TREATMENT OPTIONS FOR THORACOLUMBAR BURST FRACTURES
The mission of the Barrow Neurological Institute is to advance the knowledge and practice of medicine in the neurosciences through basic and clinical research, the education of medical professionals, and innovation in clinical techniques and technology.
Thoracolumbar burst fractures present a treatment dilemma. Whether they should be treated conservatively or by surgical stabilization has not been clearly defined. Some patients with this condition who are treated nonoperatively have excellent outcomes. Because of the potential for devastating neurological deficits, however, some clinicians prefer surgical stabilization. In this issue, Horn et al. retrospectively reviewed Barrow’s experience with conservative management of thoracolumbar burst fractures in patients with no neurological deficits. The authors found that nonoperative management of burst fractures appears to be a safe initial treatment option. They also examined the relative efficacy of posterior and anterior approaches for the treatment of burst fractures when surgical treatment is necessary.

Lekovic et al. report three cases of delayed vasospasm after clipping of incidentally discovered aneurysms and discuss possible mechanisms for vasospasm in the absence of subarachnoid hemorrhage. Prompt attention to changes in patients’ neurological condition after aneurysm clipping can help maximize the chances for a good outcome.

Three interesting case reports round out this issue. Thiex et al. report a woman with a 3-year history of pulsating bruit and dizziness who was found to have a dissecting aneurysm of the superior cerebellar artery. The authors discuss treatment options for dissecting aneurysms in this unusual location. Garrett et al. report a child whose orbit was penetrated by a small tree branch while sledding and examine the difficulties of distinguishing wooden objects on computed tomographic and magnetic resonance imaging. Finally, Zargarpour et al. discuss the unusual occurrence of a thoracic spinal cord injury without radiographic abnormality.

Such cases highlight the broad range of problems that can result from injury or other lesions of the brain and spinal cord and underscore the many challenges confronted by clinicians caring for these patients. We hope that you find these articles educational. Please consider sending a tax-deductible donation in the enclosed self-addressed stamped envelope to help us continue to share our experiences with the medical community throughout the world.

Robert F. Spetzler, MD
Editor-in-Chief
Analysis of Conservative Management and Comparison of Neurological Outcomes Between Anterior and Posterior Approaches for Thoracolumbar Burst Fractures

Eric M. Horn, Iman Feiz-Erfan, Nicholas C. Bambakidis, Stephen M. Papadopoulos, Volker K. H. Sonntag, and Nicholas Theodore

Bracing is a reasonable first-line treatment for thoracolumbar burst fractures. If conservative treatment fails, both anterior and posterior approaches are acceptable surgical options associated with no differences in neurological outcomes.

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Analysis of Conservative Management and Comparison of Neurological Outcomes Between Anterior and Posterior Approaches for Thoracolumbar Burst Fractures

We retrospectively reviewed our experience with 86 patients with thoracolumbar (T11-L2) burst fractures (1) to determine the efficacy of nonoperative treatment, (2) to determine predictors of successful nonoperative treatment, and (3) to compare outcomes of patients treated with an anterior or posterior approach. Patients treated initially with bracing were analyzed for failures and those failing bracing were compared with those treated successfully. All patients treated surgically were analyzed and those treated with an anterior approach were compared to those treated with a posterior approach. Twenty-eight patients were initially treated nonoperatively with a brace and 58 underwent surgery. Of the 23 patients initially treated nonoperatively that were available for follow-up, 8 failed bracing and underwent surgical fixation. Including the nonoperative failures, 66 patients underwent surgical fixation: 48 from an anterior approach and 18 from a posterior approach. Although the difference was not significant, patients who underwent a posterior approach who were available for follow-up had more complications (7/17) than those undergoing an anterior approach (8/43). There were no differences in neurologic outcomes between the two groups. Even with a higher than expected failure rate, nonoperative treatment of thoracolumbar burst fractures remains a safe, initial management option. If surgery is necessary for treatment of thoracolumbar burst fractures, either an anterior or posterior approach may be acceptable.

Key Words: burst fractures, paraplegia, spinal cord injury, spine, surgical approach, surgical fixation, trauma

Abbreviations Used: AO ASIF, Association for the Study of Internal Fixation; ARDS, acute respiratory distress syndrome; ASIA, American Spinal Injury Association; CSF, cerebrospinal fluid; CT, computed tomography; CVA, cerebrovascular accident; DVT, deep venous thrombosis; MR, magnetic resonance

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comparisons have found few differences in the outcomes of patients treated via an anterior or posterior approach.\textsuperscript{6,11,20,49} Furthermore, the only randomized, prospective trials comparing the two approaches included only patients without neurologic deficits.\textsuperscript{11,49} Whether there is a difference in neurologic outcomes between the two approaches is still unclear. We therefore reviewed our experience with thoracolumbar burst fractures (1) to determine the efficacy of nonoperative treatment, (2) to determine predictors of successful nonoperative treatment, and (3) to compare outcomes of patients treated with an anterior or posterior approach.

**Methods**

The cases of patients presenting at our institution with a traumatic thoracolumbar burst fracture were reviewed retrospectively. All patients with an acute fracture (less than 14 days old) at a single level from T11 to L2 were included in the study. Furthermore, only burst fractures that lacked significant distraction of the posterior element or complete subluxation were included in the study. The fractures primarily corresponded to the Association for the Study of Internal Fixation (AO ASIF) fracture types A3, B2, and B3.\textsuperscript{21} The inpatient and outpatient charts of each patient were reviewed, and their presenting computed tomographic scans were used to measure fracture parameters.

Between December 1999 and September 2005, 86 patients satisfied the inclusion criteria. Their mean age was 39 years (range, 9-77 years). The most common fracture level was L1 (62 patients) followed by T12 (21 patients), L2 (2 patients), and T11 (1 patient).

There were two stratifications for comparison. Twenty-eight patients (mean age, 37 years) initially treated with bracing alone were compared to all 58 patients (mean age, 37 years) treated with surgical fixation. All 28 nonoperative patients underwent placement of a custom-fit molded plastic thoracolumbosacral orthotic from the same manufacturer (Hanger Orthotic, Bethesda, MD). The 58 operative patients underwent either an anterior or corpectomy and internal fixation via a thoracoabdominal approach or posterior reduction and fixation using predominantly pedicle screws and rods.

Forty-eight patients (mean age, 37 years) treated via an anterior corpectomy and internal fixation (anterior approach) were compared to 18 patients (mean age, 37 years) treated via posterior reduction and fixation (posterior approach). This comparison included patients who failed nonoperative treatment and subsequently underwent surgical fixation. A corpectomy of the fractured vertebra was performed in patients undergoing anterior fixation. Then either an allograft, rigid titanium cage packed with autograft, or expandable titanium cage packed with autograft was used for fusion material. In all anterior patients, fixation was limited to the immediately adjacent vertebrae, and either a screw-plate or screw-rod system was used. Seventeen patients treated via a posterior approach had pedicle screws and rods placed between one and two adjacent levels above and below the fracture site. In some cases posterolateral fusion was performed using autograft with bone fusion extenders. In one patient, a hook and rod system was used for posterior fixation.

For each stratification, Student’s t-tests were used to compare means, and \( \chi^2 \) analysis was used to test proportions. Significance was set at \( p < 0.05 \). This study was approved by our Institutional Review Board.

### Results

Patients initially treated with bracing alone had less severe injuries, both radiographically and neurologically, than operative patients (Table 1). The mean hospital stay for the 28 nonoperative patients was 6.4 days (range, 1-21 days): 24 patients were discharged to home and 2 each were discharged to a rehabilitation or skilled nursing facility. Of these 28 patients, 23 (82%) were available for long-term follow-up (mean, 10.1 months). The other five patients lived outside Arizona or were from Mexico and could not be reached for follow-up. One month after bracing, one patient in the nonoperative group died from associated traumatic injuries.

Of the 23 patients with adequate follow-up, 8 (35%; mean age, 41 years) failed nonoperative treatment in a mean of 20 days (range, 3 to 79 days). The primary reasons for their failure were signs of instability or increased kyphosis on lateral dynamic radiographs (upright and supine). Despite these structural failures, no patient experienced neurologic injury from the failed bracing. When the severity of fractures was compared between the 15 nonoperative patients in whom bracing was successful (mean age, 46 years; Fig. 1) and those who failed (Fig. 2), the only difference was a greater extent of spinal canal compromise in the failed group (Table 2).

Including the patients who failed bracing and subsequently underwent surgical fixation, 66 patients underwent anterior or posterior fixation. Of these,

<table>
<thead>
<tr>
<th>Variable</th>
<th>Nonoperative</th>
<th>Operative</th>
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<tbody>
<tr>
<td>Vertebral height loss</td>
<td>32.6</td>
<td>40.7*</td>
</tr>
<tr>
<td>(mean %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spinal canal compromise</td>
<td>30.1</td>
<td>48.0*</td>
</tr>
<tr>
<td>(mean %)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kyphosis</td>
<td>7</td>
<td>12.5*</td>
</tr>
<tr>
<td>(mean %)</td>
<td></td>
<td></td>
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<tr>
<td>Neurologically intact</td>
<td>96</td>
<td>64*</td>
</tr>
<tr>
<td>(% ASIA E)</td>
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\( *p < 0.05. \)
48 anterior approaches were performed: 44 initially and 4 after failing bracing. In contrast, 14 patients initially and 4 patients who failed bracing underwent a posterior approach. Patients undergoing an anterior approach had a greater degree of spinal canal compromise and kyphosis than the group undergoing the posterior approach (Table 3). These data indicate that our institutional bias was to treat structurally severe injuries with anterior corpectomy and fixation. It appears that the decision was not based on the presence of neurologic compromise, which was similar in the two groups.

There were no differences in mean blood loss between anterior and posterior approaches (989 ml vs. 1338 ml, respectively) or in the length of postoperative hospital stay (9.1 vs. 7.9 days). In the anterior group, 43 of the 48 patients (90%) had adequate follow-up (mean, 10.3 months) and 17 of the 18 (94%) patients in the posterior group were available for a mean follow-up of 9.6 months. Patients undergoing an anterior approach had more structurally severe fractures and a lower rate of complications (8/43, 19%) than patients undergoing a posterior approach (7/17, 41%), but the difference in the rate of complications failed to reach significance (p > 0.05). No differences were noted in the complication rates associated with the different types of hardware used for fixation in patients undergoing either an anterior or posterior approach. Details concerning the types of complications in each group are presented in Table 4. Finally, there were no differences in the neurologic recovery (Fig. 3) of patients undergoing an anterior (7/43, 16%; Fig. 4) or posterior approach (3/17, 18%; Figs. 5 and 6).

Discussion

Compared to earlier reports, 3-5,15,32,36,41,47,48 our data show that a high rate of failure was associated with nonoperative treatment. Although most authors have reported successful treatment using a nonoperative approach, there are notable exceptions. In one cohort of 22 patients, six patients failed nonoper-
ative treatment and underwent surgical stabilization. In another early study, 17% of patients treated nonoperatively developed neurologic symptoms that necessitated surgical fixation.

**Failure of Conservative Treatment**

Why so many patients in our series failed nonoperative treatment is unclear. Overall, loss of vertebral height, degree of spinal canal compromise, and degree of kyphosis in the patients treated nonoperatively were all less than in the patients treated operatively (Table 1). This fact led to the choice of nonoperative treatment in the first place. When we compared patients who failed nonoperative treatment to those in whom it was successful, the only difference identified was a greater degree of spinal canal compromise at admission in the former (Table 2). Logically, the most important biomechanical indicators of instability are degree of kyphosis and loss of vertebral height. Therefore, the difference in spinal canal compromise was probably not a significant factor underlying the failure of bracing.

One explanation for the high rate of failure associated with nonoperative treatment in our patients could be the use of a custom-fit plastic brace rather than a cast, but it seems unlikely. Several studies have used similar types of bracing successfully. In fact, in one study patients treated nonoperatively without the use of a brace were ambulated immediately and showed excellent results. Another possibility is a low threshold for nonoperative failure and subsequent surgical treatment. Because our study was retrospective and there were no preexisting criteria for failure of nonoperative treatment, this possibility cannot be discounted. However, all patients who failed nonoperative treatment had radiographic demonstration of increased kyphosis or overt instability when placed upright in the brace. Furthermore, all patients who failed had severe, persistent pain that greatly restricted ambulation in the brace. Despite differences in the criteria used for determining failure of nonoperative bracing between our study and previ-

<table>
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<tr>
<th>Variable</th>
<th>Successful†</th>
<th>Failed‡</th>
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<tr>
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<td>30.1</td>
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<tr>
<td>(mean %)</td>
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<tr>
<td>Spinal canal compromise</td>
<td>23.8</td>
<td>38.1*</td>
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<tr>
<td>(mean %)</td>
<td></td>
<td></td>
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<tr>
<td>Kyphosis</td>
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<td>(mean degrees)</td>
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†15 patients, ‡8 patients, *p < 0.05.

<table>
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<tr>
<th>Variable</th>
<th>Anterior Approach</th>
<th>Posterior Approach</th>
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<tbody>
<tr>
<td>Vertebral height loss</td>
<td>41.6 (mean %)</td>
<td>33.5*</td>
</tr>
<tr>
<td>Spinal canal compromise</td>
<td>49.2 (mean %)</td>
<td>40.5*</td>
</tr>
<tr>
<td>Kyphosis</td>
<td>12.8 (mean degrees)</td>
<td>9.3*</td>
</tr>
<tr>
<td>Neurologically intact</td>
<td>65 (% ASIA E)</td>
<td>72</td>
</tr>
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</table>

*p < 0.05.

<table>
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<tr>
<th>Variable</th>
<th>Anterior Approach†</th>
<th>Posterior Approach‡</th>
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<tbody>
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<td>Surgical Complications</td>
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<td>Instability</td>
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<tr>
<td>Infected hardware</td>
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<td>Hardware failure</td>
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<td>Incomplete decompression</td>
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<td>Incomplete decompression</td>
</tr>
<tr>
<td>CSF leak</td>
<td>1</td>
<td>Retained drainage catheter</td>
</tr>
<tr>
<td>Medical Complications</td>
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<td>CVA</td>
<td>1</td>
<td>DVT</td>
</tr>
<tr>
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<td>1</td>
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</tr>
<tr>
<td>Pneumonia</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>DVT</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Total complications</td>
<td>19% (8/43); 41% (7/17).</td>
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ous reports, we believe that our criteria were appropriate for the management of these patients.

Despite the high rate of failure associated with nonoperative treatment in our patients, none developed a neurologic deficit, including the patients who failed bracing and ultimately underwent surgical fixation. The relative safety of nonoperative treatment is well established.\(^3\)\(^{-}\)\(^5\),\(^3\)\(^6\),\(^3\)\(^7\),\(^4\)\(^1\),\(^4\)\(^7\),\(^4\)\(^8\). In fact, only two studies have reported neurologic deterioration during nonoperative treatment.\(^9\),\(^3\)\(^2\) When the present report is included, the neurologic complication rate is less than 2% of the 360 reported patients treated nonoperatively. Thus, even if the failure rate associated with nonoperative treatment is as high as reported in the present cohort, such conservative management still represents a safe and effective initial treatment for thoracolumbar burst fractures.

**Posterior Approach**

Posterior reduction and pedicle screw-rod fixation are widely used when surgical fixation is needed for unstable thoracolumbar burst fractures. Before the early reports of transpedicular fixation for thoracolumbar burst fractures, various fixation devices, including the Luque loop and Harrington rods,\(^9\),\(^1\)\(^0\),\(^2\)\(^3\),\(^4\)\(^4\),\(^4\)\(^6\),\(^4\)\(^7\) were used. Although posterior fixation using these older techniques was efficacious, the use of pedicle screw-rod techniques has allowed shorter fixation segments with equivalent fusion rates.\(^1\),\(^2\),\(^1\)\(^1\),\(^1\)\(^7\),\(^1\)\(^9\),\(^3\)\(^3\),\(^3\)\(^6\),\(^3\)\(^9\),\(^3\)\(^\text{A}\)\(^\text{A}\)

In our series, the most common technique of posterior fixation (11 of 18 patients) was a pedicle screw-rod construct that spanned two levels above and below the fracture site. In six patients, however, the construct spanned only the immediately adjacent levels, and a hook-rod system was used in one patient.

We found no correlation between type of hardware used and complications, although hardware failures were found in one patient for each type of hardware used. Because the number of patients receiving each type of hardware was small, statistical comparison of complication rates across types of hardware is impossible. High failure rates have been associated with the use of small segment pedicle screw-rod systems (one level above and below).\(^3\)\(^6\),\(^3\)\(^2\) Thus, in most cases where posterior fixation is used to treat thoracolumbar burst fractures, the longer (two levels above and below) pedicle screw-rod construct is recommended.

One criticism of the posterior approach for thoracolumbar burst fractures is the relative inability to completely remove bone fragments compressing the thecal sac in the anterior portion of the spinal canal. Although there is little disagreement that adequate decompression can be performed from an anterior approach, it is becoming widely accepted that the posterior approach is equally effective. In fact, excellent decompression of the spinal canal has been associated with newer techniques used to access the vertebral body from a posterior approach.\(^1\)\(^7\),\(^3\)\(^8\) These transpedicular or lateral extracavitary approaches are especially needed in cases of neurological injury because distraction alone is inadequate for the reduction of most bony fragments.\(^3\)\(^1\)
Our data support the ability to achieve adequate decompression of the spinal canal from a posterior approach. After undergoing a posterior approach for residual compression, none of our patients needed anterior decompression. The one patient who underwent a second operation for inadequate decompression after a posterior approach needed only an extension of a partial laminectomy. Furthermore, most patients with an initial neurologic deficit improved significantly after posterior decompression and fixation. This finding correlates with reports that have found significant improvement of neurologic deficits in patients with thoracolumbar burst fractures after posterior decompression and fixation.2,6,20,45,47

Anterior Approach

Over the past 30 years, anterior approaches for surgical fixation of thoracolumbar burst fractures have become more common.14,16,22,24,29,34,35 Because the impingement of bony fragments on the thecal sac can be visualized directly, an anterior approach yields more complete decompression than a posterior approach.11 In several reports, patients with thoracolumbar burst fractures and neurologic deficits treated via an anterior approach recovered significant neurologic function.14,16,22,24,34,35 The question remains, however, whether anterior decompression offers a significant advantage in terms of neurologic outcome compared to a posterior approach. No prospective, randomized trials have specifically analyzed this issue. However, several retrospective studies have found no differences in neurologic outcome associated with these two approaches.6,20,35

It is well established that the degree of initial spinal canal compromise corre-

Figure 4. (A) Sagittal T2-weighted MR image of a 46-year-old patient immediately after presentation shows an L1 burst fracture. (B) Reconstructed sagittal CT scan shows a solid fusion 12 months after anterior fixation and fusion.

Figure 5. (A) Initial reconstructed sagittal CT scan of a 19-year-old patient shows an L1 burst fracture. (B) The fracture was reduced and fixated from a posterior approach, and the patient’s outcome was excellent. (C) After 9 months, however, the hardware failed due to incomplete fusion. (D) An anterior approach was performed for definitive treatment.
lates with the presence of a neurologic deficit.\textsuperscript{18,27} There is no correlation, however, between the extent of spinal canal compromise and the severity of neurologic deficits.\textsuperscript{27,30} More importantly, no correlation has been found between the initial extent of spinal canal compromise and eventual neurologic outcome.\textsuperscript{18,30} Furthermore, the extent of decompression has no effect on neurologic outcome.\textsuperscript{18,30} There is no correlation, however, between the extent of spinal canal compromise and the severity of neurologic deficit.\textsuperscript{18,27} There is no correlation,\textsuperscript{18,19} between operative and nonoperative treatment.\textsuperscript{Clin Orthop Relat Res (189): 142-149, 1984}

Conclusions

Nonoperative management of thoracolumbar burst fractures is a reasonable first-line treatment: There were no neurologic injuries in patients initially treated with bracing. Nonetheless, a significant number of these patients may eventually require operative treatment. Although a higher complication rate was associated with the posterior approach compared to the anterior approach, there were no differences in neurologic outcomes between the two approaches. We advocate nonoperative management as the initial treatment for thoracolumbar burst fractures in patients without neurologic deficits. When patients with a neurologic deficit need decompression, either anterior or posterior decompression and fixation are acceptable options.

References

2. Been HD, Bouma GJ: Comparison of two types of surgery for thoraco-lumbar burst fractures: combined anterior and posterior stabilisation vs. posterior instrumentation only. \textit{Acta Neurochir (Wien)} 141:349-357, 1999

**Figure 6.** (A) Initial reconstructed sagittal CT scan of a 46-year-old patient with a T12 burst fracture. Two years after posterior fixation, (B) a lateral radiograph showed a solid construct.


Delayed Ischemic Neurological Deficit After Elective Clipping of Incidentally Discovered Aneurysms: Three Case Reports

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P. Roc Chen, MD§
Volker K. H. Sonntag, MD
Joseph M. Zabramski, MD
Robert F. Spetzler, MD

Delayed vasospasm after subarachnoid hemorrhage (SAH) is a well-recognized phenomenon commonly attributed to the chemical irritation of intracranial vessels by blood or its breakdown products. However, delayed postoperative vasospasm in the absence of SAH is an unusual event that does not easily lend itself to popular theories about the origin of vasospasm. We present three cases of severe vasospasm that produced delayed ischemic neurological deficits after the patients underwent uncomplicated surgical clipping of incidentally discovered, unruptured aneurysms. In all three cases, the vasospasm failed to respond to hyperdynamic therapy. After treatment, however, the last two patients significantly improved both clinically and angiographically. One patient was treated by placement of cisternal catheters and continuous papaverine infusion; the other patient responded to endovascular treatment with intra-arterial nicardipine. These observations suggest that delayed vasospasm, although rare, is a significant potential complication after uncomplicated elective craniotomy for aneurysmal clipping, even in the absence of subarachnoid blood. Moreover, this phenomenon may have implications relevant to our understanding of the pathophysiology of vasospasm itself.

Key Words: aneurysm, cerebral vasospasm, ischemia, neurological deficit

Abbreviations Used: ACoA, anterior communicating artery; CSF, cerebrospinal fluid; CT, computed tomography; ICA, internal carotid artery; MCA, middle cerebral artery; MR, magnetic resonance; PCoA, posterior communicating artery; SAH, subarachnoid hemorrhage

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Cerebral vasospasm (also known as intracranial arterial spasm) is characterized by intense localized contraction of arterial smooth muscle and can be defined by both angiographic and clinical components. Angiographic vasospasm is the radiographic evidence of arterial narrowing, classically using contrast angiography, but alternatively documented by CT or MR angiography. Clinical vasospasm is the onset of neurological deficits corresponding to the regions of arterial narrowing thought to be related to cerebral ischemia or infarction.

The timing of vasospasm in relation to the causative stimulus defines the type of mechanism likely to be involved. Chemical vasospasm associated with SAH or neurotrauma occurs in a delayed fashion, persists for days to weeks, and is usually synonymous with the development of delayed ischemic neurological deficits. In contrast, mechanical vasospasm, which follows manipulation of intracranial arteries, occurs immediately and only lasts for hours. Despite the increasing amount of research and experience with cerebral vasospasm, our understanding of its pathophysiology is still incomplete, and prophylaxis or therapies are less than ideal.

Of special interest is the phenomenon of symptomatic vasospasm after an uncomplicated craniotomy for incidental aneurysms, because this entity does not fit neatly into existing theories of vasospasm. Vasospasm associated with unruptured aneurysms has been reported. Nonetheless, several factors require that these reports be interpreted with caution when compared to the type of cases represented by our pa-
tients. First, many of these reports predate the era of MR imaging and CT. Second, most reported cases involve patients with symptomatic aneurysms (i.e., manifesting with cranial neuropathy, presumably after increasing in size, or with headache). These aneurysms did not manifest with overt SAH. Nonetheless, the aneurysms were dynamic lesions that could have leaked subarachnoid blood—just not in quantities sufficient to be detected by CT or lumbar puncture. This issue has gained import as the widespread availability of noninvasive screening studies such as CT angiography and MR angiography has increased the detection rate of incidental aneurysms. Whereas vasospasm after aneurysmal SAH is routinely attributed to spasmogens contained in subarachnoid blood or to its breakdown products, the occurrence of vasospasm in patients with unruptured, incidental aneurysms requires alternative explanations.

**Illustrative Cases**

We describe two cases of delayed vasospasm after uncomplicated elective clipping of incidental aneurysms and include a patient reported previously (Table 1).6

**Case 1**

In March 2003 a 58-year-old woman with a long-standing history of multiple sclerosis presented with four incidental saccular aneurysms involving the ACoA, left PCoA, left ICA terminus, and left MCA (Fig. 1A and B). She had no family history of intracranial aneurysms. Her medical history was significant for multiple sclerosis, and her only medication was conjugated estrogen hormone replacement therapy. Neurologically, she was intact except for long-standing weakness of the proximal lower extremities, which was attributed to multiple sclerosis.

The patient underwent an elective left modified orbitozygomatic craniotomy for microsurgical clipping of the four aneurysms and wrapping of an aneurysmal dilatation of the left MCA (JMZ). Four Yasargil titanium aneurysm clips were used. At surgery no evidence of previous SAH was found. Postoperative angiography confirmed patency of the parent vessels and well-placed clips with no apparent residual aneurysm or spasm (Fig. 1C and D). The procedure was otherwise uneventful, and the patient was admitted to the intensive care unit.

The patient's initial postoperative course was unremarkable. As she neared discharge on postoperative Day 4, she suddenly developed difficulties with finding words. CT of the head showed unremarkable postoperative changes that included an extra-axial fluid collection in the left frontal lobe. The CT was negative for SAH. Angiography, however, showed severe vasospasm of the left M1 (Fig. 1E and F), which improved after treatment with hyperdynamic therapy (i.e., hypertensive hypervolemic hemodilution) (Fig. 1G and H). On postoperative Day 4, the patient's craniotomy was re-opened to remove the muslin wrap and to place cisternal catheters through which papaverine was irrigated continuously for 4 days (Fig. 1I). She made a complete clinical and angiographic recovery and was discharged home on postoperative Day 16. At the patient's 2- and 7-month follow-up visits, her only complaint was positional headache. Neurologically, she was at her baseline.

**Case 2**

In April 2004 a 53-year-old woman sought treatment after experiencing floaters and flashes of light through her left eye and occasional left retro-orbital pain for 10 months. Her medical history was negative for aneurysm or for a family history of SAH. She was taking conjugated estrogens for postmenopausal hormone replacement therapy. At presentation she was neurologically intact.

Four months after the patient's initial presentation, she underwent elective cerebral angiography, which showed a 12-mm x 8-mm aneurysm involving the paraclinoid segment of the ICA just distal to the ophthalmic artery (Fig. 2A, B,
Figure 1. Case 1. Preoperative (A) oblique and (B) lateral digital subtraction angiograms show four saccular aneurysms—one on the ACoA, one on the left PCoA, one on the left ICA, and one on the left MCA. Postoperative (C) anteroposterior and (D) lateral angiograms after initial clipping of the aneurysms confirm satisfactory placement of the clips and the absence of vasospasm. On postoperative Day 4, (E) anteroposterior and (F) lateral views of left ICA angiographic injections show severe vasospasm. On postoperative Day 6, (G) posteroanterior and (H) lateral angiographic views show that the vasospasm has improved. (I) CT of the head after the muslin wrapped around the mild proximal aneurysmal dilatation of the left MCA has been removed and after the cisternal catheters through which papaverine was irrigated continuously for 4 days have been placed.
Figure 2. Case 2. Initial preoperative (A) anteroposterior and (B) lateral angiographic views show the irregular wide-neck aneurysm on the left ophthalmic artery. (C) Preoperative lateral unsubtracted left ICA angiogram shows the location and size of the aneurysm on the left ophthalmic segment of the ICA. On postoperative Day 4 before intra-arterial nicardipine therapy was instituted, (D) posterior-anterior and (E) lateral angiographic views of left ICA injections show severe vasospasm. On postoperative Day 6, (F) posterior-anterior and (G) lateral angiographic views show radiographic improvement of the vasospasm. On postoperative Day 8, (H) anteroposterior and (I) lateral angiograms show continued improvement of vasospasm.
and C). The irregular aneurysm encompassed a 6-mm segment of the ICA and projected laterally. Due to the broad-based nature of the aneurysm and to its proximity to the ophthalmic artery, endovascular treatment was not attempted. The patient was recommended for a craniotomy and aneurysm clipping.

Subsequently, the patient underwent an elective left modified orbitozygomatic craniotomy. Treatment included temporary occlusion of the left ICA and common carotid artery and a left anterior clinoidectomy to expose the cavernous portion of the ICA. At surgery there was no evidence of prior SAH. Five aneurysm clips (RFS) were used to clip-reconstruct the ICA, and the craniotomy was closed in the usual manner. There were no complications.

The patient’s initial postoperative course was unremarkable. On postoperative Day 4, however, she suddenly developed intermittent difficulties with finding words. Hyperdynamic therapy was instituted. Angiography showed severe vasospasm of the distal left ICA (Fig. 2D and E), and intra-arterial nicardipine (9 mg) was administered. Her problems with speech improved rapidly.

Angiography performed on postoperative Day 6 showed significant improvement of vasospasm involving the distal left ICA, A1, and M1 (Fig. 2F and G). Intra-arterial nicardipine was again administered.

On postoperative Day 8, transcranial Doppler ultrasonography showed elevated values for the left MCA. The patient developed mild confusion and occasionally required reorientation to place and time. She also exhibited some inappropriate speech and subtle right pronator drift. Repeat angiography showed that mild residual vasospasm of the distal left ICA, A1, and M1 was continuing to improve (Fig. 2H and I). Hyperdynamic therapy was continued without further endovascular intervention. By postoperative Day 10, the patient’s neurological status had improved to her baseline. She was discharged on postoperative Day 16.

**Case 3**

Previously, we reported the case of a 54-year-old woman with an asymptomatic aneurysm involving the right MCA bifurcation. The aneurysm was detected incidentally on CT after she sustained a concussion during an assault. The 2-month gap between the assault and elective clipping of the aneurysm effectively ruled out neurotrauma as a cause of vasospasm. The unruptured aneurysm was clipped (VKHS). During surgery transient focal spasm of the MCA was treated successfully with papaverine. After a brief uneventful hospital course, the patient was discharged.

While at home on postoperative Day 9, the patient developed left hemiparesis. Angiography confirmed diffuse vasospasm of the right supraclinoid ICA, anterior cerebral artery, and MCA. Despite aggressive treatment with volume expansion and dexamethasone, the patient eventually developed an infarction in the distribution of the right MCA. When she recovered, mild weakness of her left hand persisted. Mechanical, chemical, and hypothalamic causes of the vasospasm were ruled out. The proposed pathogenesis was local release of chemical factors from the wall of the aneurysm or local autonomic dysfunction.

**Discussion**

We reviewed all cases of vasospasm that followed clipping of unruptured aneurysms treated at our institution (Table 1) and reported in the literature (Table 2) between 1975 and 2005 (i.e., since the advent of CT, Table 1). Including our patients, there are 19 such cases. These cases can be further divided into two groups: early vasospasm (i.e., onset within 24 hours of surgery) and delayed vasospasm (onset thereafter).

Immediate postoperative mechanical vasospasm cannot be considered the same phenomenon as delayed ischemic neurological deficits resulting from true delayed vasospasm. Mechanical vasospasm induced by the manipulation or electrostimulation of cerebral arteries is characterized by an early onset (within hours) and usually runs a transient self-resolving course that lasts no more than 1 or 2 days. True delayed vasospasm occurs as early as 3 days after initial SAH. Its occurrence peaks 5 to 10 days after SAH. It can persist 2 to 3 weeks and, if left untreated, can cause permanent neurological deficits or death.

In a previous analysis of the literature published before the advent of CT (between 1970 and 1980), Peerless found eight cases of vasospasm that followed uncomplicated clipping of unruptured aneurysms. Forty-five similar cases were eliminated due to identifiable complicating factors such as SAH from neurotrauma or aneurysmal leakage, prior SAH from aneurysmal rupture, mechanical trauma to vessels from clips or retractors, and thromboembolic events. However, these eight angiographically confirmed cases with symptomatic vasospasm occurring within hours of surgery are consistent with a mechanical cause. Of three cases described by Simeone and Peerless, two patients (Cases A and B) developed vasospasm immediately after surgery. These patients could also be categorized as instances of mechanical vasospasm. Simeone and Peerless reported three other patients (Cases C, D, and E) who developed delayed vasospasm after aneurysm clipping. One patient (Case C) had presented with headache, and two (Cases D and E) had presented with third cranial nerve palsies. Another patient (Case F) presented with both headache and vasospasm at surgery, but no frank evidence of previous SAH was observed.

Between 1991 and 1998, Kitizawa and coworkers clipped 30 unruptured paracclinoid ICA aneurysms, of which nine (33%) developed postoperative vasospasm. Of these nine cases, four patients had moderate postoperative angiographic vasospasm and had never been symptomatic. Only three patients with severe angiographic vasospasm (defined as luminal narrowing greater than or equal to 70%) were thought to have symptomatic vasospasm (delayed ischemic neurological deficits). These three patients were treated successfully.
with hyperdynamic therapy plus hyperbaric oxygen or intra-arterial papaverine.28

Kitazawa et al.28 retrospectively analyzed 11 factors involved in the development of vasospasm. They found that two factors, temporary occlusion of the ICA and the presence of multiple clips, were associated with delayed vasospasm. The authors suggested that intraoperative bleeding around the dural ring and the aforementioned factors, which increased mechanical stimulation of the vascular wall, may amplify the risk of delayed postoperative vasospasm associated with unruptured aneurysms involving the paraclinoid ICA. Other variables such as age, gender, history of prior SAH, aneurysm size or number, left or right side, operation time, exposure of the cervical ICA, and extent of dural ring incision failed to correlate with the development of delayed vasospasm. Although the small sample requires these statistical correlations to be interpreted with caution, such a high incidence of delayed vasospasm associated with uncomplicated clipping of unruptured aneurysms in the anatomic region of the paraclinoid ICA is still remarkable.

Paolini et al. described a patient who developed delayed vasospasm after uncomplicated aneurysmal clipping on postoperative Day 28.38 This interval is the longest reported delay between elective clipping and onset of symptomatic vasospasm. On postoperative Day 1, their patient had postretraction edema and a small hemorrhage in the frontal lobe. She remained asymptomatic until postoperative Day 28 when she developed left lower facial droop and left upper extremity weakness. Treated successfully with volume expansion and antiplatelet therapy, she returned to her baseline neurological status within 12 hours.

Interestingly, all 10 patients who developed delayed ischemic neurological deficits (Table 2) were women. One patient in our series had a history of multiple sclerosis, and immunological processes may contribute to the pathogenesis of vasospasm.37 As in two of our patients, two or more clips have been placed in many of the reported patients during surgical treatment of their unruptured aneurysm (Table 2). Kitazawa et al. reported that multiple clips may increase the risk of developing cerebral vasospasm.28

Implications for Pathophysiology of Vasospasm

There is some research on delayed vasospasm after skull base surgery and neurotrauma, but the mechanisms involved in cerebral vasospasm are best studied relative to SAH.5,33,55,61,65 Delayed vasospasm after SAH are most popularly attributed to chemoreceptor-mediated irritation of arteries by blood or its breakdown products such as oxyhemoglobin, especially since the amount and distribution of blood in aneurysmal SAH are proportional to the occurrence of vasospasm.12,14,35,36,55,56,66

The concept of endothelial damage alone serving as a stimulus for vasospasm is based on a primate model. The intracranial portion of the ICA of monkeys was cannulated with a 30-gauge needle using a technique that avoids SAH or occlusion of the lumen.46 Of nine monkeys undergoing this procedure, six developed severe local arterial constriction that persisted about 3 days. Within 1 day one monkey developed rapidly fatal vasospasm of all ipsilateral cerebral arteries.46

Catheter-induced vasospasm is a well-recognized complication of angiography, even in the absence of vessel rupture or dissection.11,20 These observations indicate that endothelial injury in the absence of significant subarachnoid blood is sufficient to produce severe vasospasm. The cause may be a spasmodogenic agent released from endothelial cells or direct endothelial dysfunction itself. It has been suggested that dynamic aneurysms or surgical injury can produce endothelial damage. The production of prostacyclin is thereby reduced, rendering damaged intracranial arteries susceptible to unopposed vasoconstriction by thromboxanes and prostaglandin endoperoxides in the circulating blood or CSF.7,17 Another possible cause, membrane-bound tissue factor in the CSF of patients with aneurysmal SAH, is predictive of vasospasm, brain injury, and overall outcome.23

Other proposed causes of chemical vasospasm include various cellular agents derived from injured vessels or cerebral tissue such as endothelin-1, free radicals, lipid peroxidation agents, and phospholipase C pathway components such as protein kinase C, diacylglycerol, and intracellular calcium.32,62 Oxyhemoglobin or endothelin-1 can directly prolong smooth muscle contraction. Free radicals can inhibit smooth muscle relaxation by inactivating nitric oxide. Lipid peroxidases can increase protein kinase C and intracellular calcium for smooth muscle contraction.32,50

In addition to local vasoactive mediators, generalized circulating pharmacologic agents can produce chemical vasospasm or sensitize vessels to spasmodagens, especially in regions of damaged endothelia. Certain chemical mediators such as methysergide, cocaine, and female hormones can exacerbate or produce forms of pharmacologically induced vasospasm in the intracranial vasculature.8,31,51 Females tend to develop vasospastic phenomena in vascular beds throughout the body more often than males,36,27,42 and it may be more than coincidence that two of our cases were postmenopausal women on hormone-replacement therapy.

Wilkins suggested the concept of hypothalamic or neurogenic vasospasm.56,60 He believed that damage to the anterior hypothalamus can cause sympathetic hyperactivity and increase circulating catecholamines, with subsequent dysfunction of autoregulation and autonomic nervous control of cerebrovascular tone. Because the hypothalamus is involved in the autonomic regulation of vascular tone, hypothalamic dysfunction may be the essential step in the development of vasospasm.56,60

This argument is based on four ideas. (1) Unlike vasospasm in animal models, which is based solely on subarachnoid blood, vasospasm in humans has other characteristics. (2) Not all patients with
### Table 2. Cases of Vasospasm Associated with Unruptured Aneurysms in the Literature

<table>
<thead>
<tr>
<th>Authors</th>
<th>Age/Sex</th>
<th>History</th>
<th>Aneurysm</th>
<th>No. Clips</th>
<th>Notable Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simeone &amp; Peerless 1975⁴⁶</td>
<td>40/M</td>
<td>NA</td>
<td>L MCA</td>
<td>NA</td>
<td>Dynamic w/evidence of growth over 2 y</td>
</tr>
<tr>
<td></td>
<td>30/F</td>
<td>NA</td>
<td>L ICA</td>
<td>NA</td>
<td>Rickham reservoir used due to intraop spasm of L ICA + MCA</td>
</tr>
<tr>
<td></td>
<td>32/F</td>
<td>NA</td>
<td>L ICA</td>
<td>NA</td>
<td>Aneu mainly intracavernous, filled w/copper wires to promote clot formation</td>
</tr>
<tr>
<td>Raynor &amp; Messer 1980⁴¹</td>
<td>25/F</td>
<td>NA</td>
<td>L ICA</td>
<td>NA</td>
<td>Preop (4 d) spasm of L ICA next to aneu neck, postop incidental R ophth aneu</td>
</tr>
<tr>
<td>Friedman et al. 1983¹⁷</td>
<td>30/F</td>
<td>NA</td>
<td>L PCoA</td>
<td>10</td>
<td>Preop (1 mo) spasm of L ICA and basilar a. Dynamic w/evidence of growth over 2 wks</td>
</tr>
<tr>
<td>Bloomfield &amp; Sonntag 1985⁶</td>
<td>54/F*</td>
<td>HTN, postconcussion syndrome, pyelonephritis</td>
<td>R MCA near bifurcation</td>
<td>7</td>
<td>Neurotrauma 2 mo before aneu surgery, transient intraop focal R MCA vasospasm treated w/papaverine</td>
</tr>
<tr>
<td>Day &amp; Raskin 1986⁹</td>
<td>42/F</td>
<td>MS (mild paraparesis, L optic atrophy)</td>
<td>R supracl, ICA/ PCoA junct</td>
<td>10</td>
<td>Bloodless CSF x 2</td>
</tr>
<tr>
<td>Kitazawa et al. 2005²⁸</td>
<td>29/M</td>
<td>NA</td>
<td>R paracl ICA 3 aneu)</td>
<td>7</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>39/F</td>
<td>NA</td>
<td>R paracl ICA</td>
<td>6</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>52/F</td>
<td>NA</td>
<td>R paracl ICA</td>
<td>5</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>57/M</td>
<td>NA</td>
<td>L paracl ICA</td>
<td>5</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>59/F</td>
<td>NA</td>
<td>L paracl ICA</td>
<td>5</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>62/F</td>
<td>NA</td>
<td>L paracl ICA</td>
<td>10</td>
<td>Temp occlusion of ICA, Mild EDH</td>
</tr>
<tr>
<td></td>
<td>21/F*</td>
<td>NA</td>
<td>L paracl ICA</td>
<td>4</td>
<td>Temp occlusion of ICA</td>
</tr>
<tr>
<td></td>
<td>53/F*</td>
<td>NA</td>
<td>L paracl ICA 3 aneu)</td>
<td>4</td>
<td>SAH</td>
</tr>
<tr>
<td></td>
<td>63/F*</td>
<td>NA</td>
<td>L paracl ICA</td>
<td>5</td>
<td>Temp occlusion of ICA, Mild EDH</td>
</tr>
<tr>
<td>Paolini et al. 2005²⁸</td>
<td>47/F*</td>
<td>Signif smoking</td>
<td>R MCA bifurcation “large”</td>
<td>2</td>
<td>Small R frontal hemorrhage w/postretraction edema on POD 1</td>
</tr>
<tr>
<td>Lekovic et al. (current study)</td>
<td>58/F*</td>
<td>Postmenopausal HRT, MS</td>
<td>ACoA, L PCoA, L ICA, L A1, L M1</td>
<td>NA</td>
<td>Left MCA muslin wrap</td>
</tr>
<tr>
<td></td>
<td>53/F*</td>
<td>Postmenopausal HRT, diverticulosis, cholecystectomy, appendectomy, hysterecotomy, L shoulder + bilateral knee surg</td>
<td>Ophth segment of L ICA</td>
<td>8</td>
<td>Preop narrow caliber of L ICA</td>
</tr>
</tbody>
</table>

⁠word finding difficulty, ⁴ophthalmoplegia due to incidental R ophthalmic aneurysm, ⁵mild residual weakness of L hand, ⁶cases of true delayed symptomatic vasospasm after incidental aneurysm clipping, aneu = aneurysm, ACA = anterior cerebral artery, ACoA = anterior communicating artery, angio = angiography, asympt = asymptomatic, CN = cranial nerve, CSF = cerebrospinal fluid, EDH = epidural hematoma, GOS = Glasgow Outcome Scale, GR = good recovery, HHH = hyperdynamic therapy (hemodilu-
<table>
<thead>
<tr>
<th>Neurological Deficits</th>
<th>Vasospasm onset &amp; location</th>
<th>Treatment</th>
<th>GOS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aphasia, R hemiparesis</td>
<td>Early (POD 0) clinical vasospasm, (POD 2) angio vasospasm of L ICA + MCA</td>
<td>NA</td>
<td>MD</td>
</tr>
<tr>
<td>Aphasia, RUE weakness</td>
<td>Early (POD 0) clinical vasospasm, (POD 1) angio vasospasm of L ICA + MCA</td>
<td>Papaverine + angio phenoxybenzamine through Rickham reservoir, mannitol + glycerol</td>
<td>MD</td>
</tr>
<tr>
<td>Aphasia, somnolence, R hemiparesis</td>
<td>Delayed (POD 5) L ICA, L ACA, L MCA</td>
<td>Intraarterial phenoxybenzamine, steroids, + mannitol</td>
<td>GR</td>
</tr>
<tr>
<td>Somnolence, R hemiparesis (Preop L CN 3 palsy from mass effect)</td>
<td>Preop early (POD 0) L ICA (increased spasm since 4 d preop)</td>
<td>NA</td>
<td>GR‡</td>
</tr>
<tr>
<td>Asympt vasospasm (Preop L CN 3 palsy from mass effect)</td>
<td>Preop (1 mo) L ICA, basilar artery</td>
<td>NA</td>
<td>GR</td>
</tr>
<tr>
<td>L hemiparesis</td>
<td>Delayed (POD 9) R ICA – supraclinoid segment, R ACA, R MCA</td>
<td>HHH + dexamethasone 4 mg/6hr/5d</td>
<td>GR§</td>
</tr>
<tr>
<td>Headache</td>
<td>Preop (1 wk) diffuse vasospasm</td>
<td>NA</td>
<td>GR</td>
</tr>
<tr>
<td>None</td>
<td>Delayed (POD 14) mod vasospasm</td>
<td>HHH</td>
<td>GR</td>
</tr>
<tr>
<td>Convulsions</td>
<td>Delayed (POD 5) mod vasospasm</td>
<td>HHH</td>
<td>GR</td>
</tr>
<tr>
<td>None</td>
<td>Delayed (POD 11) mod vasospasm</td>
<td>HHH</td>
<td>GR</td>
</tr>
<tr>
<td>None</td>
<td>Delayed (POD 11) mod vasospasm</td>
<td>HHH</td>
<td>GR</td>
</tr>
<tr>
<td>Aphasia, R hemiparesis</td>
<td>Delayed (POD 3) mod vasospasm</td>
<td>HHH</td>
<td>GR</td>
</tr>
<tr>
<td>Aphasia</td>
<td>Delayed (POD 16) mod vasospasm</td>
<td>HHH</td>
<td>GR</td>
</tr>
<tr>
<td>Aphasia, Gertzmann syndrome</td>
<td>Delayed (POD 12) severe vasospasm</td>
<td>HHH + intraarterial papaverine</td>
<td>GR</td>
</tr>
<tr>
<td>Aphasia</td>
<td>Delayed (POD 9) severe vasospasm</td>
<td>HHH + hyperbaric O₂</td>
<td>GR</td>
</tr>
<tr>
<td>Aphasia, R hemiparesis</td>
<td>Delayed (POD 5) severe vasospasm</td>
<td>HHH + hyperbaric O₂</td>
<td>GR</td>
</tr>
<tr>
<td>L lower facial droop, L hemiparesis</td>
<td>Delayed (POD 28) severe vasospasm</td>
<td>HHH + antiplatelet therapy</td>
<td>GR</td>
</tr>
<tr>
<td>Aphasia†</td>
<td>Delayed (POD 4) L M1</td>
<td>HHH + intradural papaverine</td>
<td>GR</td>
</tr>
<tr>
<td>Aphasia†</td>
<td>Delayed (POD 4) L distal ICA, L A1, L M1</td>
<td>HHH + intraarterial nimodipine</td>
<td>GR</td>
</tr>
</tbody>
</table>

Additional abbreviations: HRT = hormone replacement therapy, HTN = hypertension, ICA = internal carotid artery, MCA = middle cerebral artery, MD = moderate disability, mod = moderate, MS = multiple sclerosis, ophth = ophthalmic, paraclinoid = paracan, PCoA = posterior communicating artery, POD = postoperative day, RUE = right upper extremity, signif = significant, supraclinoid = supraclini, and temp = temporary.
subarachnoid blood develop vasospasm, but it most often occurs in those with aneurysms located adjacent to the hypothalamus or involving vessels that supply the hypothalamus. (3) Postmortem evidence has shown destructive lesions in the anterior hypothalamus of patients with aneurysmal SAH consistent with sympathetic dysfunction. (4) Finally, the study of postoperative and posttraumatic cerebral vasospasm provides similar anatomic support.

Selective stimulation of nerves or ganglia has restored autonomic balance in animal models of SAH-induced vasospasm. Furthermore, when applied either prophylactically or as treatment, reversible functional sympathectomy via cervical spinal cord stimulation with epidural electrodes has improved cerebral vasospasm in animal models. In a similar manner, spinal cord stimulation has been used successfully to treat cerebral vasospasm in select patients. That nitric oxide is involved as a mediator in this response suggests overlap with the mediators involved in chemical vasospasm. Thus, the two ideas of chemical and neurogenic vasospasm can be combined: It is possible that a spasmogenic substance is released from the hypothalamus in response to stimulation or damage. Wilson and Field showed that porcine hypothalamic extract, but not porcine cerebral cortical tissue extract, could produce vasospasm when injected into the cisterna magna of dogs; their finding indicates the presence of a spasmogenic substance in the hypothalamus.

Management of Delayed Ischemic Neurological Deficits After Elective Clipping of Aneurysms

In our cases, as in the other reported cases of delayed vasospasm, hyperdynamic therapy used in conjunction with dexamethasone, antiplatelet drugs, hyperbaric oxygen, and intradural or intrathecal local vasodilators resulted in good overall outcomes. Although the potential benefits of these treatment options seem to outweigh their risks, the small samples and lack of controls for comparison make it difficult to determine which therapies are actually effective. The only common treatment factor among all of these cases was hyperdynamic therapy. Similarly, all patients with moderate angiographic vasospasm in the series reported by Kitazawa et al. were treated effectively with hyperdynamic therapy alone; none developed symptomatic vasospasm. Although hyperdynamic therapy using a combination of crystalloids and colloids can exacerbate cerebral edema in patients with acute infarcts, it is useful in the prophylaxis and therapy of vasospasm in aneurysmal SAH. Despite the use of invasive and noninvasive monitoring to guide treatment, hyperdynamic therapy can also produce occasional cardiopulmonary complications such as congestive heart failure and pulmonary edema related to volume overload. Calcium antagonists, balloon angioplasty, or injection of intra-arterial or intradural vasodilators can be helpful if vasospasm is severe or when aggressive medical therapy has failed. Nonetheless, outcomes have been excellent in response to a combination of the aforementioned therapies. Consequently, current experimental treatments such as spinal cord stimulation for producing reversible functional sympathectomy and endothelin antagonists probably add no value at this time for the treatment of delayed vasospasm associated with unruptured incidental aneurysms.

Role of Routine Angiography

After aneurysms are clipped, the routine use of intraoperative (or immediate postoperative) angiography is beneficial. However, whether the additional cost and risk of complications associated with routine delayed angiography can be justified for use in asymptomatic patients is unclear. Kitazawa et al. routinely obtained postoperative angiograms in their series of unruptured aneurysms a mean of 12 days after surgery. Although these authors discovered a surprisingly high rate of angiographic vasospasm, severe vasospasm only occurred in the symptomatic group. Kitazawa et al. did not assess the cost-effectiveness of this strategy.

Rather, a high-index of suspicion and prompt evaluation with angiography are warranted for patients with symptoms consistent with delayed ischemic neurological deficits after elective clipping of aneurysms. Therefore, patients should be made aware of the rare possibility that delayed vasospasm may occur within the first two weeks of treatment so that they are prepared to return for the management of vasospasm if necessary. As experience with this population grows, it may become possible to use CT angiography to assess for delayed postoperative vasospasm in a simpler and less invasive manner than conventional angiography.

**Conclusion**

Based on our observations, we suggest that vasospasm may be a rare but significant complication of elective craniotomy for aneurysmal clipping. Moreover, the incidence of delayed vasospasm after elective clipping may be underestimated because postoperative angiograms are not routinely obtained. Therefore, a high index of clinical suspicion for delayed ischemic neurological deficits is warranted for patients undergoing elective clipping of aneurysms, particularly in the paraclinoid region and in women, or when multiple aneurysm clips are placed. These patients should be counseled about the possibility of delayed ischemic neurological deficits. After aneurysm clipping, patients with complaints consistent with delayed ischemic neurological deficits should undergo angiography to rule out vasospasm as the cause. After uncomplicated clipping of unruptured aneurysms, early recognition of vasospasm as a possible cause of changes in patients’ neurological condition may allow appropriate interventions to be instituted to maximize the chances of a favorable outcome.
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Lekovic et al: Delayed Ischemic Neurological Deficit After Elective Clipping of Incidentally Discovered Aneurysms: Three Case Reports


Dissecting Aneurysm of the Superior Cerebellar Artery: Case Report

We report the rare case of a dissecting aneurysm of the SCA that presented without SAH and that was treated by wrapping.

Case Report

A 37-year-old woman had a 3-year history of a pulsating whooshing sound in her left ear and dizziness. MR angiography suggested a vascular abnormality related to the left SCA. DSA showed that the abnormality involved the anterior pontomesencephalic segment of the SCA 1 cm distal to its origin from the basilar artery where its maximum diameter was 6 mm. The patient underwent a left-sided full orbitozygomatic craniotomy. A dissecting aneurysm of the left SCA was wrapped because the length of the aneurysm rendered revascularization impossible in case the SCA had to be sacrificed. The patient’s long-standing history of symptoms and the location of the lesion make this dissecting aneurysm exceptional.

Key Words: dissecting aneurysm, orbitozygomatic approach, superior cerebellar artery

Abbreviations Used: CN3, oculomotor nerve; DSA, digital subtraction angiography; MR, magnetic resonance; PCA, posterior cerebral artery; SAH, subarachnoid hemorrhage; SCA, superior cerebellar artery

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the lesion, providing a good view of the tortuous portion of the dissecting aneurysm and the proximal side of the parent artery (Fig. 2B-C). Consequently, it was impossible to revascularize this vessel in case it needed to be sacrificed. Therefore, the dissecting aneurysm was wrapped with cotton (Fig. 2D). The patient tolerated the procedure well. She had no postoperative deficits and was discharged 2 days after surgery.

**Discussion**

Aneurysms on the main trunk of the SCA are rare. Before 1990 only 13 cases, all of which were saccular aneurysms, had been reported. Most arose from the origin of the SCA. In 1990 Hirose et al. first described a fusiform aneurysm of the SCA in a 25-year-old patient with SAH. They clipped the SCA at a site distal to the origin of a perforating artery, and the patient recovered with mild cerebellar ataxia. Since then both endovascular and surgical techniques have been used to treat SCA aneurysms.

**Location of Dissecting Aneurysms**

Dissecting aneurysms of the SCA are extremely rare; only four such cases have been reported to date. The origin of dissecting aneurysms remains unclear but seems to be related to atherosclerosis and degenerative changes in the vessel wall. Defects of the internal elastic lamina may be the cause of dissection in the wall of intracranial arteries, particularly in patients with congenital or acquired medical abnormalities. As a dissection progresses, other contributing factors also may be involved, including hypertension, delivery, various arterial diseases, and anatomical characteristics of the artery. Most dissecting aneurysms occur in the intracranial vertebrobasilar trunk. Thus, the vertebral artery undergoes morphological changes after it penetrates the dura. For example, the reduction in the thickness of the adventitial and medial layers and the significant reduction or loss of elastic fibers in the medial and external laminae may contribute to the development of dissecting aneurysms.

Dissecting aneurysms involving the intracranial peripheral arteries predominantly occur in relatively young patients who usually lack underlying abnormalities to explain the appearance of a dissection. These vascular abnormalities have also been related to migraine, fibromuscular dysplasia, mixed connective-tissue disease, angitis, and trauma. The origin of the dissecting aneurysm of the SCA might have been associated with our patient's history of heavy smoking. Dissecting aneurysms in the peripheral arteries of the posterior circulation are primarily located in the proximal portion of arterial branches. Anatomically, the SCA can be divided into two segments. The cisternal segment extends from the anterior pontomesencephalic to the lateral pontomesencephalic to the cerebellomesencephalic segment. The cortical segment is equivalent to the hemispheric, vermian, and marginal branches. Of the 14 SCA aneurysms reported before 1990, seven involved the anterior pontomesencephalic segment. Thirteen percent of vertebrobasilar system aneurysms are fusiform. Of the cases reported before 1990, however, all but one aneurysm, which involved the main trunk of the SCA, were saccular.
Surgical Indications
The indications for surgical treatment of dissecting aneurysms remain controversial. Dissecting aneurysms of the anterior circulation tend to manifest with ischemic symptoms; those involving the posterior circulation are more likely to lead to SAH.8,37 Unlike all reported cases of dissecting aneurysms of the SCA,10,11,29our patient did not present with SAH. Instead she had a long-standing history of a bruit in her left ear. Only after syncope, which prompted MR imaging, was the aneurysm discovered incidentally. Whether irritation from the tortuosity of the dissecting segment of the SCA caused the bruit remains questionable. Lacking evidence of previous hemorrhage renders secondary pathological changes subsequent to hemorrhage unlikely.

Urgent surgical treatment for dissecting aneurysms of the posterior circulation is recommended because the rate of recurrent hemorrhage is high and these patients have extremely poor outcomes if their lesion ruptures.32,37 However, there are some arguments against surgical treatment. Spontaneous resolution of a dissecting aneurysm of the posterior cerebral artery17 and similar results in the vertebrobasilar system24 favor conservative management in the absence of neurological deterioration. The decision to administer surgical treatment in neurologically stable patients who present with ischemia or no recurrent hemorrhage is controversial. Such cases can heal spontaneously or after anticoagulant therapy or both.17,23

The need for surgery is unequivocal in patients with recurrent SAH or angiographic progression of their dissection.8,24 We offered our patient surgical exploration for several reasons. First, the patient’s angiographic findings were indefinite about the presence of a lesion. Second, the patient’s tortuous vascular anatomy was a concern. Finally, the patient was young.

Surgical Treatment
Surgical treatment of dissecting aneurysms includes excision, trapping, proximal ligation, and reinforcement with or without an intracranial-to-extracranial bypass.3,7 Still, the optimal surgical procedure has not yet been established. Aneurysmal excision or trapping seems to be the only effective means of managing a ruptured dissecting aneurysm. As in our case, revascularization procedures7,13 or preservation of the blood flow of the parent artery may be necessary.

In our patient, the dissection encompassed an extensive portion of the SCA beginning 1 cm distal to its origin. Consequently, revascularization after excision of the tortuous dissecting aneurysm or trapping was infeasible. We therefore reinforced the wall of the dissecting aneurysm by wrapping it. According to Rhoton,27 the SCA bifurcates into a rostral and caudal trunk 0.6 to 34 mm (mean, 19 mm) from the origin of the vessel. The bifurcation is near the point of maximal caudal descent of the artery on the lateral side of the brainstem.

The most appropriate surgical procedures should be selected based on the location of the aneurysm, its anatomical relation to the parent arteries, the extent of the dissection, and the collateral circulation. Regardless of the approach used, the exposure is typically deep and confined, and the vascular anatomy is complex and variable. The intimate re-
relationship between these aneurysms and cranial nerves and perforating vessels increases the potential for surgical complications.26

Aneurysms involving the cisternal segment may be approached through a full OZ approach, as in our case, or through the pterional transsylvian, subtemporal transtentorial,6,22 or occipital transtentorial approaches.21 Aneurysms involving a marginal branch of the cortical segment are usually clipped through the suboccipital approach. Those involving the hemispheric branch are treated through an infratentorial supracerebellar or occipital transtentorial approach. Finally, lesions involving the vermian branch are treated through a suboccipital, infratentorial supracerebellar,9 or occipital transtentorial approach.34

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Transorbital Penetrating Tree Branch During a Sledding Accident

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Complications related to intracranial wooden foreign bodies are well known. If wooden foreign bodies are suspected, efforts should be made to remove them surgically to avoid infection-related complications or even death. The authors present the case of a 10-year-old girl whose left orbit near the medial canthus was penetrated by a small tree branch during a sledding accident. Different imaging modalities showed that the branch had fragmented. One fragment rested along the lateral border of the pons, and one penetrated the sphenoid sinus and carotid canal. The patient underwent successful surgical removal of the wooden fragments and made an excellent clinical recovery.

Key Words: cerebral abscess, foreign body, intracranial, trauma, wood

The morbidity and mortality associated with intracranial wooden foreign bodies have repeatedly been demonstrated in the literature. The orbit is a pathway of low resistance for penetrating objects to enter the intracranial compartment. However, intracranial wood can be challenging to detect with current imaging modalities. We present the case of a 10-year-old girl who had a tree branch penetrate her orbit during a sledding accident. Despite the dangerous resting place of two branch fragments, her intracranial structures were uninjured. Surgical removal of the fragments was successful, and the patient had an excellent clinical outcome.

Case Report

A 10-year-old girl was sledding when a small tree branch penetrated the left orbit near the medial canthus. She was transferred to the trauma service at our institution. Her initial examination ruled out coexisting injuries. On admission her Glasgow Coma Scale score was 15. Her left pupil was reactive to direct light, but she complained of blurry vision. An ophthalmologic examination of that eye documented her vision as 20/200 and the presence of medial rectus palsy. The remainder of her neurologic examination was normal. The patient was placed on intravenous antibiotics shortly after admission (1 gm of vancomycin twice a day, 500 mg of metronidazole four times a day, and 1.5 gm cefotaxime three times a day).

CT angiography showed that the branch had fragmented. One piece had traversed the sphenoid sinus and lodged...
in the medial carotid canal (Fig. 1). MR angiography showed that the internal carotid artery was indented, but there was no obvious vascular injury (Fig. 2). Another piece of the branch had traveled intracranially and rested along the mesial temporal lobe. Its tip lay in the ambient cistern between the tentorial edge and pons (Fig. 3). Multiple reconstruction CT scans and MR images were obtained to determine the exact path of the branch fragments (Fig. 4).

The decision was made to remove the foreign bodies surgically through a combined approach. The entrance wound was widened, and the orbit was explored to identify the branch fragments. A modified orbitozygomatic craniotomy was performed to visualize the largest intracranial branch during removal and to gain vascular control in the event of injury. However, the wooden fragment, which was entirely contained within the leaflets of the tentorium, was never visualized through the craniotomy.

Immediately after the fragment was removed, a small amount of subdural bleeding occurred but stopped spontaneously. Although we believe that the tip of the fragment caused this bleeding as it penetrated the tentorial dura, direct visual verification could not be obtained. The bleeding stopped completely, and no further exploration was performed. After the branch that had penetrated the carotid canal was removed, intraoperative angiography confirmed the absence of vascular injury.

Postoperative CT raised concerns that pieces of wood were retained within the orbit. Because of the high risk associated with retained foreign bodies, the patient was returned to the operating room for re-exploration. No further fragments were found, and the CT findings were attributed to posttraumatic liposis.

After the first surgery, the patient had an afferent pupillary defect but her perception of light and motion was intact. After the second procedure, she lost all vision in that eye. Decadron was started and continued (4 mg every 8 hours) until she was discharged. After consultation with the infectious disease service, the antibiotics were discontinued at discharge with plans for close follow-up.

At her 1-month follow-up examination, the patient had regained near normal vision in the left eye. Only slight medial rectus palsy persisted. The patient had no signs or symptoms of infection and otherwise made an excellent recovery.

Discussion

Our patient’s case is of interest because of the dangerous path taken by the branch fragments without injuring in-
intracranial vascular or parenchymal structures. The fragments required surgical removal—the danger of intracranial wood is well known. Nishio et al., for example, reported a 13-year-old girl whose orbit was penetrated by a chopstick. Initial CT failed to show a wooden fragment, but 7 years later she developed a brain abscess. The authors also reviewed 23 case reports of wooden foreign objects that penetrated the dura published since 1984. Sixteen cases were complicated by central nervous system infections of which 12 were associated with brain abscesses. The fragment in the carotid canal placed our patient at risk not only for infection but also for blood flow abnormalities.

Our patient’s surgical reexploration highlights the dilemma of diagnosing foreign wooden objects with current imaging modalities. A thin-cut postoperative CT scan of the orbits showed a small, well-defined, iso- to hypodense area that appeared consistent with a retained fragment. Both neurosurgeons and neuroradiologists evaluated her postoperative CT scans, and the decision was made to reoperate. On CT wood is often hypointense and can be mistaken for air or even orbital fat. Consequently, some authors have argued that MR imaging is superior to CT in this setting.

However, the appearance of wood on MR imaging can be quite variable. On MR imaging of the extremities, the appearance of wooden foreign bodies can vary, but the signal intensity of wood on T1-weighted and T2-weighted MR images is usually equal to or less than that of skeletal muscle. Smely and Orszagh confirmed this variability, reporting cases that showed both hyper- and hypointensity on T1-weighted images. These findings prompted an in vitro study using CT and MR imaging to study the appearance of multiple types of wood in both fatty and soft-tissue backgrounds that resembled an orbit. In that investigation, CT was superior to MR imaging for detecting wooden foreign bodies in the eye. If findings on CT or MR imaging raise suspicion of a penetrating wooden foreign body, surgical intervention seems warranted to avoid the attendant risks.

Conclusion
This interesting clinical vignette serves as an important reminder of issues regarding intracranial wood. Specifically, significant morbidity and mortality rates are associated with re-
tained intracranial wooden objects. Although the radiographic diagnosis of wooden objects remains difficult, high suspicion based on either CT or MR imaging should prompt surgical removal of any foreign body.

References


Motor Vehicle Accident Causing Thoracic Spinal Cord Injury Without Radiographic Abnormality

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SCIWORA is most commonly caused by traumatic injury sustained in a motor vehicle accident or fall. The immature pediatric spine confers some protection against direct osseous spinal injury but makes children susceptible to ligamentous and spinal cord injury. Trauma-induced spinal cord injury usually involves the cervical spine; we report a rare case of SCIWORA involving the thoracic spinal cord. Despite negative radiographic or CT findings, pediatric patients with neurologic deficits should undergo MR imaging to rule out spinal cord injury.

Key Words: pediatric, SCIWORA, spinal cord injury, trauma

Spinal injuries are primarily sustained by adults. However, SCIWORA, defined as the occurrence of a spinal cord injury without radiographic abnormality, is predominantly found in pediatric patients. Children with SCIWORA show no evidence of an abnormality on plain film radiography or CT but may have positive neurological findings and corresponding findings on MRI imaging. SCIWORA represents fewer than 1% of all spinal cord injuries, but its diagnosis has increased with advances in MR imaging technology and with increased awareness about the phenomenon. Of the approximately 1300 new cases of spinal cord injury in the pediatric population each year, 19 to 34% are reported to be SCIWORA. This article describes a relatively rare case of SCIWORA involving the thoracic spinal cord.

Case Report

A 22-month-old boy, who was an unrestrained passenger, was ejected in a rollover motor vehicle accident. At admission he was intubated and sedated. A cursory examination showed no obvious injuries of the extremities. He was resting in a stable position in bed. His cranial nerve function was intact. He localized purposely with both upper extremities but demonstrated flaccid paraparesis and lack of strength in both lower extremities. Examination showed no rectal tone. Reflexes in his upper extremities were 2+. Reflexes were absent in both lower extremities, but his pulse was palpable bilaterally.

Initial plain film radiographs of the cervical, thoracic, and lumbar spine were

Abbreviations Used: CT, computed tomography, MR, magnetic resonance, SCIWORA, spinal cord injury without radiographic abnormality, STIR, short tau inversion recovery

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negative for acute findings (Fig. 1). Subsequent CT of the cervical, thoracic (Fig. 2), and lumbar spine showed no fractures or subluxation. CT of the head was also negative.

Based on the patient’s neurologic findings, MR imaging was ordered (Fig. 3). The study demonstrated abnormal cord signal intensity from T10 to T12. STIR sequences demonstrated no evidence of bone marrow edema.

Discussion

In terms of tautness, the pediatric spine reaches adult maturation at 8 to 9 years of age. SCIWORA is caused by ligamentous flexibility and elasticity of the immature vertebral column. Not surprisingly, two-thirds of the cases occur in patients 8 years or younger before the spine has matured. The malleability of the spine places the spinal cord under severe distortion, causing it to contact its bony counterpart, potentially leading to neurological problems. MR imaging may show compressive, treatable lesions that were not apparent on plain radiographs. Spinal cord injury in pediatric patients can include ligamentous instability, ischemia, infarction, or an evolving lesion.

In the uncommon case of a thoracic spinal cord injury, the mechanism of injury is often related to motor vehicle accidents, crush injuries, or falls from a height. Damage to the thoracic spine is
less common than damage to the cervical spine because the former is protected by the rib cage, abdomen, and thorax, which resist excessive flexion and extension.\textsuperscript{5,9} Lesions involving the lower thoracic segments, as in our patient, are usually caused by a crushing effect more than by flexion or extension.\textsuperscript{6,7} In contrast, injury to the cervical spine is commonly caused by extreme flexion or extension of the spinal cord.

When Pang coined the term SCIWORA in 1982, MR imaging had only recently been introduced. Thus, the definition was based purely on CT and plain x-rays. As the use of MR imaging has become more widespread, such lesions are increasingly detected. Therefore, the term SCIWORA is now a misnomer because most patients have an abnormality that is detectable on MR imaging.\textsuperscript{2} This fact underlines the importance of MR imaging when a child has clinical neurological deficits but no findings on CT or plain films.

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