PARAPLEGIA CAUSED BY SPONTANEOUS SPINAL EPIDURAL HEMORRHAGE*

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PARAPLEGIA resulting from spontaneous spinal epidural hemorrhage is rarely encountered. Review of the literature from the first reported case of Bain1 in 1897 to the case of Svien, Adson, and Dodge9 in 1950 reveals a total of 12 cases. Ver Brugghen10 reviewed the first 7 of these and pointed out the paucity of information on spinal epidural hemorrhage to be found in current textbooks. He emphasized the importance of prompt recognition of spinal epidural hemorrhage and recorded the only case in which early surgical removal of an epidural blood clot from the cervical region resulted in prompt recovery from quadriplegia. Kaplan and Denker6 in 1949 summarized the previous literature and reported 2 cases of their own in which the lesions were treated surgically. It was their belief that spinal epidural bleeding was of venous origin caused by a rupture in the weak, vascular wall of a pre-existing abnormal epidural venous plexus. They admitted the lack of convincing pathological evidence. Chavany et al.2 added another case of lumbar epidural hemorrhage. Svien and co-workers9 called attention to the clinical similarity of epidural hemorrhage and the lumbar disk syndrome. Our own recent experiences corroborate this.

Spontaneous spinal epidural hemorrhage is poorly understood. We have little doubt that undiagnosed cases occur frequently. Neurologic findings may be transient and bizarre, depending upon the amount of hemorrhage. When the hemorrhage is small, little or no neurologic changes ensue, and the usual symptomatic treatment of back pain suffices. There is presumptive evidence that recurrent episodes of back pain may result from repeated spinal epidural hemorrhage. When massive hemorrhage occurs, marked motor and sensory impairment follow. Only by recognition of the pathology and prompt surgical removal of the clot can satisfactory neurologic recovery be anticipated. Recently, we have had the opportunity to treat 4 patients with massive spontaneous spinal epidural hemorrhage. Our purpose of reporting these cases is to re-emphasize this grave neurologic lesion and to

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* The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration. Case 4 was reviewed in the Veterans Administration and published with the approval of the Chief Medical Director.
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encourage others to record their experiences. It is our belief that only by the reporting of individual cases will a better comprehension of the etiology and treatment occur. Furthermore, we wish to emphasize that spontaneous spinal epidural hemorrhage is apparently more common than we have heretofore believed.

Case 1. #63810. F.G.G., a 24-year-old white apprentice printer, was seen by one of us (A.C.J.) Dec. 6, 1951. According to his physician, Dr. T. F. Walker, Jr., of Great Falls, Montana, he had been in good health until the previous evening when he experienced severe, stabbing pain in the upper thoracic spine about 1 hour after heavy lifting. Flexion of the trunk aggravated the condition. Later during the night, pain radiated anteriorly in a distribution more or less corresponding to the 5th thoracic dermatome bilaterally. An analgesic was given. By morning he experienced numbness and weakness of the legs, progressing to a complete paraplegia with a transverse sensory level in the upper thoracic region.

Examination, 18 hours after onset of symptoms. He was a well-developed and nourished male, alert, oriented, and cooperative. Normal motor power was present in the upper extremities. The lower extremities were flaccid, although the deep reflexes were present, equal, and active to slightly increased. Voluntary motion had been affected in the right lower extremity initially, and on this side there was a Babinski sign. On the left this sign was absent. Deep sensation on pinching the small toe was still present on the right side but absent on the left. There was a complete loss of superficial sensation as high as the 5th thoracic dermatome on the right and the 6th on the left.

The clinical impression was that this patient had an acute transverse myelopathy, apparently originating from the right side of the spinal cord at about T5 cord level (T3 vertebral level). The two most likely possibilities appeared to be an acute extrusion of the nucleus pulposus or a spinal epidural hematoma. In either event immediate decompressive laminectomy was indicated. Because of the rarity of herniation of the nucleus pulposus at the level indicated in this case, some doubt was cast on this possibility, though it was also recognized that a spinal epidural hematoma is a very rare occurrence, at least in the absence of known specific trauma.

Roentgenograms of the upper dorsal and lower cervical spine revealed no evidence of fracture, dislocation, or erosion.

Spinal puncture at the 4th lumbar level yielded normal-appearing fluid. There was a complete block to the Queckenstedt test. One cc. of pantopaque (ethyl iodo-phenylundecylate) was inserted into the lumbar sac. With the fluoroscopic table tilted down to approximately 60° from the horizontal, there was evidence of a complete block with a concave "tumor cap" type of defect corresponding with the lower level of the 3rd thoracic vertebral body. A small specimen of CSF contained 50 mg. per cent of protein and no cells.

Operation. The patient was immediately taken to surgery, where under endotracheal anesthesia a complete laminectomy of the 2nd through the 4th thoracic vertebrae was carried out (A.C.J.). As soon as the removal of the laminae was begun a moderately large, dark, "currant jelly" epidural clot was disclosed lying dorsal over the dural sac and markedly compressing it to a depth of about 1 cm. at its deepest portion. After removing a portion of this for pathologic study, the remaining hematoma was completely removed by suction and irrigation. Complete relief of the block was immediately evident by the development of normal pulsations of the dura
mater. Intradural exploration disclosed no gross abnormality and a #8 F. catheter could be readily passed 10 to 15 cm. in either caudal or cephalic direction without encountering an obstruction. The dura mater was closed tightly with a continuous silk suture and the laminal defect was filled with Gelfoam to obliterate the dead space.

Course. There was rapid and progressive neurologic recovery. On the 1st postoperative day a flicker of voluntary contraction of the left quadriceps with extensor movements at the knee joint was noted. Good deep and superficial sensation and sensory localization were present in both legs. On the 2nd day there was movement of the toes of the left foot and physiotherapy was begun. Voluntary contractions of both flexors and extensors at the left hip, knee, and ankle were present on the 3rd day. Slight extensor movements were present at the right knee, ankle, and toes. By the 5th postoperative day he was up in a chair with continued progressive improvement in motor power in both lower extremities, and on the 6th day nearly normal motor power was present in the left lower extremity and only slightly reduced motor power in the right extremity. During the 1st postoperative week, voluntary emptying of the bladder was not possible, and an indwelling catheter was used most of the time. Because of the high urinary residuals of as much as 800 cc., he was put on a regimen of Prostigmine methylsulfate 0.0005 (1/120 gr.) with Doryl (carbamino-choline chloride) 0.00025 (1/260 gr.) subcutaneously before attempting to void. Residual urine steadily decreased.

Subsequent follow-up revealed continued recovery. On Feb. 19, 1952, he was practically symptom free, although he stated his legs felt “a little heavy” when running. He was free from any subjective limitation with usual physical activity. The only objective neurologic findings consisted of a slight briskness in the knee and ankle reflexes, particularly the latter, and these were somewhat greater on the left side. There was no ankle clonus, and an inconstant and equivocal Babinski sign was obtained on the right side only. On re-examination on Oct. 23, 1952, he was asymptomatic and neurologically normal.

Summary. An acute onset of severe back pain followed by progressive loss of function of lower extremities was found to be caused by an expanding epidural hemorrhage. Evacuation by laminectomy resulted in prompt recovery.

Case 2. #57872. S.W.R., a 79-year-old white male, was admitted to the Urologic Service of Dr. T. D. Moore at the Baptist Memorial Hospital, Memphis on Mar. 11, 1950, complaining of paralysis of the lower extremities, urinary retention, and incontinence of bowel. For the previous 2 years the patient had had urinary difficulty manifested by slowing of the stream and straining. Nocturia occurred 2 to 4 times. A few weeks before admission he had had left sciatic pain after lifting heavy lumber. This pain gradually disappeared. One week prior to admission, while standing and playing with his grandchild, he experienced a sudden onset of excruciating pain in the right hip which spread to the left hip and down the posterior aspect of both lower extremities to the feet. Within a few moments both lower extremities became weak, and numbness of the feet and legs occurred. By holding to a wall, he was able to support himself until help arrived a few moments later. He complained of numbness and tingling of both lower extremities. Voluntary motion of the extremities was lost. Urinary retention and rectal incontinence developed.

He was hospitalized in another city for 8 days where he was treated with bed rest, and an indwelling urethral catheter was inserted. Low back and lower extremity
pain was so severe that the administration of opiates was necessary. Three days after the onset of his illness slight movement returned to the left extremity, and a few days later in the right. It was because of his urinary difficulties, however, that he was transferred to the Baptist Memorial Hospital. We were asked to see him in consultation shortly after admission. Despite the recent administration of a sedative, he complained of severe back and lower extremity pain, especially when moved.

**Examination.** The patient was obese and in obvious pain. His temperature was 99°F., pulse 90, respiratory rate 18, and B.P. 136/84. Heart sounds were normal and the lungs were clear.

There were no abnormalities of the upper extremities. Both lower extremities were markedly weak. On the right voluntary plantar flexion of the foot was impossible, but all other movements of the extremity could be performed weakly. All muscle groups on the left were functioning, but strength was greatly diminished. There were hypalgesia and hypesthesia over L5-S1 dermatomes bilaterally. Position sense and vibratory sense were normal. Pressure over the sciatic nerves caused severe pain. The Lasèque sign was positive at 150° bilaterally. Compression of the veins of the neck produced low back and extremity pain. The patellar and Achilles reflexes were absent on the right. The left patellar reflex was diminished, the Achilles reflex was absent. Cremasteric reflexes on the right were diminished, on the left absent. Abdominal reflexes were normal. There was marked spasm of the lumbar paravertebral muscles and the lumbar lordotic curve was decreased. Pressure over the 4th lumbar spine produced intense bilateral sciatic pain. There was an indwelling urethral catheter. The anus appeared patulous. On digital examination, slight anal sphincter tone remained. There was a benign enlargement of the prostate to grade 2 plus.

The blood was normal. The urine was normal except for the presence of 1-10 RBC and WBC/low power field. Blood non-protein nitrogen was 42.8 mg. per cent. X-rays of the lumbosacral spine revealed a slight scoliosis to the left, moderate decalcification, and moderate osteoarthritic changes. No definite loss of curve or intervertebral space narrowing were noted.

It was our belief that the patient most probably had suffered an extrusion of a lower lumbar intervertebral disk. However, we felt that lumbar myelography was advisable because of the unusual clinical picture. This was at first refused by the patient. Three days later the procedure was permitted. The spinal puncture, done at the 5th lumbar level, revealed light-yellow CSF under 140 mm. water pressure. It contained 17 lymphocytes and 1 segmented granulocyte. Total protein was 194 mg. per cent. Kline and Queckenstedt tests were negative.

Myelography, using 3 cc. of Pantopaque, disclosed a streaking of the column of oil at the 3rd and 4th lumbar levels with almost complete block of the subarachnoid space at the 2nd lumbar space, suggesting an extradural lesion.

**Operation.** On Mar. 15, 1950, using local anesthesia, a laminectomy was done from L2 to L4 (E.C.S.). Upon opening the ligamentum flavum, a small quantity of black blood exuded from the epidural space. A well-organized hematoma was exposed, measuring approximately 12 cm. in length and 10 to 12 mm. in thickness. After removal of the clot at the 2nd lumbar level, the dural sac was found to be collapsed. The clot extended anteriorly so that the lumbar sac was completely encircled. The clot was removed by suction and cup forceps. On opening the dura mater, the arachnoid was found to be light yellow in color, slightly thickened and adherent to nerve roots which appeared red and inflamed. By lowering the head of the operating
table, globules of pantopaque could be seen to collect beneath the arachnoid at the 2nd lumbar level, where adherent arachnoid obstructed its cephalad flow. The arachnoid was opened and jugular compression produced a prompt flow of cerebrospinal fluid into the wound. The cause of the hemorrhage was not found. The dura mater was closed tightly with interrupted silk sutures. Routine closure of the wound was carried out. A small Penrose drain was left beneath the paravertebral muscles for 24 hours.

Pathological diagnosis of the tissue removed: organized blood clot.

Course. Motor power returned rapidly over a period of 10 days. Rectal sphincter tone was regained over the same period.

Suprapubic cystotomy was performed on Mar. 27, 1950, and he was discharged to his home 2 days later. He was re-admitted to the hospital ambulatory on June 12, 1950, for transurethral prostatectomy.

When seen Aug. 1, 1950, he complained of slight paresthesias of the right leg and left foot. He denied any pain. Some urinary incontinence from previous prostatic surgery remained. Rectal tone was normal. Both Achilles and patellar reflexes on the right were absent. On the left these were diminished. Straight leg raising and compression of the veins of the neck caused no discomfort. Muscle power in the extremities was normal. There were no abnormalities of sensation.

Summary. Severe low back and lower extremity pain in an elderly male was followed by immediate complete paraplegia. Over a period of several days slight muscle function returned to both extremities. Bowel and bladder incontinence persisted. Operative removal of an epidural hemorrhage resulted in prompt return of normal function.

Case 3. Hospital #186-725. F.D., a white female, aged 56 years, was seen on Sept. 7, 1952, in Le Mars, Iowa, at the request of Dr. F. C. Bendixon. On Sept. 6, 1952, as the patient climbed into an automobile and bent her head to enter the backdoor, she had experienced a sudden pain in the left flank and slight backache. During the next 15 minutes the pain became excruciating and radiated across the back into both flanks. Examination by a physician revealed only slight left upper abdominal tenderness. Morphine, gr. 3/8, was administered and all pain stopped. There was normal voluntary motion in all extremities. After the doctor left, she noticed tingling sensations in both feet, accompanied by weakness of the extremities. This gradually increased so that in 3 hours there was complete paralysis of both lower extremities. Later in the evening the patient was hospitalized. No change occurred during the night. Lumbar puncture the following morning revealed uniformly blood-tinged fluid that could be obtained only by aspiration.

Neurologic Examination, about 24 hours after the onset. The patient was mentally clear. Acute flexion of the neck with coughing produced aching pain in the flank, but the Naffziger sign was absent. The deep reflexes were normal and equal in the upper extremities. Reflexes were absent in the lower extremities. There was no plantar response bilaterally. The abdominal reflexes were absent. Complete loss of pain, temperature, vibratory and position sense to the 10th thoracic dermatome was found. Straight leg raising on the right produced backache at 115° and was normal on the left. Tenderness was present on pressure over the 12th dorsal spine and caused aching sensation into the left flank and both lower extremities. There was slight tenderness in the left upper quadrant of the abdomen, but no rebound pain was present. The dorsal pedal and femoral pulses were normal, and there was no edema of the lower extremities.
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It was learned that in 1950 the patient had had a sudden numbness of the right face and extremities without headache, nausea, or unconsciousness. Recovery was complete except for a slight residual numbness in the right leg. The B.P. averaged 160 to 180 systolic after that time. No hemorrhagic tendency had ever occurred, although she bruised easily. A history was also obtained of occasional pain in the left flank during the last 6 to 8 months. This was increased by hunger and relieved by eating, lasting not over 15 to 30 minutes.

The patient was transferred to St. Joseph Mercy Hospital, Sioux City, Iowa, where myelography was done. The Queckenstedt test revealed a complete subarachnoid block, and 2 cc. of Pantopaque introduced at the 3rd lumbar level flowed cephalad only to the upper border of the 1st lumbar vertebra. Roentgenograms of the chest and spine were normal. Blood studies, including prothrombin time and hematocrit determination, revealed no abnormalities.

Operation. Under general anesthesia, laminectomy (C.A.B.) of T10, T11, and T12 was done 5 hours after initial examination (29 hours after onset of symptoms). This revealed an acute epidural clot extending over the entire length of the exposure. Its approximate length was 7 cm. and it covered the entire dorsal half of the dura mater. This was similar to that in Case 1. The hematoma was easily removed. There was slight epidural bleeding which was controlled by coagulation and oxycel gauze. The dura mater was opened without opening the arachnoid, and normal pulsations were seen. A #10 catheter could be run in the subdural space for a distance of 10 cm. cephalad and caudad without obstruction. The dura mater was closed by a continuous silk suture.

Course. The patient made slow but progressive improvement. There was some motion of the toes of both feet within 12 hours, but she did not void spontaneously for 1 month. A spinal puncture on Sept. 24, 1952 revealed a pressure of 130 mm. Spinal fluid dynamics were normal.

At the time of hospital discharge on Oct. 11, 1952, there was an estimated 50 per cent function in all muscles of the left lower extremity and 25 per cent in the right lower extremity. Epicritic sense was present to the knees in both legs and protopathic sense in the legs and feet.

Three months after surgery the patient was able to walk freely with a stroller and could take 8 to 10 steps alone. She was free of pain and had normal function of bowel and urinary bladder.

Summary. Several hours after the onset of severe back pain, paraplegia occurred. Prompt removal of a low thoracic epidural hematoma was followed by progressive return of neurologic function.

Case 4. C.M.F., a 34-year-old white male, was admitted to the Veterans Administration Hospital, Fort Howard, Maryland, on Aug. 14, 1952. In 1938, 14 years previously, while walking he had been suddenly seized with sharp, stabbing pain in the lumbar region. There was no radiation of pain and it vanished after 1 week of bed rest. Two years later, while lifting, he again had a sudden onset of sharp, stabbing pain in the lumbar region. Following 2 weeks’ bed rest the pain subsided. In 1940 low back pain returned while riding in a vehicle. On this occasion the pain was more severe than before and lasted 10 days. Approximately 1 year later, while standing, there was still another abrupt onset of sharp, stabbing pain—worse than ever before—in the low back. Despite hospitalization, the pain remained severe for 3 weeks. Since the initial attack of pain in 1938, he had been bothered by low backache when he began to straighten up after bending over or if he stood in one position for a pro-
longed period. He found that this aching pain was relieved by assuming a squatting position or by bending over as if to touch his toes. He stated that during the 4 years preceding his present hospital admission he had been drinking on an average one pint of whiskey per day.

At 2:30 A.M. on Aug. 14, 1952, the patient awakened with severe pain in his low back which radiated to his lower abdomen and to the posterior aspect of both hips, associated with numbness along the lateral aspect of his thighs and legs. The family physician gave an injection of morphine which relieved the pain; 15 minutes later the patient tried to get out of bed. His legs gave way under him and he fell. Although he was able to walk a few minutes, pain persisted, and he was hospitalized.

Examination. On admission he was awake, aware, and cooperative. His temperature was 98.6°F, pulse 72, respiratory rate 20 and B.P. 130/70. There was diffuse tenderness over the lumbar spine with paravertebral muscle spasm on the right side. Motor power appeared normal in the lower limbs. At that time pain was limited to the low back and right lower limb. Straight leg raising was painful at 140° on the right with reference of pain to the low back and right hip. Except for a decreased Achilles reflex on the right, the deep reflexes were normal and the plantar responses were flexor.

Urinalysis on August 15 showed 3+ albumin, 6–8 WBC/hpf and a few finely granular casts. On August 19 hb. was 13. 2 gm. (91 per cent), and WBC 13,600 with 85 per cent polymorphs, 12 per cent lymphocytes, 1 per cent monocytes, and 2 per cent eosinophiles. The urea nitrogen was 25 mg. per cent, blood sugar 80 mg. per cent, and the Eagle flocculation was negative. Roentgenograms of the chest and lumbar spine showed no abnormality except an old compression fracture of the body of the 3rd lumbar vertebra. The patient later stated that he had been told of this roentgenologic finding in 1943.

Course. Two days after admission he complained of inability to move his lower limbs and inability to void. He was catheterized and 1150 cc. urine were obtained. Later the same evening he became confused and disoriented. A psychiatrist reported that he was having delusions and hallucinations which centered around his being captured and his wife and child being dead. He was moved into a locked ward and heavily sedated. Three days later he became oriented and was noted to have a flaccid paraplegia. He was seen in neurosurgical consultation on Aug. 24, 1952.

Neurologic Examination. The patient was well oriented and cooperative. Cranial nerves and upper limbs appeared normal. There was a flaccid paralysis of both lower limbs with absent patellar and Achilles reflexes bilaterally. Plantar stimulation on either side evoked no response other than a slow plantar flexion of the great toe associated with some flaring and some flexion of the lesser toes on the stimulated side. There was a sensory level at L1 below which all modalities of sensation were absent. Throughout the lower thoracic and upper lumbar regions, there was diffuse tenderness. When the neck was flexed or when the extended lower limbs were elevated, he complained of severe pain in the lower dorsal region radiating around to the anterior aspect of the lower abdomen. Compression of the jugular veins did not cause back or leg pain.

Lumbar puncture showed a complete block on bilateral jugular compression. The CSF was clear and xanthochromic, with a protein of 196 mg. per cent. There were no cells, and the spinal fluid sugar was 66 mg. per cent.

Operation. Under general anesthesia immediate laminectomy (W.H.M.) was carried out from T9 through L1, exposing a sausage-shaped mass lesion which lay
on the dorsal and right lateral aspects of the dural sac, terminating at the upper and lower ends of the operative field. The dorsal and lateral aspects of the lesion appeared to be encapsulated, whereas medially and anteriorly, the major portion of the mass consisted of black, clotted blood. The hematoma was removed in its entirety and the wound was closed. The dura mater appeared normal and was not opened.

Pathological Report. The tissue removed consisted of blood clot which showed varying amounts of fibroblastic organization. Practically no iron-like pigment was recognized. There were abundant fibroblasts and many capillary-like vessels of varying sizes. Some areas of hemorrhage appeared older than others and it appeared that hemorrhage had occurred in granulation tissue, organizing a previous hemorrhage. No neoplastic cells were seen. Histological examination of bone removed at time of laminectomy showed normal-appearing, blood-forming elements.

In light of the pathologic findings, further blood studies were carried out on Sept. 4, 1952. Hb. was 12.7 gm. (88 per cent), RBC 4,790,000, hematocrit 46, WBC 10,900 with 76 per cent polymorphs, 23 per cent lymphocytes, and 2 per cent eosinophiles, and platelets 384,000. The bleeding time was 1 minute, coagulation time 5 minutes, and the prothrombin time was 16 seconds with control 14.6 seconds.

Course. Immediately following operation, the patient was relieved of pain and from the subjective numbness in the back and lower abdomen. There was no return of motor power, and he remained incontinent of urine and feces. Two days later his sensory level had dropped to L2 and all modalities of sensation were absent below that level. Deep reflexes remained absent and since operation there had been no response to plantar stimulation on the left. Until Aug. 30, 1952, plantar stimulation on the right evoked a slow plantar flexion of the great toe, associated with some flaring and flexion of the lesser toes.

The patient is now up and about in a wheelchair and has been transferred to the rehabilitation service.

Summary. Over a period of 14 years, this 34-year-old man had recurrent backache. On 5 occasions severe back pain had confined him to bed, and he was twice hospitalized because of the pain. During the last hospitalization, a psychotic episode occurred which lasted 3 days. It was then noted he was paraplegic. Operation was carried out 5 days later, and although he was improved by surgery, paraplegia persisted. Had operative removal of the epidural hemorrhage been carried out within a short time after the appearance of extremity weakness or paralysis, a more nearly complete neurologic recovery would undoubtedly have resulted. Microscopic sections of the tissue removed revealed it to be composed of both old, well-organized clot and recent hemorrhage.

DISCUSSION

In a review of the literature, spontaneous epidural hemorrhage was found to occur at any age from 20 months to 75 years. With but one exception it occurred in the adult age group. There was no sex predominance. All patients had a history of trauma with the exception of a 20-month-old infant, in whom infection was present. It is noteworthy that in most cases in which trauma occurred, it was mild. Straining and twisting efforts seemed particularly prevalent. Severe neurologic impairment occurred in 20 minutes to several weeks after trauma. There were 3 lesions of the cervical
region: 2 confined to the cervical levels, the other extending from C3 to the lumbar area. There were 4 lesions of the upper dorsal region, and 2 of the lower dorsal region, 1 of which extended to the 1st lumbar level. In 3 cases the lesions were confined to the lumbar region. Nine of these patients were treated surgically. Of these, 5 made almost complete recovery—1 lesion was in the cervical, 2 were in the dorsal, and 2 in the lumbar regions. Three made no neurologic recovery, and 1 patient expired 2 weeks postoperatively. To this group of operated cases, we add our own 4.

REFERENCES