ELSEVIER

Journal of the Neurological Sciences 126 (1994) 88–89

JOURNAL OF THE NEUROLOGICAL SCIENCES

Short report

Release of muscle proteins in brain stroke patients

P. Secnik [a], E. Artner-Dworzak [b], S. Cifra [a], V. Parrak [c], B. Puschendorf [b], F. Gerstenbrand [d,*]

[a] Department of Clinical Biochemistry and Department of Neurology, General Hospital of Lucenec, Lucenec, Slovak Republic
[b] Institute of Medical Chemistry and Biochemistry, University of Innsbruck, Innsbruck, Austria
[c] Department of Clinical Biochemistry, Derers Hospital, Bratislava, Slovak Republic
[d] Department of Neurology, University Hospital Innsbruck, Anichstraße 35, A-6020 Innsbruck, Austria

Received 28 June 1994; accepted 6 July 1994

Keywords: Brain stroke; Muscular proteins; Myosin

We report changes of muscle protein concentrations in plasma of 16 patients (12 female and 4 male) with brain stroke (10 ischemic and 6 hemorrhagic) and with resulting grave neurological deficits (hemiparesis, hemiplegia, etc.). The patients were immobilized in bed, and were subjected to mostly passive rehabilitative measures in the first 7 days after stroke.

Patients with heart failure, septic illness and those who had received massive intramuscular injections were excluded from the study.

Blood was collected every 1–2 days for 1 week, the first sample being taken immediately after admission of the patient into hospital. We measured plasma concentrations of slow twitch skeletal (cardiac β-type) myosin, namely myosin heavy chain fragments (MHC), myoglobin, creatine kinase (enzyme activity), and creatine kinase MB isoenzyme (enzyme mass).

The values of muscle protein concentrations indicate that patients respond in two different ways to immobilization. In subgroup 1 ($n = 11$), the response to immobilization was only minimal, the levels of muscle proteins in plasma being practically within the normal range. Patients in subgroup 2 ($n = 5$), however, responded with a dramatic increase in all of the proteins measured. Figure 1 shows the time-course of MHC concentrations in plasma of both subgroups. Creatine kinase activity and CK-MB mass concentrations exhibited a closely similar pattern in plasma (data not shown). In a previous study (Artner-Dworzak et al. 1993) of changes in a similar muscle protein pattern of 5 healthy volunteers immobilized in dry water immersion, we found no increase in any of the parameters measured.

We suggest that in some brain stroke patients the autonomic nervous system ("stress axis") is intensively

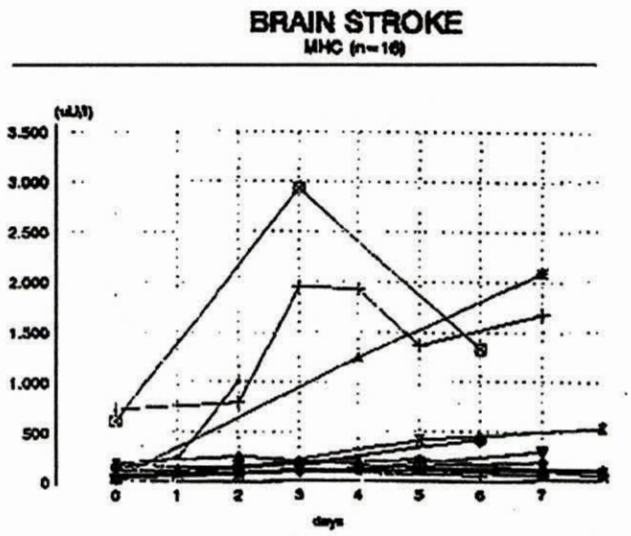

Fig. 1. Pattern of myosin heavy chain fragments: MHC, measured in plasma of 16 brain stroke patients, who were immobilized in the first 7 days after stroke and were subjected to mostly passive rehabilitation measures. Patient subgroup 1: ($n = 11$) MHC concentrations did not exceed 300 µU/l (cut-off value of healthy controls). Patient subgroup 2: ($n = 5$) MHC concentrations in plasma exceeded the cut-off value of 300 µU/l up to 10-fold. Symbols represent different patients.

* Corresponding author. Tel.: (512) 507 3500; Fax: (512) 507 2872.

SSDI 0022-510X(94)00155-3

activated, resulting in an alteration of hormonal homeostasis with increased blood levels of cortisol, thyroxine, triiodothyronine and other hormones, a process that was identified as autonomic hormonal shift (Chkhikvishvili et al. 1992). In analogy to clinical situations with intensively activated "stress axis" (Vary and Kimball 1992; Bessey and Lowe 1993; Fryburg and Barrett 199), an autonomic hormonal shift can be postulated as a plausible explanation for increased breakdown of muscle proteins observed in some of the patients with brain stroke.

However, it cannot be excluded that the dramatic, almost up to 10-fold increase of myosin concentrations in plasma of patients (subgroup 2) is the result of nonmuscular brain myosin (Helbing et al. 1993), corresponding to brain-specific creatine kinase BB (Delanghe et al. 1990) being released into the blood. In order to establish the exact causes of the observed phenomena, it will be necessary to analyze the correlations between the location and extent of brain injuries, intensity of autonomic hormonal shift and muscle protein release into blood and cerebrospinal fluid of a larger number of brain stroke patients.

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J Neurol Sci. 1994 Oct;126(1):88-9.

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Author information

PMID: 7836953

[Indexed for MEDLINE]