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Leukocyte counts in patients with cervical artery dissection: determinants and prognostic value for functional outcome

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Cervical artery dissection (CeAD) is one of the major etiologies of ischemic stroke and transient ischemic attack in young and middle-aged adults. Elevated inflammatory parameters including leukocyte counts (LCs) have been observed upon spontaneous CeAD in small study samples, but the determinants and the clinical relevance of this phenomenon remained unknown.

In the present study, we aimed to analyze the occurrence of elevated leukocyte counts in CeAD patients and to figure out its determinants and its predictive value for the short to middle-term functional outcome. For this purpose, we analyzed 190 consecutively recruited acute CeAD patients from two university hospital databases and 95 sex-and age-matched healthy controls. LCs of patients were assessed at hospital admission and demographic and clinical characteristics as well as risk factors were recorded. Genotyping of CeAD patients was performed for inflammatory gene polymorphisms of interleukin-1 receptor antagonist, interleukin-6, tumor necrosis factor alpha and selenoprotein S. Functional outcome of the patients at three to six months after the acute dissection event was measured with the modified Rankin Scale score.

CeAD patients presented with significantly higher LCs when compared to controls, and leukocytosis occurred significantly more frequently in patients. The elevation of leukocyte counts correlated positively with the NIHSS score assessed on hospital admission, negatively with the blood sampling latency and positively with the proinflammatory selenoprotein S gene promoter variation rs28665122 in women. The functional outcome of CeAD patients at 3-6 months as assessed by modified Rankin Scale was strongly associated with the initial NIHSS score on hospital admission, but not with LC levels in the acute phase.

In this work we were able to show that CeAD goes along with inflammation in its acute phase and the extent of the inflammation is influenced by the degree of ischemic damage but also by a proinflammatory promoter variant of selenoprotein S gene. But the question whether inflammation plays a pathogenetic role in CeAD development or merely represents a reactive state, remains unanswered and requires further investigation.