Acute spinal cord compression by subarachnoid and subdural hematoma occurring in association with brachial plexus avulsion

Case report

NEVILLE A. RUSSELL, M.D., F.R.C.S.(C), AND MICHAEL A. MANGAN, M.D., B.CH.

Divisions of Neurosurgery and Diagnostic Radiology, St. John's General Hospital, Health Science Complex and Memorial University of Newfoundland, St. John's, Newfoundland, Canada

The authors report a case of acute spinal cord compression caused by a subarachnoid and subdural hematoma. This occurred following traumatic brachial plexus avulsion. It is believed to be the first such case recorded.

KEY WORDS • subdural hematoma • spinal cord compression • brachial plexus avulsion

TRAUMATIC avulsion of the roots of the brachial plexus from the spinal cord was first recognized by Flaubert in 1827. Since then the clinical, radiographic, and pathological features have been well described.

Hematomas within the spinal canal, although well documented, are uncommon. They may occur in the epidural, subdural, or subarachnoid spaces, or within the spinal cord. They may occur without obvious cause, or may be due to a ruptured vascular malformation, or anticoagulant therapy. They have been reported following both major and minor spinal trauma. An extensive review of the literature reveals no record of significant intraspinal hemorrhage associated with brachial plexus avulsion. We believe that the case presented below is the first report of such an entity; an intraspinal hematoma of sufficient magnitude to cause acute spinal cord compression, occurring in association with brachial plexus avulsion.

Case Report

This 19-year-old seaman was admitted to the St. John's General Hospital on June 27, 1973. He had been injured on board ship approximately 28 hours previously. He was struck by a towing cable, which had parted while under high tension, rendering him unconscious. Further information as to the mechanism of injury was unavailable.

Examination. He was stuporous, arousing to the shouted voice. There was a contusion of his mid-forehead and a laceration of his upper lip. Weak movement was present in his right arm only; the deep tendon reflexes were present but hypoactive in this limb. His left arm and both legs were flaccid and areflexic, exhibiting no response to any stimulus. Pinprick was not perceived below the clavicles, except for the saddle area in which sensation was spared. X-ray films of the entire spine were negative. Lumbar puncture at myelography showed grossly bloody cerebrospinal fluid (CSF). The myelogram showed a normal spread of the dye column to the level of T-9, where a partial obstruction was encountered. Cephalad to this, the cord was displaced from left to right. As the dye reached the lower cervical spine, extravasation occurred along the nerve root sleeves on the left at the levels of C-7 and T-1. A complete arrest of the dye flow occurred at C-6 (Fig. 1 left).

A cisternal puncture was then performed. The
Acute cord compression by hematoma

spinal fluid here appeared even more heavily blood-stained than in the lumbar region. A small amount of dye was injected and shown to loculate in the posterior aspect of the cisterna magna. There was some flow from this area into the fourth ventricle (Fig. 1 right).

Operation. Laminctomy was performed from C-1 to C-6. The dura was tense, bluish and nonpulsating. When the dura was opened, a large hematoma was discovered covering the dorsal aspect of the cord throughout the length of the laminectomy. It lay primarily within the subarachnoid space, but at the C-5 region the arachnoid had ruptured, and a portion of the clot lay in the subdural space. A large amount of clot was present in the cisterna magna, extending upward between the cerebellar tonsils. The clot was removed in its entirety with suction and gentle saline irrigation. Cord pulsation returned. Examination of the cord revealed that both anterior and posterior rootlets of C-5, C-6, and C-7 had been avulsed. The dura was left open, the area covered with Gelfoam, and the usual layered wound closure effected without drainage.

Postoperative Course. Ten days later, just before he was transferred to a hospital in his own country, he was alert and orientated. The power in the right arm was still as described on initial examination. Voluntary movement was present in both legs, although the muscle power was not sufficient to overcome gravity. The knee and ankle reflexes were weak but present bilaterally. The left arm remained flaccid and areflexic. The sensation of pinprick was present over the legs, trunk, and right arm, but was normal only in the saddle area and above the clavicles.

The patient was lost to follow-up review after transfer.

Discussion

A number of authors have mentioned the presence of blood-stained CSF in brachial plexus avul-
This has been an incidental finding noted at lumbar puncture for myelography. Taylor reviewed the subject of brachial plexus avulsion in detail and reported on seven cases. In one case, the CSF was “blood tinged and a complete manometric block existed.” There was no clinical evidence of spinal cord compression and the CSF cleared after 11 days. Laminectomy at 26 days showed only the avulsed rootlets. Yeoman reported the myelographic findings in 60 cases of brachial plexus injury. He noted that the bleeding occurred in the subarachnoid space, that is, at the site of attachment to the spinal cord. Lister pointed out that lumbar puncture would sometimes yield blood-tinged CSF during the first 5 or 6 weeks after injury. Suchodoletz and Olscher studied the CSF in 25 patients with brachial plexus injury. Xanthochromia and hemosiderin macrophages were present as long as 2 months after injury in the 12 patients with permanent neurological deficit. They believed that blood in the CSF correlated well with brachial plexus avulsion. One of Yeoman’s patients developed neurological signs consistent with spinal cord compression 7 years after the brachial plexus injury. However, further investigation resulted in a diagnosis of multiple sclerosis. The only recorded instance of late cord injury after brachial plexus avulsion was reported by Penfield. This event occurred some 35 years after the injury, and was caused by fibrous adhesions drawing the cord into the mouth of a traumatic meningocele.

Avulsion of cervical nerve roots can be a result of a variety of injuries, all of which place tension upon these roots. If the root is stretched beyond its elastic limits, structural failure occurs where it is weakest, that is, at the site of attachment to the spinal cord. Blood supply reaches the cervical spinal cord by way of radicular arteries that arise from the vertebral and subclavian arteries and travel to the cord on some of the anterior and posterior nerve roots. Trauma sufficient to tear spinal roots from the spinal cord must also tear these arteries. Brachial plexus avulsion is not usually associated with ischemic spinal cord damage, probably because adequate anastomoses exist from adjacent levels. The vessels are small (outside diameter 0.2 mm or less). This is probably one reason for the absence of significant intraspinal hemorrhage with brachial plexus avulsion. Another reason is that the blood in the subarachnoid space usually does not clot, probably because of great dilution by the spinal fluid.

The operative findings in the case reported indicate that the bleeding occurred in the subarachnoid space, and ultimately ruptured through the arachnoid membrane into the subdural space. The large amount of blood was sufficient to cause cord compression resulting in quadripareis. It is well recognized that neurological damage caused by injuries to the spine is variable, and may not correspond to the apparent severity of the bone injury. The forces causing the injury in the patient reported were probably considerable. The resulting violent movements of the cervical spine may have contributed to both the brachial plexus avulsion and the intraspinal hemorrhage.

References

22. Jellinger K: Traumatic vascular disease of the spinal cord, in Vinken PJ, Bruyn GW (eds): Handbook of...
Acute cord compression by hematoma


Address reprint requests to: Neville A. Russell, M.D., F.R.C.S.(C), Suite 201, Wedgewood Medical Centre, 12 Gleneyre Street, Wedgewood Park, St. John's, Newfoundland, Canada A1A 2M7.