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Clinical and biochemical nutritional status among non-cancerous elderly patients with pressure sores

Abstract

Background. Identification of nutritional problems can facilitate strategies which need to be employed in the frail, non-cancerous elderly patient with bed sores at the end of their life. Aim of study was to investigate nutritional status among elderly patients with pressure sores.

Material and methods. Newly admitted 313 elderly patients to Home Hospice For Adults were examined and 42 of them were non-cancerous with pressure sores in sacral region of 10–15 cm in diameter. Pressure ulcers severity was assessed due to Torrance scale and second (PS2), third (PS3), fourth (PS4) and fifth (PS5) stages of pressure sores were categorized. There was no patient with first stage severity. Nutritional status was assessed by Mini Nutritional Assessment-Short Form (MNA-SF). Additionally we assessed complete blood count (CBC), erythrocytes sedimentation rate (ESR), plasma albumin (Alb), lipids and glucose (glu) concentrations, as well as glycated hemoglobin (HbA_{1c}) level.

Results.

1. All patients were malnourished.
2. The PS5 group had the lowest systolic blood pressure (106 ± 9 mm Hg), albumin concentration ($20,4 \pm 5,3$ g/l), the highest red division width (RDW) ($15.3 \pm 1.1\%$) and glucose concentration tendency to drop down.
3. In all 42 persons MNA-SF correlated positively only with glu ($R = 0.54$; $p = 0.0002$) and from PS2 to PS4 subgroups, separately.
4. The positive correlation MNA-SF& Alb was found in PS4 subgroup only.

Conclusions. These data suggest that metabolic status may influence the development and the severity of pressure sores in elderly, non-cancerous, frail patients.

Key words: frail elderly, pressure sores, nutritional status

Adv. Pall. Med. 2011; 10, 2: 73–78

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 Advances in Palliative Medicine 2011, 10, 73–78

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Introduction

The fastest-growing age group in Poland and around the world is the oldest group aged 70 and over [1]. Among these individuals, the prevalence of disability and morbidity is higher than in other groups. We must remember, that the definition of palliative care does not only mean patients with cancer, but also concerns patients with advanced end-stage chronic diseases [2, 3]. The concept of palliative care implicates a team approach to improve quality of life of patients and their families who meet many difficult signs and symptoms related to fatal disease. End-stage of life patients need a multidisciplinary care including nutritional assessment.

Nutritional status, both clinical and biochemical, may change along with age. We may observe clinical and biochemical changes during physiological aging, such as:

- changes in body composition: fat mass increase, muscle mass reduction and water volume depletion;
- plasma glucose concentration rises: fasting glycemia increases about 1–2 mg/dl for every 10 years and postprandial glycemia increases about 2–4 mg/dl for every 10 years after third decade of life;
- plasma lipids concentration (total cholesterol, triglycerides and low density lipoprotein cholesterol) rises in both gender, and high density lipoprotein cholesterol drops down after fifth decade in elderly women (menopause);
- plasma albumin concentration tends to decrease with age but does not reflect hypoalbuminemia [4, 5].

Malnutrition is a common problem in frail, older patients but has received little attention. Some studies suggest that appropriate nutritional support may reduce malnutrition and cachexia syndromes [6, 7]. Malnutrition is associated with an increased prevalence of complications, impaired immunologic function and a high mortality rate. The Mini Nutritional Assessment has been used extensively as a tool to evaluate nutritional status of European population, the United States and worldwide [8, 9].

Malnutrition is the one of main risk factors for the development of pressure sores especially concerning elderly immobilized severely ill persons. It is difficult to calculate the real prevalence of pressure ulcers and contributing risk factors in general. A group of experts estimated an overall prevalence of 9.2% among institutionalized patients but there

are no numbers for the home care patients [10]. Thus the problem may be underestimated.

Identification of nutritional problems can facilitate strategies which need to be employed in the frail, non-cancerous elderly patients with bed sores at the end of their life. The aim of this study was to investigate nutritional status among elderly frail patients with pressure sores.

The study was performed in the Department of Clinical Biochemistry and Laboratory Medicine, Chair of Chemistry and Clinical Biochemistry of Poznan University of Medical Sciences by cooperation with Home Hospice for Adults of Association of Volunteers of Palliative Care in Wielkopolska under the permission from local ethics group in accordance with the Declaration of Helsinki of 1975 for Human Research and the study protocol was approved by the Bioethics Committee of Poznan University of Medical Sciences in Poznan, Poland.

Material and methods

Newly admitted, 313 elderly patients, to Home Hospice for Adults of Association of Volunteers of Palliative Care in Wielkopolska (Poland) were clinically assessed and 42 of them were non-cancerous with sacral pressure sores of 10–15 cm in diameter. The Karnofsky index was calculated 20, including very sick, active support needed patients. Pressure ulcer severity was assessed according to Torrance scale [11] and second (PS2) ($n = 11$, 83 ± 7 years old), third (PS3) ($n = 12$, 83 ± 7 years old), fourth (PS4) ($n = 10$, 78 ± 10 years old) and fifth (PS5) ($n = 9$, 75 ± 15 years old) stages of pressure sores were categorized. There was no patient with first stage pressure sores. We also investigated 32 non-cancerous, elderly patients with no pressure sores who were admitted to Home Hospice.

Nutritional status

Nutritional status was assessed by Mini Nutritional Assessment-Short Form (MNA-SF) [12].

Blood sampling and biochemical analysis

Blood was collected by venous arm puncture.

Biochemical status

The concentration of fasting plasma glucose (glu) and plasma lipids: total cholesterol (T-C), high density lipoprotein cholesterol (HDL-C), triacylglycerols (TAG) were evaluated enzymatically using bio-Merieux reagent kit (France) and the UV-160A Shimadzu spectrophotometer (Japan). Low density li-

poprotein cholesterol (LDL-C) was obtained using Friedewald formula. The reference sera RANDOX Assayed Human Multi Sera Level 1 and RANDOX Assayed Human Multi Sera Level 2 (Randox, United Kingdom) were used for monitoring the accuracy of the determinations. Glycated hemoglobin, HbA_{1c}, level (only non-diabetic patients were included) was measured by High Performance Liquid Chromatography, using D-10 equipment and reagents (Bio-Rad, USA). Plasma albumin concentration was determined automatically by nephelometry on the Nephelometer II (Siemens, USA). Complete Blood Count (CBC) obtained from hematological analyzer Sysmex 4500 (Siemens, USA) and the Erythrocytes Sedimentation Rate (ESR), we measured using Sedivette- Sediplus systems (Sarstedt, Germany)

Statistical analysis

Statistica (version 6.0) for Windows was used for statistical analysis. The normality of value distribution was checked by Shapiro-Wilk's test. Non-parametric Kruskal Wallis followed by Man-Whitney U test was applied to assess the significance of differences between the studied groups. Pearson linear correlation was used to determine the relation between covariants. A $p < 0.05$ was considered statistically significant. Association between the predictor variables were assessed using multiple logistic regression to adjust for potential confounding. The strength of association was measured by the odds ratio (OR) and 95% confidence intervals (CI). The results are expressed as mean \pm standard deviations (SD) and median — in round brackets.

Results

The baseline characteristics and clinical parameters of all investigated patients divided into non pressure sores (non-PS, $n = 32$) and with pressure sores (PS, $n = 42$) are shown in Table 1.

Mini Nutritional Assessment-Short Form (MNA-SF) examination showed that all investigated patients were malnourished with the MNA-SF score lower than 7 points.

As we compared pressure sores patients with non-pressure sores, the non-pressure sores group was younger ($p = 0.023$), had higher waist circumference ($p = 0.0032$), systolic blood pressure ($p = 0.041$) and even malnourished the MNA-SF was better ($p = 0.046$). The complete blood cells investigation showed better ESR ($p < 0.01$), lower WBC count ($p = 0.032$), higher hemoglobin concentration ($p < 0.001$) and lower PLT count ($p = 0.03$) in non-pressure sores vs. pres-

Table 1. Baseline characteristics and clinical parameters of the non-pressure sores and pressure sores groups

	Non-PS (n = 32) Mean \pm SD (median)	PS (n = 42) Mean \pm SD (median)
Age [years]	72.0 \pm 7 (75)	79.9 \pm 10 (82)
Waist [cm]	97.0 \pm 7.9 (92.0)	78.0 \pm 13.0 (74.0)
SBP [mmHg]	135 \pm 15 (130)	121 \pm 20 (120)
DBP [mmHg]	80 \pm 11 (80)	71 \pm 14 (70)
MNA-SF	5.3 \pm 1.6 (5.0)	2.9 \pm 1.6 (3.0)
ESR [mm/h]	16 \pm 17 (11)	46 \pm 31 (38)
WBC [G/l]	6.4 \pm 3.8 (7.5)	9.1 \pm 4.5 (8.0)
RBC [T/l]	4.8 \pm 1.7 (4.5)	4.1 \pm 0.7 (4.2)
HCT [%]	40.5 \pm 8.6 (37.3)	35.2 \pm 5.7 (34.9)
HGB [g/dl]	13.1 \pm 2.0 (13.0)	11.4 \pm 1.9 (11.2)
MCV [fl]	88.6 \pm 7.2 (88.0)	85.3 \pm 7.4 (86.0)
RDW [%]	13.7 \pm 2.0 (14.0)	14.8 \pm 1.1 (15.0)
MCH [pg]	29.9 \pm 0.9 (28.9)	28.0 \pm 3.2 (28.4)
MCHC [g/dl]	35.4 \pm 1.8 (35.5)	32.8 \pm 2.3 (32.9)
PLT [G/l]	252 \pm 55 (240)	361 \pm 142 (358)
Glu [mg/dl]	119.0 \pm 24.5 (120.8)	109.4 \pm 36.6 (98.5)
HbA _{1c} [%]	6.2 \pm 0.3 (6.0)	5.8 \pm 0.8 (5.7)
T-C [mg/dl]	193.0 \pm 13.8 (190.0)	146.7 \pm 28.9 (150.0)
HDL-C [mg/dl]	46.6 \pm 13.2 (48.0)	41.5 \pm 15.5 (39.0)
TG [mg/dl]	88.4 \pm 12.9 (80.0)	120.2 \pm 57.0 (108.0)
LDL-C [mg/dl]	100.0 \pm 18.2 (99.5)	81.1 \pm 23.7 (77.6)
ALB [g/l]	36.8 \pm 5.6 (38.8)	26.6 \pm 6.9 (26.8)

Significant variables are highlighted

sure sores group. Metabolic status in non-pressure sores were better which included: glucose, total cholesterol, HDL-cholesterol, and LDL-cholesterol concentration, ($p = 0.01$, $p = 0.023$, $p = 0.023$, $p = 0.023$, respectively) with lower triglycerides concentration ($p = 0.004$). Plasma albumin concentration was better in non-pressure group ($p = 0.02$).

Among 42 studied persons with pressure sores, 11 of them presented second stage pressure sores (PS2) (83 \pm 7 years old), 12 of them — third stage (PS3) (83 \pm 7 years old), 10 of them — fourth stage (PS4) (78 \pm 10 years old) and 9 of them — fifth (PS5) (75 \pm 15 years old) stage pressure sores. There was no patient with first stage pressure sores. The baseline characteristics and clinical parameters of the pressure sore subgroups are shown in Table 2.

Mini Nutritional Assessment-Short Form (MNA-SF) examination showed that all 42 patients were mal-

Table 2. Baseline characteristics and clinical parameters of the pressure sores subgroups

	PS2 (n = 11) Mean ± SD (median)	PS3 (n = 12) Mean ± SD (median)	PS4 (n = 10) Mean ± SD (median)	PS5 (n = 9) Mean ± SD (median)
Age [years]	83 ± 7 (83)	83 ± 7 (84)	78 ± 10 (79)	75 ± 15 (69)
Waist [cm]	73.1 ± 9.7 (72.0)	83.2 ± 17.1 (80.0)	80.8 ± 11.4 (78.0)	74.9 ± 11.5 (68.0)
SBP [mmHg]	122 ± 17 (120)	125 ± 21 (135)	134 ± 17 (138)	106 ± 19 (100)
DBP [mmHg]	76 ± 12 (80)	77 ± 15 (80)	73 ± 13 (75)	63 ± 14 (65)
MNA-SF	4.9 ± 2.8 (5.0)	2.5 ± 1.5 (2.0)	3.4 ± 3.0 (3.0)	2.7 ± 1.1 (3.0)
ESR [mm/h]	26 ± 27 (15)	48 ± 33 (47)	48 ± 29 (38)	51 ± 25 (48)
WBC [G/l]	7.4 ± 1.8 (6.5)	10.7 ± 7.2 (8.7)	8.4 ± 3.5 (7.2)	9.1 ± 3.4 (7.4)
RBC [T/l]	4.2 ± 0.7 (4.3)	4.2 ± 0.5 (4.3)	4.2 ± 0.8 (4.2)	3.8 ± 0.7 (3.8)
HCT [%]	36.5 ± 6.6 (36.7)	35.2 ± 5.2 (34.3)	36.4 ± 6.4 (36.6)	33.1 ± 4.4 (31.4)
HGB [g/dl]	12.1 ± 2.0 (11.4)	11.4 ± 2.0 (11.6)	12.0 ± 2.3 (12.0)	10.4 ± 1.0 (10.7)
MCV [fl]	86.6 ± 2.7 (86.0)	83.9 ± 9.8 (89.0)	86.9 ± 4.3 (86.0)	85.6 ± 11.3 (85.0)
RDW [%]	14.7 ± 1.2 (14.8)	14.9 ± 0.9 (15.1)	14.2 ± 0.9 (14.0)	15.3 ± 1.1 (15.7)
MCH [pg]	28.9 ± 1.9 (28.2)	27.2 ± 4.1 (28.5)	28.6 ± 2.0 (28.6)	27.9 ± 4.5 (29.8)
MCHC [g/dl]	33.4 ± 2.8 (32.7)	32.3 ± 2.0 (32.6)	32.9 ± 1.6 (33.5)	32.6 ± 2.9 (34.3)
PLT [G/l]	286 ± 95 (251)	374 ± 131 (367)	329 ± 131 (354)	441 ± 181 (428)
Glu [mg/dl]	112.0 ± 28.5 (111.8)	110.4 ± 36.6 (103.1)	128.2 ± 56.1 (115.1)	101.9 ± 20.6 (92.5)
HbA _{1c} [%]	5.7 ± 0.6 (5.6)	6.2 ± 0.8 (6.3)	5.8 ± 0.4 (5.9)	5.6 ± 0.6 (5.4)
T-C [mg/dl]	163.4 ± 23.8 (169.0)	140.3 ± 29.7 (139.0)	151.6 ± 26.8 (153.5)	135.6 ± 24.2 (139.0)
HDL-C [mg/dl]	49.6 ± 12.3 (48.7)	44.8 ± 20.4 (39.6)	37.5 ± 13.1 (35.0)	39.1 ± 14.3 (37.0)
TG [mg/dl]	117.7 ± 41.8 (118.0)	120.3 ± 78.0 (95.0)	118.5 ± 44.2 (112.5)	112.1 ± 51.0 (104.0)
LDL-C [mg/dl]	90.34 ± 21.8 (92.5)	71.4 ± 19.5 (69.5)	90.4 ± 27.3 (92.4)	74.0 ± 14.5 (77.3)
ALB [g/l]	30.7 ± 7.6 (32.8)	26.9 ± 5.0 (26.9)	30.0 ± 7.2 (32.3)	20.4 ± 5.3 (20.8)

Significant variables are highlighted

nourished 3.4 ± 2.4 points (malnutrition criteria < 7 points) and subgroups did not differ significantly: PS2: 4.9 ± 2.8 ; PS3: 2.5 ± 1.5 ; PS4: 3.4 ± 3.0 ; PS5: 2.7 ± 1.1 points.

There were no differences concerning age, waist circumference, ESR, WBC, RBC, HCT, HGB, MCV, MCH, MCHC, PLT, glucose and HbA_{1c} levels, and lipid profile in investigated group.

Kruskal-Wallis test followed by Mann-Whitney U test showed the lowest SBP in PS5 (106 ± 9 mm Hg, $p = 0.021$), the lowest albumin level in PS5

(20.4 ± 5.3 g/l, $p = 0.013$), and changes in red division width (RDW) with the the highest level in PS5 ($15.3 \pm 1.1\%$, $p = 0.021$). Glucose concentration tends to drop down from PS2 to PS5 with the lowest level in PS5 (non significant).

In all 42 persons positive correlation only for MNA-SF&fasting glycemia ($R = 0.54$; $p = 0.0002$) was observed and from PS2 to PS4 subgroups. The positive correlation MNA-SF& Alb was found in PS4 subgroup only, all correlations are presented in Table 3.

Table 3. The important correlations between MNA-SF and metabolic parameters in different stage pressure sore-groups

	PS2	PS3	PS4	PS5
MNA & G 0'	$R = 0.54$; $p = 0.001$	$R = 0.77$; $p = 0.003$	$R = 0.69$; $p = 0.027$	NS
MNA & ALB	NS	NS	$R = 0.796$; $p = 0.006$	NS

NS — no significance

For the multivariate analysis examining associations between pressure sores category, age, nutritional status (MNA-SF) and albumin concentration we formed two groups with better stage pressure sores (PS2 + PS3) and worse pressure sores group (PS4 + PS5). The analysis for age (OR: 1.09, CI: 1.005–1.176, $p = 0.037$), MNA-SF (OR: 1.38, CI: 0.18–0.32, $p = 0.175$) and albumin concentration (OR: 1.04, CI: 0.93–1.15, $p = 0.049$) found that age and plasma albumin are independently associated with the presence of pressure sores severity.

Discussion

In the best developed countries pressure sores are prevented rather (Braden scale, Norton scale) than diagnosed and treated (Torrance scale) [11, 13, 14]. In Poland it is still the tremendous problem especially among bedridden patients at their homes.

Nutritional problems are main challenges in home hospices care. Malnutrition may increase the risk for the development of pressure sores and the duration of diagnosed pressure ulcers and the cost of treatment [15]. Our investigated patients were malnourished with the very low comparable MNA-SF score. Only few researches showed the similar problems in a large group of communities regions among elderly, bedridden patients [7, 9].

Fuoco U. et al. investigated 40 patients, mean age 53.5 years old, with neither cancer nor chronic inflammation diagnosis, with the worst stage of sacral pressure ulcers categorized by National Pressure Ulcer Advisory Panel, in order to determine the pathogenesis of these complications. All of the patients showed mild to moderate anemia with low serum iron and normal or increased ferritin and hypoproteinemia with hypoalbuminemia. Both anemia and hypoproteinemia disappeared after pressure ulcer healing (confirmed 30–60 days after pressure sores healing) [16].

Gengenbacher et al. compared patients with and without pressure sores and their metabolic status. Patients with pressure sores had lower albumin, transferin, hemoglobin, total cholesterol, ferrum and zinc concentration and higher C-reactive protein concentration [17]. Our investigation focused on patients with different pressure sore severity and in those with the worst pressure sore stage (PS5) we confirmed the lowest albumin concentration in comparison with better stages (PS2–PS4) but no other metabolic changes in different pressure sore stages (PS2–PS5) were observed.

Patients at the end of their life need a multidisciplinary overview (physicians, nutritionists, nurses and

pain specialists), followed by proper treatment according to the nutritional status. In palliative care medicine we always ask whether patient should be fed or should not be fed and it is difficult to answer definitely [18]. In palliative care an active role of patient and/or his family in feeding process seems to be crucial. When life expectancy is below 3 months, and the Karnofsky index below 50, the drawbacks of home artificial nutrition are more important than its advantages. According to the American Diabetes Association, the criteria for artificial feeding in those with end stage of their life include: choice of the patient, having previously been informed about the food administration route; evaluation of benefits and risk; and the availability of staff providing enteral and parenteral feeding [19]. The ILSA group (Italian Longitudinal Study on Aging) found that subject with low TC levels (< 189 mg/dL) are at higher risk of dying even when many related factors have been taken into account [20]. Our malnourished patients had had very low total cholesterol, especially in the worst pressure sores group.

Limitation of the study

There are several limitations to this study. First we collected data on a defined group of pressure sores, which could have led us to over/under estimate some results. There were low numbers of investigated patients with different bed sores stages. Although we asked the primary office setting, an academic setting or private practice, there were no patients with the first stage of bed sores by the Torrance scale. Thus we couldn't compare patients from the mildest to worst type of bed sores with malnourishment. Second, inconsistent documentation in the medical record may have caused us to underestimate the number patients with bed-sores with the first stage.

Third, we found no differences in nutritional MNA-SF status (all patients were malnourished) in those without pressure sores vs. with bed sores at the different stage. However, the MNA status was better in those in non-pressure sores group than that in the different stages of bed sores patients.

Conclusions

It is worth to investigate nutrition status among elderly patients at the end of their life. Our data suggest that metabolic status may influence the development and the severity of pressure sores in elderly, non-cancerous, frail patients. The MNA-SF scale seems to be better than laboratory markers,

because the poor nutritional status we may find sooner than biochemicals drop down.

Acknowledgement

We would like to thank doctor Anna Jakrzeska-Sawińska, the director of Home Hospice For Adults Of Association Of Volunteers Of Palliative Care in Wielkopolska we had possibility to performe this work.

No conflict of interest was declared with relation to this work.

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