Platelet aggregation

- Prediction Model to Optimize Long-Term Antithrombotic Therapy Using Covert Vascular Brain Injury and Clinical Features
- Tirofiban Combination Therapy for Acute Ischemic Stroke: A Systematic Review and Meta-Analysis
- Cerebral ischemia, a rare postoperative complication of cervical disc herniation: a case report
- Prophylactic administration of tirofiban prevents ischemic events in endovascular treatment of unruptured intracranial aneurysms
- Platelet aggregation rate serves as a significant predictive indicator for thromboembolic events in the context of stent-assisted embolization for unruptured arterial aneurysms
- Platelet transfusion and antiplatelet timing not associated with decreased rates of ventriculostomy hemorrhage in aneurysmal subarachnoid hemorrhage
- Nomogram for predicting delayed intraparenchymal hemorrhage after pipeline embolization device treatment in patients with intracranial aneurysms: a multicenter, retrospective model development and validation study
- Aspirin Continuation or Discontinuation in Surgically Treated Chronic Subdural Hematoma: A Randomized Clinical Trial

Platelet aggregation refers to the process by which platelets in the blood clump together to form a blood clot or plug at the site of a blood vessel injury. It is a crucial step in hemostasis, the body's natural mechanism to stop bleeding.

When a blood vessel is damaged, platelets are activated and undergo a series of changes that lead to aggregation:

Adhesion: Platelets adhere to the exposed collagen fibers and other components of the damaged blood vessel wall. This initial attachment is mediated by specific receptors on the surface of platelets, such as glycoprotein Ib-IX-V complex.

Activation: Adherent platelets become activated, resulting in shape change, release of granules from platelet cytoplasm, and exposure of receptors on the platelet surface. This activation is triggered by various factors, including thrombin, ADP (adenosine diphosphate), collagen, and thromboxane A2.

Secretion: Activated platelets release granules containing various substances, including ADP, serotonin, thromboxane A2, and platelet-derived growth factors. These substances further promote platelet aggregation and recruit additional platelets to the site of injury.

Aggregation: Platelet aggregation occurs when activated platelets bind to each other through specific receptors, primarily glycoprotein IIb/IIIa (integrin αIIbβ3). This receptor binds to fibrinogen, von Willebrand factor, and other adhesive molecules, allowing platelets to form a platelet plug.

The formation of platelet aggregates reinforces the initial platelet plug and stabilizes the clot. It provides a temporary seal over the injured blood vessel, preventing excessive bleeding until the underlying injury is repaired.

Platelet aggregation is regulated by a delicate balance between pro-aggregatory and anti-aggregatory factors. Disruptions in this balance can lead to abnormal clotting or bleeding disorders. Medications such as antiplatelet drugs (e.g., aspirin, clopidogrel) and certain diseases (e.g., von Willebrand disease, thrombocytopenia) can affect platelet aggregation.

The study of platelet aggregation plays a vital role in understanding and managing various conditions, including cardiovascular diseases, thrombotic disorders, and bleeding disorders. Laboratory tests, such as platelet aggregation studies, can assess platelet function and help guide diagnosis and treatment decisions in these conditions.

A prospective study aimed to test changes in hemostasis in patients with glioblastoma multiforme, occurring at baseline (before surgery, time 0, T0) and 2 (T2), 24 (T24), and 48-hour (T48) after surgery. Leal-Noval et al. enrolled consecutive patients subjected to GBM resection (GBR group; N = 60), laparoscopic colon cancer resection (comparative CCR group; N = 40), and healthy blood donation group (HBD group; N = 40). They performed 1. conventional coagulation tests 2. ROTEM (rotational thromboelastometry) parameters and 3. platelet function tests, including PFA-200 closure time when stimulated by collagen/epinephrine (COL-EPI) and ROTEM platelet, using three different activators (arachnoid acid in ARATEM, adenosine diphosphate in ADPTEM, and thrombin receptor-activating peptide-6 in TRAPTEM). Variables associated with unfavorable 1-year clinical outcome were investigated, too. We observed in GBR patients that platelet aggregometry, as assessed by ROTEM platelet parameters, was significantly impaired along with a shortened closure time. These changes were evident from T0 to T48. A decreased area under the aggregation curve in TRAPTEM was associated with improved survival (adjusted odd ratio (95% CI), 1.03 (1.01-1.06)). This study suggests that patients with GBM presented a decreased platelet aggregation from before surgery and thorough the postoperative period. Decreased platelet aggregation improved clinical outcome [1].

1)
Leal-Noval SR, Casado M, Palomares C, Narros JL, García-Garmendia JL, Escolar G, Cuenca DX, Görlinger K. Prospective assessment of platelet function in patients undergoing elective resection of glioblastoma multiforme. Platelets. 2023 Dec;34(1):2216802. doi: 10.1080/09537104.2023.2216802. PMID: 37246516.