

Current Concepts Targeting Antiplatelet ‘Resistance’

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The pivotal position paper of the Working Group on oral antiplatelet drug resistance of the Polish Cardiac Society published in this issue of *Kardiologia Polska* represents a high-quality, comprehensive analysis of the available evidence with regard to this controversial clinical matter [1]. The importance of the messages, balanced opinions, and perfect timing of publication is hard to underestimate. Hopefully, this publication will be a ‘cold shower’ to the advocates of uniform aggressive oral antiplatelet strategies, which are obviously so prevalent today. Doubled and even tripled clopidogrel loading regimens unjustified by randomized data, attempts to increase the maintenance clopidogrel dose based on low platelet responsiveness and even hypothetical ‘resistance’ cause extra bleeding risks with no definite evidence whatsoever of better vascular outcomes. In fact, the postulate ‘the more, the better’, while being reasonable for cholesterol lowering with statins, and which also works well for the treatment of arterial hypertension, is very questionable when applied to oral antiplatelet regimens and improved outcomes, especially considering hardcore randomized evidence. The position paper is well written, very focused, and balanced, and will be useful for the scientific and clinical community. Despite obvious industry pressure, the authors were able to keep their conclusions reasonable, and took the high road interpreting the facts fairly and honestly.

It seems very reasonable to divide ‘resistance’ after oral antiplatelet agents into clinical and laboratory phenomena. The authors are obviously right when suggesting that these two entities are not equal, especially from the prognostic viewpoint. I fully agree that ‘clinical resistance’ and ‘treatment failure’ are not appropriate terms, especially when we do not monitor compliance, and

clopidogrel may not be on-board at the time of the vascular event [2]. The authors should also be acknowledged for their intelligent estimation of the laboratory algorithms assessing response after antiplatelet therapy, and the reasonable suggestion not to overestimate the platelet test findings, and advocating using caution in extrapolation of the platelet biomarkers to the clinical arena. I also support the statement that it is currently impossible to fairly assess the prevalence of ‘resistance’ due to the lack of standard platelet tests, and the inability to routinely measure active (thiol), or inactive (carboxyl) clopidogrel metabolites in determining platelet response after thienopyridine. The authors of the position paper admit that based on the present data from randomized trials, there is no reason to monitor antiplatelet potency of oral agents, which is also in full agreement with the European and US recommendations.

In short, I am happy to endorse this index position paper and wish the authors ultimate success in their research endeavors and clinical achievements.

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References

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