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New Insights on Long-Term Aspirin Therapy in Primary Prevention for Cardiovascular Disease

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Short Communication

The atherosclerosis-related cardiovascular diseases, such as coronary artery disease, stroke and peripheral artery disease, are the leading cause of death and morbidity in the United States and Europe [1].

The pathophysiology of atherosclerotic plaque is essential to understand the criticality of these diseases and the correct functioning of pharmacological strategies [2].

The use of statins, both primary and secondary prevention, it is well established in clinical practice based on scientific evidence; however, the use of aspirin in primary prevention is widely debated.

Current ESC guidelines recommend the use of long-term antiplatelet therapy in asymptomatic carotid artery stenosis (Grade 1B) [1], and in patients suffering from stable coronary artery disease (Grade 1A) [3], but do not recommend the use of aspirin in people with diabetes who do not have clinical evidence of atherosclerotic disease (Grade 3A) [4], due to increased risk of bleeding. Interestingly, recent studies observed differences in the efficacy and safety of antiplatelet therapy; a lower platelet activity and platelet inhibitory response to therapy were described in diabetic women [5].

Results from the 10-years follow up of the JPAD study (Japanese Primary Prevention of Atherosclerosis with Aspirin for Diabetes) [6] evidenced that 81 mg to 100 mg dose of aspirin was not able to reduce the incidence of adverse cardiovascular events in a Japanese type 2 diabetes population [6].

In Saito's results were described 371 adverse cardiac events in the whole population, 151 in patients taking aspirin and 166 in patients not taking aspirin, including coronary artery thrombosis (125), cerebrovascular accidents (133), and peripheral vascular events (45) [6], finding no significant differences between the two groups regarding sudden death, coronary artery, cerebral, and peripheral artery thrombosis. Similar results were confirmed after adjustment for common cardiovascular risk factors [6].

Regarding haemorrhagic events, no differences were found in the two study groups; in particular, 80 haemorrhagic events (6%) were described in patients taking aspirin and 67 (5%) in patients not taking aspirin ($p=0.2$).

Authors reported a higher incidence of gastrointestinal bleedings in those taking aspirin (25, 2%), in comparison to the others (12, 0.9%; $p=0.03$). No intracranial hemorrhages were reported [6].

These results confirmed previous findings [7] and were in disagreeing with a previous statement [8]. A possible explanation was that the use of high efficacy statins has significantly contributed to the reduction of events in recent years in primary prevention patients [9]. This topic is still widely debated. In a recent work, Verdoia et al. [10] showed that the use of rosuvastatin was associated to a lower reactivity of clopidogrel, suggesting cautiousness in their combination. Mechanisms underlying this phenomenon could involve the cell metabolism of adenosine and intracellular cAMP levels [11], but the real impact on clinical practice is still to be quantified.

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