounced decline (compared to the baseline values) in plasma ghrelin levels 120 min after ingestion (n = 8) compared to HD-DM patients with normal absorption (n = 8) [(–8.4, 95% CI from –13.2 to –3.6% vs. –5.5, 95% CI from –11.5 to 0.6%, respectively; p = NS].

A significant inverse correlation was found between the value of postprandial plasma ghrelin decline at 60 min and serum paracetamol concentrations in C (R = –0.676, p = 0.001). A similar correlation at 120 min in this group did not reach the statistical significance (R = –0.462, p = 0.11). No correlation was observed between a reduction in postprandial plasma ghrelin at 60 and 120 min and the corresponding concentrations of serum paracetamol in both hemodialysis groups, also in combined analyses (HD + HD-DM).

In the present study fasting plasma ghrelin correlated significantly both with BMI (R = –0.390; p = 0.03) and total fat mass (R = –0.488; p = 0.006) in the combined group of hemodialysis patients (HD + HD-DM). In C, the correlation between fasting plasma ghrelin and BMI alone was close to the level of statistical significance (R = –0.497; p = 0.06). Moreover, there was a significant inverse association between fasting plasma ghrelin and serum insulin levels (R = –0.342; p = 0.05) in the combined hemodialysis patients (HD + HD-DM). In C none of those correlations were significant.

DISCUSSION In the current study we have confirmed previous findings showing that plasma ghrelin levels are elevated in patients with chronic renal failure.[7,8,9] Moreover, results of these studies suggest that the kidneys are an important site for biodegradation/elimination of circulating ghrelin. The values of plasma ghrelin levels obtained in our study were similar to those reported by Iglesias et al.[9] Lower values of plasma ghrelin levels were found by Yoshimoto et al. and Perez-Fontan et al.[7,8] These discrepancies may derive from different methods used for hormone assessment. Moreover, both groups of hemodialysis patients were about 20 years older than the control group. As a plasma ghrelin concentration is decreasing with age, the age difference may in part contribute to a smaller difference in plasma ghrelin levels between hemodialysis patients and the control group than expected.

Ghrelin is an anabolic hormone, thus hyperghrelinemia in patients with chronic kidney disease appears to be inconsistent with protein-

-calorie malnutrition, which is a common finding in uremic patients. The incidence of protein-calorie malnutrition varies from 10 to 70% of hemodialysis patients[18,19] and contributes to increased morbidity and mortality rates in this population.[12] Diminished biological activity of ghrelin in patients with chronic kidney disease may be caused predominantly by an increased concentration of desacyl ghrelin, an inactive fraction of total ghrelin in this population.[7] Another explanation might be increased receptor or postreceptor resistance to endogenous ghrelin action on target organs in patients with chronic kidney disease.

Circulating ghrelin levels have been reported to decrease after meal ingestion. The decrease in postprandial plasma ghrelin concentrations demonstrated in our study indicates that physiological mechanisms of ghrelin secretion after food stimuli are preserved in hemodialysis patients. However, there were considerable differences in postprandial changes in plasma ghrelin levels between hemodialysis patients and healthy subjects: decreased plasma ghrelin levels persisted longer (over 120 min after ingestion of a test meal) in both groups of hemodialysis patients (significant only in hemodialysis patients with diabetic nephropathy), while in healthy subjects ghrelinemia returned to the initial values at 120 min. This delayed suppression of ghrelin secretion after ingestion of a test meal may lead to the development of impaired gastric emptying (gastroparesis) in hemodialysis patients, particularly in those with diabetic nephropathy. The possible participation of disturbed ghrelin secretion in the pathogenesis of diabetic gastroparesis had been previously shown by Asai et al. who demonstrated the lack of ghrelin response to oral glucose in patients with diabetic gastroparesis and normal kidney function.[16] Impaired gastric emptying is observed even in 50% of diabetic patients.[11] Diabetic gastroparesis can be followed by a variety of metabolic complications: poor glycemic control, frequent episodes of postprandial hypoglycemia and, in advanced stage patients, with feeding difficulties and vomiting leading to the development of protein-calorie malnutrition. Prokinetic activity of ghrelin after parenteral administration has been already demonstrated both in animal models and in humans.[15,20] Therefore, ghrelin receptor agonists are a potential prokinetic agent for treatment of gastroparesis and protein-calorie malnutrition.

Plasma ghrelin concentration is inversely related to body mass. Low plasma ghrelin levels are found in obese subjects, whereas in anorectic patients they are markedly higher.[21] The present study demonstrates an inverse correlation between fasting ghrelinemia and BMI, total fat mass and serum insulin concentration in hemodialysis patients. Thus, our results confirm previous findings, suggesting preserved physiological mechanisms of ghrelin secretion in hemodialysis patients. The above mentioned relationships, as well as the inverse correlation between serum paracetamol concentration and decline in plasma ghrelin levels at 60 min postprandially (in healthy subjects) do not allow us to establish mechanisms of ghrelin suppression. The limitation of our study is the lack of assessment of the degree of autonomic neuropathy that could have an important influence on gastric emptying in patients with gastroparesis.

In conclusion, plasma ghrelin levels in hemodialysis patients are significantly higher than in healthy subjects, suggesting disturbed ghrelin biodegradation in uremic patients. Prolonged postprandial suppression of ghrelin secretion may contribute to the pathogenesis of gastric emptying disorders in hemodialysis patients, especially in those with diabetic nephropathy.

REFERENCES

1 Kojima M, Hosoda H, Date Y, et al. Ghrelin is a GH-releasing acylated peptide from stomach. Nature. 1999; 402: 656-660.

2 Wren AM, Small CJ, Ward HL, et al. The novel hypothalamic peptide ghrelin stimulates food intake and growth hormone secretion. Endocrinology. 2000; 141: 4325-4328.

3 Howard AD, Feighner SD, Cully DF, et al. A receptor in pituitary and hypothalamus that functions in growth hormone release. Science. 1996; 273: 974-977.

4 Petersenn S, Rasch AC, Penshorn M, et al. Genomic structure and transcriptional regulation of the human growth hormone secretagogue receptor. Endocrinology. 2001; 142: 2649-2659.

5 Levin F, Edholm T, Schmidt PT, et al. Ghrelin stimulates gastric emptying and hunger in normal-weight humans. J Clin Endocrinol Metab. 2006; 91: 3296-3302.

6 Ariyasu H, Takaya K, Tagami T, et al. Stomach is a major source of circulating ghrelin, and feeding state determines plasma ghrelin-like immunoreactivity levels in humans. J Clin Endocrinol Metab. 2001; 86: 4753-4758.

7 Yoshimoto A, Mori K, Sugawara A, et al. Plasma ghrelin and desacyl ghrelin concentrations in renal failure. J Am Soc Nephrol. 2002; 13: 2748-2752.

8 Perez-Fontan M, Cordido F, Rodriguez-Carmona A, et al. Plasma ghrelin levels in patients undergoing haemodialysis and peritoneal dialysis. Nephrol Dial Transplant. 2004; 19: 2095-2100.

9 Iglesias P, Díez JJ, Fernández-Reyes MJ, et al. Serum ghrelin concentrations in patients with chronic renal failure undergoing dialysis. Clin Endocrinol. 2006; 64: 68-73.

10 U.S. Renal Data System, USRDS 2006 Annual Data Report: Atlas of End-Stage Renal Disease in the United States, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD, 2006.

11 Salaheddin T, Płazińska M, Zagórowicz E, et al. [Gastric emptying disorders in diabetes]. Pol Arch Med Wewn. 2002; 108: 879-88. Polish.

12 Stenvinkel P, Barany P, Heimbürger O, et al. Mortality, malnutrition, and atherosclerosis in ESRD: what is the role of interleukin-6? Kidney Int Suppl. 2002; 80: 103-108.

13 Sokup A, Świątkowski M. [Gastric motor activity disorders in diabetic patients: pathogenesis, clinical picture, diagnosis and treatment]. Przegl Lek. 1998; 55:342-345. Polish.

14 van Vlem B, Schoonjans R, Vanholder R, et al. Delayed gastric emptying in dyspeptic chronic hemodialysis patients. Am J Kidney Dis. 2000; 36: 962-968.

15 Murray CD, Martin NM, Patterson M, et al. Ghrelin enhances gastric emptying in diabetic gastroparesis: a double blind, placebo controlled, crossover study. Gut. 2005; 54: 1693-1698.

16 Asai S, Katabami T, Obi N, et al. No ghrelin response to oral glucose in diabetes mellitus with gastroparesis. Endocr J. 2009; 56: 79-87.

17 Willems M, Quartero AO, Numans ME. How useful is paracetamol absorption as a marker of gastric emptying? A systematic literature study. Dig Dis Sci. 2001; 46: 2256-2262.

18 Cianciaruso B, Brunori G, Kopple JD, et al. Cross-sectional comparison of malnutrition in continuous ambulatory peritoneal and hemodialysis patients. Am J Kidney Dis. 1995; 26: 475-486.

19 Marckmann P. Nutritional status of patients on hemodialysis and peritoneal dialysis. Clin Nephrol. 1988; 29: 75-78.

20 Dornonvilledl C, Lindstrom E, Norlen P, et al. Ghrelin stimulates gastric emptying but is without effect on acid secretion and gastric endocrine cells. Regul Pept. 2004; 120: 23-32.

21 Otto B, Cuntz U, Fruehauf E, et al. Weight gain decreases elevated plasma ghrelin concentrations of patients with anorexia nervosa. Eur J Endocrinol. 2001; 145: 669-73.

ARTYKUŁ ORYGINALNY

Poposiłkowe wydzielanie ghreliny u chorych hemodializowanych z nefropatią cukrzycową i innymi nefropatiami

Piotr Firczyk[1], Witold Ignacy[2], Marcin Adamczak[2], Jerzy Chudek[2], Andrzej Więcek[2]

1 Oddział Nefrologii i Stacja Dializ, Szpital Śląski, Cieszyn
2 Klinika Nefrologii, Endokrynologii i Chorób Przemiany Materii, Śląski Uniwesytet Medyczny, Katowice

SŁOWA KLUCZOWE

gastropareza, ghrelina, hemodializoterapia, nefropatia cukrzycowa

STRESZCZENIE

WPROWADZENIE Ghrelina jest hormonem wytwarzanym głównie przez żołądek, przyśpieszającym jego opróżnianie. Zaburzenia wydzielania i/lub biodegradacji ghreliny mogą uczestniczyć w patogenezie gastroparezy u chorych hemodializowanych, szczególnie z nefropatią cukrzycową.

CELE Celem było porównanie poposiłkowych zmian stężenia ghreliny w osoczu u hemodializowanych chorych z nefropatią cukrzycową i innymi nefropatiami.

PACJENCI I METODY Badanie przeprowadzono u 32 pacjentów hemodializowanych: 16 z nefropatią cukrzycową i 16 z innymi niż cukrzycowa oraz u 15 osób zdrowych. Stężenie ghreliny oznaczano przed oraz po 30, 60 i 120 minutach po spożyciu posiłku testowego. Pasaż żołądkowy oceniano na podstawie pomiaru stężenia paracetamolu w surowicy, podanego podczas posiłku.

WYNIKI W obu grupach pacjentów ghrelinemia na czczo była znamiennie wyższa niż w grupie kontrolnej. Po posiłku stężenie ghreliny obniżało się do wartości najniższych po 60 minutach. W odróżnieniu od grupy kontrolnej, w obu grupach chorych hemodializowanych obniżone stężenie ghreliny w osoczu utrzymywało się jeszcze po 120 minutach od spożycia posiłku. W żadnej z grup pacjentów hemodializowanych nie wykazano zależności pomiędzy wielkością poposiłkowego obniżenia stężenia ghreliny w 60 i 120 minucie i stężeniem paracetamolu w surowicy, podczas gdy u osób zdrowych stwierdzono występowanie znamiennej ujemnej korelacji pomiędzy wielkością poposiłkowego obniżenie stężenia ghreliny w 60 minucie a stężeniem paracetamolu w surowicy.

WNIOSKI Stężenie ghreliny w osoczu u chorych hemodializowanych jest znamiennie wyższe w porównaniu do ghrelinemi u osób zdrowych. Wydłużenie czasu trwania poposiłkowej supresji wydzielania ghreliny może uczestniczyć w patogenezie zaburzeń motoryki przewodu pokarmowego u hemodializowanych chorych. Nieprawidłową poposiłkową ghrelinemię obserwowano zwłaszcza u chorych hemodializowanych z powodu nefropatii cukrzycowej.

Adres do korespondencji:
prof. dr hab. Andrzej Więcek, FRCP (Edin), Klinika Nefrologii, Endokrynologii i Chorób Przmiany Materii, Śląski Uniwesytet Medyczny, ul. Francuska 20/24, 40-027 Katowice,
tel.: 0-32-255-26-95,
fax: 0-32-255-37-26, e-mail: awiecek@spskm.katowice.pl
Praca wpłynęła: 01.03.2009.
Przyjęta do druku: 05.05.2009.
Nie zgłoszono sprzeczności interesów.
Pol Arch Med Wewn. 2009; 119 (7-8): 447-452