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Opinion

Clinical Application of Platelet Mapping Using Thromboelastography in Neurology Patients –Is It Needed?

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Stroke remains a global leading cause of death and long-term disability, highlighting the need for more effective treatment approaches. The efficacy of aspirin in both primary and secondary prevention of stroke and hence cardiovascular death is established and the addition of adenosine diphosphate (ADP) receptor inhibitor, clopidogrel, further reduces these risks. This medication is administered once per day at 75 mg. Clopidogrel works by irreversibly blocking the P2Y12 component of ADP receptors on platelets that ultimately prevents the activation of GPIIb/IIIa receptor complexes. GPIIb/IIIa aids in platelet activation through its receptor site for fibrinogen and von Willebrand factors. Stroke prevention, particularly in patients who have already experienced stroke or transient ischemic attack (TIA), is critical for reducing the burden of disease on patients, their families and society since occurrence of a first stroke or TIA is the strongest predictor of a repeat event [1]. Thromboelastography-platelet mapping (TEG-PM) assay relies on evaluation of clot strength to enable a quantitative analysis of platelet function. Standard thromboelastography (TEG) is a Point Of Care Test (POCT) that measures the rate and strength of clot formation induced by thrombin, which is used as the main platelet activator. The TEG-PM is a modification of the TEG, measuring percentage platelet aggregation in the presence of ADP or arachidonic acid (AA). It was designed to be used for evaluating the therapeutic inhibitory effect of clopidogrel or aspirin on platelet aggregation and in decision-making for timing of cardiac surgery or other procedures after discontinuation of antiplatelet therapy [2,3].

The most obvious application of this test is to assess platelet function and the contribution of P2Y12 receptor or COX pathways to the clot formation. It can be measured by the addition of an appropriate agonist, ADP or AA. Then, AA is added to activator F to measure the degree of thromboxane A2-induced platelet aggregation. It has been shown to correlate with optical platelet aggregation which was the main ex vivo assay of platelet function used in the clinical studies of clopidogrel [4,5]. As also proved by Tantry et al, [6]. platelet aspirin resistance assessed by methods that directly indicate inhibition of cyclooxygenase is rare in patients with coronary artery disease. Nevertheless, it continued for many years, this method was not successful in everyday practice, and most scientists choose impedance aggregometry in such situations. The same authors, in 2013 evaluate the usefulness of thrombelastography platelet mapping assay to measure the antiplatelet effect of P2Y12 receptor inhibitors and high on-treatment platelet reactivity. As we reported that MAADP [adenosine diphosphate (ADP)-induced platelet-fibrin clot strength] > 47 mm was significantly associated with the short- and long-term post-percutaneous coronary intervention clinical outcomes and could be used as an important predictor of ischemic events occurrence [7,8].

R Kasivisvanathan et al, [9] designed a study to identify the value of TEG-PM testing in patients taking clopidogrel within 7 days of non-cardiac surgery. These researchers determined an optimal ADP-PRI cut-off of 34 per cent, which was identified to assist with decision-making with regard to proceeding with or cancelling elective surgery. The use of TEG-PM testing could prevent significant bleeding complications and unnecessary cancellations [9]. In 2018 Chandrasekaran et al, [10] designed a study, which recruited neurology patients, to evaluate TEG-PM in patients with spontaneous intracerebral hemorrhages on aspirin and/or clopidogrel who receive platelet transfusions. TEG-PM is not an effective measure of platelet inhibition in spontaneous intracerebral hemorrhages patients who were on antiplatelet medications, and one cannot rely on this test as an indication for platelet transfusions, moreover there were no correlation between hemorrhage expansion and platelets inhibition. One year later, Arora et al, [11] checked the role of TEG-PM in acute ischemic stroke (AIS) patients and her findings support that ADP inhibition

may be involved in the pathophysiology of AIS. TEG-PM is highly sensitive for the presence of a radiographically confirmed stroke. TEG-PM may be a useful diagnostic tool in evaluation of stroke, similar to its use in other settings, particularly trauma, and should be considered to be a routine test in initial stroke evaluation.

In 2021, Li et al, [12] evaluated the benefits of individualized antiplatelet regimens based on TEG-PM parameters for patients with aneurysmal subarachnoid hemorrhage who underwent stent-assisted coiling (SAC). This study proved that the individualized antiplatelet therapy based on TEG-PM parameters could reduce the bleeding risks of patients with aneurysmal subarachnoid hemorrhage after SAC without increasing the rates of thromboembolic events and unfavorable outcomes. It seems that there are currently groups among neurological patients that could use the potential of TEG PM, especially if we are interested in platelet aggregation depending on ADP or AA. Moreover, this assay measures clot strength, maximal amplitude, reflecting maximal platelet function, and detects the reduction in platelet function, presented as percentage inhibition, by both aspirin and clopidogrel. The standard for measuring platelet inhibition is light transmission aggregometry (LTA) [13]. However, LTA method is challenged for lacking standardization, time-consuming and complicated sample preparation, which limits its clinical routine. Although TEG might be more reflective of the physiologic character of a blood clot in vivo, its correlation with currently widely used platelet function testing has not been validated. The low analytical variation of the TEG-PM assay may reflect the use of whole blood, obviating pre-analytical and analytic factors such as platelet count and size, preparation of platelet rich plasma, including centrifugation steps. It seems that this test can benefit to prevent and reduce thromboembolic events. TEG-PM could be identified as parameters for tailor-individualized antiplatelet treatment designed to reduce ischemic events and bleeding. However, there is little consensus regarding how to adjust the antiplatelet regiment according to TEG-PM parameters. It is necessary to further investigate in order to search for new possibilities of using TEG PM tests in patients in neurology.

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