Letters to the Editor

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Resumption of Anticoagulation During Hypertensive Cerebral Hemorrhage With Prosthetic Heart Valve

To the Editor:

We would like to bring to your attention our recent dilemma of when to re-anticoagulate a patient with a hypertensive cerebral hemorrhage who concurrently has a prosthetic heart valve.

The patient, a 63-year-old woman, had had her aortic heart valve replaced in 1978 with a modified Bjork-Shiley prosthetic valve and had been taking warfarin without bleeding complications. She was also hypertensive and treated with enalapril. She was admitted to a local community hospital after having been found unconscious and was later noted to have right hemiparesis with attention, language, and memory abnormalities. Computed tomography (CT scan) showed a left thalamic hemorrhage that had extended into the third ventricle, the internal capsule, and the caudate nucleus. The anticoagulant was stopped after admission, and she was transferred to our service a few days later.

We faced the difficult decision of whether to restart the warfarin and risk the possibility of rebleeding or to wait an indefinite period of time during which the risk of embolization would be great. An extensive literature search directed at guiding our strategy was fruitless. We decided to treat her with platelet antagonists for 2 weeks (empirically chosen) and then restart the warfarin. On Day 10, however, she became unresponsive to verbal stimuli, inattentive to her surroundings, and showed complete motor deficit. Repeat CT scan failed to show any new areas of bleeding, and she was therefore started on heparin i.v. drip at a rate of 800 units/hr without any preceding loading dose. In <24 hours she began to improve and eventually was switched from heparin to warfarin. No rebleeding occurred and her neurologic condition has remained stable.

Literature supporting a rationale for re-anticoagulating patients in this setting is not available. Although the natural history of intracerebral hemorrhage suggests this is a monophasic event, it is unknown whether the addition of anticoagulant drugs increases the risk of rebleeding. We suggest that reinstitution of anticoagulant therapy after the first 10 days following an intracerebral hemorrhage may be a rational option. If this form of therapy is contemplated, it appears prudent to use slowly increasing dose schedules rather than large initial doses.

Camillo R. Gomez, MD, AFACA
Jagveer Sandhu, MD
Parag Mehta, MD
Cerebrovascular Disease Service
Department of Neurology
St. Louis University School of Medicine
St. Louis, Missouri

Resumption of Anticoagulation After Intracranial Bleeding in Patients With Prosthetic Heart Valves

To the Editor:

Recipients of prosthetic heart valves are anticoagulated because they are at an increased risk for embolism.

This risk is reduced by therapy, but in 2% of patients on long-term warfarin, the treatment is complicated by serious, frequent intracranial, hemorrhage. When to resume anticoagulation has not been addressed previously. We describe our recent experience.

We reviewed our records for patients with prosthetic valves who had intracranial bleeding while on anticoagulants. Six were restarted on warfarin and had radiologic studies (computed tomography in six instances and cerebral angiography with radionuclide brain scans in two) to confirm the clinical diagnosis. The findings are summarized in Table 1. Prothrombin time (PT) was determined within a mean interval of 3 days (12 hours to 7 days) from the onset of symptoms in all cases except for Patient 3, who had been symptomatic for 2 months. Warfarin was stopped as soon as the diagnosis was established and was resumed after a mean interval of 19 days. Thromboembolic events were not observed during the period off warfarin, and no clinical deterioration was evident during the 6 months following resumption of treatment. Patient 5 died of complications of bacterial endocarditis, and postmortem examination of his brain showed residual cysts from the frontal hematoma and separate cerebellar and occipital areas of encephalomalacia.

Hematostasis and enoxin staining and Congo red staining failed to show significant vascular wall abnormalities that could be attributed to warfarin.

The decision to resume anticoagulation in patients with prosthetic heart valves who have had intracranial bleeding hinges on the balance between the relative risks of valve thrombosis or thromboembolism, if the period off anticoagulants is prolonged, and rebleeding, if warfarin is resumed too early. The incidence of thromboembolism is determined by the valve's position and mechanical characteristics, associated cardiac arrhythmia, and the interval since valve implantation, and varies between 0.36 and 11.1 episodes per 100 patient-years. Additionally, data regarding the incidence of rebleeding are, to our knowledge, not available. An assessment of the latter's risk should be based on identifying precipitating causes such as hypertension, vascular malformations, and preceding infarction. Review of our data (Table 1) shows that in six of eight instances the admission PT was prolonged to more than twice the control.

The decision to re-anticoagulate was made in each patient allowing for PT to return to normal and after the mass effect of the hematoma started to resolve.

We conclude that the timing of re-anticoagulation in this setting should be individualized and based on an assessment of both the specific risk factors related to the valve and potential causes leading to the hemorrhage. An average waiting period of 19 days in our patients proved to be safe.

V.L. Babikian, MD
C.S. Kase, MD
Department of Neurology
Boston University School of Medicine
Boston, Massachusetts
M.S. Pessin, MD
L.R. Caplan, MD
Department of Neurology
Tufts University School of Medicine
Boston, Massachusetts
P.B. Gorelick, MD
Michael Reese Hospital
Chicago, Illinois

References

Atrial Septal Aneurysm as a Cause of Cerebral Embolism in Young Patients

To the Editor:

The article by Belkin and associates,1 in which they reported a high prevalence of embolic events in a series of 36 consecutive patients with atrial septal aneurysm, has several additional important clinical implications besides those put forth by the authors. First, atrial septal aneurysm should always be considered among the cardiac causes of cerebral embolism that can be detected by echocardiography in young patients.2

Second, the consideration should be even more serious if the patient develops simultaneous embolic events in both the systemic and pulmonary circulation since biatrial myxoma and paradoxical embolization are the only two other conditions that can cause bilateral embolization.

Third, the frequent association of paradoxical embolism with right-to-left atrial shunting with atrial septal aneurysm found by Belkin and associates results from their aggressive use of contrast echocardiography. The sensitivity of contrast echocardiography in the detection of paradoxical embolism, of course, might be further enhanced had they used it in conjunction with the Valsalva maneuver.3 As a matter of fact, when requesting echocardiography of a stroke patient in the evaluation for cardiogenic embolism, the referring physician should not only request contrast study but also specify the application of the Valsalva maneuver as routine echocardiography does not employ either of these maneuvers.4

Tsang O. Cheng, MD
Department of Medicine
George Washington University Medical Center
Washington, DC

References


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TABLE 1. Summary of Cases Restarted on Warfarin

<table>
<thead>
<tr>
<th>Patient/age/sex</th>
<th>Valve type</th>
<th>AC duration (months)</th>
<th>PT (sec)</th>
<th>BP (mm Hg)</th>
<th>Hematoma location</th>
<th>Days AC stopped</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/77/M</td>
<td>Bjork-Shiley aortic</td>
<td>132</td>
<td>20/12</td>
<td>160/80</td>
<td>Parietal</td>
<td>8</td>
</tr>
<tr>
<td>2a/55/F</td>
<td>Bjork-Shiley mitral</td>
<td>23</td>
<td>27/11</td>
<td>130/80</td>
<td>Parietal (L)</td>
<td>19</td>
</tr>
<tr>
<td>2b/55/F</td>
<td></td>
<td>.48</td>
<td>24/12</td>
<td></td>
<td>Temporal (R)</td>
<td>—</td>
</tr>
<tr>
<td>3/66/M</td>
<td>Bjork-Shiley aortic</td>
<td>129</td>
<td>21/12</td>
<td>130/80</td>
<td>Subdural</td>
<td>5</td>
</tr>
<tr>
<td>4/64/F</td>
<td>Ionescu-Shiley tricuspid</td>
<td>4</td>
<td>25/12</td>
<td>130/70</td>
<td>Subdural</td>
<td>13</td>
</tr>
<tr>
<td>5a/35/M</td>
<td>Bjork-Shiley aortic</td>
<td>32</td>
<td>26/12</td>
<td>160/80</td>
<td>Frontal (L)</td>
<td>27</td>
</tr>
<tr>
<td>5b/35/M</td>
<td></td>
<td>63†</td>
<td>32/12</td>
<td>130/80</td>
<td>Frontal (R)</td>
<td>—</td>
</tr>
<tr>
<td>6/47/F</td>
<td>Starr-Edwards mitral</td>
<td>48</td>
<td>40/12</td>
<td>140/70</td>
<td>Subdural</td>
<td>42</td>
</tr>
</tbody>
</table>

AC, anticoagulation with warfarin. AC duration is also interval since valve insertion. L, left; R, right.

†Staphylococcus aureus endocarditis.

Does Transcranial Doppler Ultrasonography Provide Information About Cerebral Microcirculatory Flow?

To the Editor:

In his editorial, J.C. Grotta regrets that "easily administered bedside studies such as ... transcranial Doppler do not provide information about microcirculatory flow". According to our experience with transcranial Doppler ultrasonography, this statement cannot be made unmodified. We use transcranial Doppler to detect changes of median artery flow velocity during breathholding tests or Valsalva maneuvers.1,2 These transcranial Doppler examinations can be performed as simple bedside tests. In patients with lacunar infarctions or white matter lucencies on computed tomography or magnetic resonance imaging, the expected changes of median cerebral artery blood flow velocity during breathholding tests or Valsalva maneuvers are often diminished or absent (unpublished observations). Similar abnormalities of autonomic regulation are detectable by transcranial Doppler measurements in patients with longstanding arterial hypertension but without lacunar infarctions or white matter lucencies on computed tomography examinations. A study to describe the specificity and sensitivity of these transcranial Doppler tests in detecting cerebral microvascular disease is under way at our department.

M. Reinecke
H.D. Langhorst
B. Görzel

Departments of Neurology and Neurophysiology
Staedtische Kliniken Fulda
Fulda, Federal Republic of Germany
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V L Babikian, C S Kase, M S Pessin, L R Caplan and P B Gorelick

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