Letters to the Editor

Switching Thienopyridines: Hypothetical Versus Real Risks

I enjoyed reading the quality paper by Campo et al. (1) that tried to determine whether platelet response after thienopyridines is drug or class specific in a broad spectrum of post-stent patients. The team should be acknowledged for the effort and for realistic rates for low response after clopidogrel (21%), and ticlopidine (19%). The major take-home message conveyed to the readership is that clopidogrel-treated patients may be switched to ticlopidine if “resistance” is determined by the platelet tests. However, the practical implications of this idea are not obvious, may be dangerous, may not be supported by clinical or epidemiologic evidence, and deserve at least some clarification and/or adjustment.

In fact, low response to clopidogrel as a major risk factor for the worsened vascular outcomes has been suspected but never proven to be a real clinical phenomena, especially considering that no load 75 mg clopidogrel saved 119 lives, and provided an absolute to be a real clinical phenomena, especially considering that no load 75 mg clopidogrel saved 119 lives, and provided an absolute to be a real clinical phenomena, especially considering that no load 75 mg clopidogrel saved 119 lives, and provided an absolute to be a real clinical phenomena, especially considering that no load 75 mg clopidogrel saved 119 lives, and provided an absolute to be a real clinical phenomena, especially considering that no load 75 mg clopidogrel saved 119 lives, and provided an absolute...

On the other hand, substituting clopidogrel with ticlopidine definitely increases the bone marrow toxicity risks. Indeed, neutropenia and thrombocytopenia were 2-fold higher in the ticlopidine arm than in patients treated with clopidogrel in CLASSICS (Clopidogrel Aspirin Stent International Cooperative Study) (4). Double cytotoxicity rates after ticlopidine were confirmed in a post-stent study (5) and a recent meta-analysis of 11,668 patients (6). Therefore, the suggestion that in case of low platelet response after clopidogrel patients should be switched to ticlopidine is not valid. Unless there is proof that response after clopidogrel is indeed linked to the clinical outcomes, monitoring compliance and potential tailoring of dual antiplatelet regimens with aspirin and clopidogrel will be a safer alternative than switching thienopyridines.

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REFERENCES