Commentary

Erythrocyte sedimentation rate (ESR) represents the velocity of red cells precipitation in test tube. This parameter is determined by several acute phase proteins, being fibrinogen one of the most recognized. Although ESR is not specific, this inexpensive test may be used to estimate indirectly the presence of systemic inflammatory states.[1] Several epidemiological studies reported that raised ESR values were associated with risk of suffering a coronary event, both fatal and non-fatal, and stroke.[2,3] However, data about mechanism of stroke were not available from these studies. Considering patients with diagnosis of ischemic stroke, higher ESR values were related to severity of clinical manifestations, greater final volume of infarct, and worse outcome after a short time of follow-up.[4,5] However, these studies remarked that raised ESR may be a phenomenon secondary to vascular lesion, and they did not evaluate the mechanism of ischemia. A relatively recent study comparing 200 patients with cardioembolic, atherothrombotic, lacunar, and undetermined ischemic stroke supported the association between higher ESR values and atherothrombotic cause.[6] This etiology of stroke is generally related to internal carotid and/or intracranial stenosis. In all these patients, the main classical vascular risk factors associated with development of arterial wall pathology are hypertension and smoking. The pathologic process starts with endothelial lesion and accumulation of lipids, which may be assessed by determination of the intima-media thickness. Because this first step in arterial wall may lead to atherosclerotic plaque and finally to ischemic stroke, it is extremely relevant to find markers of disease in early stages.

In the present paper, the authors studied 92 patients with atherosclerotic ischemic stroke and found a strong correlation between raised ESR and the presence of carotid plaques and increased carotid intima-media thickness.[7] In spite of some methodological limitations, these findings support the role of low-grade systemic inflammation in the pathophysiology of atherosclerosis. When other causes of increased ESR are excluded, this parameter may be a help in the diagnosis of pathological mechanisms underlying stroke, especially when it is combined with other biomarkers, as C-reactive protein and fibrinogen. The precise evaluation of these mechanisms is basic to determine the etiology of stroke, its prognosis, and the individual risk of suffering a future event. As previously commented, older studies supported the usefulness of ESR as a marker of atherosclerotic vascular disorders and as a marker of worse prognosis after stroke. Considering the concordance of literature about the role of ESR in vascular disorders, and the low price and wide availability of this technique, clinicians should evaluate this parameter in all patients with vascular diseases. Obviously, because ESR is an unspecific marker of inflammation, its absolute value in individual patients must be always integrated with other characteristics of the global clinical profile to estimate the real contribution of atherosclerosis and concomitant vascular risk factors to ESR. With a rational use, this parameter could be useful in early diagnosis and perhaps monitoring of vascular patients, with a very low price and without any risk for patient safety.

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