EDITORIAL

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# Aspirin Resistance, True or False?

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Aspirin (acetylsalicylic acid; ASA) is one of the most commonly used drugs, Studies have showed that low dose ASA can reduce cardiovascular events by irreversibly binding to cyclooxygenase and inhibiting platelet aggregation. However, ASA has not prevented all patients from cardiovascular events [1]. Ackerman and Newman were the first to describe the phenomenon of Aspirin resistance (AR) in 1990, named "incomplete antiplatelet effect of aspirin". Afterwards, in the last two decades, several studies have demonstrated that some people do not respond to low dose aspirin as an anti-platelet [2].

Several mechanisms are believed to be involved in AR i.e. dose alternation, type of the aspirin, drug interactions, genetic polymorphisms of genes involved in thromboxane synthesis like COX-1 and COX-2 [3]. However, no subjective definition of AR is present. Furthermore, there are several in-vivo (Bleeding time and occurrence of vascular event during aspirin use) and in-vitro (PFA-100, Light transport agerogometry, thrombo-elastography, Urinary Tromboxane B2) methods to evaluate the platelet functions. Numerous studies have also used these methods to estimate AR prevalence throughout normal population or specific diseases like diabetes or coronary artery disease [4]. Most of the results were vague and we can extract a range of near "zero" to "more than 75%" of AR in different populations, which seems to be controversial [5-6].

The term "resistance" in this case is not purely an objective definition. There are cases which in-vitro studies indicate a subject as AR, but no vascular event has occurred, and vice versa, in terms of laboratory findings [7]. The fact is that there are some alternation in response to ASA in different bodies and maybe, there are different degrees of aspirin resistance in some cases, but it does not prove that all patients with a cardiovascular episode under ASA use are "Biologically" AR, or, a "Biologically suggested" AR does not respond well to ASA at all. Accordingly, different methods sometimes demonstrate different results in a single case. Therefore, in my opinion, AR is more a "research field" rather than a "clinical fact". It is not suitable to use it in daily practice, at least until there is enough evidence to provide a gold-standard method to evaluate patients with high accuracy. It seems inevitable to prescribe low dose ASA, along with other anti-platelet drugs such as Clopidogrel, for all coronary artery disease patients except those who have hypersensitivity to ASA or other contraindications.

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