OLIGODENDROGLIOMA OF THE SPINAL CORD*

JOHN R. RUSSELL, M.D., AND PAUL C. BUCY, M.D.

The Chicago Memorial Hospital and the Department of Neurology and Neurological Surgery, The Illinois Neuropsychiatric Institute, University of Illinois College of Medicine, Chicago

(Received for publication February 28, 1949)

Oligodendrogliomas comprise 3.5 per cent of the gliomas of the brain, according to the analysis of 254 histologically verified gliomas classified by Bailey and Cushing. In their discussion of oligodendroglioma of the brain, Bailey and Bucy pointed out that this variety of glioma should also occur in the spinal cord, since the oligodendroglioma are found in the spinal cord as well as in the brain.

Detailed histological analyses have been made of only a few large series of intramedullary spinal cord tumors. Kernohan, Woltman and Adson in 1931 classified 51 cases of intramedullary spinal cord tumor operated upon at the Mayo Clinic and found 2 (4 per cent) oligodendrogliomas. Up to Jan. 1, 1939, the Mayo Clinic series included 64 intramedullary tumors, 3 of which were oligodendrogliomas, an incidence of 5 per cent. The same group reported 25 gliomas of the conus medullaris and filum terminale, of which 1 was an oligodendroglioma. In 1944 Woods and Pimenta made a study of 30 histologically verified spinal cord gliomas, and classified 1 as an oligodendroglioma. Oljenick has also reported 1 case, giving a total of 6 cases of oligodendroglioma of the spinal cord and filum terminale recorded in the literature.

A careful search of the literature from 1930 to 1947 disclosed an additional 98 intraspinal gliomas, histological classification of which was attempted. The exact pathologic diagnosis is subject to question in some cases, but there was no instance of an oligodendroglioma. Thus oligodendroglioma of the spinal cord is a very rare lesion. For this reason we feel it worth while to report a case in which operation was performed on May 13, 1948. The symptoms were of long duration, and there was an associated scoliosis. This case has been briefly reported by Bucy and Heimburger.

CASE REPORT

History. A LeC., a white male railway clerk, aged 31, was referred by Dr. Harry Hodges and admitted to the Chicago Memorial Hospital on May 11, 1948.

In 1932, at the age of 15 years, the patient was in an automobile accident, but sustained no injury that was noted at the time. One month later he noticed protrusion of one of the lower left ribs, and was told that it was fractured. Two years after the accident "twitching movements" occurred in the muscles of the back, and his family noticed that his spine was crooked. At about the same time he began to have periods of clumsiness and numbness in both hands, lasting only a few minutes. Following 5 months of naprapathic treatments in 1937, the patient noted no fibrillations in the back muscles, until they returned in 1940. The "twitches" occurred at a rate of about 40 per minute, were most pronounced when the patient was sitting, and caused his entire body to jerk.

During the next 2 years there developed slowly progressive weakness and awkwardness of the legs and hands. He fell twice, due to weakness of the legs. His gait was peculiar and he carried one shoulder lower than the other.

Examination by an orthopedic surgeon in 1942 revealed a lower thoracic scoliosis with

* Presented before the Chicago Neurological Society, January 11, 1949.
convexity to the right. The left shoulder was higher than the right. Rhythmical contractions of the left sacrospinalis musculature were palpable. A questionable decrease in pain sensation in the lower extremities was noted, but a detailed neurological examination was not recorded. X-ray films of the spine made at that time showed the scoliosis (Fig. 1).

On May 20, 1942, the orthopedic surgeon performed a spinal fusion with a 6 inch tibial bone graft placed on the denuded laminae from the 10th thoracic to the 3rd lumbar vertebra.

On June 9, 1942, a 2nd operation was done to correct the malposition of the tibial graft. Following this the wound became infected and drained for 4 months. After recovery from the operation the patient was completely relieved of the twitching in the back muscles. He had no weakness or clumsiness of the extremities, except that he considered the right leg a little weak, and favored it because the tibial bone graft had been removed from that side. In due time, he was able to return to work.

In December, 1946, the patient sustained burns on the thighs from a hot radiator, but felt no pain. Following this he had several minor traumata to the legs, without any sensation of pain. In the Spring of 1947, he noticed clonus of the right ankle while playing the organ. After that he had a progressive weakness of the legs, the right being most affected. By the Summer of 1947 he was unable to climb stairs or run, and had great difficulty in walking. In the year before his admission to the hospital, the patient had fecal and urinary urgency, and was incontinent if unable to relieve his urgency at once. He also complained of aching pain in the lower abdomen and back.

Examination. When admitted to the hospital on May 11, 1948, the patient could barely walk with the aid of two canes. His gait was spastic, with a broad base. The Romberg test was positive. He was unable to hop on either foot. There was generalized weakness of both lower extremities, most marked in the hamstring muscles, and greater on the right than on the left. The thighs and calves were markedly atrophic. The tonus of the lower extremities was increased, especially on the right. The knee jerks were hypoactive, but the ankle jerks were hyperactive. Ankle clonus and Babinski's sign were present bilaterally. Spontaneous flexor spasms were noted. The abdominal reflexes were present in the upper quadrants but absent in the lower quadrants. He could not feel pin prick below the 11th thoracic dermatome. Perception of warmth was absent below the 2nd lumbar dermatome, and of cold was present but diminished below the same level. Position sense was diminished in the right foot, and the ability to recognize numbers written on the skin was absent in both legs. Tactile sensibility was normal. No neurological defect was found in the upper extremities or in the cranial nerves. General physical examination revealed a lower thoracic scoliosis with convexity to the right, and a surgical scar from the 9th thoracic to the 3rd lumbar spinous process.

On lumbar puncture the initial CSF pressure was 160 mm. fluid. Bilateral jugular compression gave a rapid rise in pressure to 300 mm., but on release of the compression the pressure fell slowly and hesitantly back to the original level. After removal of 7 cc. of fluid, the pressure

---

Fig. 1. Composite roentgenograms of spine showing scoliosis in 1942.
fell to 0 mm. The impression was that a partial CSF block was present. The CSF was clear and colorless, contained 2 lymphocytes/c.mm., and 78 mg. per cent of total protein. The Wasserman reaction was negative and the Lange colloidal gold curve was 0000000000. Examination of the blood and urine revealed no abnormality.

Roentgenograms of the spine showed a scoliosis extending from the 5th thoracic vertebra to the 3rd lumbar. A dense spinal fusion in the thoraco-lumbar region made detailed examination of the spine and of the spinal canal impossible. A chest x-ray showed no abnormality except old fibrotic scarring in the right infraclavicular area.

![Fig. 2. Photomicrograph of section of tumor stained with hematoxylin and eosin.](image)

**Operation.** On May 13, 1948 a laminectomy of the 8th, 9th and 10th thoracic vertebrae was made through the old spinal fusion. At the level of the 9th thoracic vertebra there was no epidural fat, and the dura mater was thin and distended. Incision of the dura mater revealed a greatly enlarged yellowish spinal cord. All the usual landmarks were obliterated and the surface vessels were flattened. No fluid could be aspirated from the cord. The left posterior column of the cord was then incised longitudinally for a distance of 3.5 cm. This resulted in exposure of a very gelatinous, pinkish-gray tumor, which herniated through the opening. There was no capsule, and the tumor would not shell away from the cord substance. The upper and lower limits of the tumor were not exposed. A considerable portion, but by no means all, of the tumor was removed with a pituitary rongeur and with suction. The dura mater was left open, and the other soft tissues were closed in layers as usual.

**Pathology.** Sections of the tumor stained with hematoxylin and eosin show it to consist largely of round or polyhedral cells with large vesicular nuclei, and surrounding clear cytoplasm, giving a box-like appearance to the cells. For the most part these cells are closely packed with no intervening stroma. No mitotic figures or other evidences of malignancy were seen. The walls of many of the blood-vessels are thickened and some are practically occluded by this intravascular proliferation. No calcification was seen. The microscopic appearance of the tissue (Fig. 2) is typical of an oligodendrogloma.
Postoperative Course. The patient was completely unable to move the lower extremities. The sensory level remained at the 11th thoracic dermatome until 3 weeks postoperative, when it had dropped to the 1st lumbar dermatome. On the 9th postoperative day, he began to have a spiking fever, and by the 12th day a spinal fluid fistula was apparent at the lower end of the incision. Lumbar puncture yielded CSF containing 3000 polymorphonuclear leukocytes /c.mm. On the 13th day, a secondary closure of the fistula was done, and no further drainage has occurred. The signs of meningitis subsided with streptomycin, penicillin and sulfadiazine therapy. It is thought that this meningitis was the result of the lighting-up of the old infection which had been present in 1942 and which had been lying latent in the bone graft.

On June 8, 1948, the patient was transferred to the Illinois Neuropsychiatric Institute for rehabilitation from the standpoint of the paraplegia. He has remained paraplegic, but has learned to walk with the aid of braces and crutches. On June 14, 1948 a lumbar puncture revealed an initial pressure of 90 mm. CSF. There was a rapid rise in CSF pressure on jugular compression, with a rapid fall in pressure on release of the compression. The CSF contained 22 lymphocytes/c.mm. The patient had a paradoxical urinary incontinence, with a residual of 500 cc. To correct this, a transurethral bladder neck resection was done on Oct. 2, 1948, following which he developed an automatic bladder, with no residual. He was discharged from the Illinois Neuropsychiatric Institute on Oct 26, 1948.

DISCUSSION

The association of this intramedullary spinal cord tumor with scoliosis has been discussed in another publication.1 An intramedullary spinal cord tumor that does not destroy the vertebrae probably can cause scoliosis only when it develops slowly in a growing individual. Scoliosis was first observed in this patient at the age of 17. The scoliosis is probably produced by asymmetrical spasm or weakness of the peri-vertebral musculature innervated by the involved portion of the spinal cord. It may be noted that this patient observed twitching of the muscles of the back at the onset of the scoliosis. Vertebral erosion by pressure of an intraspinal tumor can lead to scoliosis, but this is very unlikely in the case of intramedullary lesions.

Allen and Kahn1 reported a case of scoliosis produced by an upper thoracic and cervical intramedullary astrocytoma. Their patient, who was 7 years old at the onset of symptoms, had an 8-year history of scoliosis and back pain, with weakness of the left leg for 10 months.

Our patient has an unusually long history of signs and symptoms, dating back 14 years prior to operation. In Kernohan's8 cases, the duration of symptoms was 8 years, 4 years, and 6 months (personal communication). In the case reported by Oljenick,5 symptoms were present for 2 years. The patient of Woods and Pimenta14 had symptoms for only 4 months, and died 4 ½ months after operation, but the tumor tissue showed “relatively malignant cellular characteristics.”

Ependymoma is by far the most common intramedullary spinal cord tumor. In the largest recorded series, ependymomas comprise 40 to 50 per cent of the gliomas. All other types of glioma are found in the spinal cord, but the incidence of any one type, such as oligodendroglioma, is low.

Oligodendrogliomas occurring in the brain frequently contain calcareous deposits visible by x-ray examination. No such calcification was present in our case, or in any of the 6 other recorded cases. Weaver10 reported a lower cervical glioma “moderately cellular and with considerable calcification,” but did not specify the cell-type. Gray4 reported a case in which the roentgenogram showed a spotty calcification within the spinal canal. At operation an intramedullary tumor was found, but no biopsy was taken. Jefferson6 thought that this lesion was a hemangioblastoma or possibly a very vascular oligodendroglioma.
Kernohan, Woltman and Adson\textsuperscript{7} pointed out that in their large series of intramedullary tumors of the spinal cord, conus medullaris and Flum terminale, only 3 of the tumors invaded the subarachnoid space, and 2 of these were oligodendrogliomas. No invasion of the subarachnoid space had occurred in our case.

**SUMMARY**

1. A case of oligodendroglioma of the spinal cord occurring in a 31-year-old white male is presented. There were symptoms of scoliosis for 14 years, followed by progressive motor and sensory loss in the lower extremities.

2. The scoliosis in this case is thought to be due to the intramedullary spinal cord tumor.

3. A review of the literature is given; 6 other cases of primary intraspinal oligodendroglioma have been reported.

**REFERENCES**