Safety of Hemodynamic Augmentation in Patients Treated With Guglielmi Detachable Coils After Acute Aneurysmal Subarachnoid Hemorrhage

Venkatesh Aiyagari, DM; DeWitte T. Cross III, MD; Ellen Deibert, MD; Ralph G. Dacey, Jr, MD; Michael N. Diringer, MD

Background and Purpose—Guglielmi detachable coils (GDC) used in the treatment of intracranial aneurysms do not always completely occlude the aneurysm. Thus, after an acute subarachnoid hemorrhage (SAH), there is a theoretical risk of rebleeding from coiled aneurysms, especially when blood pressure is elevated. The aim of this study is to determine whether use of hemodynamic augmentation (HA) to treat delayed ischemic deficits (DID) will increase the risk of rebleeding in these patients.

Methods—Delayed ischemic deficits developed in 12 (7 women and 5 men, aged 31 to 64 years) of 51 patients treated with GDC for acute SAH over a 4-year period. Aneurysms in all 12 patients were ≥80% obliterated with GDC, and there was ≥90% obliteration of 78% of the aneurysms. Hemodynamic augmentation with fluids, phenylephrine, dopamine, and/or dobutamine was used to treat DID for a mean duration of 3 days (range 1 to 11 days).

Results—With HA, mean arterial blood pressure (MAP) rose 15% (range 0 to 30%) and systolic blood pressure (SBP) rose 13% (range 0 to 29%) above baseline. MAP was maintained at >10% above baseline for 65% of the treatment period. The maximum MAP was 104 to 170 mm Hg (mean 140 mm Hg), and maximum SBP was 154 to 261 mm Hg (mean 210 mm Hg). No patient had rebleeding or any significant complication during the course of therapy.

Conclusions—Based on this limited series of patients, we believe that it may be safe to use HA in patients treated with GDC for SAH. (Stroke. 2001;32:1994-1997.)

Key Words: radiology, interventional • subarachnoid hemorrhage • vasospasm, intracranial

Endovascular treatment with Guglielmi detachable coils (GDC) is being increasingly used as an alternative to surgical clipping of intracranial aneurysms.1–3 This method appears to have a lower rate of complete obliteration of the aneurysmal lumen.3 Delayed ischemic deficits (DID) are a leading cause of death and disability after aneurysmal subarachnoid hemorrhage (SAH).4 The treatment of DID involves (1) hemodynamic augmentation (triple-H therapy; HA), in which patients are treated with pharmacological agents to increase blood pressure or cardiac output, and (2) endovascular therapy with angioplasty or intra-arterial papaverine.5–7 When patients with acutely ruptured aneurysms treated with GDC develop DID and are treated with HA, there is concern that increased hemodynamic stress on the incompletely obliterated aneurysm may cause rebleeding.

The safety of HA in patients with ruptured aneurysms treated with GDC has not been well studied. The purpose of our study was to determine whether use of HA in this group of patients would cause rebleeding.

Subjects and Methods

We treated a total of 51 patients with acute ruptured intracranial aneurysms over a period of 4 years (December 1995 to August 1999) using GDC. Twelve of these patients (23%) were treated with HA for DID. Data were retrospectively collected from the medical records of all 12 patients.

Patients were managed in the neurology/neurosurgery intensive care unit of a tertiary care hospital. Aneurysms were treated by a team of interventional neuroradiologists. All patients received standard care for SAH and underwent hourly neurological examinations and recording of vital signs, including blood pressure monitoring by an automated cuff or an indwelling arterial catheter. Central venous pressure, pulmonary artery wedge pressure, and cardiac index (CI) were monitored at least every 4 hours with a Swan-Ganz catheter when clinically indicated.

Delayed ischemic deficit was diagnosed when decreased level of consciousness or new/worsened neurological deficits occurred 2 to 12 days after aneurysmal rupture in the absence of other causes. All patients underwent confirmatory angiography. In patients with a severe neurological deficit, DID was diagnosed on the basis of vasospasm on surveillance angiogram.

Delayed ischemic deficits were treated with volume expansion combined with pharmacological agents such as phenylephrine,
A total of 14 aneurysms in 12 patients (aged 31 to 64 years, 7 women) were treated with GDC (Table 1). Eleven aneurysms were obliterated by 90%. Five patients had other untreated aneurysms.

The clinical signs of DID were decreased level of consciousness in 5 patients, decreased level of consciousness with a new focal deficit in 4 patients, and a new focal neurological deficit in 1 patient. Two patients (cases 6 and 9) had only angiographic evidence of vasospasm and were untreated aneurysms. Two patients (cases 6 and 9) showed no improvement, and 1 patient worsened despite treatment. No patient had aneurysmal rebleeding during therapy (95% confidence limits 0% to 25%).

Six patients improved by at least 2 points on the Glasgow Coma Scale (GCS), and 2 improved by 1 point. Three patients showed no improvement, and 1 patient worsened despite treatment. No patient had aneurysmal rebleeding during therapy (95% confidence limits 0% to 25%). All patients received substantial amounts of intravenous fluids (9.8 ± 2.8 L/d). HA was effective in raising the SBP (average and maximum) and MAP (average and maximum) in most of the patients. The MAP was maintained at >10% of the baseline for an average of 65% of the duration of therapy. One patient treated with dopamine alone had a 15% increase in cardiac index. The mean duration of treatment was 4 days (range 1 to 12 days; Table 3).

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Discussion

Surgical clipping of aneurysms is effective in preventing rebleeding and allowing safe use of HA. Residual “aneurysmal rests,” which may predispose to aneurysm regrowth and rebleeding, are infrequently seen. On the other hand, the persistence of a small neck remnant is not uncommon after endovascular treatment. A recent review reported that of all ruptured aneurysms treated with GDC, 48.1% had <100% obliteration and 6.3% had >90% obliteration of the aneurysm. In addition, persistent flow within the interstices of the coil mass may be present. These issues are worrisome when a patient needs HA.

Gruber et al. noted an increased incidence of vasospasm-related infarctions in patients treated endovascularly. This is of concern, because a larger population would require HA and be exposed to a risk of rebleeding. Other authors have not found an increased risk of vasospasm.

A few reports mention the use of HA in patients treated with GDC. Bernardini et al. treated 7 patients with pressors.
for 1 to 9 days for symptomatic vasospasm. Five patients had ≥90% obliteration of the aneurysm. The mean blood pressure was 118 mm Hg, and peak SBP was 195 to 250 mm Hg. The authors did not mention how long hypertension was maintained or the percent increase over baseline. There were no episodes of rebleeding. Charpentier et al11 treated vasospasm in 25 patients using hypervolemic therapy and induced hypertension to keep the SBP above 150 mm Hg. Details of the efficacy and side effects of this treatment were not mentioned. Eleven of 83 patients with a ruptured basilar tip aneurysm treated with GDC were treated for vasospasm using medical management and angioplasty. No episodes of rebleeding were mentioned.2 Graves and colleagues15 reported 4 patients treated with medical therapy and 4 with balloon angioplasty and papaverine for vasospasm. Target blood pressure goals were described, but individual or mean patient data were not included. Murayama et al12 treated 12 patients with HA and 4 patients with HA and balloon or chemical angioplasty without complications. Yalamanchili et al13 reported 4 patients who improved with induced hypertension to maintain an MAP of >120 mm Hg.

Our study is the first report in the literature to specifically address the risk of HA in the treatment of DID in patients with acute aneurysmal SAH treated with GDC. In our patients, MAP was increased by an average of 15% and was at least >10% of the baseline over an average duration of 63 hours (range 6 to 225 hours). The average maximum SBP was 210 mm Hg (range 154 to 261 mm Hg). Neurological improvement was noted in 67% of the treated patients. There was no rebleeding in any patient, although only 3 patients (25%) had complete obliteration and only 5 patients (42%) had ≥90% obliteration of the aneurysm.

### Table 2. Radiographic Findings and Endovascular Treatment of Delayed Ischemic Deficit

<table>
<thead>
<tr>
<th>Case No.</th>
<th>DID, No. Days After SAH</th>
<th>Angiogram Results</th>
<th>Endovascular Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11</td>
<td>Mild to severe spasm</td>
<td>Angioplasty and papaverine</td>
</tr>
<tr>
<td>2</td>
<td>6</td>
<td>Mild to moderate spasm</td>
<td>Papaverine</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>Mild to severe spasm</td>
<td>None</td>
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<tr>
<td>4</td>
<td>4</td>
<td>Mild to severe spasm</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>12</td>
<td>Mild to moderate spasm</td>
<td>Papaverine</td>
</tr>
<tr>
<td>6</td>
<td>5</td>
<td>Mild to moderate spasm</td>
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</tr>
<tr>
<td>7</td>
<td>4</td>
<td>Mild to moderate spasm</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>3</td>
<td>Mild to moderate spasm</td>
<td>None</td>
</tr>
<tr>
<td>9</td>
<td>20, 6</td>
<td>Mild to moderate spasm</td>
<td>Angioplasty and papaverine</td>
</tr>
<tr>
<td>10</td>
<td>4</td>
<td>No spasm</td>
<td>None</td>
</tr>
<tr>
<td>11</td>
<td>5</td>
<td>Severe spasm</td>
<td>None</td>
</tr>
<tr>
<td>12</td>
<td>6, 3, 1</td>
<td>Mild to severe spasm</td>
<td>Papaverine</td>
</tr>
</tbody>
</table>

### Table 3. Results of Hemodynamic Augmentation

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Duration of Therapy, h</th>
<th>Drug Used for HA</th>
<th>Average During Therapy, mm Hg (% increase above baseline)</th>
<th>Maximum During Therapy, mm Hg (% increase above baseline)</th>
<th>Average of Hg During Therapy, mm Hg (% increase above baseline)</th>
<th>Maximum of Hg During Therapy, mm Hg (% increase above baseline)</th>
<th>Hours MAP was &gt;10% mean (% of duration of therapy)</th>
<th>Change in GCS With Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>61</td>
<td>PE</td>
<td>187 (17)</td>
<td>223 (18)</td>
<td>128 (17)</td>
<td>146 (9)</td>
<td>53 (87)</td>
<td>+2</td>
</tr>
<tr>
<td>2</td>
<td>167</td>
<td>PE</td>
<td>151 (12)</td>
<td>203 (36)</td>
<td>106 (26)</td>
<td>135 (26)</td>
<td>145 (87)</td>
<td>−2</td>
</tr>
<tr>
<td>3</td>
<td>77</td>
<td>PE, DB</td>
<td>137 (20)</td>
<td>154 (24)</td>
<td>91 (24)</td>
<td>104 (20)</td>
<td>76 (99)</td>
<td>+2</td>
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<tr>
<td>4</td>
<td>142</td>
<td>PE, DB, DP</td>
<td>194 (21)</td>
<td>256 (34)</td>
<td>137 (30)</td>
<td>168 (32)</td>
<td>126 (89)</td>
<td>+2</td>
</tr>
<tr>
<td>5</td>
<td>23</td>
<td>PE</td>
<td>169 (14)</td>
<td>205 (18)</td>
<td>118 (20)</td>
<td>142 (20)</td>
<td>19 (82)</td>
<td>+5</td>
</tr>
<tr>
<td>6</td>
<td>24</td>
<td>DB</td>
<td>188 (5)</td>
<td>225 (0)</td>
<td>124 (0)</td>
<td>154 (1)</td>
<td>6 (25)</td>
<td>0</td>
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<tr>
<td>7</td>
<td>80</td>
<td>PE, DP</td>
<td>183 (10)</td>
<td>230 (10)</td>
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<td>148 (9)</td>
<td>47 (59)</td>
<td>+3</td>
</tr>
<tr>
<td>8</td>
<td>268</td>
<td>PE, DP</td>
<td>171 (29)</td>
<td>224 (35)</td>
<td>116 (26)</td>
<td>143 (36)</td>
<td>225 (84)</td>
<td>+1</td>
</tr>
<tr>
<td>9</td>
<td>21</td>
<td>PE</td>
<td>140 (10)</td>
<td>156 (1)</td>
<td>95 (14)</td>
<td>111 (10)</td>
<td>13 (62)</td>
<td>0</td>
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<tr>
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<td>33</td>
<td>PE, DP</td>
<td>211 (0)</td>
<td>261 (11)</td>
<td>137 (2)</td>
<td>170 (5)</td>
<td>8 (24)</td>
<td>+1</td>
</tr>
<tr>
<td>11</td>
<td>69</td>
<td>PE</td>
<td>144 (9)</td>
<td>175 (22)</td>
<td>99 (10)</td>
<td>119 (24)</td>
<td>37 (54)</td>
<td>+2</td>
</tr>
<tr>
<td>12</td>
<td>20</td>
<td>PE</td>
<td>162 (6)</td>
<td>208 (−14)</td>
<td>118 (2)</td>
<td>148 (−10)</td>
<td>6 (30)</td>
<td>0</td>
</tr>
</tbody>
</table>

DB indicates dobutamine; DP, dopamine; and PE, phenylephrine.
GDC is probably sufficient to prevent immediate rebleeding and allow HA.

This study has limitations. The number of patients is small, primarily because treatment of aneurysms with GDC is a relatively recent advance. The study needs to be replicated with a larger patient population before the results can be generalized. The MAP was not increased in all patients by an average of 20% to 25%, as recommended by some authors. Despite this, the end result of treatment, an improvement in the neurological deficit, was achieved in 67% of patients. Four patients did not improve despite treatment. In 3 of these patients, the presence of associated untreated aneurysms limited escalation of therapy.

In summary, we conclude that in patients with SAH treated with GDC, DID could be safely treated with HA with or without adjuvant endovascular therapy. In our patients, an increase in MAP of up to 25% of baseline was achieved without significant side effects and specifically without rebleeding, even when the aneurysm had not been completely occluded.

Acknowledgment

The authors thank Tracy Dobbie for assistance in the preparation of this manuscript.

References

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doi: 10.1161/hs0901.094621

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