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Letter to the Editor

Is erythrocyte sedimentation rate a useful inflammatory marker independently of the hematocrit? Comparison results with plasma viscosity

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To the Editor,

Plasma viscosity (PV) and erythrocyte sedimentation rate (ESR) are tests which can be used as inflammation and tissue injury markers [2, 11]. ESR usage is more extended than PV in clinical laboratories. However ESR may be haematocrit (Ht) dependent and is related with red blood cell (RBC) rheological properties [1], whereas RBC are not involved in PV. We aimed to evaluate retrospectively the impact of Ht on ESR as an inflammatory marker and compare the results with those of PV in 333 out- and in-patients at our hospital. Our study is a human non-interventional retrospective review and written consent is not required for human non-interventional studies.

We evaluated both markers in two patient groups of i.e., the first one did not present other inflammatory components measured as C-reactive protein (CRP) below 10 mg/L and fibrinogen below 450 mg/dL ($n = 211$). The second one presented CRP over 10 mg/L and/or fibrinogen values higher than 450 mg/dL ($n = 122$).

PV and ESR were determined in a capillary plasma viscosimeter (Fresenius GmbH, Germany) at 37°C and using a Ves matic 30 Plus Analyzer (Diesse Diagnostica Senese, Siena, Italy), respectively. CRP was determined by immunoturbidity in an AU5430 autoanalyzer (Beckman Coulter, Lexington, KY, USA) and fibrinogen analysis was performed in an ACL-TOP 700 autoanalyzer (Instrumentation Laboratory, Bedford, MA, USA).

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Our results showed that ESR correlated negatively with the Ht in both patient groups (Non Inflammatory group: rho: -0.528, $p < 0.001$ and Inflammatory group: rho: -0.727, $p < 0.001$). As it was be expected, PV did not correlate with Ht because it do not contain RBC (Non Inflammatory group: rho: -0.079, $p = 0.255$ and Inflammatory group: rho: -0.099, $p = 0.276$).

Moreover, we studied ESR and PV in both patient groups according to the different hematocrit values (below 36%, between 36–45% and higher than 45%). We found significant diferences in ESR between the Ht groups ($p < 0.001$ in both groups), but not in PV ($p = 0.480$ in the non-inflammatory group and $p = 0.161$ in the inflammatory group) (Fig. 1).

Finally, we studied the probability of PV and ESR discriminating both inflammatory states (CRP below 10 mg/L and fibrinogen under 450 mg/dL vs. CRP over 10 mg/L and/or fibrinogen values over 450 mg/dL) across the three hematocrit patient groups. The PV cut-off value was 1.33 mPa.s, and the ESR cut-off points were 30 mm/h for women and 20 mm/h for men. Our results showed that PV has a higher predictive negative value (PNV) and predictive positive value (PPV) than ESR to discriminate the inflammatory state in patients with Ht below 36% or higher than 45% (Table 1).

Our results suggest that ESR may be influenced by Ht unlike PV, which may be useful as an inflammatory marker independently of the Ht values. Different authors have suggested that ESR is dependent of red blood cell factors, such as Ht, red cell aggregability and deformability and influenced by demographic data [8, 10]. Even others authors have suggested that ESR should be replaced with PV because of its

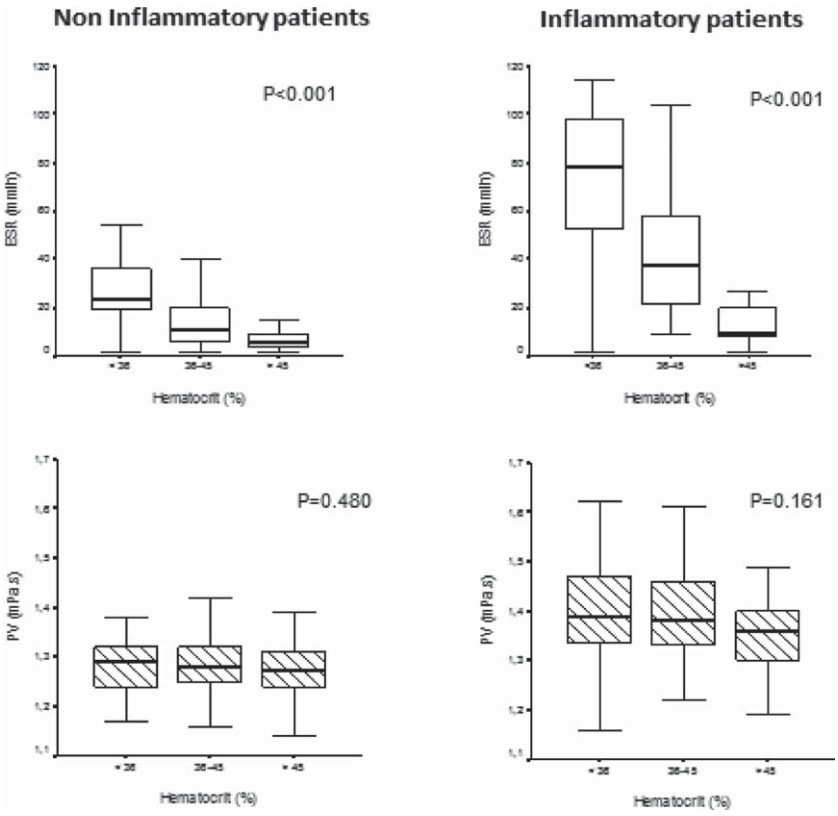

Fig. 1. Erythrocyte sedimentation rate and plasma viscosity in patients with positive and negative inflammatory markers according to the hematocrit.

Table 1
Predictive negative and predictive positive values of erythrocyte sedimentation rate and plasma viscosity to discriminate inflammatory states

	PNV % (CI 95%)	PPV % (CI 95%)
Hematocrit <36% ($n = 41$)		
PV	84 (65–100)	61 (31–91)
ESR	81(54–100)	45 (22–68)
Hematocrit 36–45% ($n = 160$)		
PV	89 (82–96)	44 (26–62)
ESR	92 (86–98)	47 (31–63)
Hematocrit >45% ($n = 132$)		
PV	89 (81–96)	43 (21–65)
ESR	82 (74–90)	33 (2–64)

independence of red cell changes, slight variation with demographic factors and proven measure of disease activity [4–8]. Although PV offers advantages over ESR, different studies have generally shown a reasonable correlation between PV and ESR in a variety of organic diseases, such as rheumatic disease, chronic infections, and malingnancy [5, 9]. However our results show that PV has greater discrimination power for the inflammatory state than ESR in patients with Ht under 36% or over 45% in the studied population. Despite our results, PV and ESR could display a different behaviour in different clinical settings. So, there are discrepancies in the choice of PV or ESR in several pathologies where ESR has been traditionally used such as rheumatic disorders or temporal arteritis [4, 6, 9]. In this sense the election of one of both tests could be conditioned by the presence of ESR influencing factors. Therefore, further studies on specific diseases that take into account the factors that can modify both parameters are required. In any case, the two tests can be used together to improve PNV and PPV.

In summary, our results imply that PV is an inflammatory marker that is independent of the Ht in contrast to ESR, which is mainly related with the Ht and RBC rheological behaviour. The usefulness of ESR should be reviewed in pathologies which imply reductions or increases in the Ht.

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