Coated-platelets, formerly known as COAT-platelets, represent a subpopulation of cells observed after dual agonist stimulation of platelets with collagen and thrombin. This class of platelets retains on its surface high levels of several procoagulant proteins, including fibrinogen, von Willebrand factor, fibronectin, factor V and thrombospondin. Coated-platelets also express surface phosphatidylserine and strongly support prothrombinase activity. Retention of alpha-granule proteins on the surface of coated-platelets involves an unexpected derivatization of these proteins with serotonin and an interaction of serotonin-conjugated proteins with serotonin binding sites on fibrinogen and thrombospondin¹.

Prothrombotic states of early brain injury (EBI) and delayed cerebral ischemia (DCI) after aSAH determine morbidity and mortality. To understand how platelet activation might contribute to such prothrombotic states, Ray et al. studied trends in coated platelets during EBI and DCI periods. Serial blood samples from a prospective cohort of aSAH patients were collected and assayed for coatedplatelet levels. Patient's coated-platelet level during post-hospital discharge follow-up served as an estimate of baseline. Occurrence of DCI, Montreal cognitive assessment (MOCA) score of < 26, and modified Rankin scale (mRS) of 3-6 were considered poor clinical outcomes. Non-linear regression analysis detected a transition between periods of rising and declining coated-platelet levels at day 4. Additional regression analyses of coated-platelet trends before day 4 showed differences among patients with modified Fisher 3-4 [4.2% per day (95% CI 2.4, 6.1) vs. - 0.8% per day (95% CI - 3.4, 1.8); p = 0.0023 and those developing DCI [4.6% per day (95% CI 2.8, 6.5) vs. - 1.9% per day (95% CI - 4.5, 0.5); p < 0.001]. Differences between peak coated-platelet levels and baseline levels were larger, on average for those with DCI [18.1 ± 9.6 vs. 10.6 ± 8.0 ; p = 0.03], MOCA < 26 [17.0 ± 7.8 vs. 10.7 ± 7.4 ; p = 0.05] and mRS 3-6 [24.8 ± 10.5 vs. 11.9 ± 7.6; p = 0.01]. Coated-platelet trends after aSAH predict DCI and short-term clinical outcomes. The degree of rise in coated-platelets is also associated with adverse clinical outcomes²⁾.

1)

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