Spontaneous spinal subarachnoid hemorrhage and subdural hematoma

Report of two cases

KARL W. SWANN, M.D., ALLAN H. ROPPER, M.D., PAUL F. J. NEW, M.D., AND CHARLES E. POLETTI, M.D.

Neurological/Neurosurgical Intensive Care Unit, and Departments of Neurosurgery, Neurology, and Radiology, Massachusetts General Hospital, Boston, Massachusetts

Two patients with spontaneous spinal subarachnoid hemorrhage are presented to emphasize the clinical and radiological features of this uncommon illness. Both had severe back pain at the onset. One patient had a subdural hematoma that compressed the conus medullaris and cauda equina, and was drained percutaneously; the other had clots in the subarachnoid space. The cerebrospinal fluid showed a polymorphonuclear pleocytosis that simulated septic meningitis. Complete spinal angiography failed to reveal a cause for the hemorrhages.

KEY WORDS □9 spinal subarachnoid hemorrhage □9 spinal subdural hematoma □9 back pain □9 meningitis □9 spinal hemorrhage

Spinal subarachnoid hemorrhage is unusual, accounting for less than 1% of all cases of subarachnoid hemorrhage (SAH). It is important to recognize this disease early because hematomas can cause spinal cord damage, the clinical syndrome can mimic intracranial SAH, and the source of hemorrhage (for instance, arteriovenous malformation (AVM), spinal cord tumor, or aneurysm) is frequently treatable with surgery.

This report details some unusual features in two patients with spontaneous spinal SAH. One had a spinal subdural hematoma (SDH) that was recognized on computerized tomography (CT). The hematoma caused spinal cord and cauda equina compression, and was drained percutaneously. The clinical data, radiographic findings, and cerebrospinal fluid (CSF) features that aid in diagnosis of this disease are emphasized.

Case Reports

Case 1

This 46-year-old female nurse developed bifrontal headache while bending over a patient's bed. A few minutes later, she suddenly had intense pain dorsally at the thoracolumbar junction while standing at the nurses' station. The pain radiated to the mid-abdomen, both buttocks, and posterior aspects of the thighs. She had noted a mild generalized headache the day before that had diminished on taking aspirin. There was no prior back pain, trauma, or use of anticoagulant drugs. She had experienced monthly menstrual headaches since her teens.

Forty minutes after the onset of back pain, she reported paraparesis and loss of sensation in both legs extending cephalad to the umbilicus. A mid-abdominal sensory level was found, but the leg weakness resolved. A lumbar puncture revealed bloody CSF, with xanthochromia of a spun specimen, and an opening pressure of 180 mm H2O. There were 15,000 red blood cells (RBC's) and 200 white blood cells (WBC's)/cu mm, with 75 polymorphonuclear cells (PMN's) and 25 mononuclear cells; glucose and protein levels were 89 and 9.2 mg/100 ml, respectively. The numbness resolved shortly after the spinal tap was performed. The following day, leg strength and sensation were normal but severe back pain, stiff neck, and headache persisted. In addition, the patient intermittently behaved inappropriately and was drowsy and uncooperative, perhaps due to pain medications.

Examination. A complete spinal arteriogram, including radiculomedullary and radicular arterial injections, from T-1 to the hypogastric artery was normal. The artery of Adamkiewicz arose on the left side at T-9; injection of contrast material at that level repro-
FIG. 1. Case 1. A and B: Initial metrizamide myelograms. A: Lateral view, showing the tapering mass effect and displacement of the contrast column and upper cauda equina posteriorly. Irregularities in the ventral aspect of the subarachnoid space are inconsistent with an epidural mass. B: Frontal view, showing compression of the cauda equina and complete obstruction at the upper margin of L-2. There is no evidence of extension of the mass to the lateral surfaces of the subarachnoid space. C and D: Frontal (C) and lateral (D) projections of a myelogram 6 weeks after percutaneous drainage of the subdural hematoma. There is a very minor filling defect ventral to the subarachnoid space in the upper lumbar region. There is no longer compression of the cauda equina or spinal cord.

duced the severe pain that heralded the onset of illness. Severe pain hindered initial attempts at myelography, but the CSF that was obtained was dark red-brown in color, with a protein content of 1020 mg/100 ml, a glucose level of 20 mg/100 ml, a WBC count of 154/cu mm (15 PMN's, 85 lymphocytes), and no RBC's. A lumbar metrizamide myelogram showed a tapered narrowing of the sagittal diameter of the contrast column beginning anteriorly at the lower edge of L-3 and extending cephalad to a complete obstruction at the upper aspect of L-2 (Fig. 1A and B). This ventral filling defect had the appearance of a subdural rather than an epidural mass because of focal irregularities in the anterior contour of the subarachnoid space. An upper lumbar and lower thoracic spinal CT scan followed shortly after the myelogram and provided confirmation of an anterior symmetrically compressive mass, consistent with a subdural collection of blood (Fig. 2).

Operation. The following day the mass was drained percutaneously. Under fluoroscopic guidance, a No. 17 Tuohy needle was directed through the L2-3 interlamellar space in the midline and advanced to the vertebral body. A total of 18 ml of dark brown fluid that had the appearance of liquefied hematoma was aspirated from the location of the subdural mass. When fluid could no longer be withdrawn a catheter was inserted through the Tuohy needle. With Trendelenburg positioning and positive-pressure ventilation of the patient, an additional 2 ml of fluid was obtained. When the catheter was withdrawn, several millimeters of xanthochromic CSF was easily aspirated. The RBC concentrations from the subdural and subarachnoid spaces were 16,850/cu mm and 4280/cu mm, respectively. Multiple cytological examinations of both the CSF and the aspirated subdural clot showed no malignant cells.

Postoperative Course. Immediately following the procedure the patient's back pain resolved, the deep tendon reflexes became less hyperactive, and shortly thereafter neck stiffness and inappropriate behavior improved. A metrizamide myelogram and spinal CT scan 6 weeks after discharge demonstrated only a small amount of residual subdural clot lying behind the L-2 vertebra (Fig. 1C and D). Pernicious anemia was found during her hospitalization.

Case 2

This 72-year-old man had the sudden onset of excruciating dorsal thoracolumbar back pain without headache 6 days prior to his transfer to the Massachusetts General Hospital. The pain radiated into both flanks. At another hospital, he was found to be hypertensive and agitated. There was no history of back pain, trauma, use of anticoagulant drugs, or neurological or medical illness. An intravenous pyelogram showed bladder stones. The back pain resolved spontaneously after 2 days. Three days later the patient became confused and developed a stiff neck and a temperature of 102°F. A lumbar puncture showed 875,000 RBC's/cu mm, 11,600 WBC's/cu mm (83 PMN's and 17 mononuclear cells), a protein level of 1000 mg/100 ml, and

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FIG. 2. Case 1. Post-myelographic computerized tomography scan at the L-2 level. The isodense ventral mass is smoothly convex and symmetrical, and does not extend to the intervertebral foramina or the lateral surfaces of the subarachnoid space. The compressed cauda equina is visible within the metrizamide.

FIG. 3. Case 2. Initial metrizamide myelograms. Left: Lateral view showing elongated filling defects associated with the nerve roots at L-5, S-1, and S-2. Right: Frontal view showing a number of small, oval, and irregular filling defects within the subarachnoid space at L4-5.

A glucose content of less than 10 mg/100 ml. He was treated with antibiotics and epsilon-aminocaproic acid.

When examined at our hospital his temperature was 101°F; he was lethargic and mildly confused, and had meningismus. There was mild proximal leg weakness. Antibiotic drugs were discontinued. A cerebral angiogram showed mild atheroma but no aneurysm. A lumbar and thoracic metrizamide myelogram showed contrast medium flowing slowly through viscous CSF, in which clots were seen; no block or mass was identified (Fig. 3). A selective spinal angiogram performed as in Case 1 was normal. The patient was given analgesics and kept in bed for 8 days. Cultures of CSF were negative. A lumbar puncture 2 weeks after the initial hemorrhage produced xanthochromic CSF, with 1326 RBC's/cu mm, 42 WBC's/cu mm (88 lymphocytes and 12 mononuclear cells), a glucose level of 38 mg/100 ml, and a protein content of 100 mg/100 ml.

The patient's legs remained mildly weak, but the confusion and neck stiffness improved. A second thoracic and lumbar metrizamide myelogram prior to discharge was normal.

Discussion

The paroxysmal onset of severe back or neck pain, termed "le coup de poignard" or "the strike of the dagger" by Michon, is the hallmark symptom of spinal SAH. Sudden pain is frequent at the level of the lesion, and pain may radiate into one or both legs, the flanks, or occasionally into the abdomen, thus mimicking a visceral catastrophe. There is a frequently a history of previous episodes of back pain, suggesting intermittent hemorrhage. One report identified a patient with symptoms of 12 suspected hemorrhages in a 2-month period and another with 25 hemorrhagic episodes over 1½ years. Neurological findings referable to the spinal cord or cauda equina are frequently absent; however, sudden paraparesis, urinary retention, and leg numbness may occur at the onset. Meningismus, with Brudzinski's and Kernig's signs, is usually prominent soon afterward. Opisthotonic posturing has been reported in one patient. Occasionally, auscultation of a bruit over the spine signifies excessive blood flow in an underlying AVM. This sign may be enhanced by exercising the patient prior to auscultation.

Cerebral symptoms after spinal SAH may be conspicuous and confound diagnosis. The severity of intracranial symptoms is probably related to whether blood reaches the cerebral hemispheres. The more cephalad the source of bleeding, the more frequently blood reaches the brain. In one review, 80% of patients with spinal SAH had intracranial symptoms; headache was present in 70%, and mental status changes were found in 22%. Fincher noted that headache and other cranial complaints may be the first symptoms of spinal SAH and are occasionally as severe as in intracranial SAH; others have reported that headache improves rapidly after spinal SAH. One review reported loss of consciousness in 12% of patients with spinal SAH, but others have suggested that this symptom is rare or delayed in onset. Papilledema, diminished visual acuity, nystagmus, diplopia, seizure, oculomotor paresis, and tinnitus have all been observed occasionally. It
is noteworthy that subhyaloid hemorrhages have not been reported.32

The polymorphonuclear pleocytosis and hypoglycorrhrea observed in both of our patients simulated septic meningitis and led to early treatment with antibiotic therapy. We have encountered similar, but not as prominent, CSF findings in the occasional patient with cranial SAH and herpes encephalitis. These CSF findings should not delay the appropriate investigations, and antibiotic treatment is probably unnecessary if other clinical features suggest spinal SAH.

The most common cause of spinal SAH appears to be an AVM of the spinal cord.27,43 Spinal AVM’s usually arise in the thoracolumbar region and are more common in men than women.13,48 Approximately 10% of spinal AVM’s have spinal SAH as their presenting syndrome.49 Herdt, et al.9 reported that 6% of their 50 cases of angiographically documented spinal AVM’s had an associated spinal artery aneurysm. Since the only three patients who bled had coincidental aneurysms, the authors concluded that, in the setting of spinal SAH, an AVM frequently coexists with a spinal artery aneurysm. Caroscio, et al.,5 have corroborated this association.

Spinal arterial aneurysms alone have also been reported to cause spinal SAH and occur at a number of locations,4.7,9,15,21,28,30,34,36,49 most commonly the anterior spinal artery.7,15,30,34,49 The artery of Adamkiewicz,9,21 the posterior spinal artery,4,27 and the cervical radicular arteries30 are other reported sites. Aneurysm formation and subsequent spinal SAH has also been attributed to enlargement of the anterior spinal artery in aortic coarctation.7,12,30,42,52 The diagnosis of spinal artery aneurysm must be made by applying rigid angiographic criteria. In particular, the sac should arise from an artery and be visible in multiple projections during the early arterial phase, since vascular tortuosities within AVM’s may mimic aneurysmal sac formation.28

Spinal tumors, especially in the region of the cauda equina and conus medullaris, have been reported to cause spinal SAH. Ependymoma is most frequently encountered,18,31,39,43 but neurofibroma,11,24,43 meningioma,41,45 cavernous hemangioma,26 hemangioblastoma,11,35 astrocytoma,6 schwannoma,22 and meningeal sarcoma24 have been reported. Spinal endometriosis has led to repeated episodes of spinal SAH.27

A number of other entities, usually mechanical or hematological in nature, such as extreme physical exertion45 or external trauma,25 have been reported to cause spinal SAH. Coagulopathy, either iatrogenic or pathological, has been associated with spinal SAH, either spontaneously,23 after lumbar puncture,23 or following minor trauma.8 Collagen vascular disorders, such as polyarteritis nodosa,27 systemic lupus erythematosus,19 Sjogren’s syndrome,2 and pseudoxanthoma elasticum,24 have also been associated with isolated cases of spinal SAH.

The use of water-soluble myelographic contrast ma-

terial and the development of safe and accurate spinal arteriographic techniques have probably decreased the number of cases of spinal SAH with undetermined sources; however, as illustrated by the two cases presented in this report and others in the literature,27,32,42 a number remain undiagnosed even after extensive evaluation. Our first patient had a discrete and obstructive spinal SDH. The myelographic features and the nature of the fluid aspirated from the mass when compared to the CSF suggested a separate SDH. Subdural hematomas of the spinal cord are rare; one recent review stated that only 30 cases have been reported in the world literature.50 The rarity of spinal SDH contrasts with the more common spinal epidural hematoma that is a well known complication of lumbar puncture or anticoagulation therapy. There is a relatively large space, which contains an extensive venous network,5 separating the spinal epidural surface from the surrounding vertebrae, in contrast to the paucity of vessels in the spinal subdural space. Veins that bridge the subdural space intracranially and give rise to intracranial SDH’s are not present in the spinal canal.51 Blood vessels apparently do not traverse the anterior or posterior spinal subdural surfaces, but have been observed to run longitudinally along the lateral margins of the dura.17

Most spinal SDH’s have occurred in patients with coagulopathy after lumbar puncture,16,23,50 or with trauma.46,54 We are aware of only two previous patients with spontaneous spinal SDH similar to ours.1,3 Both had spinal SAH without antecedent trauma, lumbar puncture, anticoagulant drugs, or other coagulopathy. The hematoma extended from T-9 to T-11 in one patient1 and from T-8 to T-12 in the other.3 In the patient presented by Anagnostopoulos and Gortvai,3 the CSF formula was similar to that in our patient.

The distinction between spinal subarachnoid hematoma and spinal SDH has been emphasized by Diaz, et al.10 They described a discrete subarachnoid hematoma at T-6 that caused a myelopathy after a difficult lumbar puncture in an anticoagulated patient. At operation, they discovered a hematoma confined by arachnoid adhesions underneath a distinct arachnoid membrane. True subarachnoid hematomas such as this are rare, probably because the CSF tends to dilute the blood or prevent the formation of clots. The hematoma in that case10 was probably protected from dissolution by the arachnoid adhesions.

In view of the paucity of blood vessels traversing the subdural space, it has been suggested that spinal SDH’s may originate in the more vascular subarachnoid space and dissect subdurally.38,50 Rader44 postulated that the vessels traversing the subarachnoid space are subjected to rapid increases in intraluminal pressure transmitted from intrathoracic and intra-abdominal pressures. Cerebrospinal fluid pressure lags momentarily behind the intravascular pressure, thus leading to vessel rupture.44 Masdeu, et al.38 further suggested that blood is subsequently cleared from the CSF, leaving behind an
isolated SDH. This would explain the cleared subarachnoid space found at operation in the two previously reported cases,1,3 and the xanthochromic CSF with low RBC count obtained after drainage of the SDH in our patient.

The myelographic distinction between subdural, epidural, and subarachnoid spinal masses is occasionally difficult. Frager, et al.,20 described the myelographic appearance of a subarachnoid hematoma; the clot enveloped the cord, causing widening of the cord shadow and an abrupt but smooth termination of the contrast column (“capping”). Initially, the myelogram of our first patient suggested an extradural mass, but it was noted that there were multiple irregularities of the ventral aspect of the subarachnoid space, more consistent with a subdural mass, or possibly with adherent subarachnoid blood clot (Fig. 1A). The myelogram showed an elongated mass ventral to the subarachnoid space that displaced the contrast column posteriorly. The CT scan (Fig. 2) confirmed that the mass did not extend to the region of the intervertebral foramen and lateral aspects of the subarachnoid space. On the other hand, epidural masses, including epidural hematomas: 1) often extend beyond the confines of the spinal canal; 2) usually cause asymmetrical deformity of the thecal sac over shorter distances; and 3) cause displacement of nerve root sleeves.

The multiple small and medium-sized oval and irregularly elongated filling defects in the lumbar sacral subarachnoid space of the myelogram of our second patient were thought to represent blood clots. However, such filling defects in and of themselves cannot be differentiated with confidence from CSF seeding of neoplastic deposits. The fact that they were blood clots was confirmed by their disappearance on follow-up myelography.

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References

25. Harris W: Two cases of spontaneous haemorrhachis, or intrameningeal spinal haemorrhage, one cured by laminectomy. Proc R Soc Med 5:115–122, 1911
30. Hopkins CA, Wilkie FL, Voris DC: Extramedullary an-


Manuscript received March 21, 1984. Address reprint requests to: Karl W. Swann, M.D., Neurological/Neurosurgical Intensive Care Unit, Massachusetts General Hospital, Boston, Massachusetts 02114.