Hereditary prothrombotic states of clinical importance include factor V Leiden, the prothrombin 20210A mutation, deficiencies of protein C, protein S, or antithrombin, sickle cell disease, and hyperhomocysteinemia. Major acquired prothrombotic states include cancer, myeloproliferative disorders, the antiphospholipid syndrome, and heparin-induced thrombocytopenia. Because most of the hereditary prothrombic states are not established risk factors for arterial thrombosis, routine laboratory testing in most patients with ischemic stroke should be limited to complete blood count, lupus anticoagulant, anticardiolipin antibodies, and plasma total homocysteine. Additional testing for factor V Leiden, prothrombin 20210A, antithrombin, protein C, and protein S may be indicated for patients under the age of 50 or those with paradoxical cerebral embolism. The treatment of acute ischemic stroke in patients with prothrombotic states is similar to that in patients without an identifiable prothrombotic condition, and may include antiplatelet agents, anticoagulants, or thrombolytic therapy in patients who otherwise meet eligibility criteria. The potential benefit of chronic anticoagulation therapy for the primary or secondary prevention of stroke in patients with prothrombotic states has not been addressed in controlled clinical trials. Specific therapeutic approaches for the prevention of stroke are established for patients with sickle cell disease, myeloproliferative disorders, and heparin-induced thrombocytopenia, and are under investigation for hyperhomocysteinemia and the antiphospholipid syndrome [1].

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 coated-platelet 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 outcomes [2].

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2)
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