CASE REPORTS

EXTRADURAL HEMATOMA OF THE SPINAL CANAL

REPORT OF A CASE

POMEROY NICHOLS, JR., M.D.,* AND L. O. J. MANGANIELLO, M.D.*

Department of Neurosurgery, Medical College of Georgia, Augusta, Georgia

(Received for publication April 30, 1956)

Since Baine in 1897 first described extradural hemorrhage of the spinal canal, a total of 14 cases have been found in a review of the English literature. These cases have been well documented, individually, by Sadka.2

A positive etiology remains obscure, although in 10 of the 15 known cases, trauma seems to have played a role. The most logical explanation, and this is supported by the present case, is that an epidural venous abnormality or hemangioma is present which may rupture either spontaneously or traumatically.

It is felt that the diagnosis and treatment of these lesions may be difficult unless the progressive nature of the lesion is determined. It is obvious that the earlier surgery is instituted, the better the chance for recovery.

Guthkelch,1 in reporting his 8 cases of extradural hemangiomas, had 1 patient with a remitting paraplegia. In the case presented below, there was also a remitting paraplegia.

REPORT OF CASE

A 15-year-old white male was referred by Dr. Wallace McNair of Aiken, S.C. on Feb. 1, 1956. His past history was significant only in that he had been having intermittent attacks of pain in the chest bilaterally for the past 7 months. This pain was not too bothersome, and he did not have to seek medical help. On Dec. 27, 1955, he was struck on the back of his head by the handle of a fertilizer spreader. This blow was sufficient to knock him down, but he did not have any sequelae from the accident itself. However, he noticed that the pains in his chest were more severe after the blow on the head. When the pain came it lasted approximately 5 hours and then would disappear. On the morning of admission to the hospital, he awoke with severe pain in his chest and marked weakness in his legs. He sought medical attention for this and was quickly referred to the University Hospital in Augusta, Georgia.

On arrival at the University Hospital, the patient stated that the weakness in his legs had passed away and that the pain in the chest was much improved.

* Medical Arts Building, Augusta, Georgia.
Examination. A mild symmetrical weakness in both legs was noted. The Babinski sign was positive bilaterally. There were no sensory changes and there were no posterior column signs. A complete blood count and urinalysis were normal. Temperature, pulse rate, and respiration were normal. Roentgenograms of the cervical and thoracic spine were normal. A lumbar puncture was performed which showed a spinal fluid pressure of 150 mm. of water. The fluid was clear and colorless. The Queckenstedt test was mildly suggestive of a block. The spinal fluid contained 3 white cells, a trace of Pandy, and a total protein of 28 mg. per cent.

Course. For the next 3 days, the patient showed gradual improvement, but continued to have a positive Babinski sign bilaterally.

At 4:00 P.M. on Feb. 3, 1956, severe pain in the chest again developed in the same distribution as he had been having for the past 7 months. This was accompanied by a rapid onset of paraplegia. There was a sensory level at T4. All modalities of spinal cord function were absent below this level.

Lumbar puncture revealed a pressure of 160 mm. of water. The Queckenstedt test showed a complete block. The spinal fluid contained 9 white cells, with a negative Pandy and a total protein of 40 mg. per cent. A myelogram revealed a complete block at the level of the 1st thoracic interspace (Fig. 1). The patient was taken immediately to the operating room.

Operation. A laminectomy was performed, starting at T1 and continuing up to C6 (Fig. 2). As the lamina was removed from T1, a dark bluish semisolid clot extruded under pressure. This hematoma was found to extend up to C6. While removing the hematoma, large tortuous veins were noted which bled freely. The mass was removed and hemostasis was secured. The dura mater was then opened and the spinal cord was visualized. The spinal cord was seen to be indented the length of the hematoma. This appeared to be of long standing. Complete

Fig. 2. Operative sketch showing the extradural hematoma and the compression of the cord beneath it.
inspection of the spinal cord and spinal canal failed to reveal any other evidence of disease.

Pathologic Examination. The hematoma grossly consisted of blood enmeshed in extradural fat. The majority of the specimen was hematoma, but there was definitely a tangled mass of veins entering the clot. Microscopic preparation showed an organizing hematoma contained within fatty tissue, and a scattering of veins which were normal in appearance (Fig. 3).

Course. The patient made a rapid and uneventful postoperative recovery with complete return of spinal cord function. He was dismissed from the hospital ambulatory on the 9th postoperative day. Complete laboratory studies for any disturbance in bleeding and clotting mechanisms disclosed no abnormalities.

SUMMARY

It is felt that extradural hematoma should be considered in the differential diagnosis in any case of acute cord compression with absence of inflammatory signs or symptoms, particularly if there is a history of an injury.

REFERENCES