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Iron deficiency in patients with acute ischemic stroke and 1 year after stroke

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Introduction The role of iron homeostasis in stroke is unclear. Iron deficiency (ID) and anaemia contribute to the functional performance and quality of life [1]. The aim of this study was to evaluate the role of anaemia and ID in patients with acute ischemic stroke (AIS) and their role in the functional outcome and neurological deficits. Serum ferritin and transferrin saturation (TSAT) were used for diagnosis of ID.

Patients and methods We consecutively evaluated 142 patients (age 69 ± 13 years, BMI 27.7 ± 4.5 kg/m², mean \pm SD) admitted to stroke unit with AIS of the middle cerebral artery. Forty-four patients (31%) were treated with thrombolytic agent Actilyse on admission. Baseline study examinations were completed within 3 ± 2 days after acute event. The neurological status was elevated according to the modified Rankin scale (mRS). Muscle isometric strength was assessed by handgrip test and knee extension leg test. Blood parameters were measured from venous blood samples after overnight (>8 h) fasting. Sixty-four patients were available for follow-up examination 383 ± 26 days after stroke. Anaemia was diagnosed in patients with serum haemoglobin levels <12 g/L for females and <13 g/L for males. Iron deficiency was diagnosed with serum ferritin levels

<100 μ g/L or <300 μ g/L and TSAT $<20\%$ for both genders. Iron deficiency anaemia (IDA) was diagnosed in patients with anaemia and ID.

Results The prevalence for ID was 37% (48%) at baseline examination and increased up to 45% (67%) at follow-up ($P < 0.05$), while the prevalence for IDA doubled from 11% in acute stroke to 22% at follow-up ($P < 0.05$). Patients with IDA showed a more severe stroke according to mRS compared with patients with ID only and patients without ID (3.1 ± 1.2 vs. 2.2 ± 1.5 and 2.2 ± 1.4 , respectively, $P < 0.05$). However, there was no difference in stroke severity according to mRS at follow-up examination. Handgrip strength and quadriceps strength of the non-paretic side were reduced in patients with IDA and ID compared with patients without ID at baseline and at follow-up examination.

Conclusions The prevalence for ID and IDA increased 1 year after stroke. IDA was associated with more severe stroke. Muscle strength was reduced in stroke patients with IDA and ID.

Reference:

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Serum levels of Myostatin as a biomarker of muscle wasting in patients with chronic heart failure: results from the Studies Investigating Co-morbidities Aggravating Heart Failure (SICA-HF)

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Background and aims: Skeletal muscle wasting (sarcopenia) affects around 20% of heart failure (HF) patients and is related to poor exercise tolerance.^{1, 2} A biomarker to detect muscle wasting is required. Myostatin (a negative muscle regulator) could be a promising biomarker to screen for muscle wasting in serum. We investigated the diagnostic properties of soluble myostatin in ambulatory patients with chronic HF.³

Methods: We measured serum myostatin levels by enzyme-linked immunosorbent assay in 220 HF patients [reduced ejection fraction (HFrEF, 69.5%) or preserved EF (HFpEF, 30.5%)], 79% male, mean age 67 ± 11 . NYHA 2.4 ± 0.7 . Dual-energy X-ray absorptiometry was used to detect muscle wasting, defined as an appendicular skeletal muscle mass 2 SD below the mean of a healthy young reference group.

Results: Muscle wasting was identified in 39 patients (17.7%). Patients with muscle wasting showed higher myostatin levels than those without (29 ± 14 ng/mL vs. 27 ± 9 ng/mL, $P = 0.03$). Using receiver operating characteristics curve (ROC curve), we calculated the optimal myostatin value to identify patients with muscle wasting as >32 ng/mL, which had a specificity of 78.4% and a sensitivity of 46.1%. The area under

the ROC curve was 0.61 (95% CI 0.54–0.68). Using simple regression, serum myostatin higher than 32 ng/mL was associated with muscle wasting ($R = 0.20$, $P = 0.003$) and type of HF ($R = -0.16$, $P = 0.01$). The logistic regression model showed that myostatin >32 ng/mL (OR 3.00, 95% CI 1.27–7.08, $P = 0.01$), age (OR 1.06, 95% CI 1.01–1.11, $P = 0.01$) and BMI (OR 0.78, 95% CI 0.69–0.87, $P < 0.001$) were predictors of muscle wasting.

Conclusions: Myostatin is an important regulator of skeletal muscle mass in HF, and it shows reasonable specificity for the detection of muscle wasting.

References:

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