

PLASMA GFAP LEVELS IN PATIENTS WITH MIGRAINE: LACK OF ASSOCIATION WITH CLINICAL PHENOTYPE AND DISEASE BURDEN

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Background. Growing evidence highlights the role of astrocytic activation and subsequent neuroinflammation in migraine pathophysiology, particularly in driving trigeminovascular sensitization. As a major intermediate filament protein of astrocytes Glial Fibrillary Acidic Protein (GFAP) directly reflects glial response and neuroinflammatory changes. Although GFAP is widely utilized to monitor neurodegenerative conditions, evidence regarding its correlation in migraine patients remains uncertain. This study aimed to evaluate plasma GFAP levels in migraine patients compared to healthy controls, and to investigate its association with specific clinical phenotypes, disease burden, and attack phases.

Methods. We conducted a cross-sectional observational study in patients with migraine and controls. Plasma GFAP concentrations were quantified using the fully automated Lumipulse® G1200 platform (Fujirebio, Ghent, Belgium). Clinical variables included monthly migraine days (MMD), pain intensity, migraine-related disability (MIDAS), headache impact (HIT-6), disease duration, migraine subtype (episodic or chronic), migraine with aura, medication-overuse headache (MOH), and ictal/interictal status. Group comparisons, Spearman correlation analyses, and multivariable linear regression analyses were performed.

Results. Seventy-one patients with migraine and 26 healthy controls were included. Plasma GFAP concentrations showed no statistically significant difference between cases and controls (18.40 [IQR 11.80–28.70] vs 18.10 [12.05–20.60] pg/mL; p=0.372); with no difference after adjustment for age. No significant differences were observed according to migraine chronicity, presence of aura, MOH, attack phase, sex or disease severity indices. A positive correlation was observed between plasma GFAP levels and age both in patients with migraine (ρ=0.470, p<0.001), and in the overall population (ρ=0.422, p<0.001). In multivariable analysis, age was the only independent predictor of plasma GFAP levels.

Conclusions. Our study showed that Plasma GFAP concentrations did not differ between migraine patients and controls, and were not associated with migraine phenotype, attack phase, or disease burden. The consistent association with age, observed both in patients and in the overall population, indicates that age is the main determinant of circulating GFAP levels, consistent with the established age-related increase of this marker. Overall, our findings do not support plasma GFAP as a biomarker of migraine phenotype or severity.