

SHOULD ASPIRIN-RESISTANCE BE ROUTINELY APPLIED TO TAILOR SECONDARY STROKE PREVENTION? NO

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In the field of stroke prevention, the commonest mistake I see being made in emergency departments is doubling the dose of aspirin in a patient who has had a TIA or stroke. The second commonest is spending too much time fussing about what antiplatelet agent should be given if aspirin failed.

Antiplatelet agents will only prevent ~ 25-30% of strokes, because they will only prevent strokes that are caused by embolization of platelet aggregates (white thrombus).

There are two main kinds of aspirin resistance: clinical and laboratory. Clinical aspirin resistance (the occurrence of TIA or stroke in the face of aspirin therapy) is not usually due to laboratory aspirin resistance; it occurs because the stroke was caused by a mechanism other than embolization of platelet aggregates (white thrombus).

Laboratory aspirin resistance is too complicated to be useful clinically, and the testing is not widely available. When a patient presents with a TIA or stroke in the face of aspirin, instead of fussing about the dose of aspirin, we should be asking what was the cause of the event? If it was a hypertensive lacunar infarction, the blood pressure needs to be controlled. If it was an embolus of atheromatous debris from carotid stenosis, the patient needs intensive statin therapy and an endarterectomy. If it was cardioembolic, the patient needs anticoagulants, and if it was from giant cell arteritis, the patient needs high-dose corticosteroids.

If, after considering other diagnoses, antiplatelet therapy still seems to be the primary treatment needed, it is more useful to add an antiplatelet agent of a different class. With recent advances, it seems likely that the best choice will be ticagrelor, with low-dose aspirin.

References:

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