Antithrombotic therapy use for patients with atrial fibrillation undergoing percutaneous coronary intervention: new data in an area of limited evidence

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Atrial fibrillation (AF) is the commonest sustained cardiac arrhythmia, which confers a risk of stroke and thrombo-embolism, and thromboprophylaxis is recommended with oral anticoagulation for those at moderate-to-high risk of stroke. Antiplatelet therapy is clearly less effective for stroke prevention than vitamin K antagonists (VKAs).

Dual antiplatelet therapy, in contrast, is recommended after coronary artery stenting to reduce the increased risk of stent thrombosis. Oral anticoagulant therapy is less effective after stenting, reflecting the pathophysiology of platelet-rich thrombus in stents, and so the need to inhibit platelet aggregation to prevent stent thrombosis.

Given that AF commonly co-exists with vascular disease, increasing numbers of AF patients undergo percutaneous coronary artery interventions (PCI). However, there are limited data on the optimal antithrombotic management strategy in such patients. Triple antithrombotic drug therapy (i.e. warfarin, clopidogrel, and aspirin) appears to be the best treatment option in these patients, although such a strategy is associated with an increased bleeding risk.

Current guidelines only briefly address this management problem and advise clinicians to discontinue VKA therapy before the procedure—which increases thrombo-embolic risk—and post-PCI to use bridging therapy with heparin until international normalized ratio (INR) levels reach therapeutic range, but this is often accompanied with an increase in peri-procedural bleeding risk. Achieving a balance between causing thrombotic events as much as possible and not causing major bleeds is even more delicate for long-term therapy, for example after acute coronary syndromes.

Halbfass et al. report a single-centre experience of their patients with AF undergoing PCI. The authors confirm the high risk of both thrombotic and bleeding events in this population, which (as expected) consisted of usually elderly patients with multiple co-morbidities. Other studies have already identified high rates of events (both thrombotic and bleeds) in similar populations. In general, discontinuation of oral anticoagulants causes a high stroke risk. Interestingly, triple therapy was not associated with major haemorrhage episodes. Thrombo-embolic events, in contrast, were associated with no or ineffective (INR <1.8) anticoagulation.

Although the study has the same limitations as other published series, that is, a small sample size and retrospective analysis, this new study supports that a strict evaluation for both thrombotic and haemorrhagic risk should be performed before PCI and coronary stenting in order to determine the best antithrombotic therapy management option.

Clinical factors associated with the risk of ischaemic complications (mainly stroke) are well-known (e.g. described in the CHADS2 and similar scores). Factors that may help identify an increased risk of bleeding include the use of Gp IIb/IIIa inhibitors, chronic kidney disease, or a high INR value (>2.6). An important opportunity for individualized therapy may be the duration of combination therapy in anticoagulated patients undergoing PCI: longer durations of ‘triple therapy’ are associated with markedly higher bleeding risk as shorter (1 month) durations in prior studies. Large, prospective registries with consecutive patient enrolment and prospective clinical studies are necessary in order to determine the best management of these patients.

Until such data become available, the debate on the optimal antithrombotic strategy for anticoagulated patients undergoing PCI/stenting will continue to echo in pages of learned journals. A position paper generated by several organizations within the ESC will hopefully provide an integrated basis for educated guessing of optimal antithrombotic therapy in anticoagulated patients undergoing PCI in the near future. The paper by Halbfass et al. adds valuable information to this underexplored area of clinical cardiology.
References


