



Serum adiponectin upon admission to the intensive care unit may predict mortality in critically ill patients ☆

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Abstract

Purpose: Adiponectin has been proposed as an important regulator of glucose metabolism influencing obesity and insulin resistance, which are important risk factors for the outcome of critically ill patients. Moreover, experimental models of inflammation suggest protective anti-inflammatory properties of adiponectin. We therefore investigated the potential pathogenic role and prognostic value of circulating adiponectin levels in critical illness.

Materials and methods: One hundred seventy critically ill patients (122 with sepsis and 48 without sepsis) were prospectively studied at admission to the medical intensive care unit (ICU) and compared with 60 healthy controls. Patients' survival was followed for approximately 3 years.

Results: Adiponectin serum concentrations did not differ between healthy controls and critically ill patients, neither in patients with nor in patients without sepsis. However, patients with decompensated liver cirrhosis had significantly elevated serum adiponectin levels. Likewise to non-critically ill subjects, ICU patients with preexisting diabetes or obesity displayed significantly reduced circulating adiponectin. Inflammatory cytokines did not correlate with serum adiponectin. Interestingly, low adiponectin levels at ICU admission were an independent positive predictor of short-term and overall survival.

Conclusions: Although serum concentrations did not differ in critically ill patients from controls, low adiponectin levels at admission to ICU have been identified as an independent predictor of survival. © 2011 Elsevier Inc. All rights reserved.

1. Introduction

Obesity and diabetes mellitus have been suggested to be prominent risk factors for mortality in critically ill patients and especially in sepsis [1-3], but a recent multicenter study and a meta-analysis did not reveal an impact of obesity on mortality [4,5]. However, beyond the role of adipose tissue as a store of excess energy, it is regarded as a hormonally active system, influencing inflammation, metabolism, body weight, and lipid and glucose metabolism [6]. Active mediators released by adipose tissue are summarized as adipocytokines and include adiponectin, leptin, retinol binding protein 4, and resistin. We previously demonstrated that serum resistin levels are elevated in critically ill patients; associated with inflammation, organ

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dysfunction, and metabolism, and predict survival of nonseptic patients [7].

Adiponectin is a 30-kDa peptide hormone secreted exclusively by adipocytes and is involved in lipid and glucose metabolism as well as insulin resistance [8,9]. Although generally circulating at high concentrations in human plasma, adiponectin was found reduced in obese and diabetic patients [10] and has been regarded as a potential link between adiposity and increased cardiovascular risk in these patients [11]. In nonobese subjects without severe illness, serum adiponectin has been demonstrated to be negatively correlated to diastolic blood pressure, insulin resistance, low-density lipoprotein cholesterol, and triglycerides and positively correlated to high-density lipoprotein cholesterol [12]. Beyond antiatherogenic effects, experimental data suggest anti-inflammatory properties of adiponectin, promoting protective functions in animal models of sepsis [13,14]. Altered release patterns of adiponectin have been reported in patients with chronic hepatitis C virus infection and experimental endotoxinemia in humans, suggesting a role as a regulator of the immune response [15,16].

Little is known about the regulation of adiponectin in critical illness. Because of the proposed physiologic functions of adiponectin, serum concentrations are most likely influenced by essential therapeutic measurements such as nutrition and insulin therapy. During the course of critical illness, low to normal circulating levels of adiponectin have been reported [17-19], but the potential initial dysregulation of adiponectin at admission to the

intensive care unit (ICU), before therapeutic interventions, is currently unclear.

We therefore determined serum adiponectin concentrations in a large cohort of well-characterized critically ill patients at the time of admission to the ICU in different circumstances of critical disease. In addition, we aimed to identify possible pathogenic functions of adiponectin in obese and nonobese and diabetic and nondiabetic patients by correlations with a wide number of markers of inflammation, organ dysfunction, and metabolism and to investigate the impact of adiponectin as a prognostic marker in critically ill patients.

2. Patients and methods

2.1. Study design and patient characteristics

The present study was approved by the local ethics committee, conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki, and written informed consent was obtained from the patient, his or her spouse, or the appointed legal guardian. We enrolled 170 patients (111 males and 59 females, with a median age of 62 years; range, 18-86 years) consecutively upon admission to the medical ICU of the RWTH (Rheinisch-Westfaelische Technische Hochschule) University Hospital Aachen [7]. Details about the study cohort are given in Table 1. Patients who were expected to have a short-term (<72 hours)

Parameter	All patients	Patients with sepsis	Patients without sepsis
Number	170	122	48
Sex (male/female), n	111/59	81/41	30/18
Age, median (range) (y)	63 (18-86)	64 (20-86)	60 (18-79)
APACHE II score, median (range)	14 (0-31)	14 (0-31)	15 (0-31)
SAPS2 score, median (range)	44 (0-80)	45 (0-79)	41 (13-80)
ICU days, median (range)	8.5 (1-137)	10* (1-137)	6* (1-45)
Hospital days, median (range)	27 (2-151)	30** (2-151)	14** (2-85)
Death during ICU, n (%)	54 (32)	42 (34)	12 (25)
Death during follow-up, n (%)	88 (52)	64 (53)	24 (50)
Mechanical ventilation, n (%)	113 (67)	82 (67)	31 (65)
Ventilation time, median (range) (h)	66 (1-2966)	127.5** (1-2966)	31** (1-755)
Preexisting diabetes, n (%)	56 (33)	39 (32)	17 (35)
BMI, median (range) (m ² /kg)	25.8 (14.0-59.5)	26.0 (14.0-59.5)	25.1 (17.5-53.3)
GFR/cystatin C, median (range) (mL/min)	30 (0.8-379)	25 (4-379)	52 (0.8-379)
Albumin, median (range) (g/L)	28 (5-47)	26* (5-47)	30* (14-44)
Bilirubin, median (range) (mg/dL)	0.8 (0.1-40.4)	0.8 (0.1-40.4)	1.1 (0.2-39.1)
Procalcitonin, median (range) (μg/L)	1.0 (0.1-224.5)	2.2** (0.1-224.5)	0.2** (0.1-36.5)
IL-6, median (range) (ng/L)	110 (2-1000)	140** (2-1000)	40.5** (2-1000)
IL-10, median (range) (ng/L)	13 (5-1500)	18* (5-1500)	5.7* (5-750)
TNF-α, median (range) (ng/L)	22.0 (4.1-430)	26.0* (4.8-430)	16.0* (4.1-100)
Adiponectin, median (range) (μg/L)	11300 (820-25000)	10386 (820-25000)	12500 (2373-25000)

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