Atrial fibrillation (AF) is a common sustained cardiac arrhythmia afflicting more than 6 million inhabitants in Europe. It bears an increased risk of stroke and higher mortality than sinus rhythm since it predisposes to the formation of atrial thrombi and may cause embolization to the systemic circulation and hitting the brain with and increased 4 – 5 fold risk of stroke. About 50% of victims of embolic stroke will die within one year. Age is an important risk factor for development of AF afflicting about 2.5% of population by the age of 50 years and increasing sharply thereafter being present in about 25% of people of 80 years of age and older.

Anticoagulation proved to be superior to antiaggregation and so warfarin and other anti-vitamin K antagonists have long been considered the therapeutic gold standard to prevent embolism in this situation. Vitamin K antagonists were the only available medication for about 60 years. Although a significant number of patients do not take medication or do not accomplish with the therapeutic INR index, being either above or below the therapeutic range. New oral anticoagulants (NOAC) appeared in recent years and are an attractive alternative because of their stability, no interaction with vitamin k dietary intake, fewer interactions with other medications and no need to monitoring. Additional advantages are rapid onset and offset of effect, lower risk of hemorrhage, particularly cerebral hemorrhage. In fact, meta-analysis including NOAC showed a large reduction in hemorrhagic stroke by half, all-cause mortality was also significantly reduced with NOAC versus warfarin while ischemic stroke and myocardial infarction were not.

Nevertheless, some questions remain. First, the lack adequate experience in some situations and second the absence of an antidote to reverse their action.

One particular case is history of cerebral hemorrhage. Clinical trials showed that NOAC have a smaller risk of intracranial hemorrhage compared to vitamin K antagonists. However the risk of using NOAC in patients with antecedents of cerebral hemorrhage is not known. In fact NOAC were not tested in patients with history of cerebral hemorrhage since they were excluded from clinical trials. This fact along with the lack of an antidote to reverse their effect precludes, in our opinion, its use until robust clinical data will be available.

References


