Subdural Hematoma of the Spinal Cord and Widespread Subarachnoid Hemorrhage Complicating Anticoagulant Therapy

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SUMMARY A patient developed paraparesis and signs of meningeal irritation spontaneously while on anticoagulant therapy. At autopsy, a subdural hematoma of the thoracic cord and evidence of widespread subarachnoid hemorrhage were found. The possible mechanism for these combined hemorrhages is discussed.

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SUBDURAL HEMATOMA of the spinal cord is an exceedingly rare pathologic entity. To date, only some 30 cases have been reported in the world literature, the single largest series being that of Edelson et al. (1974).1 These authors state that in 5 of their 7 patients examined pathologically, some subarachnoid blood was also present adjacent to the subdural hematoma. It has been rare indeed to find a spinal cord subarachnoid hematoma in association with a subdural hematoma, though sporadic reports of this have appeared.2 in the article by Masdeu et al. (1979) this was taken to imply a specific etiology for both types of intraspinal hemorrhage. Also of interest, though without unequivocal pathologic documentation, is the suggestion that disturbances of the sensorium, headache, vomiting, photophobia, and signs of generalized meningeal irritation that accompany spinal subarachnoid hemorrhage result from intracranial extension of bleeding.3

The following report documents a unique case in which a patient on long-term anticoagulant therapy developed a subdural hematoma of the low thoracic spinal cord, and, at autopsy, was discovered to have evidence of widespread old subarachnoid hemorrhage elsewhere in the cranium.

Patient Report

The patient, a 73-year-old white male, was admitted to University Hospital Aug. 22, 1978. He had a history of pulmonary embolism following gallbladder surgery in 1974, and at that time had been placed on anticoagulants. These were discontinued briefly following a trans-urethral resection of the prostate gland in April, 1978, but were given again in July, 1978, after a recurrence of thrombophlebitis and pulmonary embolism. At the time of his final hospital admission, he was on Warfarin 5 mg once a day.

Seven days prior to admission, he had gradual onset of low back pain. Despite adequate analgesia, this pain worsened over 4 days and then came to be associated with leg weakness, greater on the left side. On admission to a peripheral hospital, radiographs of the lumbosacral spine were reported as normal. On the day of transfer to University Hospital, he was noted to be confused and complaining of a headache. Nuchal rigidity was present and an attempt at lumbar puncture was not successful.

Following transfer, physical examination revealed the patient to be drowsy, confused and irritable. Petechiae and bruising were noted over the legs. The abdomen was distended and diffusely tender. Rectal tone was normal. A urinary catheter had been inserted at the peripheral hospital. There was localized tenderness over the lumbar spine. Neurologic examination revealed marked nuchal rigidity; slurring of speech; arteriolar narrowing in the fundi, but no evidence of papilledema; impaired right lateral rectus muscle function but otherwise intact cranial nerves; diminished perception of light touch in the left leg and decreased pinprick sensation in the right leg; markedly diminished power in all muscle groups in the legs, greater on the left; absent deep tendon reflexes in the arms; and downgoing plantar responses. Hemoglobin was 15.7 g/dl, white blood count 19.1 × 10⁶/l, and platelets 240 × 10⁶/l. The prothrombin time was 13 sec (control 10.5 sec) and partial thromboplastin time 33 sec (within normal limits).

Attempts at lumbar puncture were again unsuccessful, yielding bloody fluid. Radiologic examination of the spine showed degenerative changes in the cervical, thoracic, and lumbar regions. On the second hospital day, a myelogram was carried out via a cisternal puncture. This showed a complete extradural block in the region of the twelfth thoracic and first lumbar vertebrae, compatible with an extradural hematoma. Repeat myelography on the tenth hospital day, using metrizamide, showed an extradural lesion of the cord from the first to the fourth lumbar vertebrae, with a smooth contour, larger on the left, again compatible with extradural hematoma.

Dexamethasone therapy was instituted with minimal improvement in the patient's strength. His hospital course was further complicated by hematemesis, aspiration pneumonia, septicemia, bowel obstruction, and several hypotensive episodes. Signs of meningeal irritation remained prominent for several days, but began to subside by the third week.
Weakness of the lower extremities showed only modest improvement, and the left leg remained the weaker of the two. The patient remained confused. Computerized tomography of the head on 2 occasions revealed only mild cortical atrophic changes and slight ventricular dilatation. Terminally, the patient developed marked respiratory distress and septicemia unresponsive to antibiotic therapy. He died 56 days after admission to the University Hospital.

Pathologic Findings

General necropsy findings included phlebothrombosis of the left lower extremity; multiple pulmonary emboli and infarcts with massive cystic necrosis of the lungs as well as bilateral extensive pneumonia; acute vascular congestion of the liver and spleen; and bilateral arteriolonephrosclerosis.

Gross neuropathologic findings included a fresh brain weight of 1,160 grams; slight greenish brown discoloration of cerebrospinal fluid over the convexities, but otherwise clear leptomeninges; moderate diffuse cortical atrophy, especially of the frontal lobes, symmetrical on both sides of the brain; and from the base, slight greenish brown discoloration over the brainstem and adjacent orbital and medial temporal cortical surfaces. The circle of Willis was free of significant atheroma and an aneurysm was not seen despite a careful search.

Coronal sections of the cerebral hemispheres confirmed the impression of frontal atrophy, and revealed slight enlargement of the lateral and third ventricles, as well as a small (1.5 cm) old cystic infarct in the left lateral parieto-occipital cortex. Sections of the brainstem and cerebellum were normal.

The spinal cord (removed by dorsal laminectomy) showed no abnormality in the cervical region. The dura mater appeared dark and slightly distended over a distance of 14 cm in the lower thoracic and upper lumbar areas. On opening the dura, it was noted to be rather firmly adherent to an underlying thick, greenish material apparently encircling the cord and outside the subarachnoid space (fig. 1). Transverse sections of the fixed cord showed an old subdural hematoma overlying the dorsal and lateral aspects of the thoracolumbar cord (fig. 2). It varied in thickness from 1 to 3 mm and, grossly, had a layered appearance in some regions.

No abnormality of the bony vertebral canal was noted and no vascular anomaly of the cord was seen.

Microscopic Findings. In the leptomeninges overlying all regions of the cerebral hemispheres, brainstem, and cerebellum, there was extensive diffuse infiltration by macrophages containing old blood pigment, as well as old blood pigment lying free in the meninges (see fig. 6). Sections of the frontal cortex showed an occasional senile 'neuritic' plaque, and in the left parieto-occipital cortex the infarct observed grossly was seen to contain abundant compound granular corpuscles, with proliferation of fibrous astrocytes in the overlying molecular layer. The right hippocampus contained a focus of anoxic encephalopathy and scanty neuro-
fibrillary tangles were noted in both hippocampi. Sections of the deep central gray matter, brainstem, and cerebellum were unremarkable apart from the meningeal findings described above.

In cross-sections of the cervical and upper thoracic cord, there were large numbers of macrophages bearing old blood pigment in the meninges. There was secondary degeneration of the gracile fasciculi. In the lower thoracic and uppermost lumbar regions, a large subdural hematoma (fig. 3) and less extensive old subarachnoid hemorrhage were seen. The hematoma covered the posterior and lateral aspects of the cord. It contained regions of old hemorrhage, including hemosiderin-laden macrophages and hematoidin (fig. 4). There was organization of portions of the hematoma adjacent to the cord and more recent hemorrhage superimposed on this. Extensive deposits of old blood pigment surrounded and infiltrated the dorsal roots (figs. 5, 6). There was degeneration of the gracile fasciculi, greater on the left side, presumed to be secondary to the involvement of dorsal roots by the hematoma.

Discussion

At the time of the most extensive recent review of subdural hematoma of the spinal cord by Edelson (1976), only about 25 cases not associated with major trauma had been reported. Most had occurred in patients with underlying hematologic disorders (e.g., classic hemophilia, leukemia) or in thrombocytopenic patients following lumbar puncture. But other associations were with lumbar puncture alone, minor trauma, and anticoagulant therapy (two patients). The well-known patient of Cloward and Yuhl (1955) involved a combined subarachnoid, subdural and epidural hematoma of the thoracolumbar cord in a patient on dicumarol therapy.

Since the review by Edelson, a handful of new patients has come to light. These include thoracolumbar subdural hematoma in a 56-year-old man with ankylosing spondylitis following minor trauma; thoracolumbar hematoma in a 23-year-old woman with meningococcal septicemia, treated with anticoagulants; and further examples of lesions related to lumbar puncture or occurring spontaneously. Unusual intraspinal (though not strictly subdural) hematomas, some related to previous surgical intervention, have received attention. Though most cases have involved the middle to low thoracic region, a phenomenon for which there exists no adequate explanation, recently a dorsal hematoma overlying the cervical cord has been described.

Barnett (1969) has noted that subarachnoid hemorrhage resulting from rupture of a berry aneurysm in the circle of Willis may extend into the subdural space. Although this happens infrequently—in 8.8% of cases in his large autopsy series—it may result in subdural hematoma around the spinal cord, as shown by the remarkable example illustrated in his paper.

Epidural hematoma of the spinal cord, by no means a frequent occurrence, is, nevertheless, more commonly encountered, with approximately 200 examples now in the literature. The anatomical reasons for such a disproportion in numbers have been thoroughly dis-

Figure 3. Cross-section of the thoracic region of spinal cord. The hematoma overlies the dorsolateral aspects of the cord and is partly recent (dark arrow) and partly old (white arrow). It compresses the dorsal roots. H and E/Luxol fast blue, X 12.6.
FIGURE 4. Dorsal roots in the thoracic region enveloped in fibrous tissue containing deposits of hemosiderin (arrows) and extending into the subarachnoid space. Perl's Prussian blue, × 31.5.

cussed,1-15 and probably relate to the relatively larger and more vascular epidural space occupied by loose fatty tissue and Batson's vertebral plexus. Unusual recent examples of spinal extradural hematoma include one presenting without pain in a patient on anticoagulant treatment;16 a hematoma secondary to aspirin-induced prolonged bleeding;17 a spontaneously remitting hematoma, also in a patient on anticoagulant therapy;18 and a post-lumbar puncture hematoma in a patient with liver disease.19 The association with deranged hemostasis and abnormal clotting mechanisms is obvious from these examples.

FIGURE 5. Closeup of an area in figure 4. Note hemosiderin pigment in fibrous tissue (white arrow) and on the outer part of the roots (dark arrows). Perl's Prussian blue, × 126.
Subarachnoid hemorrhage in the spinal cord rarely presents as a symptomatic solid hematoma. This is postulated to result from the 'diluting' effect of cerebrospinal fluid. However, several authors have shown that this 'diluting' effect does not always function and that indeed subarachnoid hemorrhage may result in a space-occupying lesion, e.g. in the cauda equina.

Anticoagulant therapy is a known predisposing factor for intracranial subdural hematoma. Wiener and Nathanson (1962) reported that 6 of 50 consecutive cases (i.e. 12%) treated at the Mount Sinai Hospital had been on anticoagulants. Sreerama et al. (1973) have described 11 patients on anticoagulant therapy presenting with either intracranial subdural hematoma or spinal epidural hematoma, comprising over a third of all subacute and chronic subdural hematomas seen over a 3 year period. Bret et al. (1976) have stated that previous anticoagulant treatment has a role in causing 4.8% to 14% of subdural hematomas. Lizuka (1972) has described 12 intracranial (mainly subdural) and 2 spinal extradural hematomas associated with anticoagulant therapy. Not surprisingly, anticoagulated patients undergoing subdural or epidural anesthesia are at increased risk for developing hematomas in the epidural or subdural compartment around the cord.

The most logical conclusion as regards the present patient is that the intraspinal hematoma had its origin in the subarachnoid space, with intracranial extension and dissection into the subdural space. There, it remained as a solid mass producing cord compression and causing the patient's paraparesis.

Masdeu et al. (1979) have shown in at least one patient that subarachnoid hematoma of the cauda equina had its origin in a radicular vessel that was lacerated at the time of lumbar puncture. They have further postulated that many subdural hematomas of the cord may have their source in the subarachnoid space. The blood is then thought to be cleared from the cerebrospinal fluid, leaving hematoma in the more "sealed" subdural space to become organized. This may explain why, when autopsy is carried out weeks or months after hemorrhages related to lumbar puncture, little trace of subarachnoid blood remains, while a subdural clot may be found.

Symptoms referable to spinal cord compression in our patient clearly antedated attempts at lumbar puncture. In view of the absence of pathologic evidence for coarctation of the aorta, polyarteritis nodosa, a spinal angioma, aneurysm or neoplasm — all known causes of spinal subarachnoid hemorrhage — we must conclude that it and the subsequent subdural hematoma were related to anticoagulant therapy. This constitutes a most unusual set of complications of such therapy.

References
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