

Solid cancers after antiplatelet therapy: Confirmations, controversies, and challenges

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Abstract

© Schattauer 2015. The role of anticoagulants and antiplatelet agents in tumour growth and prognosis is not new, and currently under intense investigation. Some randomised data strongly suggest that this association exists, but it is complex, and not necessarily pointed at the same direction. The potential mechanisms responsible for such harmful association include a direct hazard of novel antithrombotics on cancer, indirect promotion of tumour growth, easier metastatic dissemination due to instability of platelet-tumour cell aggregates, or/and inability to keep cancer cells locally in situ are considered. The latest randomised evidence ultimately rejected the drug-specific cancer risks, clearly indicating the class-effect. In lay terms “cancers follow bleeding”, which seems to be true for antithrombotic agents in general. Significant excess of solid cancers which was similar after prasugrel in TRITON, and with vorapaxar in TRACER trials was confirmed by the FDA reviews. Later, extra cancer deaths reported following clopidogrel and prasugrel in DAPT, and after ticagrelor in PEGASUS are also of concern. However, there are remaining controversies with regard to published cancer risks after ticagrelor (PLATO), or another vorapaxar trial (TRA2P), while full disclosure of separate clopidogrel and prasugrel cancer data in DAPT is still lacking. In short, if we apply moderate antiplatelet strategies for over two years, or aggressive regimens including triple therapy for much less than one year, the solid cancer risks emerge. Currently, more delicate platelet inhibition, and shorter exposure to dual oral antiplatelet agents should prevail.

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Keywords

Aspirin, Cancer, Clinical trials, Clopidogrel, Duration of antiplatelet therapy, Prasugrel, Ticagrelor, Triple antiplatelet therapy, Vorapaxar