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ACTIVITY OF α-FUCOSIDASE AND β-GLUCURONIDASE IN SERUM AND URINE OF PATIENTS ADMINISTERED PARENTERAL NUTRITION

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ABSTRACT

Background. In hospital patients suffering from adverse clinical and biochemical symptoms of malnutrition, it is often necessary to employ parenteral nutrition to avoid the body's tissue becoming broken down by being metabolised. Thus, the patient's welfare and survival can be supported throughout any periods of medical crisis. Two of the enzymes responsible for metabolising glycoconjugates are α-fucosidase (FUC) and β-glucuronidase (GLU), present in lysosomes. They release fucose or glucuronic acid from the non-reducing end of oligosaccharide chains.

Objective. To determine the effect of parenteral nutrition administered to ill patients, on glycoconjugate metabolism, by measuring serum and urinary activities of FUC and GLU.

Material and methods. Blood samples and the daily urine collection were taken from 23 patients' who had been undergoing parenteral nutrition for either 5 or 10 days, as well as from a baseline sample. Enzyme activities in serum and urine were determined by the method of *Zwierz* et al.

Results. Serum FUC activities were significantly lower after 10 days compared to 5, ($p< 0.0172$), whereas GLU activities were significantly lower after both 5 and 10 days, ($p< 0.0007$ and $p< 0.0208$ respectively), compared to levels before starting parenteral nutrition. GLU activities were however higher after 10 days than those after 5 days, ($p< 0.0023$). In urine, FUC activities were significantly decreased after 10 days compared to 5 days after starting parenteral nutrition, ($p< 0.0245$). Urine GLU activities were unaffected by parenteral nutrition nor was any effect seen on FUC or GLU activities when calculated per 1mg creatinine.

Conclusions. Serum FUC and GLU activities can be used for assessing the effect of parenteral nutrition on glycoconjugate metabolism. The significant decreases of serum GLU activity observed after 5 and 10 days, may serve to indicate that the components of parental nutrition are appropriate and that the body has become suitably adapted to this form of nutrition.

Key words: *serum, urine, α-fucosidase, β-glucuronidase, parenteral nutrition*

STRESZCZENIE

Wprowadzenie. Kliniczne lub biochemiczne objawy niedoborów pokarmowych stwarzają konieczność wdrożenia żywienia pozajelitowego w celu ograniczenia katabolizmu własnych tkanek i stworzenia warunków umożliwiających choremu przetrwanie krytycznego okresu. α-fukozydaza (FUC) i β-glukuronidaza (GLU) są enzymami lizosomalnymi uczestniczącymi w katabolizmie glikokoniugatów. Odcinają fukozę (FUC) lub kwas glukuronowy (GLU) od nieredukującego końca łańcuchów oligosacharydowych.

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Cel . Celem badań było zbadanie wpływu żywienia pozajelitowego na katabolizm glikokoniugatów poprzez ocenę aktywności FUC i GLU w surowicy krwi i moczu chorych żywionych pozajelitowo.
Materiał i metody. Krew z żyły łokciowej oraz mocz z dobowej zbiórki pobrano od 23 pacjentów żywionych pozajelitowo trzykrotnie: przed rozpoczęciem żywienia pozajelitowego, w piątej oraz dziesiątej dobie alimentacji dożylnej. Aktywność FUC i GLU w surowicy krwi i moczu oznaczano metodą kolorymetryczną *Zwierza* i wsp.
Wyniki. W trakcie żywienia pozajelitowego, stężenie aktywności FUC w surowicy krwi uległo istotnemu obniżeniu w dziesiątej dobie ($p<0,0172$), w porównaniu do doby piątej, żywienia pozajelitowego. Stężenie aktywności GLU istotne obniżyło się ($p<0,0007$) w piątej oraz ($p<0,0208$) dziesiątej dobie, w porównaniu do aktywności przed zastosowaniem żywienia pozajelitowego. Stężenie aktywności GLU istotnie rosło ($p<0,0023$) w dziesiątej dobie, w porównaniu do piątej doby żywienia pozajelitowego. Stężenie aktywności FUC w moczu uległo istotnemu obniżeniu ($p<0,0245$) w dziesiątej dobie, w porównaniu do piątej doby żywienia pozajelitowego. Żywienie pozajelitowe nie wpływa istotnie na stężenie aktywności GLU w moczu oraz moczowe aktywności FUC i GLU przeliczane na 1 mg kreatyniny.
Wnioski. Stężenia aktywności FUC i GLU w surowicy mogą być użyte do oceny wpływu żywienia pozajelitowego na katabolizm glikokoniugatów. Istotne obniżenie aktywności GLU w surowicy krwi w 5 i 10 dniu pozajelitowego żywienia, może świadczyć o prawidłowym doborze składników i adaptacji organizmu do żywienia pozajelitowego.

Słowa kluczowe: *surowica krwi, mocz, α-fukozydaza, β-glukuronidaza, żywienie pozajelitowe*

INTRODUCTION

One of the most important achievements in modern medicine has been the widespread use of parenteral nutrition, as a part of the patient treatment [7] whenever dictated by clinical need. Indeed, since the discovery of blood circulation made by *William Harvey* in 1628, the anatomical basis for vein transfusion has now included supplying patients with an adequate nutrition through parenteral means [36]. This type of nutritional support is thus based on providing all the key dietary elements, in the correct proportions, necessary for rapid cellular assimilation in maintaining adequate nutritional status of the human body, in terms of both quality and quantity [24]. In patients undergoing liver transplants, with short bowel syndrome, parenteral nutrition constitutes a life saving treatment [18, 23].

One of the important benefits in adopting parenteral nutrition is in limiting the breakdown of the body's own tissue in providing an energy source, together with assuring appropriate conditions for patients to survive the most critical moments of their illness [29].

Glycoconjugates such as glycoproteins and glycolipids, form an integral part of cellular membranes and interstitial tissue that are metabolised [38, 10] by various lipases, proteases and glycosidases [5, 16]. Exoglycosidases in lysosomes metabolise glycoconjugates by release simple sugar residues starting from the non-reducing ends of oligosaccharide chains [35]. The enzyme α-fucosidase, (FUC - E.C. 3.2.1.51) releases α-fucose [31] from oligosaccharides, whilst β-glucuronidase, (GLU - E.C. 3.2.1.3), in similar fashion, liberates glucuronides through hydrolysis of glucuronic acids linked to alcohols, phenols and carboxylic acids, which take part in the detoxification [3, 40], and hydrolysis of glycosaminoglycans in connective tissue. FUC demonstrates broad substrate specificity including degrading cell and membrane glycoprotein, mucin [1] and the oligosaccharide chains that constitute the surface antigens in blood groups A, B and H [5]. Indeed, lysosomal exoglycosidases have now been extensively studied due to their functions and widespread presence in tissues and bodily fluids [25, 28, 33, 34].

The study aims are to determine the effect of parenteral nutrition on the activity of FUC and GLU enzymes in serum and urine as well as on glycoconjugate metabolism. These could therefore serve as a potential parameters/markers of assessing metabolic changes during parenteral nutrition.

MATERIAL AND METHODS

Subjects were 23 sick hospitalised in-patients, (8 women, 15 men) aged 22-82 years, (mean 57.1 ± 19.37 years), receiving parenteral nutrition at the Department of Surgery and Endocrinology, Bialystok Medical University Hospital. To avoid false positives, patients with kidney or liver disease, diabetes, obesity and alcoholics were excluded as these conditions are known to effect the activity of lysosomal exoglycosidases.

Parenteral nutrition was intravenously administered in an all-in one 24 hour procedure using a infusion pump for maintaining a constant rate of nutrient delivery. The nutrient admixture was made up at the hospital's dispensary and patients received 4 types of nutritional formulae as follows:

a) 15% Aminoplasmal E (1000 ml), 10% Intralipid (500 ml), 20% Glucose (1000 ml), Gensulin R (36j), 15% KCl (40 ml), 20% MgSO4 (10 ml), Addamel (1 vial), Addiphos (1 vial) and Cernevit (1 vial);
b) 10% Aminoplasmal hepa (1000 ml), 10% Intralipid (500 ml), 10% Glucose (500 ml), Gensulin R (36j), 15% KCl (40 ml), 20% MgSO4 (10 ml), Addamel (1 vial), Addiphos (1 vial) and Cernevit (1 vial);

c) 15% Aminoplasmal E (500 ml), 20% Clinoleic (100 ml), 20% Glucose (1000 ml), Gensulin R (36j), 15% KCl (40 ml), 20% MgSO4 (10 ml), Addamel (1 vial), Addiphos (1 vial) and Cernevit (1 vial);
d) 10% Aminosteril KE (500 ml), 20% Clinoleic (100 ml), 20% Glucose (1000 ml), Gensulin R (40j), Vit. B1 (100 mg), 20% MgSO4 (10 ml), Addamel (1 vial), Addiphos (1 vial) and Cernevit (1 vial).

Samples of serum and urine were obtained from patients at 3 time points; before starting parenteral nutrition, (baseline) and 5 and 10 days thereafter. The samples were then spun for 20 minutes at 4000 x g, 4ºC and supernatants were subsequently suitably aliquotted and stored in eppendorf tubes at -80ºC ready for use. Comparing FUC and GLU activities after 5 and 10 days of parenteral nutrition with the baseline thus allowed any effects of the patient's illness *per se* on these enzyme activities to be excluded.

The times of sampling were in the first instance based on recommendations for monitoring parenteral nutrition at its early stages (ie. after 3-5 days) and comparing these with baseline results, thereby allowing organ function to be assessed as well as any modifications to the nutrient formulations to be made whenever required [19]. In addition, studies have shown that after 9-10 days of parenteral nutrition in rats, the activity of lysosomal exoglycosidases changes when measured in liver, kidney and spleen homogenates [20, 22, 32].

Activities of FUC and GLU were measured, in duplicate, by the method of Zwierz et al [39] adapted to the microtitre plate format, (NUNC). For the FUC test, the following were added to each well; 40 µL of 0.1 M phosphate-citrate buffer pH=4.3 and 30 µL 20 mM p-nitrophenyl α-fucopyranoside substrate. In the case of the GLU test, the following were added; 40 µL 0.1 M acetate buffer pH=4.4 and 30 µL 20 mM 4-nitrophenyl β-D- glucuronide substrate, (Sigma St Louis Mo, USA). For both tests, the sample volume of serum or urine was 10 µL. The microplate mixtures were then incubated and mixed for 60 minutes at 37ºC, (Varishacker Incubator, Dynatech). The reaction was stopped by adding 200 µL of 0.2 M borate buffer pH=9.9. The released p-nitrophenol was detected by its absorbance at 405 nm and the actual amounts were calculated from a calibration curve using a microplate reader, (Elx 800 TM, Bio-Tek Instruments, Inc. Vermont, USA). The study had received approval from the Bioethical Commission at the Bialystok Medical University, (No. R-I-003/320/2006).

Statistical analyses were performed by *ANOVA* and results were expressed as means and standard deviations. A value of $p \leq 0.05$ was taken as being significant.

RESULTS

The mean serum FUC activity, (nmol/ml/min) in patients before receiving parenteral nutrition was 11.106 ± 3.136 compared to 10.805 ±4.168 and 9.142 ±3.798 after 5 and 10 days respectively, (Figure 1); the difference between days 5 and 10 being significant at $p< 0.0172$. Also the activities after both 5 and 10 days were decreased when compared to the baseline, (Figure 1). Mean GLU activities in patient's serum were found to be 14.524 ±3.186 at baseline which was significantly higher when respectively compared to 11.791 ± 2.924 ($p< 0.007$) and 14.009 ±2.962 ($p< 0.0208$) after 5 and 10 days of receiving parenteral nutrition, (Figure 2). The GLU activity after 5 days was found to be significantly lower than after 10 days, ($p< 0.0023$), also shown in Figure 2.

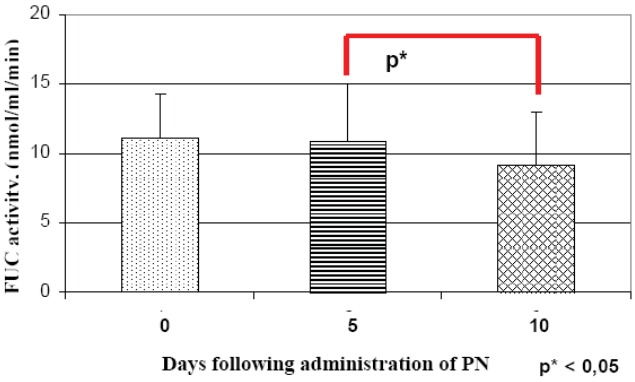

Figure 1. Serum FUC activity in patients receiving parenteral nutrition, (PN).

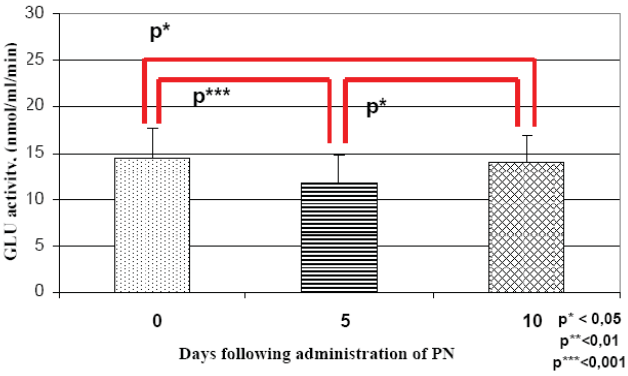

Figure 2. Serum GLU activity in patients receiving parenteral nutrition, (PN).

The corresponding results for urine were a FUC activity, (nmol/ml/min) of 4.644 ±1.961 at baseline and 5.495 ±3.758 and 4.203 ±2.064 after 5 and 10 days respectively of parenteral nutrition, (Figure 3), where the activity after 10 days was significantly lower than after 5 days, ($p < 0.0245$). Urinary GLU activities did not show any significant differences between any of the time points although a small upward trend was noticed as follows 12.609 ±4.893 (baseline), 13.082 ±6.626

(5 days) and 13.722 ±4.975 (10 days); Figure 4. When FUC activity in urine was corrected for creatinine then also there were no significant differences between time points, (nmol/ml/min/mg creatinine) ie. 10.498 ±6.401 (baseline), 11.672 ±9.46 (5 days) and 8.809 ±5.504 (10 days); Figure 5. Furthermore, similar findings for urinary GLU activity were seen with the creatinine correction as follows 28.11 ±13.765 (baseline), 27.435 ±17.074 (5 days) and 28.594 (10 days) as shown in Figure 6. Parenteral nutrition had no effect on urinary creatinine concentration; Figure 7.

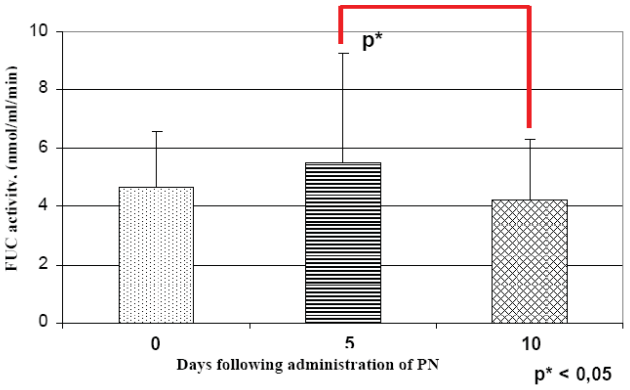

Figure 3. Urine FUC activity in patients receiving parenteral nutrition, (PN).

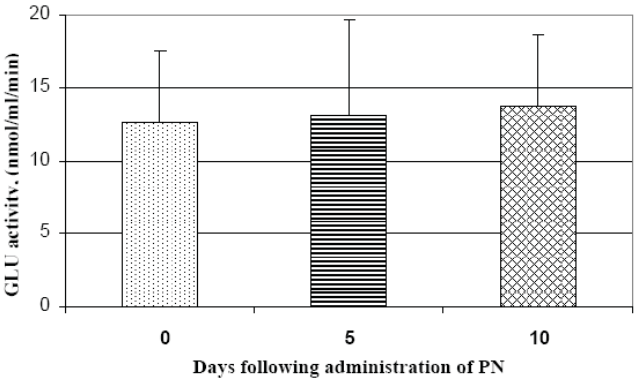

Figure 4. Urine GLU activity in patients receiving parenteral nutrition, (PN).

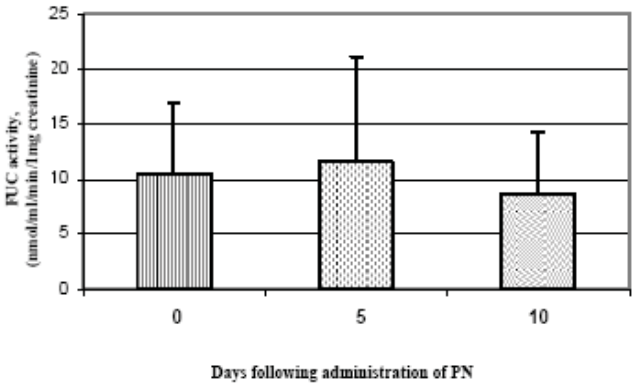

Figure 5. Urine FUC activity per 1mg creatinine in patients receiving parenteral nutrition, (PN).

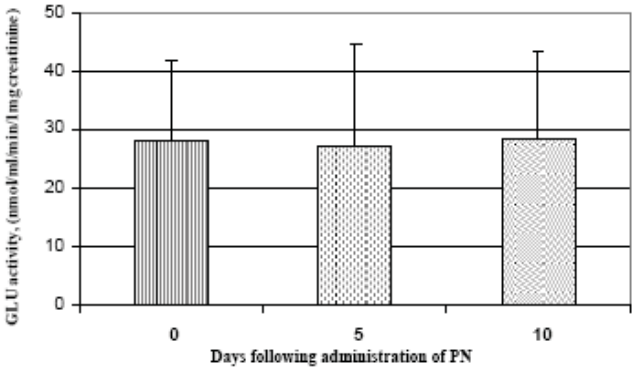

Figure 6. Urine GLU activity in patients per 1mg creatinine receiving parenteral nutrition, (PN).

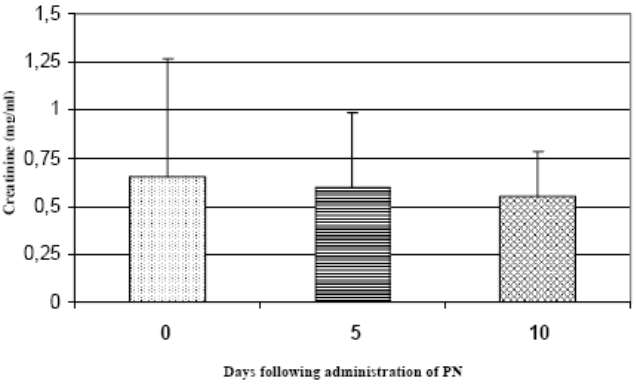

Figure 7. Urine creatinine concentrations in patients receiving parenteral nutrition, (PN).

DISCUSSION

Parenteral nutrition is a relatively new method of clinical treatment if patients are malnourished or at risk of malnutrition whenever intravenous administration of appropriate nutrients are medically required [9]. For instance, it is adopted after surgery when metabolic activity becomes elevated due to the body's response to injury and associated trauma. These factors contribute to an increased basal energy expenditure and protein requirement, mediated by both the immune and neuroendocrine systems [15]. In reaction to pain, blood loss and organ damage, the hypothalamic-pituitary-adrenal axis becomes activated with an increased release of ACTH, growth hormone and prolactin. As a consequence of injury, metabolic changes also occur with increased levels of cortisol, catecholamines and glucagon reflecting the increased basal expenditure of energy [6, 7, 15]. Part of the inflammatory response to surgery and trauma is proteolysis of structural protein, (eg. muscle) effected by the increased secretion of interleukins, (Il-1α, 1β, 6). Many studies have confirmed the relationship between malnutrition and untoward outcomes following surgery. It is seen that patients receiving adequate nutrition are able to better tolerate surgical procedures and recover more rapidly than those that are malnourished [15, 29].

Understanding the biochemical basis of complications arising from parenteral nutrition is vital for ensuring either that prevention is effective, an early diagnosis can be made and that such complications can be treated [2]. Measuring lysosomal exoglycosidase activity of the FUC and GLU enzymes that metabolise glycoconjugates, such as glycoproteins, glycolipids and proteoglycans, [35] can allow changes in their metabolism to be evaluated in sick patients receiving parenteral nutrition. Indeed, the presented study demonstrated some significant changes in serum FUC and GLU activities of such patients, where significant decreases of FUC activity was observed after 10 days of parenteral nutrition compared to 5 days. The study also showed significantly lower serum GLU activity after 5 days of parenteral nutrition compared to baseline, whereas GLU activity after 10 days was significantly higher than after 5 days, (Figures 1 and 2).

These observed changes in lysosomal exoglycosidase activities found in serum may be the result of structural changes in tissue and organs. Animal studies on the short-term effect of parenteral nutrition on rat livers, showed that blood vessels contained stagnant blood, agglomerated leucocytes in capillaries, increased numbers of lysosomes and macrophage fat storage as well as increased activities of enzymes in homogenates such as N-acetyl-β hexose-aminidase (HEX), β galactosidase (GAL), GLU and Cathepsin D [20, 22, 32]. According to studies by *Poriadkova* and *Vasileva* [20, 32], the increased activity of lysosomal enzymes may reflects the body's way of adapting to a deficiency of certain nutrients or is a reflection of lysosomal enzymes involved in the reconstruction of tissues and organs.

Thanks to recent studies, the relationships between pathological changes in cellular structure and biochemically defined disorders of metabolism are now better understood. There has also been an increase in the number of non-invasive and high sensitive methods used for assessing kidney function [12]. Thus, one of study aims was to determine whether changes in urinary FUC and GLU activity occur whenever parenteral nutrition is administered to patients. In normally functioning kidneys, exoglycosidases do not cross the glomerular membrane filter barrier, where proteins > 68 kD are retained in the blood circulation [30]. Nevertheless, in this case both FUC and GLU activity has been detected in urine [26, 27], demonstrating that these enzymes are released therein by other means than cellular breakdown or membrane damage, as resulting from any diseased states. The presence of these enzymes in urine may be due to the natural process of urethral sloughing or an exocytosis of these enzymes from kidney tubules into the urine.

Animal studies on rats receiving parenteral nutrition have shown that after 9 days blood becomes stagnant in vessels, leukocyte infiltrates into connective tissue and glomerular dystrophy and partial sclerosis occurr [20]. These morphological changes may be considered as causing the activities of lysosomal enzymes to change. Studies on human kidney function based on HEX, (N-acetyl-β-D-hexosaminidase) activity in urine, [11, 37] reveal that long-term parenteral nutrition, (> 6 months) lead to increased HEX activity in the urine which is a marker of renal tubule dysfunction. The aetiology of kidney dysfunction during parenteral nutrition can be by necrosis of the glomeruli, excessive protein intake, bacterial infection and the nephrotoxicity caused by certain nutrient components such as chromium and cadmium [4, 14].

A significant decrease in urinary FUC activity was found in the presented study after 10 days of receiving parenteral nutrition compared to 5 days, (Figure 3). In order to account for the state of diuresis affecting enzyme activities, the FUC and GLU results were corrected for urinary creatinine [12]. Concentrations of the latter are directly related to muscle mass and the efficiency of renal function which significantly decrease during fasting as well as in acute or chronic kidney dysfunction [7]. The current study showed that renal excretion of creatinine does not significantly change during when parenteral nutrition is administered at 10 days, (Figure 7). It was found that when FUC and GLU activities are thence so corrected, (per mg creatinine), then activities of these enzymes over this time period are independent of whether parenteral nutrition is administered or not, (Figures 5 and 6). Thus, those patients receiving parenteral nutrition in the short term, exerts only a small effect on renal filtration and the lysosomal metabolism of exoglycosidases present in kidney tubules. At the same time, changes in other organ function in which these enzymes are synthesised, eliminated or degraded cannot be ruled out which may thereby affect their activities in serum and subsequently urine [12].

For surgical operations, the principal aim of nutritional therapy/treatment around this time is to decrease the risk of complications so arising; chiefly post-operative infections, providing metabolic support during surgical induced metabolic stress, accelerating wound healing and a return to normal gastro-intestinal function [17]. When assessing the metabolism of glycoconjugates it might therefore be useful to determine the activities of lysosomal exoglycosidases present in the serum.

CONCLUSIONS

1. Parenteral nutrition decreases glycoconjugate metabolism which is reflected by decreased serum FUC activity after 10 days of its administration. It also significantly decreases serum GLU activity in pa-

tients receiving parenteral nutrition after both 5 and 10 days compared to baseline, where GLU activity was significantly higher after 10 days than 5 days. Urinary FUC activity significantly fell after 10 days of receiving parenteral nutrition compared to 5 days.
2. In order that a return is made to baseline levels of serum GLU activity after 10 days from receiving parenteral nutrition, an appropriately balanced mixture of nutrient components needs to be administered. The body also needs the ability of being able to adapt to this form of nutrition.
3. The administration of short-term parenteral nutrition has little effect on renal filtration and lysosomal exoglycosidases present in kidney tubules.

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Conflict of interest

The authors declare no conflict of interest.

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