Starting anticoagulants in post-stroke AF patients: how soon?

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Abstract

Point of view: No

It is an unusual opportunity to be tasked with the request to take the negative position on time in of starting anticoagulants in post-stroke atrial fibrillation patients. I usually begin the anticoagulant program earlier than later.

I have two hedge my comments based on the assumption of the stroke type; its location, size, isolated event, or the most recent of several events; coexisting brain lesions contraindicating anticoagulation, and systemic factors that may influence anticoagulation safety.

Most atrial-fibrillation-related strokes are infarct in type, the most common path up the internal carotid to the circle of Willis; from there to the middle cerebral artery stem and assuming a typical bifurcation, passing into the lower division; the final lodgment occurring in the posterior sylvian region and posterior temporal lob. The most common syndrome is a Wernicke type aphasia in the dominant hemisphere, and behavior disturbance with hemineglect in the nondominant. The severity of the syndrome reflects the degree to which one or more of the usual three branches of the lower division are affected, and whether collateral from the posterior cerebral artery minimizes the extent of temporal, parietal, and lateral occipital infarction.

Assuming the infarct is confined to the posterior portion of the sylvian fissure with good collateral, there should be no hesitation in starting anticoagulants as soon as the syndrome is clinically evident and the extent of the injury documented by imaging. Relying on the syndrome alone was the classical approach before imaging. I have several painful examples of patients with primary hematoma or major hemorrhagic infarction, neither the diagnosis or stroke severity obvious on initial clinical examination alone.

In other territories, early trials with thrombolytics demonstrated even hemorrhagic conversion for infarcts of one gyrus size seemed well-tolerated and were not unduly made worse by early anticoagulation.

Obstructions in the distal intracranial internal carotid or major circle of Willis vessels have so often been followed by hemorrhagic changes in the lenticulostriates that those in our group have been reluctant to recommend early anticoagulation for fear of exaggerating the already major hemorrhagic conversion.

Few would argue against withholding anticoagulants when major intracranial hemorrhagic disease coexists. In many the first imaging ever performed on the patient was after a stroke, because anticoagulants were instituted when atrial fibrillation was discovered without brain imaging beforehand.

The tolerance of the brain for simultaneous anticoagulation, with intracranial aneurysms, arteriovenous malformations, or even amyloid angiopathy seems quite remarkable.

The per-day recurrent embolic risk in a setting of atrial fibrillation is static and low. Early institution of anticoagulants is
not required in the initial days after a stroke event. However, a prothrombotic state justifies early intervention.

The common practice of delaying anticoagulants for a week or more seems outmoded, now that the features of the stroke can be well-characterized by modern imaging.

Our practice is to use a flat dosing program (no loading dose) if warfarin is the choice. Aspirin is given daily during the running phase can be discontinued when the INR appears in a therapeutic range. Recalling how long it takes the intravascular coagulation status to be reflected by the INR means that most cases are likely not to be fully anticoagulated even when the clinicians take the INR at face value.

The introduction of oral thrombin inhibitors are changing the anticoagulant management program and when successful could mean early therapeutic anticoagulant values will become common and allow the testing of whether hemorrhagic conversion will prove more or less common compared with the traditional oral warfarin programs. Aspirin or other antiplatelet agents were initially assumed to be safe with these new oral thrombin inhibitors, but less so at present.