

Comment on "Hyperresponsiveness of platelets in ischemic stroke" by Fateh-Moghadam et al.

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Dear Sir,

We would like to comment on the recent publication by Fateh-Moghadam et al. concerning the 'hyperresponsiveness' of platelets following an acute ischemic stroke (1).

Our first point relates to the aggregation results immediately following stroke compared with those at three months. Although a significant fall in ADP- and epinephrine-induced aggregation was found, the most striking change occurs with arachidonic acid-induced aggregation (Figure 1 in [1]); the authors' conclude that "systemic platelet activation is enhanced in patients with acute stroke or temporary ischemic attack (TIA) and returns to baseline

levels at three months follow-up". Our interpretation of these data would be 'that it is aspirin therapy alone, or together with an inherent fall in platelet activity, that has resulted in decreased platelet hyperresponsiveness at three months following an acute stroke'; the dramatic decrease seen with arachidonic acid would suggest that aspirin has a noteworthy effect in this situation.

Our second point relates to the lack of consistency between different platelet markers; in this study PAC-1 appears to relate to long-term risk of recurrent events, whereas the aggregation data identifies early changes in function. This suggests that we must 'tailor' the platelet test used to the specific question being posed, making straightforward "point-of-care" testing more difficult. Although attempts have been made to standardise techniques for platelet analysis, there is no agreed consensus on which tests should be used for which purpose (2).

Finally, the study demonstrates that PAC-1 measured at three months has a predictive value for future cerebrovascular events. The highest risk of recurrent events following a stroke is in the immediate days and weeks (3). These events therefore would not have been predicted by a test at three months post event; from the data it is not entirely clear if the events 'predicted' all took place after the three-month time point. If not, it is not surprising that PAC-1 levels were elevated in patients acutely recovering from recurrent strokes.

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