

Total Parenteral Nutrition, Metabolic Consequences, Liver Complication and Role of Some Natural Extracts

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Abstract: Liver is negatively affected by using of total parenteral nutrition (T) for long time. It has most recently been found that T is associated with recognizable loss of body weight when diuretic drug (TD) is used to minimize water retention. This has been maximally resolved when green tea was added to parsley extract (TPG). A negative role for T administrations on liver health statues included a remarkable histopathological subclinical disorder such as local monocular inflammatory and vacuolations for application of T alone in addition to dissociation hemolysed RBCs, deposition of golden brown hemosidrine pigment, necrosis of hepatocytes with psychosis of the nuclei and necrosis of sporadic were recorded when TD used. These hepatic cells were partially or even totally recovered upon the intervention with natural extract. This additional extract was also relatively stronger in controlling blood sugar, blood cholesterol, Fe level in serum, liver enzymes, WBCs as well as RBC and kidney function whose reversely affected by T administration alone. In fact, this internal metabolic homeostatic role powered by the natural mixture of extract is an added bioingredient value could be attributed to the complementary action of a unique combination of polyphenols, flavones and vitamins provided by this mixture. In essence, the highly mutual biological intervention appeared for parsley is of important medical role, but the presence of tea solution has magnified its curing effect. Yet, it may concluded that T application cannot be improved using lasix except for water retention, but health complications, especially those of liver, can be avoided by an especial mixture of the natural preparation of parsley and green tea since, tea + parsley extract was an excellent dietary therapy recorded here. Further biological evaluation for T complication recorded for other organs using its effect on their histopathology will appear soon.

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1. Introduction

The concept of feeding patients entirely parenterally by injecting nutrient substances or fluids intravenously was advocated and attempted long before the successful practical development of total parenteral nutrition (T) four decades ago. Realization of this 400 year old seemingly fanciful dream initially required centuries of fundamental investigation coupled with basic technological advances and judicious clinical applications (Dudrick, 2009). Most clinicians in the 1950's were aware of the negative impact of starvation on morbidity, mortality, and outcomes, but only few understood the necessity for providing adequate nutritional support to malnourished patients if optimal clinical results were to be achieved. The prevailing dogma in the 1960's was that, "Feeding entirely by vein is impossible; even if it were possible, it would be impractical; and even if it were practical, it would be unaffordable." Major challenges to the development of T included: (1) formulate complete parenteral nutrient solutions (did not exist), (2) concentrate substrate components to 5–6 times isotonicity without precipitation (not easily

done), (3) demonstrate utility and safety of long-term central venous catheterization (not looked upon with favor by the medical hierarchy), (4) demonstrate efficacy and safety of long-term infusion of hypertonic nutrient solutions (contrary to clinical practices at the time), (5) maintain asepsis and antisepsis throughout solution preparation and delivery (required a major culture change), and (6) anticipate, avoid, and correct metabolic imbalances or derangements (a monumental challenge and undertaking). This presentation recounts approaches to, and solution of, some of the daunting problems as really occurred in a comprehensive, concise and candid history of parental nutrition. (Dudrick, 2009). Practically, and in connection of some organ health, the influence of TPN was studied in 67 patients with severe acute pancreatitis having three or more criteria. Although TPN has been reported to not be of benefit in the progress and severity of the disease, it has found that the time T is started is important in influencing the course of the disease and in the development of local complications, as well as in the mortality rate. Patients whose TPN was started within the first 72 hours of the disease had a 23.6%

complication rate and 13% mortality, in comparison with patients whose TPN was started later in the course of the disease, who had a 95.6% complication rate and a mortality rate of 38% (Kalfarentzos et al., 1991). The nutritional status of the patients during T administration of 28.4 days was maintained either steady or was improved, as assessed by nitrogen balance, serum levels of transferrin, and albumin. The administration of fat solution derangement in protein metabolism with the standard T solution in current use suggests that either a modification of amino acid composition or an increase in total energy to protein energy ratio in T solution may be necessary to obviate such a consequence either to prevent essential fatty acid deficiency or to provide part of the caloric requirements, was found to cause neither clinical nor laboratory worsening of the disease. All pancreatic fistulae that developed during the course of the disease spontaneously closed in patients

receiving TPN without operation in a mean period of 33.3 days, and all pseudocysts subsided in an average of 18.3 days. Those who died (overall mortality rate 24%) had had uncontrollable sepsis, which resulted in hypercatabolism and multiple system organ failure (Kalfarentzos et al., 1991). Therefore, to assess the effect of (TPN) on macronutrient metabolism in obstructive jaundice, forty adult mongrel dogs were equally divided into four groups: group I (PO-control) received sham ligation of common bile duct (CBDL) and was fed dog chow and water ad libitum; group II (PO-CBDL) underwent CBDL and was fed dog chow; group III (T-control) received sham CBDL and TPN ; and group IV (T-CBDL) underwent CBDL and received TPN. Blood chemistries, plasma amino acids and liver histologies were studied before (Day 1) and at the end (Day 14) of the experiment.

Table (1): The chemistry and usage of T (Cited from Hayes et al. 2000).

Nutrient	Amount
Water (kg body wt/day)	30–40 mL
Energy* (kg body wt/day): Medical patient	30 kcal
Postoperative patient	30–45 kcal
Hypercatabolic patient	45–60 kcal
Amino acids (kg body wt/day): Medical patient	1.0 g
Postoperative patient	2.0 g
Hypercatabolic patient	3.0 g
Minerals: Acetate/gluconate	90 mEq
Calcium	15 mEq
Chloride	130 mEq
Chromium	15 µg
Copper	1.5 mg
Iodine	120 µg
Magnesium	20 mEq
Manganese	2 mg
Phosphorus	300 mg
Potassium	100 mEq
Selenium SOME TRADE NAMES	100 µg
Sodium	100 mEq
Zinc	5 mg
Vitamins : Ascorbic acid	100 mg
Biotin	60 µg
Cobalamin	5 µg
Folate (folic acid)	400 µg
Niacin some trade names niacorniaspanslo-niacin	40 mg
Pantothenic acid	15 mg
Pyridoxine	4 mg
Riboflavin	3.6 mg
Thiamin	3 mg
Vitamin A	4000 IU
Vitamin D	400 IU
Vitamin E	15 mg
Vitamin K	200 µg

*Requirements for energy increase by 12% per 1° C of fever.

A significant elevation of bilirubin and alkaline phosphatase was observed in dogs with

CBDL. Blood glucose was not changed significantly in any group. Significant increases in triglyceride and

cholesterol were present in CBDL dogs. Significant differences in the concentrations of a few plasma amino acids, including an elevation of phenylalanine, were found in TPN dogs. A significant increase in aromatic amino acids (AAA) and a noticeable depression of the molar ratio of branched-chain amino acids (BCAA) to AAA was present in T-CBDL dogs, as was a significant increase in blood ammonia. In the presence of obstructive jaundice, TPN does not significantly affect carbohydrate or lipid metabolism (Chuang et al., 1995). However, a derangement in protein metabolism with the standard T solution in current use suggests that either a modification of amino acid composition or an increase in total energy to protein energy ratio in T solution may be necessary to obviate such a consequence (Chuang et al., 1995). Amino acids, as

mentioned before, are essential for enterocytes, but the luminal supply is compromised with changes in dietary intake. The importance of maintaining amino acid supply for intestinal mucosal cells is illustrated (Howard et al., 2004). Previously lipid emulsions were given separately but it is becoming more common for a "three-in-one" solution of glucose, proteins, and lipids to be administered (Rollins et al., 1990 and Didier et al., 1998). In most hospitals, clinical pharmacists evaluate the patient's individual data and decide what T formula to be used. The use of standardized T is cost effective and may provide better control of serum electrolytes, e.g. in Tables 1 and 2 (Hayes et al., 2000). Standardized solutions may also differ between developers (Howard et al., 2004).

Table 2. Examples of T solutions (after Hayes et al., 2000)

Substance	Normal patient	High stress	Fluid-restricted
Amino acids	85 g	128 g	75 g
Dextrose	250 g	350 g	250 g
Lipids	100 g	100 g	50 g
Na ⁺	150 mEq	155 mEq	80 mEq
K ⁺	80 mEq	80 mEq	40 mEq
Ca ²⁺	360 mg	360 mg	180 mg
Mg ²⁺	240 mg	240 mg	120 mg
Acetate	72 mEq	226 mEq	134 mEq
Cl ⁻	143 mEq	145 mEq	70 mEq
P	310 mg	465 mg	233 mg
MVI-12	10 mL	10 mL	10 mL
Trace elements	5 mL	5 mL	5 mL

Still TPN is an important way of nutrition when no food is given by other injection routes (Kozier et al., 2004) and long-term TPN is occasionally used to treat people suffering the extended consequences of an accident, surgery, or digestive disorder. TPN has extended the life of children born with nonexistent or severely deformed organs. Surgically, the effect of preoperative TPN on morbidity and mortality was studied in medical records of discharged surgical patients. Nutrition parameters measured included serum albumin, total lymphocyte count, hemoglobin, weight, and percent weight loss. Major septic complications (MSC) considered were intra-abdominal sepsis, wound dehiscence, septicemia, and pneumonia (Grimes et al., 1987). Other complications included respiratory failure, congestive heart failure, fistulas, urinary tract infection, shock, and death. The experimental group showed improvements after surgery in the nutritional parameters listed and had a lower incidence of morbidity and mortality. Although TPN is sometimes a must, Ahmed et al., (2009) stated that the TPN by its slandered solution complication on rat's main blood constituents is vital. In connection to liver

health, a recent small-scale study at Children's Hospital Boston on the cause of liver failure suggests it may be due to a large difference in omega-6 to omega-3 ratio and some patients were able to recover from their condition (Gura et al., 2006). Two related complications of TPN are venous thrombosis and rarely priapism TPN, in addition, increases the risk of acute cholecystitis due to complete abusage of gastrointestinal tract, which may result in bile stasis in the gallbladder. Other potential hepatobiliary dysfunctions include steatosis, steatohepatitis, cholestasis, and chole-lithiasis (Quigley et al., 1993). Such complications are suggested to be the main reason for mortality in people requiring long-term T, such as in short bowel syndrome (Vanderhoof and Langnas, 1997). Although TPN was considered to be a dangerous form of therapy, critical review of the data suggests that, in humans, TPN does not cause mucosal atrophy or increase bacterial translocation. Increased sepsis with TPN can be ascribed to overfeeding and the dangers of T-induced complications have been exaggerated. TPN is an equally effective alternative to enteral nutrition (EN) when a risk of malnutrition is present and EN is not

tolerated or when gut failure is present (Jeejeebhoy, 2001). As a background, food in the intestine drives the enterohepatic circulation of bile components. To investigate whether parenteral or enteral delivery of nutrients alters serum and biliary lipids in critically ill patients in an intensive care unit (ICU), patients who had received ≥ 5 d of T were compared with those who had fasted for ≥ 5 d. Both groups were studied before and after 5 d of EN. Each patient served as his or her own control. Duodenal bile was analyzed for biliary lipid content and serum lipids were determined simultaneously. Duodenal bile samples from 18 healthy persons were served as controls. As a result, bile salt concentrations in all ICU patients were 17% of control values before EN and 34% of control values after 5 d of EN. Phospholipids concentrations were 12% of control before EN, but increased almost 4-fold after EN. Biliary cholesterol concentrations were 20% of control values before EN and did not improve afterward. No difference in bile composition was observed between fasted ICU patients and those who received T. The inverse correlation between the severity of illness and biliary lipid concentrations observed before EN disappeared with enteric stimulation. The low serum concentrations of HDL cholesterol and apolipoprotein A-I increased significantly with EN in all ICU patients. In conclusion, lack of EN during critical illness was associated with profound decrements in biliary lipid concentrations that normalized partially after 5 d of EN. Therefore, it hypothesize that loss of enteric stimulation in ICU patients impairs hepatic lipid metabolism (De Vree *et al.*, 1999). Moreover, Raina *et al.* (2000), in an animals treated with tumor necrosis factor α (TNF- α), noticed developed severe metabolic abnormalities despite receiving sufficient protein and energy by T. It has been sought that it is the nutritional and metabolic effects of bacterial lipopolysaccharide (LPS) in rats. In brief, biochemical abnormalities and plasma corticosterone concentrations were greater in the T+LPS group than in the other groups. These data suggest that provision of sufficient protein and energy by T does not prevent general carcass wasting induced by LPS but may protect individual muscles. However, compared with an oral ad libitum diet, T providing sufficient protein and energy worsens the biochemical abnormalities induced by LPS. More rapid clearance of TNF- α and low corticosterone concentrations in weight-losing animals may help reduce the severity of the metabolic effects of LPS. Earlier, hyperglycemia was noticed as common at the start of therapy, but can be treated with insulin added to the T solution. Most serious of which is liver failure, often related to fatty liver that may sometimes occur. Liver dysfunction can be limited to a

reversible cholestatic jaundice and to fatty infiltration demonstrated by elevated transaminases. Severe hepatic dysfunction is a rare complication (Horattas, *et al.*, 2001). Typical T induced fatty liver tissue is shown in the figure below.

From the nutritional point of view, although T is a nutritional tool to keep someone who is under critical condition alive, there is great need to biologically control its clinical and nutritional complications. As a result, hence T responses on rat's main blood chemistry, organs functions and blood cells were explored to be somewhat biologically remarkable Ahmed *et al.*, (2009), the reverse action on some organs histology is necessary to measure these organs longevity upon T long term application. Interaction of some plant extract rich of polyphenols was assessed. As an example, diets rich in fruits and vegetables (FV) have been associated with a reduced risk of chronic disease, including cardiovascular disease. Unfortunately, public health campaigns to increase FV intake have had limited success. None of the studies reported any serious adverse effects. However, health advantages on markers of inflammation, immunity, and endothelial function are promising. Limitations of the available studies were related to the diversity of studies conducted with respect to design and study population and the variability in the measured outcomes and assays utilized. While mixed FV supplements may serve as an efficacious complement for individuals who have difficulty achieving their daily FV intake requirement, further research on additional retail preparations is warranted (Esfahani *et al.*, 2011). Based on the antioxidant activity of tea, which was recognized earlier, effect of green tea and black tea have been shown to reduce blood glucose and blood triglycerides. The green tea was higher than that of black tea in the aged rats but that the antioxidative ability of black tea was better than that of green tea in the aged rats (Zeyuan *et al.*, 1998). Furthermore, antioxidant Activity of various tea extracts in relation to their antimutagenicity was conjugated to the electrocatalytic oxidation of (-)-epigallocatechin gallate (EGCG), the main monomer flavanol found in green tea (Yen and Chen 1995). It seems that natural antioxidants such as phenols and flavones may have strong health capacity in controlling T complications. Although parsley is well known as diet of highly therapeutic potential, its companion with tea may considered a real therapy in case of T administration.

2. Materials and Methods

Experimental animal design: To test the biological effect of TPN, 30 male Sprague-Dawley aged rats of initial body weight 301 ± 3.0 g in a 5 X 6 animal's group system were conducted (Table 3).

The animals were fed on basal diet for 10 days for adaptation, and then divided into those groups taking casein as negative control (*Bowman et al., 1990*). Rats have been kept individually in plastic cages oriented to the experimental procedures described as the established guidelines for the care and handling of laboratory animals. The preferred method of normal patient's TPN delivering (*Hayes et al., 2000*) was infusion procedure with a medical infusion pump used. Sterile bag of the nutrient solution, a 50 mL each animal groups from 2 to 5, were used. The pump infused a small amount (0.15 to 0.25 ml/hr) continuously in order to keep a rate of around 4 ml/d and the vein open. TPN was performed through a central intravenous catheter, through the subclavian vein, with the tip of the catheter at the superior vena

cava without entering the right atrium. Animal movements were totally minimized up to the end of experiment including the control group. The entire time of this trail was 5 days, then animal was killed after blood collection, and then organs were separated, weighed and kept to be treated afterwards. The blood analysis of total cholesterol, Fe and glucose were performed according to *Allian et al., (1974)*; *AOAC, (1984)*; and *Dacie and Lowis (1984)*, respectively. The blood cell counts were counted according to the method of Van Der Zijpp and Leenstra (1980). The liver enzyme function: GPT and GOT in rat serum were determined according to the method described by *Reithman and Frankel (1957)*. A mean of six samples was used as a final figure.

Table (3): Systemic administrations of T (G2 to G5).

#	Group	Description
G1	NC	Basel diet (Bowman et al., 1990).
G2	T	50 ml of T solution (Hayes et al., 2000) each animal.
G3	TP	G2 + 1% Parsley curly 40% extract in cold water.
G4	TPG	G3 + 1% green tea 20% extract in hot water.
G5	TD	G2 + 1% an artificial common diuretic drug.

Where: NC negative control basel diet; T, total parental nutrition; P, Parsley curly; G, green tea; and D, Lasix.

Histopathological examination of liver: The organs were collected and post-mortal examination was done as soon as possible. Fixation was carried out in 10% of natural formalin, dehydrated, cleared, and ended paraffin then sectioned at (4-6 mm), and stained with harris hematoxylin, and casein for histopathological examination (*Frankel and Reitman, 1963*).

3. Results and Discussion

Total parental nutrition (T) response on rat's liver is recorded in Table (3). Exceptional T alone, all other treatment causes 20% reduction in liver masse; surprisingly this was around to be 40% under the effect of TD.

Table (4): Effect of T administration on body and liver weights as percent control group.

Group	BW changes		Liver changes	
	g	%	g	%
NC	304.0 ± 3.52	100	8.36 ± 0.23	100
T	295.2 ± 2.69	97.1	8.45 ± 0.19	101.1
TP	282.1 ± 3.06	92.8	6.80 ± 0.32	81.3
TPG	285.2 ± 2.87	93.8	6.85 ± 0.28	81.9
TD	278.8 ± 2.59	91.7	5.15 ± 0.24	61.6

Where: NC, Basel diet; P, Parsley curly; PG, Parsley curly + green tea; D, Lasix; T, total parental nutrition and BW, body weight.

This, however, was correlated with body weight loss that ranged between 3 to 9% for T to TD, respectively. In accordingly, most probably, most of this total weight reduction is water.

More biological evaluation for T complication related to liver tissues health is appeared in Table (5). As seen, the nearest liver health profile to the control was TPG and the most reversely affected one was

TD. However, Fig 1 is showing some liver histological complication caused by T application. Here, these histological findings summary are clearly pictured in Figure 2 where liver of control, untreated rat (Fig. 2.1) showing the normal histology of liver parenchyma; small local mononuclear inflammatory cells infiltration that has been caused by T (Fig. 2-2). Additional vacuolation of hepatocytes was occurred

under the effect of T alone, small and large arrows, respectively (Fig. 2-3). Comparatively, TP treatment only made a dissociation of hepatocytes (Fig. 2-4), meanwhile the TPG has appeared to be normal hepatocytes parenchyma (Fig. 2-5). Concurrently, TD section included serious histopathological disorders such as hemolysed RBCs and deposition of golden brown hemosidrine pigments, as seen in Fig. 2-6, in addition to necrosis of hepatocytes with pyknosis of their nuclei (Fig. 2-7).

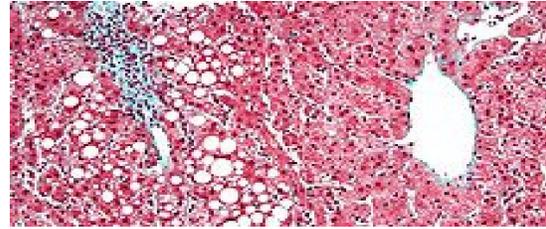


Figure (1) Micrograph of periportal fatty liver as may arise due to T (Gura et al., 2006).

Table (5): Effect of T administrations on liver histopathology as compared with control group

Liver complication	NC	T	TP	TPG	TD
1- Local monocular inflammatory	-	++	-	-	+
2- Vacuulations	-	++	-	-	++
3- Dissociation hemolysed RBCs	-	-	++	-	++
4- Deposition of golden brown hemosidrine pigment	-	-	-	-	++
5- Necrosis of hepatocytes with pyknosis of the nuclei	-	-	-	-	++
6- Necrosis of sporadic	-	-	-	-	+
Ranking No.	1	3	2	1	4

Where: NC, Basel diet; P, Parsley curly; PT, Parsley curly + green tea; D, Lasix and T, total parental nutrition.

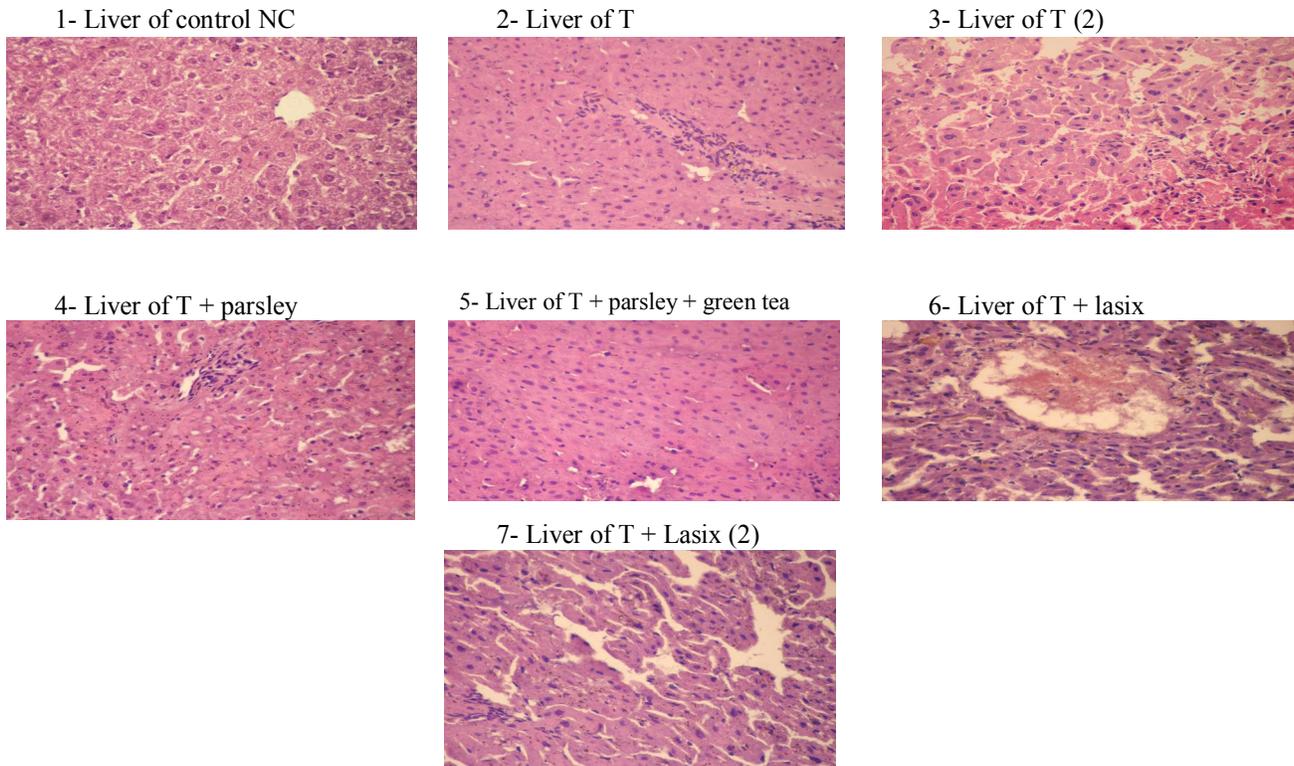


Fig. 2, 1: Liver of control, untreated rat showing the normal histology of liver parenchyma, 2: Liver of rat treated with T showing local mononuclear inflammatory cells infiltration, 3: Liver of rat treated with TP showing small local mononuclear inflammatory cells infiltration (small arrow) and vacuulations of hepatocytes (large arrow), 4: Liver of rat treated with TP showing dissociation of hepatocytes, 5: Liver of rat treated with TPG showing apparent normal hepatocytes, 6: Liver of rat treated with TD showing hemolysed RBCs and deposition of golden brown hemosidrine pigments, 7: Liver of rat treated with TD showing necrosis of hepatocytes with pyknosis of their nuclei. (H and E ×400)

Again, Table (5) and Figure 2 showed the exact negative role of T administrations on liver histopathology where several clinical remarks included local monocular inflammatory and vacuulations for application of T alone in addition to dissociation hemolysed RBCs, deposition of golden brown hemosidrine pigment, necrosis of hepatocytes with pyknosis of the nuclei and necrosis of sporadic in case of adding artificial drug to solution, e.g. TD treatment were occurred. These hepatic cells are partially or even totally recovered when the drug intervention was replaced with P or actually PG treatments, in the same respect. As an example to assess the liver response to T application, endotoxemia effect on hepatic lipid, male experimental rats received T or T plus a continuous infusion of *E. coli* lipopolysaccharide (LPS), liver weight increased significantly by 60% from 7.5 ± 0.6 g. Therefore, endotoxin, when given concomitantly with T, increases hepatic lipid accumulation and thus augments the development of T-associated fatty liver in rats (Dickerson and Karwoski 2002). Moreover, pathohistologic and electron microscopy examination discovered liver damage, similar to that caused by TD in the present study, with typical congestive changes mainly manifested local erythremia and a reduced fluid content of the blood in the liver with blockage in the sinusoidal pole of hepatocytes. There were also focal micronecrosis, considerable reduction of glycogen and slight centrolobular steatosis. The possibility is discussed for usage of hepatotoxicity, induced by furosemide, in examining the effects of some drugs with potential hepatoprotective activity (Klouček and Popv 1989). Likewise drugs, localization of some important nutrients upon T administration were estimated in thin sections at higher magnification to show that most of the lipid droplets containing vitamin A were localized near the sinusoidal side. There were very few lipid droplets in the stellate cells and none in the Kupffer cells. The results indicated that vitamin A infused in T solutions is stored in the same cellular location as orally ingested vitamin A (McKenna et al., 1983). Liver health is sensitive to several T content but postnatal

hepatic fatty acid oxidative capacity of preterm pigs receiving T does not differ from that of term pigs and is not affected by supplemental arachidonic and docosahexaenoic acids (Campbell et al., 2010). In harmony with what recorded in the present study, physicians claimed that parsley is very effective in remedying liver disease. It enriches the liver and nourishes the blood. Parsley helps reduce liver congestion, clearing toxins and aiding rejuvenation. In women, parsley improves estrogen and nourishes and restores the blood of the uterus. Conditions like delayed menstruation and the menopause, dry skin, irritability, depression and hair loss can often improve (McKeith, 2000). Furthermore, the present results and some earlier one suggested that the drinking of green tea with high catechin content may help to prevent and/or attenuate the development of a certain type of hepatitis (Abe et al., 2005). In a rats fed 2.5% green tea leaves, for 27 and 63 weeks trail; the changes of GOT, GPT, γ -GT, and creatinine were not significant in the treated group as compared with the control. These results suggested that long-term feeding of green tea leaves was not toxic to the liver or kidney. Serum total cholesterol, triglyceride, and LDL-C were decreased in the tested group. Interestingly, the dietary intakes of the two groups were approximately the same, but the body weights of the tea-fed group were decreased 10-18% compared with those of the control (Lin et al. 2003). This data is going in line with that tabulated in Table6. Similar to what given in Table 6, treatment with green tea extract significantly prevented the increases in the GOT, GPT and ALP activities in a dose-related manner. It also significantly prevented the decreases in serum albumin and total cholesterol, although not in a dose-related manner. A tendency to prevent the increase in Lecithin: cholesterol acyltransferase (LCAT) activity and the decrease in liver microsome P-450 were also noted. Little effect was found on the other abnormal changes in the serum lipids and proteins and the organ weights. These results suggest that green tea may have an ameliorating effect on hepatic dysfunction (Hayashi et al., 1992).

Table (6): Effect of TPN administration on rat's BW, blood constituents and liver functions as % control.

T Solution	BW	T C mg/dl	Glucose mg/dl	WBCs	RBCs	Iron mg/dl	GOT	GPT
Control	+	93.50	77.500	1155	17.55	184.9	326.0	114.0
T	—	132.5	104.5	29.9	97	80.6	168.9	70.6
TP	—	128.3	100.1	98.3	94.7	95.3	177.3	102.2
TPG	—	102.1	87.7	103.9	100	101.1	102.3	102.2
TD	—	146.4	114.2	33.3	93.9	71.6	187.7	127.2

Where: NC, Basel diet; P, Parsley curly; PG, Parsley curly + green tea; D, Lasix and T, total parental nutrition, body weight (BW changes; TC, total cholesterol, and W and R blood cells.

Parsley, on the other hand, prevents salt from being reabsorbed into the body tissues; thus parsley literally forces debris out of the kidneys, liver and bladder. It helps improve oedema and general water retention, fatigue and scanty or painful urination and used in conjunction with complete nutritional programmes to aid the dissolving of gall stones and in cases of gout (McKeith *et al.*, 2000). In most cases, as seen also in Table 6 and Fig. 2, dietary therapy stood as a promising health agent. Moreover, the above mentioned data went in line with some former studies on biological complication for T including rat's body weight (BW), Total Cholesterol (TC), glucose, blood cell counts. A recognizable loss of body weight; similar to what happened to organ size, reached 8.3% in 5 days using the artificial diuretic drug (TD) that used to minimize water retention. Water retention was maximally resolved also in the presence of G. In this regard, it has been suggested that patients with small-bowel syndrome who are currently on T may be at greater risk for atherosclerosis. Since T has restored a reasonably normal life expectancy for these patients, long-term follow-up will likely provide answers (Badimon *et al.*, 1987).

As general discussion and conclusion, addition to these histological impairments of liver caused by TD, the blood conservative power showed a great iron sweeping off. This was noticed under the same treatment; meanwhile, a noticeable reverse action was accomplished when T was applied. Although phenolic-rich extracts used as antioxidants in foods reduce the utilization of dietary iron, this data goes in reverse way and tea seems to synergist parsley action on Fe retention. Furthermore, blood enzymes and cell count, liver enzymes, as seen also in Table (6), were in better condition under TPG application. Moreover, artificial drug highly caused a severe reduction of WBCs and RBCs. Better results for these blood constants were found also for TPG. Although a better result was recorded for TP, TPG was the best over all including the control. Seemingly, a combined phenolic compound of the two plants is considering the main factor in this positive health role. In fact, this internal metabolic homeostatic power of PG mixture special extract is an added medicinal value to be further studied in the area of nutritional biochemistry and dietary therapy. However tea is reach of specific polyphenols. The native occurrence of tea polyphenols, namely, (-)-epicatechin, (+)-catechin, (-)-epigallocatechin 3-gallate, (-)-epicatechin, and (-)-epicatechin 3-gallate, and caffeine in tea flowers was assessed by an isocratic HPLC procedure. The levels of total catechins ranged from 10 to 38 mg/g, whereas the level of caffeine ranged from 3 to 8 mg/g. Levels of catechins and

caffeine in tea leaves and various teas were also determined and ranged from 2 to 126 mg/g and from 23 to 49 mg/g, respectively. Both tea flower and tea leaf extracts exert their strong hydroxyl radical scavenging effects in the Fenton reaction system and nitric oxide suppressing effects in LPS-induced RAW 264.7 cells. Most tea flowers contain less caffeine, but comparable amounts of total catechins, compared to tea leaves and teas (Lin *et al.*, 2003). Likewise, greater polyphenols content are determined in parsley (Halvorsen *et al.*, 2002). Tea also is an important dietary source of flavanols and flavonols. In vitro and animal studies provide strong evidence that tea polyphenols may possess the bioactivity to affect the pathogenesis of several chronic diseases, especially cardiovascular disease and cancer. However, the results from epidemiological and clinical studies of the relationship between tea and health are mixed. International correlations do not support this relationship although several, better controlled case-referent and cohort studies suggest an association with a moderate reduction in the risk of chronic disease. Conflicting results between human studies may arise, in part, from confounding by socioeconomic and lifestyle factors as well as by inadequate methodology to define tea preparation and intake. Clinical trials employing putative intermediary indicators of disease, particularly biomarkers of oxidative stress status, suggest tea polyphenols could play a role in the pathogenesis of cancer and heart disease (McKay and Blumberg 2002). Hormonal imbalance followed T application induces biliary dilatation, sludge and formation of gallstones. Cholecystokinin (CCK) induces gallbladder (GB) contraction. During thyrotropin-releasing hormone (TRH) testing for thyroid function patients felt a strong micturition reflex attributable to smooth muscle contraction of the bladder. The possibility of GB contraction after TRH administration was studied compared to cholecystokinin-octapeptide (CCK-OP) and/or fatty meal administration. The effect of intravenous (IV) CCK-OP, TRH and a combination of the two on GB volume was studied in normal volunteers without GB or liver disease. Gallbladder contraction was estimated by ultrasound prior to and after administration of the fatty meal; in the other 36 subjects, GB contraction was calculated prior to and after administration of CCK-OP, TRH, or both. Results are expressed as a percentage of the GB basal volume using each subject as his or her own control (Kalfarentzos *et al.*, 1992). The overall biological concept existed here and in several other experiments can be facilitated by the theory of hormonal oxidative balance for food and drug (Ahmed *et al.*, 2003). The naturals are always support health comparing to

synthetic drugs that may impair the oxidative hormonal balance.

References

- Abe K, Ijiri M, Suzuki T, Taguchi K, Koyama Y, Isemura M. (2005). Green tea with a high catechin content suppresses inflammatory cytokine expression in the galactosamine-injured rat liver. *Biomed Res.* 26(5):187-92.
- Ahmed, A I S Fathia, K S Abo Zeid And Adel A. El-Bagoury (2003): Hormonal oxidative balance of modified diets and uncontrollable cholesterol. *Egy. J Of Biomedical Sciences.* 13: 106-115.
- Ahmed; A I S; Fathia K Abo-Zeid; Neven M Mahmoud and Fadl E El-Deeb (2009). Complication of total parenteral nutrition (TPN) 1. A rat's main blood chemistry responses. *J. Biol. Pharm. Sci.* 7 (1): 11-20.
- Allain CC, Poon LS, Chan CS, Richmond W, Fu PC. (1974). Enzymatic determination of total serum cholesterol. *Clin Chem.* 20, 4:470-5.
- AOAC (1984). Official Methods of Analysis of the Association of Official Analytical Chemists. 14th edition, S. Williams, ed., Arlington, Virginia, 114 p.
- Badimon JJ, Fleming CR, Patton J, Mao SJ. (1987). Changes of plasma levels of apolipoproteins A-I, A-II, and B and their isoforms in patients with intestinal failure receiving long-term parenteral nutrition. *Am J Clin Nutr.* 45(2):414-22.
- Bowman TA, Goonewardene IM, Pasatiempo AM, Ross AC, Taylor CE. (1990). Vitamin A deficiency decreases natural killer cell activity and interferon production in rats. *J Nutr.* 120(10):1264-73.
- Campbell JA, Martin JE, Melendez K, Stout MB, Lyvers-Peffer PA. (2010). Postnatal hepatic fatty acid oxidative capacity of preterm pigs receiving TPN does not differ from that of term pigs and is not affected by supplemental arachidonic and docosahexaenoic acids. *J Nutr.* 140 (4):752-9.
- Chuang JH, Shieh CS, Chang NK, Chen WJ, Lo SK. (1995). Metabolic effect of parenteral nutrition in dogs with obstructive jaundice. *J Am Coll Nutr.* 14(2):197-201.
- Dacie, JV. and Lewis, SM. (1984). Practical hematology 'Fifth' ed. Pp.32 and 34 Churchill Living Stone: Edinburgh, London and New York.
- De Vree JM, Romijn JA, Mok KS, Mathus-Vliegen LM, Stoutenbeek CP, Ostrow JD, Tytgat GN, Sauerwein HP, Oude Elferink RP, Groen AK. (1999). Lack of enteral nutrition during critical illness is associated with profound decrements in biliary lipid concentrations. *Am J Clin Nutr.* 70 (1):70-7.
- Dickerson RN, Karwoski CB. (2002). Endotoxin-mediated hepatic lipid accumulation during parenteral nutrition in rats. *J Am Coll Nutr.* 21(4):351-6.
- Didier ME, Fischer S, Maki DG. (1998). Total nutrient admixtures appear safer than lipid emulsion alone as regards microbial contamination: growth properties of microbial pathogens at room temperature. *J Parenter Enteral Nutr.* 22(5):291-6.
- Dudrick SJ. (2009) History of parenteral nutrition. *J Am Coll Nutr.* Jun;28(3):243-51.
- Esfahani A, Wong JM, Truan J, Villa CR, Mirrahimi A, Srichaikul K, Kendall CW. (2011): Health effects of mixed fruit and vegetable concentrates: a systematic review of the clinical interventions. *J Am Coll Nutr.* 30(5):285-94.
- Frankel, S., and Reitman, S. (1963). *Gradwohl's Clinical Laboratory Methods and Diagnosis.* Vol. 1, p. 231, 6th ed. C. V. Mosby, St. Louis.
- Grimes CJ, Younathan MT, Lee WC. (1987). The effect of preoperative total parenteral nutrition on surgery outcomes. *J Am Diet Assoc.* Sep;87(9):1202-6.
- Gura KM, Duggan CP, Collier SB, Jennings RW, Folkman J, Bistran BR, Puder M. (2006) Reversal of parenteral nutrition-associated liver disease in two infants with short bowel syndrome using parenteral fish oil: implications for future management. *Pediatrics.* 118(1):e197-201.
- Halvorsen BL, Holte K, Myhrstad MC, Barikmo I, Hvattum E, Remberg SF, Wold AB, Haffner K, Baugerød H, Andersen LF, Moskaug Ø, Jacobs DR Jr, Blomhoff R. (2002). A systematic screening of total antioxidants in dietary plants. *J Nutr.* 132(3):461-71.
- Hayashi M, Yamazoe H, Yamaguchi Y, Kunitomo M. (1992). Effects of green tea extract on galactosamine-induced hepatic injury in rats. *Nihon Yakurigaku Zasshi.* 100(5):391-9.
- Hayes EM, Cohen KR, Pinard BE, Lauletta J, Ruggiero R. (2000) Standardized versus individually customized parenteral nutrition solutions: a comparison of serum electrolyte values. *P & T.*; 25(2):78-80, 83, 87.
- Horattas MC, Trupiano J, Hopkins S, Pasini D, Martino C, Murty A. (2001). Changing concepts in long-term central venous access: catheter selection and cost savings. *Am J Infect Control.* 29(1):32-40.

23. Howard A, Goodlad RA, Walters JR, Ford D, Hirst BH.(2004). Increased expression of specific intestinal amino acid and peptide transporter mRNA in rats fed by TPN is reversed by GLP-2. *J Nutr.* 134(11):2957-64.
24. Jeejeebhoy KN.(2001). Total parenteral nutrition: potion or poison? *Am J Clin Nutr.* Aug;74(2):160-3.
25. Kalfarentzos F, Spiliotis J, Chalmoukis A, Vagenas C, Vagenakis A (1992). Gallbladder contraction after hormonal manipulations in normal subjects and patients under total parenteral nutrition. *J Am Coll Nutr.* 11(1):17-20.
26. Kalfarentzos FE, Karavias DD, Karatzas TM, Alevizatos BA, Androulakis JA.(1991). Total parenteral nutrition in severe acute pancreatitis. *J Am Coll Nutr.* 10(2):156-62.
27. Klouchek E, Popov A.(1989). [Experimental liver damage by furosemide for studying drugs with hepato-protective activity]. *Eksp Med Morfol.* 1989; 28(3): 47- 50.
28. Kozier, B., & Erb, G., & Berman, A.J., & Burke, K., & Bouchal, S. R., & Hirst, S. P.. (2004). *Fundamentals of Nursing: The Nature of Nursing Practice in Canada.* Canadian Edition. Prentice Hall Health: Toronto.
29. Lin YS, Wu SS, Lin JK.(2003). Determination of tea polyphenols and caffeine in tea flowers (*Camellia sinensis*) and their hydroxyl radical scavenging and nitricoxide suppressing effects. *J Agric Food Chem.* 51(4):975-80.
30. McKay DL, Blumberg JB.(2002). The role of tea in human health: an update. *J Am Coll Nutr.* 21(1):1-13.
31. McKeith I, Del Ser T, Spano P, Emre M, Wesnes K, Anand R, Cicin-Sain A, Ferrara R, Spiegel R.(2000). Efficacy of rivastigmine in dementia with Lewy bodies: a randomised, double-blind, placebo-controlled international study. *Lancet.* 16;356(9247):2031-6.
32. McKeith IG.(2000). Clinical Lewy body syndromes. *Ann N Y Acad Sci.* ;920:1-8.
33. McKenna MC, Robison WG Jr, Bieri JG(1983). Cellular localization of liver vitamin A in rats given total parenteral nutrition (TPN) solutions intravenously or orally. *J Nutr.* 113(6):1176-86.
34. Quigley EM, Marsh MN, Shaffer JL, Markin RS. (1993). Hepatobiliary complications of total parenteral nutrition. *Gastroenterology.* 104(1):286-301.
35. Raina N, Matsui J, Jeejeebhoy KN.(2000). Nutritional and metabolic effects of the endotoxin bacterial lipopolysaccharide in orally and parenterally fed rats. *Am J Clin Nutr.* 71(3):835-43.
36. Reithman S. and Frankel S.(1957) A colorimetric method for the determination of serum glutamic oxalacetic and glutamic pyruvic transaminases. *Am J Clin Pathol.* 28(1):56-63.
37. Reversal of parenteral nutrition-associated liver disease in two infants with short bowel syndrome using parenteral fish oil: implications for future management. *Pediatrics.* 118(1):e197-201.
38. Rollins CJ, Elsberry VA, Pollack KA, Pollack PF, Udall JN Jr. (1990). Three-in-one parenteral nutrition: a safe and economical method of nutritional support for infants. *JPEN J Parenter Enteral Nutr.* 14(3):290-4.
39. Roy CC, Belli DC. (1985). Hepatobiliary complications associated with TPN: an enigma. *J Am Coll Nutr.* 4(6):651-60.
40. Van Der Zijpp AJ, Leenstra FR.(1980). Genetic analysis of the humoral immune response of White Leghorn chicks. *Poult Sci.* 59(7):1363-9.
41. Vanderhoof JA, Langnas AN.(1997). Short bowel syndrome in children and adults. *Gastroenterology.* 113(5) :1767-78.
42. Wu LY, Juan CC, Ho LT, Hsu YP, Hwang LS. (2004). Effect of green tea supplementation on insulin sensitivity in Sprague-Dawley rats. *J Agric Food Chem.* 52(3):643-8.
43. Yen G, Chen H. (1995). Antioxidant Activity of Various Tea Extracts in Relation to Their Antimutagenicity. *J. Agric. Food Chem.*,43 (1): 27-32
44. Yokozawa T, Nakagawa T, Kitani K.(2002). Antioxidative activity of green tea polyphenol in cholesterol-fed rats. *J Agric Food Chem.* 50(12):3549-52.
45. Zeyuan D, Bingying T, Xiaolin L, Jinming H, Yifeng C. (1998). Effect of green tea and black tea on the metabolisms of mineral elements in old rats. *Biol Trace Elem Res.* 65(1):75-86.