Mona maghraby

بسم اللّه الرحمن الرحيم

مركز الشبكات وتكنولوجيا المعلومات

قسم التوثيق الإلكتروني

Mona maghraby

جامعة عين شمس

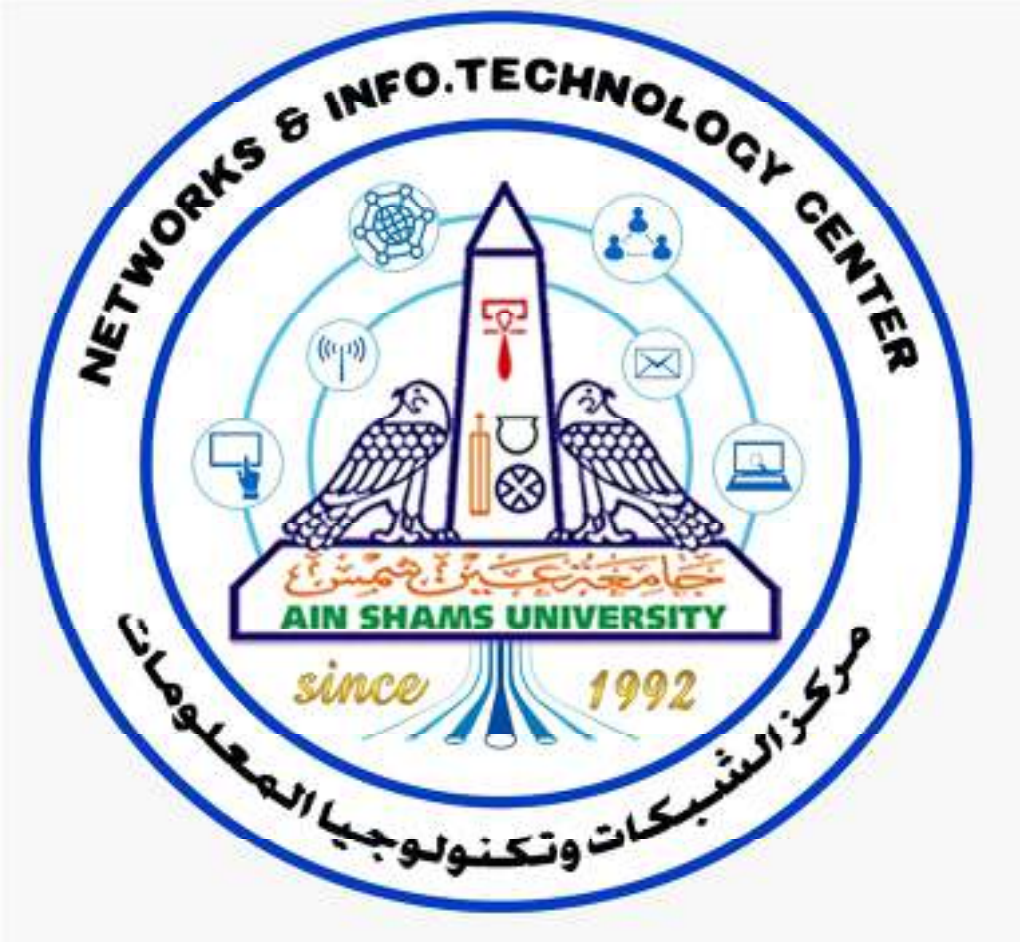
التوثيق الإلكتروني والميكروفيلم

قسم

نقسم باللّه العظيم أن المادة التي تم توثيقها وتسجيلها

علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

Mona maghraby



بعض الوثائق الأصلية تالفة
وبالرسالة صفحات لم ترد بالأصل

A study of the factors affecting adequate enteral tube nutrition in the Intensive Care Unit

Thesis

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Abbreviations

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(CI)	Catabolic Index
(r)	Correlation Coefficient
AEE	Activity Energy Expenditure
Alb	Serum albumin level
ARDS	Adult Respiratory Distress Syndrome
BMI	Body Mass Index
BMR	Basal Metabolic Rate
CCU	Critical Care Unit
CHI	Creatinine Height Index
CI	Critically Ill
COPD	Chronic Obstructive Pulmonary Disease
CPN	Central Parenteral Nutrition
DIT	Diet Induced Thermo genesis
DTH	Cutaneous delayed hypersensitivity
EN	Enteral Nutrition
GICs	Gasterointestinal complications.
GIT	Gastero-intestinal tract
ICU	Intensive Care Unit
MAC	Mid upper Arm Circumference
PDH	Pyruvate dehydrogenase
PEG	Percutaneous endoscopic gastrostomy
PN	Parenteral Nutrition
PNI	Prognostic Nutritional Index
PPD	Purified Protein Derivative
PPN	Peripheral Parenteral Nutrition
PRG	Percutaneous radiologic gastrostomy
RD	Registered Dietitian
REE	Resting Energy Expenditure
RME	Resting Metabolic Expenditure
RQ	Respiratory quotient
SD	Standard deviation
SGA	The Subjective Global Assessment
TEE	Total Energy Expenditure
TEN	Total Enteral Nutrition
TFN	Serum transferrin level
TIBC	Total Iron Binding Capacity
TPN	Total Parental Nutrition
TSF	The triceps skinfold thickness
$\dot{V}CO2$	The carbon dioxide production
$\dot{V}O2$	The oxygen consumption

Introduction

Introduction

Nutrition is the combination of processes by which the living individual receives and utilizes the food. Food is necessary to provide energy for the body activities, to build and maintain body tissues and to regulate body processes. [1]

Critically ill patient is the sickest patient with high degree of instability necessitating intensive monitoring and treatment in a specialized critical care department. [2] Critically ill patients are at high risk to develop protein-energy malnutrition as well as micronutrient deficiencies. Adequate nutritional support must be provided to these patients in order to improve survival and to decrease the period of hospital stay. [3]

Malnutrition refers to the clinical condition in which there is a dysequilibrium between nutrient intake and requirements as a result of insufficient intake, exaggerated loss of nutrients, or increased catabolism. Traditionally, malnutrition is divided into kwashiorkor, associated with edema resulting from low protein intake, or marasmus, in which protein and calorie intakes are deficient and there is no edema. [4] The malnutrition that is encountered in hospitalized patients is usually due to a deficiency of carbohydrates, proteins, and fats referred to as protein-energy malnutrition. Deficiencies in vitamins, minerals, and trace elements often accompany the protein-energy deficits. [4,5]

The prevalence of malnutrition in hospitalized patients is high and the nutritional status worsens during the course of hospitalization in multiple patient populations. [6,7,8] Malnutrition affects clinical status,

morbidity, and mortality. Weight loss associated with illness adversely affects muscle, respiratory and cardiovascular functions and decreases resistance to infection. [9,10,11] Malnutrition increases hospital cost by prolonging patient stay, and it ultimately can increase mortality. [12,13,14]

In the critically ill patient population, those who suffer traumatic injury, sepsis, burns, flare up of chronic disease or major surgery, the resolution of an acute disease process is associated with a change in neuroendocrine activity and in the cytokine repertoire that mediate the response to stress. As a result of these changes, those patients develop adult kwashiorkor-like malnutrition, metabolic bone disease, hypothalamic–pituitary dysfunction, and various micronutrient deficiencies. Regardless of the initial insult, type of acute critical illness, or natures of pulmonary dysfunction present, critically ill patients represent a convergence of disparate pathophysiologies into this discrete metabolic syndrome. [15]

Pathophysiology of malnutrition in critically ill patient

Marasmus-type malnutrition

The term marasmus refers to the simple starvation, or protein–calorie malnutrition, which develops when energy intake fails to meet metabolic demand. Carbohydrate deprivation induces physiologic decreases in insulin, and therefore, glucose uptake by the liver. Consequently, pyruvate dehydrogenase PDH activity and Krebs cycle activity are slowed, thereby reducing the metabolic rate and respiratory quotient. Concomitantly, fatty acids are liberated from adipose tissue, due to low levels of insulin, leading to increased mitochondrial fatty acid oxidation and acetyl CoA production. This further reduces PDH activity. Acetyl CoA cannot enter the Krebs

cycle and is diverted into ketogenesis; ketone bodies are the principal fuels during simple starvation and can retard muscle proteolysis. Because some organs cannot utilize ketone bodies, and remain dependent on glucose for cellular respiration [erythrocytes, brain (early in starvation), renal medulla and hepatobiliary epithelial cells], some gluconeogenesis from muscle protein must occur. [15]

Marasmus is recognized by ketosis and decreased muscle and fat mass. Normal albumin levels persist until very late stages when hypoalbuminemia and "third spacing" result from compromised hepatic function due to visceral organ autodigestion. Operationally, patients must be less than 90% of their usual body weight, with a normal serum albumin, to be considered "marasmic". Nutritional therapy is rather simple: provision of necessary calories and protein, accompanied by fluid and micronutrient support, to reverse starvation (primary nutrition).

Because virtually every critically ill (CI) patient has hypoalbuminemia and anasarca, this type of malnutrition is poorly represented in this population. [15]

Adult kwashiorkor-like, or hypoalbuminemic-type malnutrition

The majority of critically ill patients harbor cytokine-mediated responses to stress that are associated with deranged metabolic pathways. In this condition, the problem is not carbohydrate deprivation, but stressors leading to a particular set of humoral factors (chiefly, but not limited to, interleukins 1, 2, and 6; tumor necrosis factor-α and γ-interferon) that activate the hypothalamic–pituitary–adrenal axis, the sympatho-adrenal axis, and muscle proteolysis. [16]

This leads to futile substrate cycling, hepatic gluconeogenesis, and rapid reduction of muscle mass and visceral organ dysfunction.

These patients with kwashiorkor-type malnutrition may be without significant weight loss due to increased third spacing and fluid retention. Fat mass can remain intact (patients may actually remain obese) and muscle mass loss can be difficult to detect. Hypoalbuminemia is typically present and ketosis typically absent. Patients with this form of malnutrition have not lost more than 10% of their usual body weight. Mixed marasmic–kwashiorkor states with weight loss and hypoalbuminemia are treated as pure kwashiorkor patients.

Although serum albumin is a poor nutritional marker due to its long half-life (20 days), it has been demonstrated to be a very good prognosticator for weaning outcome in CI patients [17]. Hypoalbuminemia is associated with a greater risk of hospital morbidity and mortality. 30% increased length of stay; 40% increased cost of parenteral nutrition (PN); 2.5 times increased odds of sepsis and nosocomial infection; and four times increased odds of death [15].

Nutritional therapy for these patients is supportive, but primary nutrition alone is ineffective. These patients are very sensitive to caloric overfeeding, which can lead to volume expansion, hyperglycemia, and steatocholestasis. The focus should be on nitrogen retention and avoidance of specific nutrient deficiencies. Adjuvant pharmacological agents and specialized enteral formulas are frequently indicated because of the dysmetabolism. [17]

General strategy of nutritional support in critically ill patients

The primary care is removal of the stress through procedures such as repairing the wound, draining the abscess, coverings the burns, treating infections…etc.

Nutritional support begins as soon as vital functions are stable, fluid and electrolyte and acid-base balance are achieved, and tissue perfusion is adequate to allow transport of oxygen and fuel. Nutritional support during the catabolic phase will probably not result in positive nitrogen balance, but it will lower the loss of body protein. Everyday that this critically ill patient does not receive adequate support may require 3 to 5 days of such support to "catch-up". [18]

Unless exogenous protein is introduced to patient diet within 5 to 7 days, protein synthesis declines, weakens of immunocompetence, hypoalbuminemia, failure of wound healing, further infections, decubitus ulcers from skin breakdown, respiratory insufficiency from respiratory muscle weakness, and eventually multiple organ failure and death may result. [18]

A nutritional assessment should be performed once a critically ill patient is admitted to the critical care unit. Usually, a hospital Registered Dietitian (RD) will be notified of the patient's admission and this assessment will follow. A physician nutrition specialist will be consulted to directly manage nutritional issues in collaboration with the RD. Various tools are available to formulate a nutritional assessment.