Trace Elements in Critically Ill Patients

An Essay

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By

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Mohamed Abdraboh Abdel-aziz

List of abbreviations

BMI	:	Body mass index
CCI	:	Chronic critical illness
Ch	:	Chromium
CHO	:	Carbohydrates
CI	:	Critically ill
CKD	:	Chronic kidney disease
CP	:	Ceruloplasmin
Cu	:	Copper
DIT	:	Diiodothyronine
EFA	:	Essential fatty acids
ETF	:	Enteral tube feeding
Fe	:	Iron
GI	:	Gastrointestinal
GSH-Px	:	Glutathione peroxidase
GTF	:	Glucose tolerance factor
ICU	:	Intensive care unit
IV	:	Intra venous
MIT	:	Monoiodothyronine
Mn	:	Manganese
MODS	:	Multi-organ dysfunction
PN	:	Parenteral nutrition
RDA	:	Recommended daily allowances
RNOS	:	Reactive nitrogen-oxygen species
ROS	:	Reactive oxygen species
Se	:	Selenium

SIRS	:	Systemic inflammatory response syndrome
T3	:	Triiodothyronine
T4	:	Thyroxin
TPN	:	Total parenteral nutrition
TSH	:	Thyroid stimulating hormone
Zn	:	Zinc

List of tables

List of figures

Contents

Introduction

Critical illness is any disease process which causes physiological disturbance leading to disability or death ***(Antonelli et al, 2007).***

Critically ill patients, almost by definition, are catabolic; often hypermetabolic and can become malnourished. Mal-nutrition has been associated with increased morbidity and mortality in these patients, particularly in the surgical population ***(McClave et al, 2009).***

Therefore, the generally accepted goals of nutritional delivery in critically ill patients are to provide nutritional therapy consistent with the patient's condition, prevent nutrient deficiencies, avoid complications related to delivering nutrition, and improve patient outcomes ***(McClave et al, 2009).***

Nutritional support includes supplementation of the daily requirements of macronutrients and micronutrients including vitamins and trace elements which defined as those existing in less than one part per million body weight e.g., zinc, copper, manganese, selenium, chromium, etc ***(Sriram and Lonchyna , 2009).***

Geriatric patients, especially those in long-term care facilities are often trace element deficient and need early supplementation. Certain disease processes alter trace element status as patients with malignancies, those on hemodialysis or peritoneal dialysis and those who have gastrointestinal losses e.g., post-surgical patients with fistulas, diarrhea ***(Sriram and Abraham, 2012).***

So supplementation of trace elements in critically ill patients should be considered as a crucial component of ICU care. This will not only correct the deficiencies present during critical illness but also their anti-oxidant potential and immunomodulatin properties may improve the patient outcomes in severe sepsis. Future research in ICU nutrition should emphasize on the pharmaco-nutrient aspect of trace elements so as to determine the appropriate route, dose and timing of the intervention ***(Agarwal et al, 2011).***

Aim of work

This work aimed at highlighting the important role of trace elements in the metabolic support of patients and also as pharmaco-nutrients in intensive care units.

Chapter 1

Nutrition in intensive care patients: an overview

There is no specific definition of critically ill (CI) patients. In general, the term CI is used to refer to patients who remain in intensive or intermediate care units for weeks to months."The majority of patients with this syndrome fail to be weaned from mechanical ventilation."Many are in the "recovery" phase of multi-organ dysfunction (MODS) due to sepsis (***Scheinhorn et al, 2002).***

Other characteristics of CI include prolonged stay in the hospital (14, 21, and 29 days or more), immobilization, hypoalbuminemia, malnutrition, neuroendocrine exhaustion, metabolic, bone disease, myopathy, and neuropathy. There is chronic depletion of physiologic reserve, leading to accelerated aging. Regardless of the perspective, these patients represent 5% to 10% of the population in intensive care units ***(Carson et al, 2009).***

Nutritional support of intensive care patients is important, and aims to prevent the development of malnutrition. Malnutrition in these patients is under-recognized and under-treated problem. It has been shown to be strongly linked to increased length of stay in hospital, morbidity and mortality ***(McClave et al, 2009).***

Metabolic changes in critical illness

Although the human body has evolved a series of immune and hormonal responses to acute injuries manifested by the stress or systemic inflammatory response, in some cases the response becomes exaggerated. That characteristic response, geared toward repair of damaged tissue via activation and metabolic support of leukocytes exceeds what is actually required ***(Nunnally et al, 2005).***

Malignant hyperinflammation is the basis of systemic inflammatory response syndrome (SIRS), sepsis, or multi organ dysfunction (MODS). The latter occurs when the uncontrolled response evolves to produce clinically recognizable abnormalities in organ function. These take the form of lung injury (the acute respiratory distress syndrome), ileus and malabsorption, renal insufficiency, hepatic synthetic failure, coagulopathy, myopathy and neuropathy, encephalopathy, refractory vasodilatation, and myocardial depression ***(Nunnally et al, 2005).***

The metabolic changes that accompany these syndromes include glucose and lipid intolerance, increased reliance on amino acids and lactate for peripheral metabolism, thus progressive acidosis. Ultimately, patients develop immune paralysis and acquire infections involving organisms that are not normally pathogenic ***(Teno et al, 2002).***

Despite these metabolic changes, the support of this state is possible. Part of this involves metabolic support. In recent years, it has become clear that with current methods we are unable to meet the metabolic demand of these hyper inflammatory states. Thus, from a caloric point of view, patients are purposely underfed. Aggressive attempts to meet demand with protein alone have been marginally successful. Attempts to modify metabolic support, using (omega-3 fatty acids, glutamine, modified amino acid formulas, trace elements, and hormonal supplementation) have shown some promise. However, none has been fully effective. Nonetheless, the ability to sustain patients in this severely compromised state has led to the emergence of a subgroup of critically ill patients that enter a chronic, relatively stable, phase of illness. This has been termed prolonged or chronic critical illness (CCI) ***(Teno et al, 2002).***

A) Neuroendocrine dysfunction:

Acute and chronic critical illness should be seen as separate neuroendocrine paradigms. In the acute phase response there is hyper secretion of pituitary hormones, with peripheral insensitivity, presumably to favor the catabolic effects of hormones and maintain substrate delivery. Conversely, in CCI, there is restoration of peripheral hormonal sensitivity, but loss of central drive. From this perspective, CCI should be considered a state of neuroendocrine exhaustion clearly (figure1) ***(Nunnally et al, 2005).***

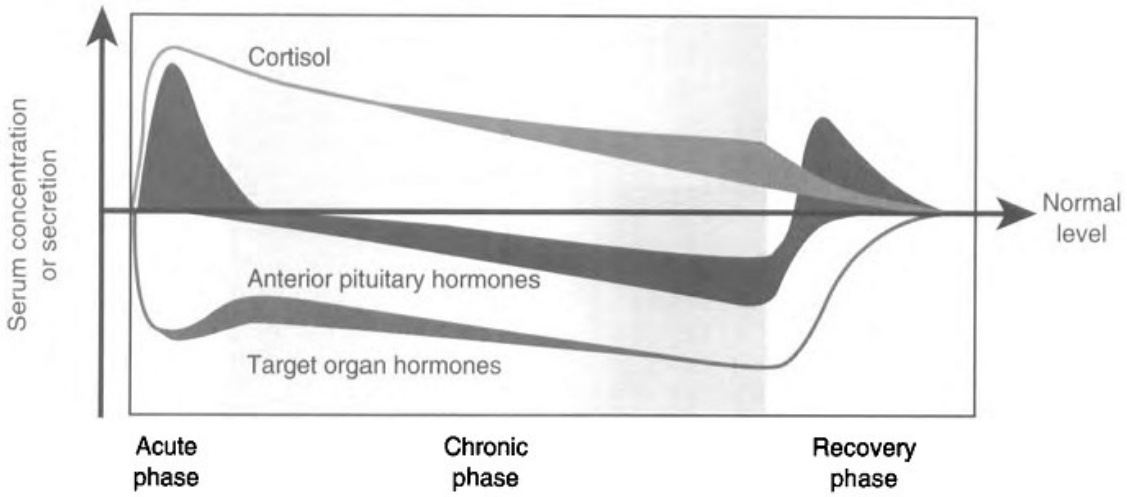

Figure 1: Simplified concept of the pituitary dependent changes during the course of critical illness anterior pituitary hormonal secretions increase, but there is peripheral resistance to anabolic activity. In chronic critical illness, anterior pituitary secretion is uniformly reduced. Cortical levels are maintained by a peripheral drive, but this may ultimately fail. The onset of recovery is characterized by restored sensitivity of the anterior pituitary to reduced feedback control ***(Berghe et al, 1998)***.

B) Bone changes:

The majority of patients with CCI develops metabolic bone disease, principally osteomalacia and bone hyper resorption. This results from prolonged immobilization, vitamin D deficiency, malnutrition, malabsorption, and renal and liver dysfunction.

Secondary hyperparathyroidism is present; In addition, iatrogenic osteopenia is common owing to glucocorticoid and heparin administration. Renal failure accelerates secondary hyperparathyroidism, hypocalcemia, and hyperphosphatemia ***(Nunnally et al, 2005).***

C) Neuromuscular changes:

Muscular atrophy is universally seen in prolonged critical illness due to hyper catabolism.

Critical illness myopathy and critical illness polyneuropathy (CIP) do not respond to nutritional therapy. CIP represents an acute axonal neuropathy that develops during treatment of severely ill patients and remits spontaneously when the critical illness resolves ***(Latronico and Bolton, 2011).***

Clinical manifestations of both critical illness myopathies and CIP include delayed weaning from mechanical ventilation, muscle weakness, and prolongation of the mobilization phase. The pathogenesis of these neuromuscular complications is incompletely understood. Administration of steroids and neuromuscular blocking agents may act as triggers. No specific strategies have been discovered to prevent or treat critical illness myopathy and CIP, except source control and avoidance of risk factors such as high dose steroids, neuromuscular blockers (particularly when used together), hyper osmolar syndromes, and hyperglycemia ***(Latronico and Bolton, 2011).***

D) Fluid and electrolyte abnormalities:

Electrolyte and acid base abnormalities are common in CCI. Hypernatremia occurs because of free water loss. Patients with CCI are particularly vulnerable to insensible water loss from open wounds, pressure ulcers, and prolonged mechanical ventilation, particularly when the ventilation liberation strategy involves spontaneous breathing ***(Nunnally et al, 2005).***