Spontaneous cervical epidural hematoma

A consideration of etiology

ROBERT M. BEATTY, M.D., AND KEN R. WINSTON, M.D.

Department of Surgery (Neurosurgery), Brigham and Women’s Hospital, and Department of Neurosurgery, Children’s Hospital and the Harvard Medical School, Boston, Massachusetts

The medical literature relating to spontaneous cervical epidural hematoma has been reviewed, and two new cases have been added. In total, 43 cases have been analyzed in detail, and the authors present some new hypotheses as to the pathophysiology of this disorder.

KEY WORDS • cervical epidural hematoma • spinal epidural artery • cervical spine

In this paper we review the 41 cases of acute spontaneous cervical epidural hematoma that have previously been reported in the literature, and add two new cases of our own. The source of bleeding in spontaneous epidural hematoma has never been clear, but has been assumed to be venous. The reason for this assumption, although never defended critically, was probably based upon the unstated opinion that there are few, if any, arteries in the normal epidural space. In 1977, Crock and Yoshizawa published photographs of beautifully dissected specimens showing an organized arterial system in the epidural space (Fig. 5). Using the available evidence, we have addressed the problem of why and from where spontaneous hemorrhage occurs in the cervical epidural space. We have suggested that our conclusions can logically be applied to the entire spinal epidural space.

Case Reports

Case 1

This 65-year-old woman experienced an acute onset of severe pain in the back of her neck with radiation into both upper extremities. Within minutes her left upper extremity became weak, and over the next 12 hours she became hemiplegic. She had been taking naproxen for headache for 1 month.

Examination. The patient had a left Horner’s syndrome, left hemiplegia with hypalgesia, and thermohyposesthesia in the right lower extremity and trunk. There was diminished perception of position and pallanesthesia in both lower extremities. Deep-tendon reflexes were hyperactive on the left with a left Babinski reflex. She had several unexplained bruises on her upper extremities.

Bleeding time was 10.5 minutes (normal 9 minutes), platelet count 200,000, prothrombin time 11.6 minutes (normal 12.8 minutes), and partial thromboplastin time 27.4 minutes. Computerized tomography demonstrated blood in the epidural space from C-2 to C-7 with displacement of the spinal cord and dura to the right (Fig. 1).

Fig. 1. Case 1. A representative computerized tomographic section through the cervical region. The arrows outline a crescent-shaped mass displacing the spinal cord.
Fig. 2. Case 2. A representative computerized tomographic section through the cervical region after metrizamide has been instilled into the thecal sac. A mass with the density of blood is seen displacing the spinal cord.

Operation. A left hemilaminectomy from C-3 to C-6 revealed a hematoma covered by a net of epidural veins. The veins had to be incised to allow removal of the clot. On pathological examination of the clot, no abnormal vessels and no neoplastic cells were found. Six months later, the patient had mild residual weakness of the left deltoid muscle and no other neurological abnormalities.

Case 2

This 37-year-old man acutely developed posterior cervical and interscapular pain, which radiated into both upper extremities. Over the next 3 hours, he became triparetic (right upper and both lower extremities). Bowel and bladder function were normal. He had an 8-year history of systemic lupus erythematosus manifested by mild renal impairment and cutaneous vasculitis, and was taking 20 mg/day of prednisone. He was admitted with a working diagnosis of vasculitis of the cervical cord and the prednisone was increased to 60 mg/day. His strength improved.

Examination. Physical examination 48 hours later revealed a mild right hemiparesis involving the upper extremity more than the lower, mild hypesthesia in the right C-6 dermatome, intact perception of joint position and vibration, symmetrical deep-tendon reflexes, and flexor plantar responses. The skin on the face and upper extremities appeared abnormally thin with a readily visible capillary pattern. A metrizamide myelogram followed by CT scanning demonstrated an extradural intraspinal mass of blood density displacing the dura to the left (Fig. 2).

Operation. A right hemilaminectomy from C-3 to C-7 revealed an epidural clot covered by a network of veins. As the hematoma was removed, some active bleeding from the lateral margin of the spinal canal, particularly near the intervertebral foramina, required coagulation. The clot was adherent to the dura. On pathological examination, no abnormal vessels or neoplastic cells were found. Except for a mild weakness of the right deltoid muscle, the patient's neurological examination was normal 6 months later.

Discussion

Acute spontaneous cervical epidural hematoma is uniformly fatal if not surgically evacuated (all six reported patients without surgery have died). The first surgically treated patient was reported in 1946 by Ver Bruggen, and this patient recovered. Of the 43 patients (41 plus our two cases) operated on for spontaneous cervical epidural hematoma, seven have died, giving an overall mortality rate of 16% (95% c.i. = 7% to 31%). Twenty-one patients developed paralysis within 3 hours of the first symptom, and 19 of these 21 were operated on. Four of these 19 patients died, giving a treatment mortality rate of 21% (95% c.i. = 6% to 46%) for this rapidly deteriorating group. Respiratory failure was the most common cause of death. Better results from treatment can probably be achieved only through earlier surgical intervention.

Spinal epidural hemorrhage has been associated with coagulopathy, arteriovenous malformation (AVM), and neoplasia. We have defined the term “spontaneous” as meaning “without identified etiology.” This definition excludes hemorrhages caused by hemophilia, neoplasia, AVM, trauma, or postoperative complications, and also patients who had received anticoagulation therapy. We have included patients with a history of very minor trauma (which would not be expected to cause significant bleeding), patients taking an occasional aspirin or other medication (not for anticoagulation), and those with a minor prolongation of bleeding time. Our first patient had a mildly elevated bleeding time, possibly related to the ingestion of naproxen, and our second patient may have had capillary fragility secondary to systemic lupus erythematosus and the chronic use of steroids. Ainslie also described a patient with spontaneous cervical epidural hematoma and capillary fragility.

Patients with spontaneous cervical epidural hematomas most commonly present with sudden cervical or interscapular pain, often following minimal physical exertion. Radicular pain into the upper extremities is usual. Typically the increasing compression of the spinal cord leads to paralysis within minutes to hours after the onset of symptoms but, less commonly, the course can progress slowly for days. The clinical features of spontaneous cervical epidural hematoma can resemble those of an acutely ruptured cervical disc, epidural neoplasia, transverse myelitis, dissecting aortic aneurysm, congenital cysts, spondylitis, or infection such as epidural abscess. Our two patients and the report of
Spontaneous cervical epidural hematoma

Post, et al., demonstrate that CT scanning can reliably identify spinal epidural hematomas.

Sixty-five percent of reported spontaneous cervical epidural hematomas have been in females, and this entity has occurred in patients as young as 21 months and as old as 79 years (Fig. 3). The hematoma spanned three to four vertebrae in 49% of patients (Fig. 4 left), and extended from C-3 to S-1 in one case. Regardless of the total length of the hematoma, it involved lower cervical levels (C5-7) in 95% of cases, and upper levels (C1-4) in only 44%. Many of the hematomas in the lower cervical region also involved thoracic levels (Fig. 4 right). Of patients with hematomas involving levels above C-5, 37% have died (95% c.i. = 16% to 62%). Only two patients have been reported with spontaneous epidural hematomas confined above the C-5 vertebral level, and neither survived. Perhaps other patients with high cervical hematomas did not survive long enough to be diagnosed and reported.

Venous pressure in the epidural veins is low in the cervical region if a person is upright and relaxed, but, regardless of position and degree of relaxation, the pressure in these veins is less than intrathecal pressure at the same level. Therefore, hemorrhage from an epidural vein would not significantly compress the spinal cord. This is confirmed by the common observation that the normally expanded dural sac may tamponade epidural venous bleeding encountered during surgical procedures. Let us consider then the possibility that the bleeding is arterial. This idea is supported by the rapidity of development of cord compression and by the location of the hematoma within the epidural space. Scott, et al., reported a patient who became paraplegic 30 minutes after onset of pain, and Tsai, et al., described a patient who became hemiparetic “minutes” after onset of pain that began while urinating. The rapidity with which myelopathy often follows the onset of symptoms in the spontaneous cases is similar to that of cases of AVM’s, and in the latter the pressure within the bleeding vessel is almost certainly greater than normal venous pressure — perhaps arterial.

The normal and pathological anatomy is also consistent with the hypothesis that the hemorrhage originates from an arterial source. The blood supply for the cervical meninges comes from branches of the ascending and deep cervical arteries. These branches run along the posterior superior borders of the cervical nerve.
roots. Just inside the spinal canal, they leave the nerve and bifurcate to form a longitudinal channel in the epidural space (Fig. 5). From this channel and midway between adjacent nerve roots, free bridging vessels pass dorsally and medially to the side of the dural sac and then run transversely toward the midline where they anastomose with their counterpart from the opposite side. The free or bridging arteries that run from the nerve sheath to make up the laterally positioned longitudinal channel and from this longitudinal channel to the arterial plexus on the spinal dura appear to be anatomically vulnerable to mechanical disruption. The spinal epidural veins from anterior and posterior internal venous plexuses form an arcuate pattern overlying the epidural arteries.

We believe that hemorrhage from one of the free epidural arteries is a likely source of acute spontaneous cervical epidural hemorrhage. The pressure of arterial bleeding is sufficient to tightly compress the spinal cord and such bleeding would not be tamponaded by the counterpressure of the thecal sac. The hematoma in each of our patients was predominantly lateral, and surrounded by a net of epidural veins which had to be incised to remove the clot. Fourteen (33%) of 43 reported patients had radicular pain as a first symptom and this suggests that the hemorrhage began in close approximation to a nerve root. The typical sequence of development of the syndrome (pain first in the neck, then in the shoulder, then the arm, followed by evidence of myelopathy) is identical to that of a large ruptured cervical disc but with an accelerated time scale. Proof of the arterial source which we propose would require either angiographic documentation (a difficult if not impossible challenge) or direct observation at operation. A bleeding epidural artery probably would thrombose long before it was surgically exposed and therefore would not be detected. Lowrey described a small artery at L-5 which began to bleed as an epidural clot was removed. The artery was clipped and postoperative angiography revealed no evidence of an AVM.

The cause of the spontaneous rupture of a small epidural artery remains a mystery, but perhaps a clue lies in Fig. 4 right, which demonstrates that these arteries, which enter the spinal canal, leave the nerve sheaths and travel as free vessels in the epidural space to form a laterally placed longitudinal channel (LC), and from this channel branches travel medially to the dura and form a plexus in the surface of the dura. Drawings were made from photographs of dissected specimens photographed by Crock and Yoshizawa.

---

**Fig. 5.** Epidural arteries and their sources are shown as seen from a posterior view (left) and from a left posterolateral view (right). The epidural veins which would envelop most of the epidural arteries are not shown. The cervical epidural arteries receive blood from the subclavian artery (SCL) via two routes. The deep cervical artery (DC) arises from the costocervical trunk (CCT) and usually enters the spinal canal on the surface of the C-7 or C-8 root. Branches of the ascending cervical arteries (AC), which arise from the thyrocervical trunk (TCT) via the inferior thyroid artery (IT) or directly from the thyrocervical trunk, enter the spinal canal on the surface of the cervical nerves above the eighth nerve. These arteries, which enter the spinal canal, leave the nerve sheaths and travel as free vessels in the epidural space to form a laterally placed longitudinal channel (LC), and from this channel branches travel medially to the dura and form a plexus in the surface of the dura. Drawings were made from photographs of dissected specimens photographed by Crock and Yoshizawa.
Spontaneous cervical epidural hematoma

spontaneous hemorrhages most commonly involve the lower cervical region. This portion of the cervical spine is most commonly affected by cervical disc disease and osteoarthritids, and is the region most active in non-rotatory movements and the region where the brachial plex                        us arises. It is normal for the spinal nerves with surrounding dural sheaths to move within the neural foramina as the extremity moves.13 Nerves in the lower cervical region might be expected to have a greater normal excursion than upper cervical nerves. Furthermore, it is reasonable to think that any stretching of the upper extremities, particularly if symmetrical, could apply traction to the spinal epidural arterial system. Therefore, a minor aberration in the local vascular, bone, or ligamentous anatomy, or something unusual about the movement itself could be critical and lead to stretching these free arteries beyond their tolerable limits. The logical result would be a hemorrhage beginning laterally in the epidural space and deep to the venous plexus and spreading longitudinally. Since spontaneous cervical hemorrhage is a rare malady, the arterial system must be constructed to tolerate normal movements, and the photographs of Crock and Yoshizawa16 seem to demonstrate redundancy in the free arteries. Of course, we cannot discern the amount of artifact in these photographs related to fixation and dissection, and we have no knowledge of individual variation or relationship to age. The mechanical explanation finds support in the observation that many of the patients were involved in minor motor activity when the first symptom appeared.

The hypotheses as to the origin and pathophysiology which we have derived from consideration of spontaneous cervical epidural hematoma can be extrapolated directly to spontaneous epidural hematomas at all spinal levels. Perhaps even the epidural hemorrhages related to anticoagulant therapy or lumbar puncture can be better understood as arterial hemorrhages.

References

22. Jackson R: Case of spinal apoplexy. Lancet 2:5-6, 1869
L’hématome extra dural rachidien spontané. Neurochirurgie 7:298–313, 1961 (see p 298)


Manuscript received December 20, 1983.
Address reprint requests to: Ken R. Winston, M.D., 300 Longwood Avenue, Boston, Massachusetts 02115.