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LETTER

Evaluation of inflammatory conditions associated with aspirin resistance

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We read with great interest the recently published article by Tasdemir et al. (1) entitled 'Aspirin resistance in patients with type II diabetes mellitus'. In that well-described study, the authors (1) investigated the prevalence and predictors of aspirin resistance in diabetic patients. They found that presence of diabetes mellitus had no effect on aspirin response, and hypercholesterolemia was the only predictor of aspirin resistance in multivariate analysis in diabetic patients. Although this study provides us with extensive information, and we commend the authors for the excellent data that they have provided, some comments may be of interest.

The authors have mentioned that high levels of cholesterol diminish aspirin responsiveness in diabetic patients due to the reduced membrane fluidity associated with the excessive accumulation of cholesterol in platelet membranes, and infusion of reconstituted high-density lipoprotein (HDL) cholesterol is highly effective in reversing the excessive accumulation of cholesterol in platelet membranes. Nonetheless, Kotani et al. (2) have reported that decreased aspirin responsiveness was related to the increased activity of aspirin esterase in older type II diabetics, and greater aspirin hydrolysis was associated with the decreased levels of HDL cholesterol as well as increased levels of total cholesterol, thereby linking activity of aspirin esterase to cholesterol metabolism.

Inflammation and oxidative stress are usually accompanied by increased platelet activation and aggregation (3–6). Increased expression of cyclooxygenase-2 associated with inflammation may induce generation of thromboxane A2, thereby resulting in a prothrombotic state (4–6). Inflammatory conditions such as hypertension, acute coronary syndrome, heart failure, stroke, connective tissue disease, Crohn's disease, ulcerative colitis, psoriasis, and end-stage renal failure are associated with an increased platelet reactivity, and could be related with the development of inadequate response to aspirin or aspirin resistance (4–6).

In conclusion: since inflammation, oxidative stress, endothelial dysfunction, and insulin resistance are essential parts of the whole, and associated with the aspirin response, it could be conceivable that inflammation and insulin resistance could increase the development of impaired platelet activation, thus playing an important role in the pathogenesis of aspirin resistance (7,8). Evaluating insulin resistance and inflammatory status more comprehensively (more than sedimentation rate), and excluding patients with inflammatory conditions in addition to end-stage renal disease, could add more consistency to the results and help in elucidating the mechanism of the observed effects. Since Pulcinelli et al. (9) have mentioned that platelets become less sensitive in patients taking aspirin for a long time, period of aspirin treatment should also be provided in detail in studies associated with aspirin resistance.

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