ATRIAL FIBRILLATION AND STROKE PREVENTION – IS WARFARIN STILL AN OPTION? YES

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There is no doubt that under-anticoagulation of patients with atrial fibrillation is an important cause of stroke[1]. Gladstone et al.[2] found that among patients admitted with stroke and atrial fibrillation, only 10% of those who should have been anticoagulated had a therapeutic INR. There is also no doubt that if the newer anticoagulants were inexpensive, they would soon replace warfarin. Unfortunately, they are too expensive, so we need to learn to do a better job with warfarin.

Using warfarin better should be a priority for physicians whose work is stroke prevention. The INR matters; a better percent of time spent in the target range for INR reduces stroke risk from 2.3% to 1.2% [3]. Better use of warfarin includes understanding of drug interactions[4], factors that affect warfarin response[5], and methods for reducing turbulence of the INR. A daily dose of oral vitamin K will increase the required dose of warfarin, while reducing turbulence of the INR due to dietary changes[6].

Stroke prevention in atrial fibrillation is not only about warfarin. Elevated levels of plasma homocysteine (a clotting factor) quadruple the risk of stroke in atrial fibrillation[7]; a major unrecognized cause of elevated homocysteine is deficiency of vitamin B12[8]. Besides better anticoagulation and treatment of unrecognized B12 deficiency there are important options for patients who refuse anticoagulation, or cannot safely take anticoagulants. Although the trials of devices to occlude the atrial appendage have to date been fraught with rather high complication rates, another option is less invasive endoscopic surgery to remove the left atrial appendage.

Reference List

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