Predicting Stroke Risk in Patients With Atrial Fibrillation

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See related article, pages 2459–2463.

Predicting which patients with atrial fibrillation will have a stroke or systemic embolic event is not an easy task. In this issue of Stroke, Lawrence Baruch and colleagues retrospectively compared 7 risk stratification schemes in a large clinical trial–based program of patients with atrial fibrillation (the Stroke Prevention using an ORal Thrombin Inhibitor in atrial Fibrillation [SPORTIF] III and IV studies).1 As assessed by the concordance (or C) statistic, which measures the area under a test’s receiver-operating characteristic curve, all prediction schemes performed rather modestly, with the best C statistic belonging to the CHADS2 scheme (C=0.65).

At first glance, these data might suggest caution regarding the use of formal risk stratification schemes to predict stroke in patients with atrial fibrillation. However, many potential caveats apply. In much of the original validation work for such schemes, patients were not selected by the presence of additional risk factors such as hypertension or heart failure, whereas in the SPORTIF program, only patients with atrial fibrillation judged to be at high risk because of the presence of concomitant stroke risk factors were included.2,3 Therefore, as recognized by the authors, the present study included few patients at low risk for stroke, thereby hampering the ability of the stratification schemes to separate patients on the basis of predicted risk. Clinical trials are often criticized because they include patients at the extreme lower end of the risk spectrum, but the converse is actually true in this case, because of the deliberate selection criteria of SPORTIF.

Another limitation is that the risk stratification schemes were developed and are still most often applied to atrial fibrillation patients not yet on oral anticoagulants, whereas all patients in SPORTIF received effective antithrombotic therapy from inception. The effect of such therapy is to blunt risk across the board, again compressing the range of risk in the present study. It has been shown that noncardioembolic subtypes of stroke predominate in patients with atrial fibrillation who are well-controlled on oral anticoagulants,4 and it is likely that other risk factors—not included in the risk stratification tools studied here—are of greater importance in such patients.5 Finally, a number of statistical concerns have been expressed regarding the use of the C statistic for gauging and comparing the effectiveness of risk prediction tools.6,7

These limitations should be balanced against the strengths of this study. Clinical trials provide a high degree of quality control, including the formalized adjudication of individual outcomes such as stroke, hemorrhage, and systemic embolus. Such adjudication is almost certainly more accurate than reliance on administrative healthcare databases although a combination of both techniques is probably more optimal than either technique used alone. As well, side-by-side comparisons of the predictive capabilities of multiple risk stratification schemes in the world of atrial fibrillation are relatively uncommon; the investigators should, therefore, be congratulated for attempting such an analysis.

So what if anything do these data tell us? Notwithstanding the caveats noted above, the study by Baruch and colleagues does provide evidence that in patients with atrial fibrillation who are already well-treated with anticoagulation, existing risk stratification schemes may not work as well as we might have thought. Confirming this finding would require prospective validation in a community-derived sample of patients with atrial fibrillation who have a broader spectrum of risk, preferably both treated and not treated with anticoagulants. In the meantime, physicians need to pay attention to the entire panoply of treatable risk factors in patients with atrial fibrillation; it is likely that vasculoprotective therapies such as statins and antihypertensive agents play an important additive role to coumarin anticoagulants in this setting.8,9

Disclosures

None.

References


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