The clinical impacts of early using glutamine/arginine enriched high protein density formula at trophic dose in intolerant enteral nutrition cachectic hypoalbuminemic hospitalized patients

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Background. We sought to evaluate the clinical impacts of the early administration of trophic doses of a glutamine/arginine enriched enteral nutrition formula (ENF) with a high protein density to cachectic hypoalbuminemic hospitalized patients intolerant to enteral nutrition.

Methods. A retrospective analysis was conducted using the nutritional and non-nutritional data of patients admitted to our institution from April 2017 through August 2019. Patients who died or were discharged before completing ≥ 1 weeks of hospital admission, or those whose data could not be obtained were excluded. Among other variables, percent changes in serum albumin levels (% Δ ALB), C – reactive protein (CRP) and their ratios were expressed as Mean $\pm$ SD using the Independent Samples T-test, while categorical variables were expressed as numbers with percentages by using χ^2 test. Two tested groups were determined based on the use of ENF: Group I received trophic doses of ENF, while Group II received no enteral nutrition.

Results. The overall hospital length of stay (LOS) and overall 28-day hospital mortality were significantly lower in Group I when compared with Group II with Means $\pm$ SDs of (11.32 $\pm$ 2.19 days vs 23.49 $\pm$ 4.33 days) and (13.13% vs. 28.16%), respectively. Also, significantly higher (% Δ ALB) for Group I compared with group II (43.48% $\pm$ 7.89% vs. 33.45% $\pm$ 6.18%), respectively was observed.

Conclusion. In malnourished hypoalbuminemic patients suffering from feeding intolerance, early trophic administration of glutamine/arginine enriched high protein density ENF was well tolerated and may be associated with increased plasma albumin levels, reduced LOS, and overall 28-day mortality, and hence may be considered in such patients.

Key words: enteral nutritional formulas, high protein density, enterocyte nutrients, trophic feeding.

Clinical relevancy statement

The major clinical outcomes of overall length of stay days and mortalities may be improved by early initiation of high protein density formulas that were enriched by conditionally-essential immune-enhancing amino acids of glutamine and arginine. Then results of our study may force the care of severe malnourished critically ill patients and assist in the course of effective treatment.

INTRODUCTION

Feeding intolerance (FI) is commonly encountered in hospitalized patients and has been associated with poor clinical outcomes [1]. The provision of adequate alimentation is most commonly hindered by FI especially in critically ill patients [2, 3]. FI is most commonly defined as gastric residual

volume (GRV) > 150 ml, along with additional gastrointestinal (GI) symptoms [4]. FI prevalence in hospitalized patients is widely variable with approximately of 1/3 in whom receiving enteral nutritional formulas (ENFs) and it is reported as a predictor of mortality which mandates both early and appropriately management [5–9].

The ASPEN, ESPEN, SCCM, and other guidelines on clinical nutrition recommend the early use of enteral nutrition (EN) over parenteral nutrition (PN), when possible, for hospitalized malnourished patients who require nutritional support [10–11]. The premise of this recommendation is that EN reduces infectious morbidity (e.g. PN-associated central line infections and GI associated sepsis) and most other major clinical outcomes [12]. EN, especially in early stage, sustains an intact enteric mucosal barrier by supporting its structural, functional, and immunological elements and prevents dysbiosis which ultimately impedes bacterial translocation

and sepsis [13–19]. Intestinal starvation, with or without PN, has been shown to dramatically increase mucosal permeability and impair mucosal defenses in both animal and human models [20].

EN sustains gut barrier integrity. Structurally, EN maintains mucosal mass by stimulating enterocyte proliferation, preserving villus height, and stimulating the physiologic production of brush border enzymes [17–19]. EN prevents dysbiosis and stimulates secretory immunoglobulin A production, tight junction synthesis and mesenteric blood flow, which results in higher and more efficient mucosal functionality [17]. Early administering EN at TF rate of 10–20 mL/h was found to be sufficient in achieving most of aforementioned positive clinical impacts with less FI when compared with partial or full-energy EF [21–23].

Enterocytes are primarily fed on two major nutrients; short chain fatty acids (SCFAs) and glutamine. SCFAs are the end active metabolite of bacterial fermentation for non-digestible, non-absorbable, but fermentable soluble fibers. These SCFAs production are largely dependent on probiotic fermentation step which is significantly affected by broad spectrum antibiotics that are commonly used in hospitalized patients [24–28]. The ‘conditionally essential’ amino acids (cAAs), glutamine and arginine, have been shown to be effective in the setting of an accelerated catabolism that causes an increase in the cellular consumption of glutamine (the main source of energy for rapidly proliferating cells such as enterocytes, colonocytes, and lymphocytes and the most important enterocyte-nutrient which is directly utilized independent on bacterial fermentation processes) and arginine (An immune-enhancing cAAs in addition to glutamine) [29–34].

ArgiMent® is a new specialized ready to mix (RTM) modular formula (MF) available in our institution and is recently introduced in our nutritional formulary list. When one sachet of ArgiMent® (42.75 g) reconstituted with 120 ml water is yielded approximately a moderate caloric density of 1.2 Cal/ml and very high protein density of 15 g/100 Cal (≈30% from glutamine, ≈30% from arginine, and ≈40% from whey protein) in addition to other Immuno-enhancing and wound healing nutrients of vit C, Zinc, and Copper. Hence, a glutamine/arginine enriched ENF was used in our cohort who would otherwise remain NPO due to FI. We hypothesized that the early administration of 10 ml of glutamine/arginine enriched ENF would attenuate albumin and overall protein catabolism, maintain GI integrity and impeding bacterial translocation, and consequently, improve positive major clinical outcomes of interest.

MATERIAL AND METHODS

We conducted an observational retrospective study at the King Hussein Medical Center (KHMC) of the Royal Medical Services (RMS) in Amman, Jordan. Institutional Review Board (IRB) approval was obtained. Nutritional and non-nutritional data of patients admitted to KHMC from April 2017 through Aug 2019 were collected via our electronic health record (Hakeem®). Inclusion criteria comprised of adult, moderate-severe malnourished patients based on subjective global assessment (SGA) who were intolerant to ENFs regardless of the reason for admission. Exclusion criteria comprised of patients who died or were discharged before completing at least one week of hospital admission, and those whose data could not be obtained. Intolerance to ENF in this study was defined as: A GRV exceeding 150 mL before ENF intake; a GRV that persisted above 150 mL after ENF intake despite the administration of prokinetic (metoclopramide 5–10 mg IV or erythromycin 250 mg Suspension); or intolerable abdominal distention, abdominal cramping, or diarrhea. Patients were divided into a glutamine-arginine enriched ENF group (Group I) and a control group (Group II). The ENF was administered in trophic doses over the course of 16 hours daily for at least 3 days; rates were started at 5 mL/hr and titrated, as tolerated, up to a of maximum 20 mL/hr. Our available ENF that met the aforementioned criteria (ArgiMent®) was used within 3 hours after reconstitution with 120 ml water due to glutamine stability concern. The control, Group II, received no enteral feeding. Primary endpoints were patients’ overall 28-day mortality and hospital LOS. Secondary endpoints included patients’ signs of GI intolerance, changes in GRVs before and after ENF administration, requirements for prokinetics, fistula and overall fluid outputs, hemodynamic parameters, respiratory quotients, changes in Sequential Organ Failure Assessment (SOFA) scores, and blood chemistries of CRP, ALB, and BUN. Quantifiable variables were expressed as Mean ± SD and used the independent samples t-test for analysis, while categorical variables were expressed as percentage (number) and used chi-squared test for analysis. Values were compared for the two tested groups. We conducted all statistical analyses using IBM SPSS v. 25 (IBM Corp., Armonk, NY, USA) and considered P-values ≤0.05 as statistically significant.

RESULTS

Our total study population was 202 adults; 69.8% were males and 30.2% were females. The age of the total sample was 59.05 ± 10.74 years.

Group I had significantly reduced mortality compared with Group II (13.13% vs. 28.16%), respectively. The overall hospital LOS was also significantly shorter in Group I with 11.32 ± 2.19 days vs 23.49 ± 4.33 days for Group II. There were no significant differences in age, body mass index (BMI), body weight (BW), total daily caloric intake (TCI), or fluid outputs (Σ FLUD) between the two tested groups. At baseline, patients in Group I demonstrated significantly lower plasma albumin levels (ALB_1) than Group II (2.27 ± 0.15 g/dl vs. 2.47 ± 0.13 g/dl), respectively. While the plasma albumin levels after receiving trophic doses of ENF (ALB_2) in Group I increased to 3.25 ± 0.06 g/dl, ALB_2 in Group II was also increased to 3.29 ± 0.02 g/dl in Group II, since they received no enteral feeding: yielding significantly higher percent changes in albumin levels (% Δ ALB) for Group I compared with Group II ($43.48\% \pm 7.89\%$ vs. $33.45\% \pm 6.18\%$), respectively. Higher % Δ ALB was associated with significantly lower IV human albumin intake (H.ALB) and significantly lower percent changes of blood urea nitrogen (% Δ BUN) (19.69 ± 1.72 g/day vs 21.17 ± 3.51 g/day) and ($26.29\% \pm 44.16\%$ vs $29.83\% \pm 49.41\%$), for Group I vs. Group II, respectively. The albumin levels are to be considered despite the significantly higher CRP levels in Group I at all stages with % Δ CRP:ALB ratio of $95.27\% \pm 35.49\%$ for Group I vs. $74.68\% \pm 22.08\%$ for Group II. Concerning gastrointestinal (GI) symptoms, there were no significant differences in

the incidence of GI symptoms; measured as the occurrence of ≥ 2 symptoms (e.g. bloating, cramping, elevated GRV, or dyspepsia). Hemodynamically, no significant differences were measured in systolic blood pressure (SBP), diastolic blood pressure (DPB), mean arterial pressure (MAP), heart rate (HR), or shock index (SI). However, modified shock index (mSI) and norepinephrine (NE) requirements were significantly higher in Group II (1.41 ± 0.33 bpm/mmHg vs. 1.68 ± 0.51 bpm/mmHg) and (5.86 ± 5.63 μ g/min vs. 7.42 ± 7.01 μ g/min) for (Group I vs. Group II), respectively. Pertaining to glycemic control, Group II exhibited significantly higher average serum glucose readings (BG_{avg}) than Group I (185.2 ± 3.61 mg/dL vs. 192.3 ± 6.80 mg/dL) with a subsequently higher total daily insulin (TDI) dose (27.25 ± 6.14 IU/day vs. 43.62 ± 15.97 IU/day) yielding higher serum glucose variability (% BG_{var}) $44.14\% \pm 11.49\%$ in Group I and $56.11\% \pm 19.19\%$ in Group II. Although there were insignificant differences in GRVs between the tested groups, the percentage of patients who received prokinetics was significantly lower in Group I 12.1% when compared with Group II 35.9%. Patients in Group I had significantly lower PaCO₂ (37.90 ± 0.77 mmHg vs. 51.92 ± 9.85 mmHg) and a significantly higher PaO₂ (72.92 ± 0.84 mmHg vs. 50.52 ± 10.46 mmHg) than Group II, respectively. Demographics, anthropometrics, nutritional indices, and follow-up comparison data of the study's malnourished hypoalbuminemic hospitalized patients are summarised in Tables 1–2.

Table 1

Baseline and follow-up categorical data of the comparative studied malnourished patient

Variables		Total (N = 202)	Group I (N = 99)	Group II (N = 103)	P-value
Sex	Female	61 (30.2%)	29 (29.3%)	32 (31.1%)	0.452 (NS)
	Male	141 (69.8%)	70 (70.7%)	71 (68.9%)	
Ward	Non-Critical care	94 (46.53%)	46 (46.46%)	48 (46.60%)	0.312 (NS)
	Critical care	108 (53.46%)	53 (53.53%)	55 (53.39%)	
Medical Dx	Medical	88 (43.56%)	43 (43.43%)	45 (43.69%)	0.556 (NS)
	Surgical	114 (56.44%)	56 (56.57%)	58 (56.31%)	
SGA	Moderate wasting	93 (46.04%)	44 (44.44%)	49 (47.57%)	0.412 (NS)
	Severe wasting	109 (53.96%)	55 (55.55%)	54 (52.43%)	
Prokinetic administration	No	153 (75.7%)	87 (87.9%)	66 (64.1%)	0.000 (S)
	Yes	49 (24.3%)	12 (12.1%)	37 (35.9%)	
GI Sx	<2	173 (85.6%)	87 (87.9%)	86 (83.5%)	0.246 (NS)
	≥ 2	29 (14.4%)	12 (12.1%)	17 (16.5%)	
Δ SOFA	<2	135 (66.83%)	74 (74.75%)	61 (59.22%)	0.031 (S)
	≥ 2	67 (33.17%)	25 (25.25%)	42 (40.78%)	
Overall 28-day Mortality	No	160 (79.21%)	86 (86.87%)	74 (71.84%)	0.001 (S)
	Yes	42 (20.79%)	13 (13.13%)	29 (28.16%)	

Data are presented as number (%) using Chi-square test

Group I: Moderate-severe malnourished hospitalized patients used trophic feeding strategy.

Group II: Moderate-severe malnourished hospitalized patients used nothing by mouth strategy.

Dx: Diagnosis

GI Sx: Gastro-intestinal symptoms.

SGA: Subjective global assessment tool.

Δ SOFA: Changes in sequential organ failure assessment.

Table 2

Baseline and follow-up non-categorical data of the comparative studied malnourished patient

Variables	Total (N = 202)	Group I (N = 99)	Group II (N = 103)	P-value
Age (Yrs.)	59.05 ± 10.74	58.81 ± 10.22	59.29 ± 11.27	0.381 (NS)
BW (Kg)	73.97 ± 10.23	74.96 ± 10.08	73.02 ± 10.32	0.178 (NS)
BMI (Kg/m ²)	25.86 ± 3.98	26.43 ± 3.93	25.31 ± 3.96	0.572 (NS)
CRP ₁ (mg/dl)	13.25 ± 4.19	15.40 ± 4.54	11.01 ± 2.14	0.000 (S)
ALB ₁ (g/dl)	2.37 ± 0.17	2.27 ± 0.15	2.47 ± 0.13	0.043 (S)
CRP ₁ : ALB ₁	5.75 ± 2.40	6.95 ± 2.69	4.51 ± 1.11	0.000 (S)
H.ALB (g/day)	20.45 ± 2.87	19.69 ± 1.72	21.17 ± 3.51	0.000 (S)
CRP ₂ (mg/dl)	34.11 ± 13.37	42.38 ± 13.72	25.49 ± 4.88	0.000 (S)
ALB ₂ (g/dl)	3.27 ± 0.05	3.25 ± 0.06	3.29 ± 0.02	0.000 (S)
CRP ₂ : ALB ₂	10.49 ± 4.41	13.12 ± 4.68	7.76 ± 1.51	0.000 (S)
%Δ ALB	38.57% ± 8.68%	43.48% ± 7.89%	33.45% ± 6.18%	0.005 (S)
%Δ CRP	155.8% ± 40.85%	178.2% ± 40.04%	132.5% ± 26.08%	0.000 (S)
%Δ CRP:ALB ratio	85.18% ± 31.37%	95.27% ± 35.49%	74.68% ± 22.08%	0.000 (S)
BUN ₁ (mg/dl)	15.06 ± 5.28	12.59 ± 3.98	17.44 ± 5.31	0.002 (S)
BUN ₂ (mg/dl)	17.60 ± 4.11	14.63 ± 2.37	20.46 ± 3.32	0.018 (S)
%Δ BUN	28.09% ± 46.83%	26.29% ± 44.16%	29.83% ± 49.41%	0.271 (NS)
TCI (Cal/kg/day)	9.48 ± 0.69	9.88 ± 0.52	9.09 ± 0.62	0.045 (S)
TCI (Cal/day)	650.4 ± 78.59	679.1 ± 77.29	622.8 ± 69.78	0.780 (NS)
Σ FLUD	2707.6 ± 415.3	2817.7 ± 406.5	2601.7 ± 397.6	0.984 (NS)
GRV (ml)	144.2 ± 8.34	146.5 ± 7.00	142.1 ± 8.97	0.090 (NS)
SBP (mmHg)	97.80 ± 10.24	100.61 ± 9.52	95.11 ± 10.21	0.743 (NS)
DBP (mmHg)	57.63 ± 7.36	61.27 ± 5.52	54.14 ± 7.24	0.531 (NS)
MAP (mmHg)	68.74 ± 11.45	71.86 ± 10.66	65.74 ± 11.42	0.916 (NS)
HR (bpm)	101.7 ± 10.86	98.23 ± 9.55	105.06 ± 11.02	0.512 (NS)
SI (bpm/mmHg)	1.06 ± 0.23	0.99 ± 0.18	1.13 ± 0.25	0.107 (NS)
mSI (bpm/mmHg)	1.55 ± 0.45	1.41 ± 0.33	1.68 ± 0.51	0.017 (S)
NE rate (μg/min)	6.66 ± 6.41	5.86 ± 5.63	7.42 ± 7.01	0.000 (S)
BG _{min} (mg/dl)	141.2 ± 15.46	144.39 ± 12.57	138.11 ± 17.32	0.084 (NS)
BG _{max} (mg/dl)	236.5 ± 21.49	225.98 ± 9.73	246.64 ± 24.67	0.000 (S)
BG _{avg} (mg/dl)	188.8 ± 6.53	185.x2 ± 3.61	192.3 ± 6.80	0.009 (S)
%BG _{var}	50.25% ± 16.95%	44.14% ± 11.49%	56.11% ± 19.19%	0.024 (S)
TDI (IU/day)	35.52 ± 11.34	27.25 ± 6.14	43.62 ± 15.97	0.000 (S)
Morphine rate (mg/hr)	2.32 ± 1.77	2.48 ± 1.74	2.17 ± 1.79	0.724 (NS)
PaO ₂ (mmHg)	61.49 ± 13.49	72.92 ± 0.84	50.52 ± 10.46	0.000 (S)
FiO ₂	0.53 ± 0.12	0.42 ± 0.01	0.63 ± 0.09	0.000 (S)
PaO ₂ /FiO ₂ ratio	128.5 ± 50.34	172.9 ± 3.98	85.74 ± 34.93	0.000 (S)
PaCO ₂ (mmHg)	44.91 ± 11.45	37.90 ± 0.77	51.92 ± 9.85	0.000 (S)
Hospital (LOS)	17.52 ± 7.00	11.32 ± 2.19	23.49 ± 4.33	0.001 (S)

Data are presented as Mean ± SD using independent t-test.

Group I: Moderate-severe malnourished hospitalized patients used trophic feeding strategy.

Group II: Moderate-severe malnourished hospitalized patients used nothing by mouth strategy.

Bw: Body weight.

BMI: Body mass index.

CRP: C-reactive protein.

ALB: Albumin level.

SI: Shock index.

mSI: Modified shock index.

1: baseline at admission.

2: 1 week after admission.

LOS: Length of stay.

H.ALB: Human albumin 20%.

BUN: Blood urea nitrogen.

TCI: Total calorie input.

GRV: Gastric residual volume.

NE: Norepinephrine.

TDI: Total daily insulin.

PaO₂: partial pressure of oxygen.FiO₂: Fractional inspired of oxygen.PaCO₂: partial pressure of CO₂.

SBP: Systolic blood pressure.

DBP: Diastolic blood pressure.

MAP: Mean arterial pressures.

HR: Heart rate.

BG: Blood glucose.

Min: minimum.

Max: Maximum.

Avg: Average.

Var: Variation.

DISCUSSION

GI impairment often manifests as FI in hospitalized patients, which is associated with malnutrition and poor clinical outcomes [13, 36–38]. In this retrospective analysis, we aimed to explore

the impacts of the early trophic administration of a glutamine/arginine enriched ENF to malnourished ward and ICU patients. Accumulating data investigating the individual roles of EN, early EN (EEN), enteral glutamine, and enteral arginine supplementation on major clinicals outcome have been published.

But this is, to the best of our knowledge, the first study to explore the outcomes of TF using wound healing specialized modular ENF (ArgiMent®) compared to not feeding for at least 3 days and up to 7 days before total parenteral nutrition was initiated.

Our primary findings were a significant reduction in all cause 28-day mortality and hospital LOS for patients who received the trophic doses of glutamine/arginine enriched EEN. Levels of daily caloric input have been reported to influence morbidity and mortality in some patients [37–39] but since our studied groups showed no significant differences in BMI nor TCI (which was balanced by the administration of IV dextrose to Group I) the improved survival could not be attributed to the caloric input associated with EN. Thus, we speculated that the improved outcomes were due to the protective effects of EEN on the GI mucosa and its integrity; impeding bacterial translocation. Our methods did not allow us to measure the rates of bacterial translocation, however, considering that: 1) Group II had a higher percentage of patients who exhibited a ≥ 2 -point increase on the SOFA score over one week 2) Group II had higher rates of vasopressor use, we deduced that Group II may have suffered a higher incidence of sepsis. This comes in accordance with reports of increased rates of Enterobacteriaceae sp. translocation in patients with impaired GI tracts suggesting the impaired GI tract as a possible source of sepsis in patients receiving PN [40–41]. The role of EEN in reducing infectious morbidity by maintaining GI barrier function has been well established. For instance, a meta-analysis of 7 RCTs that investigated the effect of EEN as opposed to early PN found EEN to significantly reduce the risk of infectious morbidity; however, no effect on mortality was recorded in that study [13]. When comparing EEN to delayed EN, a meta-analysis of 21 RCTs reported EEN to significantly reduce mortality, which was likely due EEN's role in maintaining components of the GI mucosal [11]. Glutamine deprivation is detrimental to entero- and colonocytes, since glutamine is their main source of energy [30]. Oudemans-van Straaten *et al.* [33] reported that low plasma glutamine predicted mortality in a cohort of ICU patients. Using glutamine and arginine may have enhanced the speculated protective effects of EN on components of the GI mucosa. A double-blind RCT by Buijs *et al.* [42] investigated the outcomes of the perioperative administration of arginine-enriched EN versus standard EN to severely malnourished patients with head and neck cancer. They reported significant reductions

in both long-term all-cause mortality and disease specific mortality.

Aside from maintaining GI mucosal integrity and immunity, glutamine and arginine have been proven to play a vital role in modulating inflammatory responses and protein metabolism. Glutamine appears to be a key regulator of protein synthesis through its actions on the GCN2 and mTOR pathways. Glutamine also lengthens the half-life of long-lived proteins and stimulates the expression of heat-shock proteins (HSP), HSP-25 and HSP-70, which in turn exert anti-inflammatory effects [30–43]. Arginine also enhances protein In a recent review, Mochamat [44] *et al.* noted that administering perioperative glutamine and arginine in combination with other supplements improved overall survival, reduced infectious morbidity, and increased serum albumin levels in cachectic cancer patients. Our data demonstrated similar outcomes regarding albumin levels. Group I exhibited significantly higher $\% \Delta \text{ALB}$ than Group II despite having significantly higher CRP levels, and receiving significantly lower doses of IV human albumin. However, we observed no significant differences in $\% \Delta \text{BUN}$. While these data suggest positive impacts on protein turnover, further studies are needed to confirm these findings.

Although arginine supplementation has been reported to cause nausea, vomiting, and abdominal cramping [44], the glutamine/arginine-enriched formula was well tolerated by our cohort: First, there were no significant differences in the incidence of GI symptoms among the studied groups. RCTs that reported GI symptoms as an adverse effect of arginine used it in combination with omega-3 fatty acids and RNA; however, when used with glutamine, arginine did not appear to cause such symptoms [44]. Second, Group I had a significantly lower rate of prokinetic use, possibly indicating improved GI motility. This comes in agreement with previous evidence that found both trophic feeding and enteral arginine supplementation to be associated with improved GI motility [14–21]. However, although there were no significant differences in the rates of morphine administration, Group II had significantly higher BG_{avg} and rates of norepinephrine use, possibly delaying gastric emptying and increasing their requirement for prokinetics [37]. Further investigations are required to control for these confounders. Furthermore, administering trophic EEN caused no increments in the GRVs of Group I, which remained, on average, below 150 mL in both groups with no significant differences between them. Regardless

of the differences between the two groups, GRVs below 150 mL do not indicate FI and do not warrant withholding EN [10].

In summary, malnourished hypoalbuminemic patients suffering from FI, early trophic administration of glutamine/arginine enriched high protein density ENF was well tolerated and may be associated with increased plasma albumin levels, reduced risk of bacterial translocation and GIT associated sepsis,

reduced LOS, and overall 28-day mortality, and hence may be initially considered in moderate-severe wasting ENF's intolerant patients. This study is limited by its retrospective design, and using single-centre data. Nonetheless, our centre is an experienced and high-volume unit, so our data may be useful in other centers. A larger, multisite, and prospective study is needed to control for multiple confounders.

Introducere. Obiectivul studiului a fost de a evalua impactul clinic al administrării dozelor trofice de glutamină și arginină pe nutriție enterală (ENF) cu o densitate mare proteică la pacienții cașectici și hipoalbuminemici spitalizați și care nu tolerează nutriția enterală.

Materiale și metode. A fost realizat un studiu retrospectiv pe pacienții eligibili internați între aprilie 2017 și aprilie 2019. Pacienții care au decedat sau care au fost externați în mai puțin de o săptămână de la internare sau cei la care datele au fost incomplete au fost excluși. Au fost analizate modificările albuminei procentual (% Δ ALB) și proteina C reactivă (CRP) iar raporturile acestora au fost evaluate prin medie și deviație standard și comparate folosind teste Student-t. Variabilele nominale au fost analizate utilizând testul χ^2 test. Au fost evaluate două grupuri de pacienți – cei care primeau doze trofice ENF și grupul II care nu primea nutriție enterală.

Rezultate. Durata spitalizării și mortalitatea la 28 de zile a fost semnificativ mai mică la pacienții ENF (11.32 ± 2.19 zile vs 23.49 ± 4.33 zile) și (13.13% vs. 28.16%). (% Δ ALB) a fost semnificativ mai mare la pacienții ENF ($43.48\% \pm 7.89\%$ vs. $33.45\% \pm 6.18\%$).

Concluzii. La pacienții malnutriți și hipoalbuminemici administrarea ENF a fost bine tolerată și s-a asociat cu creșterea nivelului albuminei și scăderea duratei de spitalizare și a ratei mortalității și astfel poate fi luată în considerare pentru acești pacienți.

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