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Unspecific metabolic blood parameters as used in clinical routine may differentiate malignant from benign cerebral tumors

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Abstract

The investigation of rather insensitive metabolic parameters (protein, fibrinogen, blood urea nitrogen (BUN), blood glucose) reveals significant differences between tumor-bearing and tumor-free patients as well as benign and malignant neoplasms. Whereas metastases and glioblastomas (GBM) show significantly elevated BUN levels (21.9 ± 1.7; 20.8 ± 2.2 mg/dl) compared to benign tumors (meningioma WHO I, astrocytoma I, II) (16 ± 0.9; 14.3 ± 0.9 mg/dl) and tumor-free matched controls (e.g. 13.9 ± 1.4 mg/dl) only metastases depict higher glucose (141.7 ± 11 mg/dl) counts. Fibrinogen, significantly elevated in malignancy (395 ± 25.2; 397.2 ± 25.9 mg/dl) is without difference between meningioma, astrocytoma (253.2 ± 16.6; 271.5 ± 16.5 mg/dl) and controls (e.g. 270.1 ± 10.8 mg/dl). Correlating BUN with total protein reveals a metabolic mismatch in nearly all tumor patients, regardless of dignity, as compared to tumor-free patients. Neuroendocrinoimmunological changes are the most likely reason for these overt as well as occult findings, making investigation of more sensitive metabolic parameters a rewarding task.

Keywords: Brain tumors; Host catabolism; Acute-phase protein

1. Introduction

Tumor patients are known to be in metabolic distress even in situations not related to trauma, surgery or infection [3,4,7–9,20,22]. The reasons for the metabolic alterations of these chronically consuming processes, which can progress to cachexia are poorly defined. Since oropharyngeal and esophageal passage is undisturbed in patients suffering from brain tumors as seen in patients with neoplasms along the digestive tract, other reasons than simple mechanical obstruction must be considered. Cerebral homeostasis can be disturbed directly through compression of hypothalamic structures, or nuclei of the vagal nerve or indirectly through altered functional circuits. Furthermore, immunologic dysregulation shows an increasing impact [5,6,12,15]. Whatever underlying mechanisms may be responsible for the metabolic distress, our aim was to see if cerebral tumor patients could be identified as tumor patients when investigating routine blood parameters (protein, blood urea nitrogen (BUN), fibrinogen, glucose, creatinine, hemoglobin,

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