Intracranial hemorrhage after spine surgery

Clinical article

PAUL E. KALOOSTIAN, M.D., JENNIFER E. KIM, B.S., ALI BYDON, M.D., DANIEL M. SCIUBBA, M.D., JEAN-PAUL WOLINSKY, M.D., ZIYA L. GOKASLAN, M.D., AND TIMOTHY F. WITHAM, M.D.

Department of Neurological Surgery, The Johns Hopkins Hospital, Baltimore, Maryland

Object. The authors describe the largest case series of 8 patients with intracranial hemorrhage (ICH) after spinal surgery and identify associated pre-, intra-, and postoperative risk factors in relation to outcome.

Methods. The authors retrospectively reviewed the cases of 8 patients treated over 16 years at a single institution and also reviewed the existing literature and collected demographic, treatment, and outcome information from 33 unique cases of remote ICH after spinal surgery.

Results. The risk factors most correlated with ICH postoperatively were the presence of a CSF leak intraoperatively and the use of drains postoperatively with moderate hourly serosanguineous output in the early postoperative period.

Conclusions. Intracranial hemorrhage is a rare complication of spinal surgery that is associated with CSF leakage and use of drains postoperatively, with moderate serosanguineous output. These associations do not justify a complete avoidance of drains in patients with CSF leakage but may guide the treating physician to keep in mind drain output and timing of drain removal, while noting any changes in neurological examination status in the meantime. Additionally, continued and worsening neurological symptoms after spinal surgery may warrant cranial imaging to rule out intracranial hemorrhage, usually within the first 24 hours after surgery. The presence of cerebellar hemorrhage and hydrocephalus indicated a trend toward worse outcome.

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KEY WORDS • intracranial hemorrhage • spine surgery • cerebrospinal fluid leak

Intracranial hemorrhage is an extremely rare complication of spinal surgery, with fewer than 35 individual cases reported in the literature and a proposed incidence rate of 0.8%.5,11 The etiology of remote ICH remains unclear, but evidence suggests that it is caused by excessive CSF loss, which results in cerebral dehydration causing stretching and eventually tearing of the bridging veins. The postoperative hemorrhage may be classified as cerebellar hemorrhage (CBH), subarachnoid hemorrhage (SAH), subdural hemorrhage (SDH), or intraventricular hemorrhage (IVH). Dural tear resulting in a CSF leak has been strongly implicated as a risk factor for postoperative ICH. Additionally, drain placement and continued suction at the site of dural repair have also been proposed as a mechanism for sustained cerebral hypotension.1,2,10,13,14,24,32 Left unrecognized or untreated, this complication can have debilitating and even fatal consequences. Thus, early detection and diagnosis is critical for implementing appropriate treatment and ensuring the best possible outcome.

The purpose of this report is to present the largest case series to date of ICHs after spinal surgery and to identify associated preop-, intra-, and postoperative risk factors in relation to outcome. With knowledge of these risk factors, the surgeon may better appreciate perioperative factors associated with ICH after spinal surgery, and the surgical team caring for the patient may be able to undertake a more focused evaluation in the postoperative period. We describe 8 cases of ICH after spine surgery that were seen at our institution between 1996 and 2012 and discuss the possible predisposing and precipitating factors.
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Methods

After institutional review board approval, we retrospectively reviewed the clinic notes, operative notes, and discharge summaries of 8 patients who presented with ICH after spine surgery from 1996 to 2012. Data regarding the following variables were collected: general demographics, medical history, initial spinal procedure, location and treatment of dural tear, drain placement (7 placed in the epidural space, 1 in the lumbar region), presenting symptoms, location of ICH, surgical and non-surgical treatment strategies, and outcome (Tables 1 and 2). We also reviewed the existing literature and collected demographic, treatment, and outcome information from 33 unique case reports of remote ICH after spinal surgery.

Results

The results of the data collection and analysis are summarized in Tables 1–3.

Initial Presentation

Our 8 patients ranged in age from 42 to 81 years old (median age 63.5 years). Four patients presented with scoliotic deformity, 2 with disc herniation, 1 with failed instrumentation, and 1 with left pelvic osteosarcoma. Three of the 8 patients had undergone previous spinal surgeries. Medical histories were remarkable for preoperative daily intake of aspirin, which was stopped well prior to surgery (n = 2), anemia and factor V Leiden thrombophilia (n = 1), hyperlipidemia (n = 3), clinical hypertension (n = 1), and Type II diabetes mellitus (n = 1). None of the patients were smokers (n = 0). In each of these patients all conservative therapies had failed.

Surgery and Durotomy

In our 8 patients, the spinal levels operated on ranged from C-2 to S-3. Seven cases involved deformity or mechanical instability. Specific procedures included posterior cervicothoracic instrumentation and fusion with decompression (Case A), lumbar corpectomy followed by lumbar posterior instrumentation and fusion (Case B), lumbar laminectomy and instrumentation with fusion (Cases C–E and H), lumbosacral instrumentation and fusion followed by anterior hemisacrectomy (Case F), and thoracolumbar instrumentation and fusion (Case G). Operative notes mentioned intraoperative durotomy and primary repair (n = 8). Dural repair was achieved with suture alone (n = 4), suture and fibrin glue (n = 2), or with duraplasty using collagen-based dural graft matrix and bovine pericardium for a deliberately created durotomy (n = 1). Drains were placed in the epidural space in all 8 patients, but output data were unavailable for 3 patients due to new documentation system implementation that was not existent at the time of those encounters. Average hourly drain output before symptom onset was 9.2 ml per hour, or a daily average of 221 ml per day. The total volume of intraoperative CSF loss was not recorded, but all CSF leaks were repaired immediately upon detection.

Intracranial Hemorrhage

All 8 patients were symptomatic postoperatively. Symptom onset ranged from immediately after surgery to postoperative Day 3, and symptoms included headache (n = 1), aphasia (n = 1), seizure (n = 1), and altered mental status (n = 4). No patients had documented hemodynamic instability, such as hypertensive or hypotensive episodes, in the intraoperative or postoperative period. All patients underwent head CT, which revealed CBH (n = 5), IVH (n = 2), or SDH (2). Two of the patients with CBH also presented with concomitant hydrocephalus (n = 2).

Treatment and Outcome

Five patients were treated conservatively, while 3 patients required more acute neurosurgical intervention. Surgical management consisted of craniotomy for ICH evacuation (n = 2) and/or ventriculostomy placement for hydrocephalus management (n = 2). One patient returned to the operating room for a continued CSF leak requiring reexploration and repair of a dural tear. Ultimately, 5 patients achieved a full recovery with minimal or no residual neurological deficits. One patient died 48 hours postoperatively, one was declared brain dead on the 5th postoperative day, and 1 was discharged to a rehabilitation facility with severe cognitive impairment, where she died of aspiration pneumonia.

Case Series

Case A

This 45-year-old man with a history of renal osteodystrophy and hypertension presented with acute-onset neck pain, and scans demonstrated a spontaneous compression fracture of the C-6 vertebral body. He underwent a 2-stage procedure that involved an anterior 2-level corpectomy followed by placement of posterior segmental instrumentation for stabilization. He presented again 6 weeks later with failure of cervical instrumentation and pseudarthrosis. He was taken back to the operating room for a revision of the posterior instrumentation. Surgery was complicated by a small durotomy that was repaired immediately intraoperatively. An epidural drain was placed postoperatively for 4 days. The patient received a tracheostomy on postoperative Day 13 because he could not be weaned off the ventilator. The patient’s neurological examination status continued to decline while he exhibited significant confusion. Computed tomography scanning demonstrated a small cerebellar hematoma but no hydrocephalus. The patient was managed conservatively. His cognition improved, and he was discharged to rehabilitation with minor speech deficits. At 4-year follow-up no neurological deficits were demonstrated.

Case B

This 55-year-old woman with a history of cadaveric kidney transplantation, lupus, and chronic immunosuppression therapy presented with severe lumbar stenosis and spondylolisthesis. She had undergone 2 previous lumbar decompressions with fusion but had pseudarthrosis and worsening spinal deformity. She underwent a 2-stage anterior L-3 corpectomy followed by a posterior segmental revision decompression with extension of posterolat-
# TABLE 1: Summary of perioperative data

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yrs), Sex</th>
<th>Diagnosis</th>
<th>DM</th>
<th>HTN</th>
<th>Hyperlipidemia</th>
<th>Anticoagulation†</th>
<th>Bleeding Disorder</th>
<th>Prior Spine Op</th>
<th>Other Risk Factors</th>
<th>Procedure</th>
<th>Levels</th>
<th>Op Position</th>
<th>Approach</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>45, M</td>
<td>compressionFx</td>
<td>yes</td>
<td>ESRD</td>
<td>Leiden</td>
<td>anemia, factor V</td>
<td>reop extension of instrumentation/fusion</td>
<td>C2–T5</td>
<td>prone</td>
<td>pst</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>55, F</td>
<td>kyphoscoliosis, spinal stenosis</td>
<td>yes</td>
<td>ESRD, kidney transplant</td>
<td>yes</td>
<td>ESRD</td>
<td>2 stages: ant &amp; pst decomp w/ instrumentation/fusion</td>
<td>L2–4, L-3 corpectomy</td>
<td>prone; supine</td>
<td>ant-pst (stage 1; stage 2)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>61, F</td>
<td>disc herniation, spinal cord compression</td>
<td>yes</td>
<td>yes</td>
<td>Ca-channel inhibitors</td>
<td>LIF</td>
<td>T6–10</td>
<td>prone</td>
<td>pst</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>63, M</td>
<td>kyphoscoliosis, stenosis</td>
<td>yes</td>
<td>yes</td>
<td>325 mg aspirin</td>
<td>LIF</td>
<td>L2–5</td>
<td>prone</td>
<td>pst</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>E</td>
<td>64, F</td>
<td>scoliosis, spinal stenosis</td>
<td>warfarin postop</td>
<td>LIF</td>
<td>L1–S1</td>
<td>prone</td>
<td>pst</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F</td>
<td>76, M</td>
<td>osteosarcoma spine</td>
<td>yes</td>
<td>CAD, beta-blockers</td>
<td>2 stages: pst decomp followed by hemivertebrectomy</td>
<td>L2–S3</td>
<td>prone; supine</td>
<td>pst-ant (stage 1; stage 2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G</td>
<td>77, M</td>
<td>kyphoscoliosis, ankylosing spondylitis</td>
<td>yes</td>
<td>325 mg aspirin</td>
<td>pst instrumentation/fusion w/ osteotomy</td>
<td>T11–S1</td>
<td>prone</td>
<td>pst</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H</td>
<td>81, F</td>
<td>spondyloolisthesis, disc herniation</td>
<td>Lipitor</td>
<td>LIF</td>
<td>L4–5</td>
<td>prone</td>
<td>pst</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

* ant = anterior; CAD = coronary artery disease; decomp = decompression; DM = diabetes mellitus; ESRD = end-stage renal disease; Fx = fracture; HTN = hypertension; LIF = laminectomy, instrumentation, and fusion; pst = posterior.† History of use; medication was withheld for at least 10 days prior to surgery to decrease bleeding.
TABLE 2: Summary of intra- and postoperative data*

<table>
<thead>
<tr>
<th>Case</th>
<th>Durotomy Method</th>
<th>Repair Method</th>
<th>Drains Placed (no.)</th>
<th>Postop CSF Leak</th>
<th>Drain Output Prior to ICH Detection</th>
<th>Complaint, Symptom, Finding</th>
<th>Symptom Onset</th>
<th>ICH Type</th>
<th>ICH Location</th>
<th>Hydrocephalus</th>
<th>Op Intervention</th>
<th>Status at Discharge</th>
<th>Long-Term FU</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>incidental</td>
<td>suture</td>
<td>none</td>
<td>data unavailable</td>
<td>altered mental status</td>
<td>immed CBH</td>
<td>rt cerebellar hemisphere</td>
<td>none</td>
<td>minor speech deficits</td>
<td>4-yr FU: no neurological deficits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>incidental</td>
<td>suture</td>
<td>yes (1)</td>
<td>235 ml (3 days)</td>
<td>aphasia</td>
<td>3 days SDH</td>
<td>lt frontal, parietal, &amp; temporal lobes</td>
<td>craniotomy</td>
<td>full recovery</td>
<td>8-yr FU: no neurological deficits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>deliberate</td>
<td>suture</td>
<td>yes (2): lumbar drain</td>
<td>176 ml (1 day)</td>
<td>motor seizure</td>
<td>immed ICH</td>
<td>rt temporal, parietal lobes</td>
<td>none</td>
<td>near full recovery w/ minimal cognitive deficits</td>
<td>6-mo FU: neurologically stable</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>incidental</td>
<td>suture</td>
<td>yes (2)</td>
<td>478 ml (2 days)</td>
<td>somnolence, altered consciousness</td>
<td>2 days CBH, IVH</td>
<td>bilat cerebellar hemispheres</td>
<td>none</td>
<td>full recovery</td>
<td>none</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>incidental</td>
<td>suture</td>
<td>yes (3)</td>
<td>660 ml (2 days)</td>
<td>headache, loss of consciousness</td>
<td>2 days CBH</td>
<td>bilat cerebellar hemispheres</td>
<td>none</td>
<td>brain death 48 hrs postop</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F</td>
<td>none</td>
<td>NA</td>
<td>yes (2)</td>
<td>250 ml (17 hrs)</td>
<td>somnolence, altered consciousness</td>
<td>immed SDH</td>
<td>bilat cerebral hemispheres</td>
<td>none</td>
<td>residual cognitive deficits</td>
<td>9-mo FU: resolving short-term memory loss</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G</td>
<td>incidental</td>
<td>suture</td>
<td>none</td>
<td>data unavailable</td>
<td>somnolence</td>
<td>immed CBH, IVH</td>
<td>bilat cerebellar hemispheres, 4th &amp; lateral ventricles</td>
<td>yes</td>
<td>ventriculostomy</td>
<td>significant cognitive deficits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>H</td>
<td>incidental</td>
<td>suture</td>
<td>none</td>
<td>data unavailable</td>
<td>somnolence</td>
<td>1 day CBH</td>
<td>lt cerebellar hemisphere</td>
<td>yes</td>
<td>craniotomy, ventriculostomy</td>
<td>brain death, ex-tubated 6 days postop</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* FU = follow-up; immed = immediately; NA = not applicable.
eral instrumentation and fusion. Due to the significant scar tissue intraoperatively, the surgery was complicated by a durotomy that was repaired immediately. A Hemovac drain was placed in the epidural space for 6 days. Postoperatively, the patient developed postural headaches and expressive aphasia. A CT scan demonstrated a 1.2-cm subdural hematoma along the left temporoparietal region with midline shift. A craniotomy was performed to evacuate the hematoma, after which the patient returned to baseline cognition. The patient was discharged on postoperative Day 15 with no further complications or neurological deficits. Her 8-year follow-up showed no neurological deficits.

Case C

This 61-year-old woman presented with acute onset of bilateral lower-extremity weakness after an aborted posterolateral decompression for treatment of a T6–7 disc herniation done at an outside hospital. This procedure was complicated by a dural tear that was repaired. Postoperatively, the patient developed gram-negative meningitis and lower-extremity numbness and paresis, which were treated with antibiotic agents. She presented to our hospital 1 month after the aborted procedure with a large T6–7 disc herniation, severe spinal cord compression, and near paraplegia. She underwent a T6–7 transpedicular corpectomy, T6–8 laminectomies, and T3–10 instrumentation with fusion. Intradural exposure was required due to the complexity of the disc herniation requiring complex dural reconstruction and duraplasty with bovine pericardium. A lumbar drain was placed and maintained for 6 days. The patient tolerated the procedure well but had a noted motor seizure upon waking from anesthesia. A CT scan showed ICH in the right temporal and parietal lobes. The hemorrhages were stable in size on repeat scans, and the patient’s status on examination improved to baseline. On Day 15, she was discharged to a rehabilitation facility after experiencing a full recovery of consciousness with residual dysphagia and neurogenic bladder. The 6-month follow-up showed no neurological deficits.

Case D

This 63-year-old man with a history of diabetes mellitus presented with worsening kyphoscoliosis and severe symptomatic neurogenic claudication due to lumbar stenosis. He was taken to the operating room for decompressive laminectomy and posterolateral instrumentation and fusion. Surgery was complicated by a dural tear that was repaired immediately. Two epidural drains were placed. On Day 2, the patient became combative and somnolent. A head CT scan revealed bilateral SAH over the cerebellar folia without significant mass effect or brainstem compression. The bleeding stabilized and the patient experienced a full recovery by postoperative Day 15.

Case E

This 64-year-old woman with a history of intractable back pain presented with lumbar scoliosis and severe neurogenic claudication due to spinal stenosis. She was taken to the operating room for posterior decompression, with instrumentation and fusion from L-1 to S-1. Surgery was complicated by durotomy that was repaired immediately. Three epidural drains were placed. On postoperative Day 2, the patient complained of a severe headache. Two hours later, she was noted to be unresponsive, and her pupils were 5 mm in diameter and nonreactive. Brainstem reflexes were absent. A CT scan demonstrated a large cerebellar hemorrhage, brainstem compression, and hydrocephalus. She was declared brain dead soon thereafter.

Case F

This 76-year-old man presented with left pelvic osteosarcoma extending to L-4. He had completed 3 cycles of chemotherapy and elected to undergo resection of the malignant lesion. Surgery was performed in 2 stages. Stage 1 involved a posterior decompression and long-segment instrumentation, and stage 2 involved a hemisacrectomy and anterior reconstruction. Surgery was complicated by an iliac vein injury that was repaired intraoperatively. No dural tear was encountered. Two epidural drains were placed postoperatively due to oozing tissue. The patient was taken back to the operating room on postoperative Day 5 for posterior wound drainage, during which a washout was performed. The patient developed decreasing strength on the right side and exhibited increasing confusion. A CT scan demonstrated chronic bilateral subdural hematomas with minimal mass effect. The patient was managed conservatively, and neurological examination status improved to baseline. He was discharged to rehabilitation on postoperative Day 42. His 9-month follow-up showed no residual deficits.

Case G

This 77-year-old man with a history of ankylosing spondylitis presented with worsening thoracic deformity. He underwent a T11–S1 posterior instrumentation and fusion with an L-2 pedicle subtraction osteotomy. An incidental durotomy occurred during posterior dissection and was repaired immediately. An epidural drain was placed for 4 days. Motor function was intact upon awakening, but within 2 hours, the patient became somnolent and hemodynamically unstable. A CT scan was taken at postoperative Hour 3, which revealed IVH in the slightly enlarging fourth and lateral ventricles as well as subarachnoid blood in the cerebellar folia. A ventriculostomy was placed and the patient’s neurological examination status improved. The bleeding stabilized, the patient was weaned off the ventriculostomy, and his neurological examination status improved. He was discharged to a rehabilitation facility 9 weeks after surgery with residual cognitive defects and impaired functional mobility. He died 9 months later of aspiration pneumonia.

Case H

This 81-year-old woman presented with intractable left-sided L-4 and L-5 radiculopathy due to Grade 1 spondylolisthesis at L4–5. She was taken to the operating room where L4–5 decompression and posterolateral instrumentation and fusion were performed. The disc was tightly adherent to the dura, which resulted in a du-
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rotomy that was repaired immediately. An epidural drain was placed. On postoperative Day 1, she became acutely somnolent. A CT scan revealed a large, left-sided CBH with acute hydrocephalus. The patient was taken back to the operating room for CBH evacuation and placement of a ventriculostomy. The patient did not recover neurologically and died on postoperative Day 5.

Discussion

Postoperative ICH is a rare complication of spine surgery. An incidence rate of less than 0.8% has been attributed to CBH after lumbar spine surgery, which is close to the 0.6% incidence rate associated with remote CBH after craniotomy. However, the presenting symptoms may be nonspecific, and since obtaining head CT scans is not part of the usual routine after spine surgery, many hemorrhages may have been overlooked. Left unrecognized, this complication can have debilitating or even fatal consequences.

The most common etiology for spontaneous cerebral hemorrhage is hypertension, where long-term high blood pressure induces subsequent rupture of the deep perforating vessels. The most common locations include the basal ganglia, thalamus, brainstem, and cerebellum. In individuals older than 70 years, spontaneous ICH is most commonly due to primary amyloid angiopathy, rather than hypertension. None of our patients’ vital signs showed any sustained hemodynamic instability intraoperatively or postoperatively. Other causes of intracerebral hemorrhage include hemorrhagic infarction, septic embolism, mycotic aneurysm, tumoral hemorrhage, coagulopathy, anticoagulant drug use, infection, and trauma. However, these common etiologies are rarely present in previously published case studies for remote ICH after spine surgery. Instead, the most consistent contributing factor is a dural tear, with 30 of 33 previously reported cases confirming the presence of a durotomy (Table 3).

The pathophysiological mechanism of ICH after CSF loss is unclear, but evidence suggests that the cranial bleeds are venous in origin. In contrast to focal arterial hemorrhages, these venous bleeds tend to present with bilateral hemorrhage. In one case reported of remote ICH after spinal surgery, Kim et al. described the presence of venous oozing below the tentorium without any specific arterial bleeding source. Such findings support this sagittal model hypothesis, in which a decrease in CSF volume or pressure causes the brain to descend, resulting in stretching and therefore tearing of the cerebral and cerebellar veins. Further neurological decline may occur through caudal displacement of the cerebellum with brainstem compression and development of cranial neuropathies, as described by Gul et al.

Cerebrospinal fluid hypovolemia and cerebral hypotension have also been cited as the probable cause of remote ICH after supratentorial surgery and lumbar puncture. Several authors have suggested that the expansion of the CSF space, creation of a resection cavity, and tentorial exposure may play a role in the development of CBH after temporal lobe surgery. Furthermore, multiple case reports have related traumatic lumbar puncture and even epidural steroid injection to remote ICH.

Intraoperative positioning may or may not be a relevant risk factor, since patients operated on in the sitting and lateral positions have been reported to have experienced postoperative ICH as well. However, it has been argued that while the overall body position may not make a difference, tilting the head slightly downward could counteract the sagging effect caused by dural opening and CSF drainage.

Recent reports reflect a growing concern over the placement of drains at the surgical site, which has emerged as one of the most consistently confirmed variables in patients with ICH after spine surgery. This trend is also reflected in our own cohort, all 8 patients having had subfascial or subcutaneous drains placed during closure. In 2010, Sasan et al. reported a case in which the onset of CBH symptoms did not occur immediately after spinal arteriovenous malformation surgery but instead after the postoperative placement of a lumboperitoneal shunt. That same year, Miglis and Levine described the case of a patient who was neurologically intact after anterior cervical discectomy and fusion but who experienced acute onset of headache, vomiting, and visual defects 15 hours after the insertion of a lumbar drain. Such reports implicate drain placement, whether subcutaneous or subarachnoid, as a critical precipitating factor in the onset of ICH after spine surgery, possibly through CSF diversion. One author found that the subgaleal suction system exerted enough negative pressure to remove CSF from between the stitches of a watertight dural repair. Another noted that the suction drain maintained a high volume of CSF flow through the dural layer and that the dura did not seal until after the drain was removed. Our study demonstrates that an average daily drainage of 221 ml (9.2 ml/hour) of serosanguineous fluid (possible combination of postoperative fluid and CSF) was associated with these particular intracerebral hemorrhages, without a clear estimate of intraoperative CSF loss recorded. Despite these results, drains remain a critical aspect of postoperative care in the vast majority of neurosurgical patients, both with or without an intraoperative CSF leak. The trends reported here do not justify completely avoiding use of drains in patients with a CSF leak. They merely report the association and may guide the treating physician to keep in mind drain output and timing of drain removal while noting any changes in a patient’s neurological examination findings in the meantime. At our institution the vast majority of patients with intraoperative CSF leaks in whom drains have been placed have not had any neurological symptoms to warrant intracranial imaging, and those who have had intracranial imaging have not had a documented intracerebral hemorrhage.

The authors of a review of remote CBH after supratentorial and spinal surgery reported that symptoms began within the first 10 hours after surgery for 46% of patients and later than 10 hours for 54%. Our own data collection for spinal cases alone shows that 33% of patients were symptomatic within the first 10 hours postoperatively. Common clinical signs are headache, nausea, vomiting, delayed emergence from anesthesia, somnolence, altered consciousness, dysarthria, ataxia, and motor or visual deficits. Headache is the most frequently cited symptom
<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs), Sex</th>
<th>Problem</th>
<th>Procedure</th>
<th>Drains Placed</th>
<th>Symptom Onset</th>
<th>ICH Type</th>
<th>ICH Location</th>
<th>Op Intervention</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Andrews &amp; Koci, 1995</td>
<td>36, M</td>
<td>lumbar scoliosis</td>
<td>LIF</td>
<td>DT unnoticed</td>
<td>yes</td>
<td>36 hrs</td>
<td>CBH bilat cerebellar hemispheres</td>
<td>ventriculostomy, drain removal</td>
<td>quadripareisis</td>
</tr>
<tr>
<td>Beier et al., 2009</td>
<td>39, F</td>
<td>herniated disc</td>
<td>microdiscectomy</td>
<td>incidental</td>
<td>7 days</td>
<td>SDH</td>
<td>rt frontoparietal cortex</td>
<td>DT repair in 2nd op</td>
<td>full recovery</td>
</tr>
<tr>
<td>Calisenaller et al., 2007</td>
<td>67, F</td>
<td>spondylothesis</td>
<td>LIF</td>
<td>incidental</td>
<td>8 days</td>
<td>CBH</td>
<td>bilat cerebellar hemispheres</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Cevik et al., 2009</td>
<td>79, F</td>
<td>herniated disc, stenosis, spondylothesis</td>
<td>LIF</td>
<td>incidental</td>
<td>3 days</td>
<td>CBH</td>
<td>bilat cerebellar hemispheres</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td></td>
<td>68, F</td>
<td></td>
<td>LIF</td>
<td>incidental</td>
<td>7 days</td>
<td>CBH</td>
<td>bilat cerebellar hemispheres</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Chadduk, 1981</td>
<td>59, M</td>
<td>stenosis</td>
<td>laminectomy</td>
<td>incidental</td>
<td>1 day</td>
<td>CBH</td>
<td>rt cerebellar hemisphere</td>
<td>craniotomy, ventriculostomy</td>
<td></td>
</tr>
<tr>
<td>Chalela et al., 2006</td>
<td>62, F</td>
<td>lumbar stenosis</td>
<td>LIF</td>
<td>incidental</td>
<td>4 hrs</td>
<td>CBH</td>
<td>bilat cerebellar hemispheres</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Farag et al., 2005</td>
<td>43, M</td>
<td>traumatic lumbarFx</td>
<td>spinal reexploration, lat fusion, iliac crest autograft</td>
<td>DT unnoticed</td>
<td>26 hrs</td>
<td>CBH</td>
<td>bilat cerebellar hemispheres</td>
<td>DT repair in 2nd op</td>
<td>full recovery, mild motor deficits</td>
</tr>
<tr>
<td>Fernandez-Jara et al., 2011</td>
<td>58, F</td>
<td>stenosis</td>
<td>LIF</td>
<td>incidental</td>
<td>36 hrs</td>
<td>CBH</td>
<td>rt cortex &amp; bilat cerebellar hemispheres</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Friedman et al., 2002</td>
<td>43, M</td>
<td>herniated disc</td>
<td>discectomy</td>
<td>incidental</td>
<td>12 hrs</td>
<td>CBH</td>
<td>rt cerebellar hemisphere</td>
<td>full recovery, mild motor deficits</td>
<td></td>
</tr>
<tr>
<td></td>
<td>56, F</td>
<td>lumbar spondylothesis</td>
<td>LIF</td>
<td>DT unnoticed</td>
<td>yes</td>
<td>2 days</td>
<td>CBH bilat cerebellar hemispheres</td>
<td>DT repair in 2nd op</td>
<td>full recovery, mild motor deficits</td>
</tr>
<tr>
<td>Gul et al., 2010</td>
<td>64, F</td>
<td>herniated disc, stenosis</td>
<td>LIF</td>
<td>incidental</td>
<td>yes</td>
<td>24 hrs</td>
<td>CBH rt cerebellar hemisphere</td>
<td>suboccipital de-comp</td>
<td>full recovery, mild motor deficits</td>
</tr>
<tr>
<td>Hempelmann &amp; Mater, 2012</td>
<td>61, F</td>
<td>intradural extramedullary metastasis</td>
<td>laminectomy for tumor resection</td>
<td>incidental</td>
<td>yes</td>
<td>1 day</td>
<td>CBH rt cerebellar hemisphere</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td></td>
<td>69, F</td>
<td>stenosis, instability</td>
<td>LIF</td>
<td>incidental</td>
<td>yes</td>
<td>2 days</td>
<td>CBH, ICH rt cerebellar hemisphere, temporooccipital cortex</td>
<td>DT repair in 2nd op</td>
<td>full recovery</td>
</tr>
<tr>
<td></td>
<td>62, F</td>
<td>stenosis</td>
<td>LIF</td>
<td>incidental</td>
<td>immed</td>
<td>CBH, ICH</td>
<td>rt cerebellar hemisphere</td>
<td>DT repair in 2nd op</td>
<td>full recovery</td>
</tr>
<tr>
<td>Karaeminogullari et al., 2005</td>
<td>73, F</td>
<td>stenosis</td>
<td>laminectomy, facetectomy</td>
<td>incidental</td>
<td>yes</td>
<td>2 days</td>
<td>CBH rt cerebellar hemisphere</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Khalattbari et al., 2012</td>
<td>53, M</td>
<td>herniated disc</td>
<td>discectomy</td>
<td>incidental</td>
<td>yes</td>
<td>8 hrs</td>
<td>CBH bilat cerebellar hemispheres</td>
<td>ventriculostomy</td>
<td>full recovery, mild motor deficit</td>
</tr>
</tbody>
</table>

(continued)
### TABLE 3: Summary of 33 previously reported cases* (continued)

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Problem</th>
<th>Procedure</th>
<th>Durotomy†</th>
<th>Drains Placed</th>
<th>Symptom Onset</th>
<th>ICH Type</th>
<th>ICH Location</th>
<th>Op Intervention</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Khatlabari et al., 2012</td>
<td>75, M</td>
<td>stenosis</td>
<td>laminectomy</td>
<td>incidental</td>
<td>yes</td>
<td>immed</td>
<td>CBH, IVH</td>
<td>bilat cerebellar hemispheres, 4th ventricle</td>
<td>ventriculostomy</td>
<td>died due to PCs &amp; sepsis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>34, F</td>
<td>extruded disc fragment</td>
<td>discectomy</td>
<td>no DT noted</td>
<td>yes</td>
<td>5 days</td>
<td>SDH</td>
<td>rt frontoparietal cortex</td>
<td></td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td></td>
<td>29, M</td>
<td>herniated disc</td>
<td>discectomy</td>
<td>incidental</td>
<td>yes</td>
<td>unknown</td>
<td>EDH</td>
<td>lt parietal lobe</td>
<td>craniotomy</td>
<td>full recovery, severe motor, neurological deficits</td>
<td></td>
</tr>
<tr>
<td>Kim et al., 2010</td>
<td>56, F</td>
<td>spondylothesis, herniated disc</td>
<td>LIF</td>
<td>incidental</td>
<td>yes</td>
<td>43 hrs</td>
<td>CBH, IVH</td>
<td>bilat cerebellar hemispheres, 4th ventricle</td>
<td>evacuation, ventriculostomy</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Konya et al., 2006</td>
<td>48, F</td>
<td>herniated disc, stenosis</td>
<td>LIF</td>
<td>incidental</td>
<td>yes</td>
<td>12 hrs</td>
<td>CBH</td>
<td>bilat cerebellar hemispheres</td>
<td></td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Lu et al., 2002</td>
<td>59, F</td>
<td>FBS</td>
<td>LIF</td>
<td>incidental</td>
<td>yes</td>
<td>4 days</td>
<td>SDH</td>
<td>parietal lobe</td>
<td>craniotomy</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Miglis &amp; Levine, 2010</td>
<td>45, F</td>
<td>spondylosis</td>
<td>LIF</td>
<td>DT unnoticed</td>
<td>yes</td>
<td>1 days</td>
<td>ICH</td>
<td>temporal lobe</td>
<td>neurological defects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mikawa et al., 1994</td>
<td>75, M</td>
<td>atlantoaxial subluxation</td>
<td>LIF</td>
<td>incidental</td>
<td>yes</td>
<td>36 hrs</td>
<td>SAH, CBH</td>
<td>pst fossa, rt cerebellar hemisphere</td>
<td>suboccipital decom</td>
<td>died due to pneumonia</td>
<td></td>
</tr>
<tr>
<td>Morandi et al., 2001</td>
<td>34, M</td>
<td>extramedullary schwannoma</td>
<td>laminectomy for tumor resection</td>
<td>incidental</td>
<td>immed</td>
<td>CBH, ICH</td>
<td>bilat cerebellar hemispheres, lt temporal</td>
<td></td>
<td>full recovery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Morofuji et al., 2009</td>
<td>51, M</td>
<td>ossification of ligamentum flavum</td>
<td>laminectomy with fusion</td>
<td>incidental</td>
<td>yes</td>
<td>immed</td>
<td>CBH</td>
<td>bilat cerebellar hemispheres</td>
<td>suboccipital decom, ventriculostomy</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Nakazawa et al., 2005</td>
<td>74, F</td>
<td>cervical intradural extramedullary tumor</td>
<td>laminectomy for tumor resection</td>
<td>incidental</td>
<td>immed</td>
<td>CBH</td>
<td>rt cerebellar hemisphere</td>
<td></td>
<td>full recovery, mild motor deficits</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ozturk et al., 2006</td>
<td>23, F</td>
<td>thoracolumbar scoliosis</td>
<td>LIF</td>
<td>incidental</td>
<td>yes</td>
<td>3 hrs</td>
<td>CBH, ICH</td>
<td>rt cerebellar hemisphere, lt temporal lobe, rt putamen</td>
<td>DT repair in 2nd op</td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Sasani et al., 2010</td>
<td>47, F</td>
<td>spinal AVM</td>
<td>laminectomy for AVM resection</td>
<td>incidental</td>
<td>yes</td>
<td>12 days</td>
<td>CBH</td>
<td>bilat cerebellar hemispheres</td>
<td></td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>Satake et al., 2000</td>
<td>62, M</td>
<td>intramedullary tumor</td>
<td>laminectomy for tumor resection</td>
<td>incidental</td>
<td>yes</td>
<td>18 hrs</td>
<td>CBH</td>
<td>bilat cerebellar hemispheres</td>
<td>neurological deficits</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thomas et al., 2002</td>
<td>38, M</td>
<td>intradural, extramedullary tumor</td>
<td>laminectomy for tumor resection</td>
<td>incidental</td>
<td>yes</td>
<td>immed</td>
<td>CBH, ICH</td>
<td>rt cerebellar hemisphere, temporal &amp; petrous</td>
<td></td>
<td>full recovery</td>
<td></td>
</tr>
<tr>
<td>You et al., 2012</td>
<td>63, M</td>
<td>herniated disc</td>
<td>discectomy</td>
<td>DT unnoticed</td>
<td>immed</td>
<td>CBH, ICH</td>
<td>bilat cerebellar hemispheres, rt temporal lobe</td>
<td></td>
<td>full recovery</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* AVM = arteriovenous malformation; decomp = decompression; DT = dural tear; FBS = failed back syndrome; PCs = pulmonary complications.
† All durotomies were repaired unless unnoticed.
and is easily attributed to low CSF pressure. Other
commonly misattributed signs include somnolence, altered
consciousness, and dysarthria, all of which have been
mistakenly ascribed to opioid overmedication. Several
preoperative cases report acknowledgment that this assumption prolonged
the time between symptom onset and the ordering of a
head CT scan. Thus, ICH must be included in the differential
examination for postoperative confusion and declining
mental status after spinal surgery, with opioid reversal being
a possible option to ensure accurate diagnosis.\(^{1,9,10,17,26}\)

Computed tomography scanning can reveal a characteristic bleeding pattern in cases of postoperative remote
ICH. Whereas hypertensive cerebellar hematomas are
generally found near the dentate nucleus, remote CBHs
are localized subcortically.\(^ {22}\) Brockmann et al. presented
3 cases of CBH after spinal, supratentorial, and thoracic
surgery respectively and noted a streaking pattern of sub-
arachnoid blood along the superior aspect of the cerebel-
lar folia.\(^ {4}\) The presence of this so-called “zebra sign” has
been confirmed and reported in several subsequent pa-
pers.\(^ {6,8,10,17}\) A similar bleeding pattern has also been found
in cases of CBH after supratentorial durotomy, suggest-
ing a shared mechanism of acute CSF loss after dural
opening leads to cerebellar herniation and tearing of cer-
ebellar bridging veins. Physicians are therefore advised
to relate this unique pattern of hemorrhage to a prior or
ongoing loss of CSF.\(^ {4}\)

Treating the complication of ICH varies depending
on location and extent of hemorrhage and clinical exami-
nation status of the patient. Patients who have a small
hemorrhage without significant mass effect and whose
neurological status is appropriate may be managed con-
servatively. Those with large hemorrhages who are suffer-
ing rapid deterioration must undergo surgical evacuation
of the bleeds with or without ventriculostomy placement
to manage hydrocephalus. Drains may be removed, but
this risks causing additional leakage through the dural
opening left behind. Branching off or intermittent clamp-
ing of the drains may be a safer option to control aspira-
tion.\(^ {10}\) Ventriculostomy has been shown to help relieve
symptoms in a few cases, but a persistent CSF leakage
may warrant a return to the operating room for dural re-
pair.\(^ {2,9,11–13,30}\)

Mortality rates reported for remote CBH after supra-
tentorial surgery range from 10% to 25%.\(^ {2,24}\) A review
of the literature and the cases of all patients in our study
who had ICH after spinal surgery reveals a mortality rate
of 14% (Tables 1–3). In our series of 8 patients, 2 were
declared brain dead during their postoperative hospital stay
(Cases E and H), and 1 expired of aspiration pneumonia
after having been discharged to a rehabilitation facility
(Case G). We are unable to draw significant conclusions
regarding possible risk factors given such a small cohort
of patients. However, we believe it is important to note
a few consistent variables among those who died. All 3
of these patients had an incidental durotomy that was re-
paired during the initial spinal surgery, along with drain
placement. Two of the patients had a history of anticoagu-
lant therapy prior to surgery, though therapy was stopped
well before the surgical procedure. All 3 presented with
altered consciousness by the 1st or 2nd postoperative day.

Head CT revealed CBH in 3 patients, with concomitant
hydrocephalus in 2. The patient in Case E died before
intervention was possible, whereas the patients in Cases
G and H received a ventriculostomy and both craniotomy
and ventriculostomy, respectively.

Of the surviving patients, 5 achieved full recovery
with mild or no residual neurological or motor deficits as
many as 4 years postoperatively. The fifth patient (Case
F) had significant cognitive impairment, but this has been
attributed to prolonged intraoperative hypoxia as opposed
to postoperative SDH. Only 1 of the 5 survivors required
acute intervention. The remaining 4 were managed con-
servatively and had satisfactory resolution of the hemato-
mas, further supporting the theory that conservative treat-
ment and close observation is a safe option for patients
with milder symptoms and subacute bleeds. Furthermore,
only 1 of the surviving patients had a cerebellar bleed,
whereas the rest had a subdural hemorrhage or SAH, and
none had hydrocephalus. These findings suggest that both
the neurological examination at diagnosis of ICH and the
location of the ICH, namely within the cerebellum, as
well as the presence of hydrocephalus, may have impor-
tant implications regarding the final outcome.

Given the small cohort, statistical analysis was not
appropriate in determining causal relationships for ICH
after spinal surgery or risk factors associated with out-
come after ICH. Intraoperative CSF loss, which may also
be a contributing factor for remote ICH, was not record-
ed, although the dural tears were repaired immediately
intraoperatively. Additionally, postoperative drain output
is not entirely CSF in composition, and so the hourly es-
imates of drain output cannot all be attributed to a CSF
leak. However, we reviewed the largest case series of ICH
after spinal surgery and have reported trends that may
prove helpful in managing patients with neurological
changes after spinal surgery and in counseling patients—
and their families—postoperatively as they are being
treated for ICH. Additionally, we provide a more focused
look at the use of drains postoperatively in patients with
CSF leaks so that the treating physicians are more aware
of a possible association between CSF diversion and ICH,
warranting closer correlation of neurological examination
findings in these cases.

Conclusions

Although rare, remote ICH can occur after spinal
surgery. Based on our review of the largest case series
of 8 patients, the presence of intraoperative CSF leakage
and the use of drains postoperatively, with aforemen-
tioned moderate outputs, were all common factors shared
among our patients; this does not indicate a causal rela-
tionship, because of the lack of statistical validation,
but merely that these are factors that show an associa-
tion. Furthermore, the trends reported here do not justify
a complete avoidance of drain placement in patients with
CSF leakage. They merely underscore the association and
may guide the treating physician to keep in mind drain
output and timing of drain removal while in the mean-
time noting any changes in patient neurological examina-
tion. The vast majority of ICH was localized within the
Intracranial hemorrhage after spine surgery
cerebellum and identified based on changes in neurologi-
cal status unexplainable by any other cause. Patients with
unexplainable and severe headaches postoperatively, as
well as those with acute confusion and lethargy relat-
ed to recent medical or laboratory abnormalities, should
be sent for an immediate CT scan. Patients with mild to
moderate headache or confusion after surgery should
be monitored in a subacute setting with more frequent
neurological examinations to detect any deterioration,
especially while the epidural or lumbar drains are still in
place. Management is based on the severity of findings on
a patient’s neurological examination as well as the extent
of ICH. In cases of severe neurological decline in patients
with a large ICH causing mass effect, emergency surgery
may be warranted. However, this subset of patients had
the worst prognosis. In all cases, early detection and in-
tervention are essential to ensure the best possible chance
for full recovery.

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Dr. Gokaslan reports the following: Direct stock ownership in
US Spine and Spinal Kinetics, clinical or research support from AO
North America/AOSpine/AO International, and is a member of the
board of AOSpine. Dr. Sciubba reports that he is a consultant for the
following: DePuy, Medtronic, Globus, and NuVasive. Dr. Bydon
reports support from DePuy Spine of a non-study-related clinical or
research effort overseen by the author.

Author contributions to the study and manuscript preparation
include the following. Conception and design: Kaloostian, Witham.
Acquisition of data: all authors. Drafting the article: Kim. Critically
revising the article: all authors. Reviewed submitted version of
manuscript: all authors. Approved the final version of the manuscript
on behalf of all authors: Kaloostian.

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Address correspondence to: Paul E. Kaloostian, M.D., Depart-
ment of Neurological Surgery, The Johns Hopkins Hospital, Mey-
er 7-109, 600 N. Wolfe St., Baltimore, MD 21287. email: paul
kaloostian@hotmail.com.