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Oxidative stress and acute-phase response in patients with pressure sores

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Abstract

Objective: We investigated the relation between oxidative stress and the occurrence of the acute-phase response with serum ascorbic acid and α-tocopherol levels in patients with pressure sores.

Methods: The following groups of patients were studied: 1) those who had patients with pressure sores, 2) those who had pneumonia, and 3) those who did not develop pressure sores or any type of infection (control). Concentrations of total proteins, albumin, creatinine, iron, ferritin, transferrin, C-reactive protein, α_1-acid glycoprotein, total iron-binding capacity, ascorbic acid, α-tocopherol, and malondialdehyde were measured during the first days of hospitalization.

Results: Albumin concentrations were significantly lower ($P < 0.05$) and C-reactive protein concentrations were significantly higher ($P < 0.05$) in patients with pressure sores compared with controls. Concentrations of ascorbic acid and α-tocopherol were significantly decreased ($P < 0.05$) in patients who had pressure sores or infection, whereas malondialdehyde concentrations were significantly increased ($P < 0.05$) compared with control patients. Five of 11 patients (55.56%) with pressure sores and 10 of 12 patients (83.33%) with pneumonia presented serum ascorbic acid concentrations below the reference value (34 to 91 μmol/L). Concentrations of ascorbic acid and α-tocopherol versus malondialdehyde were significantly correlated in the three patient groups ($r = -0.44$, $P < 0.05$; $r = -0.55$, $P < 0.01$, respectively).

Conclusion: Patients with pressure sores and acute infection present a systemic inflammatory response accompanied by an increase in lipid peroxidation that is associated with decreased serum ascorbic acid and α-tocopherol levels, suggesting that these patients may be at risk for important nutritional deficiencies. © 2005 Elsevier Inc. All rights reserved.

Key words: Pressure sores; Acute-phase response; Oxidative stress; Ascorbic acid; α-Tocopherol

Introduction

Pressure sores are considered to be one of the most important immobility complications among hospitalized patients [1,2] and occurs more frequently among elderly and/or undernourished individuals [3]. Patients with pressure sores are at higher risk for morbidity and mortality because these ulcers are associated with a larger number of complications, with infection being among the most critical [4,5].

In addition, as is the case for other severe inflammatory stimuli, pressure sores may result in a systemic inflammatory response, denoted by the acute-phase response (APR), a process mediated by cytokines such as tumor necrosis factor-α and interleukin-1 that promote protein catabolism, interfere with central nerve system, and thus lead to anorexia [6–8]. Clinically, it is characterized by anorexia, fever, increased neutrophil numbers in blood, hyperglycemia, and anemia [9]. In this respect, changes occur in APR

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that include alterations of concentrations of many plasma proteins known as acute-phase proteins, including C-reactive protein and α_1-acid glycoprotein, in addition to many behavioral, physiologic, biochemical, and nutritional alterations [10]. A compromised nutritional status with regard to certain nutrients, such as energy-protein [11], iron, zinc, [12], ascorbic acid, and α-tocopherol [13–15] deficiencies, may contribute to the development of pressure sores and to impaired wound healing.

Further, continuous pressure results in increased damage to the parenchyma and vascular system [16] that is related to ischemia and reperfusion, a process triggered by mechanisms involving an increase in lipid peroxidation products. Ischemia and reperfusion may be among the factors that strongly contribute to the pathogenesis of pressure sores, and antioxidant agents such as vitamins C and E play an important role in decreasing the injury induced by pressure [17]. Vitamin E decreases cellular oxidative damage in an effective manner after the period of ischemia and reperfusion, and vitamin C is recognized to be one of the most potent stable oxygen reducers and to act against free radicals in blood [4,16,18].

Clinical and epidemiologic studies have demonstrated a significant decrease in concentrations of ascorbic acid [14,19] and α-tocopherol [14] in patients with pressure sores. Low concentrations of ascorbic acid and α-tocopherol appear to be associated with subsequent development of pressure sores [2].

Clinical trials in subjects with pressure sores and the presence of low serum levels of ascorbic acid and α-tocopherol in relation to the APR and the increase in free radical concentration have not been reported.

Thus, the objective of the present study was to determine the relation between oxidative stress and the occurrence of the systemic inflammatory response with serum ascorbic acid and α-tocopherol levels in patients with pressure sores.

Material and methods

Patients

A prospective study was conducted in patients who were being seen at the School Hospital, School Medicine of Triângulo Mineiro (FMTM; Uberaba-MG, Brazil). The patients were selected at the time of hospital admission and were included in the following groups: 1) patients who had pressure sores (PS), 2) those who had infectious pneumonia (IC), and 3) those who did not develop pressure sores or any type of infection (control, C). Patients who had difficulty in swallowing, frequent vomiting, osteomyelitis in the sore area, renal failure, severe congestive heart failure, hypoalbuminemia attributed to hepatic insufficiency or to glomerular disease, injury to the spinal cord, or taking vitamin C supplementation and/or systemic glucocorticoids were excluded from the study. Median times of hospitalization before assessment were 4.79 $\pm$ 3.13 and 5.07 $\pm$ 3.13 d for patients who had a normal hospital diet and those who had enteral feeding, respectively. The study was approved by the medical ethics committee of FMTM and informed written consent was obtained from each patient before the beginning of the study.

All patients were evaluated during the first week of hospitalization, when they underwent clinical examination (medical history and clinical examination), anthropometric measurements, a food ingestion inquiry, and urine and blood collection for laboratory tests.

Clinical examination

The clinical condition of each patient was assessed by applying a specific protocol that was developed by the Nutrology Subject of the FMTM School Hospital, which included information about personal data, medical history (changes in the oral diet, presence of gastrointestinal symptoms, and functional level), and physical examination.

Anthropometric and dietary parameters

The anthropometric parameters measured in patients were arm circumference, triceps skinfold thickness, and arm muscle circumference. Triceps skinfold thickness was measured in triplicate in the posterior part of the arm with the aid of a Lange skinfold caliper and a uniform pressure of 0.1 g/mm [20]. The criteria and classification established by Frisancho [21] were adopted as reference standards for arm circumference, triceps skinfold thickness, and arm muscle circumference.

Food ingestion was determined by the method of daily food weighing [22], with all the foods and drinks offered by the hospital for 24 h/d being weighed and measured for 3 consecutive days before being offered to the patients. The food left in the patient's dish after the end of each meal was also collected and weighed as food leftover and deducted from the respective food portions. The total intake of the main nutrients was calculated with the aid of software developed by the Federal University of São Paulo (version 1).

Laboratory tests

Venous blood was collected into sterile 10-mL Vacutainer tubes (Becton Dickinson, Juiz de Fora, MG, Brazil) with no anticoagulant and into 5-mL tubes containing the anticoagulant ethylene-diaminetetra-acetic acid. Blood samples were collected between 9:00 and 11:00 AM after a 12-h fast. In addition, 24-h urine was collected from each patient and an aliquot was transferred to polypropylene tubes for determination of urinary creatinine.

A 10-mL Vacutainer tube was used for serum separation and measurement of antioxidant vitamins (ascorbic acid and α-tocopherol) and malondialdehyde (MDA). Tubes were centrifuged at 1000*g* for 10 min at 10°C. For determination

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