Spontaneous Multiple Intramedullary Hemorrhages

Case Report

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"S"pontaneous hematomyelia” of unknown origin usually manifests itself in an abrupt onset of pain and rapidly developing pyramidal signs in patients who have hitherto been asymptomatic. A hemorrhagic diathesis and vascular malformation as well as inflammatory processes affecting the spinal cord and neoplasms have occasionally been reported as causative factors, but malformation of intramedullary vessels and vascular tumors have been found to be the most frequent causes of spontaneous hematomyelia.

Etiologically, there are three groups of spontaneous intramedullary hemorrhages:

Group 1. Spontaneous hemorrhage into the substance of the spinal cord due to malformation of intramedullary vessels; in these cases injuries do not have any etiologic significance.1,2,4,6,8,9,11,15,40 Spontaneous hematomyelia in patients with intramedullary angiomas, in which minor injuries may be a factor, is included in this group.8

Group 2. Spontaneous hematomyelia with underlying syringomyelia. In these cases, the onset may either be spontaneous3,7 or due to minor injuries.10 Rarely, intramedullary hemorrhage in the presence of syringomyelia has been associated with concomitant hemophilia.10,14

Group 3. Sporadic cases of spontaneous hematomyelia during the course of hemophilia.1,13

Case Report

An 11-year-old boy fell while skiing and sustained an external rotation of his left foot. The child was able to walk without discomfort, but 4 days later he experienced transient pains in the left knee and 3 weeks later developed claudication and progressive weakness in the left leg. He was seen at the neurosurgical department 2 months after injury, on February 28, 1968.

Examination. There was paralysis of flexion and extension of the left foot, as well as a flexor and extensor paresis in the knee joint. Neither knee nor ankle jerks could be elicited; there was no sensory loss or pyramidal tract signs and bladder function was normal. Reexamination 1 week later showed paresis of the left leg more pronounced distally with absence of knee and ankle jerks; reflexes in the right leg were weak. Deficits of all sensory modalities of the left leg were observed ranging over the L-2 to S-2 dermatomes; sensory function of the hips and thighs was, however, retained. Sensation in the right leg was normal. On electrical stimulation, accommodation was found to be impaired in the quadriceps, extensor communis digitorum, tibialis anterior, peroneus, and gastrocnemius muscles of the left leg. Chronaxie was prolonged in the extensor digitorum communis muscle. The gluteal as well as ischio-crural muscles gave normal responses. Apart from revealing a spina bifida, plain x-rays of the lumbar spine were noncontributory. Myodil myelography was normal.

On March 18, the patient was admitted to the Wilhelminen-Hospital, Vienna. Due to the peripheral palsy in the left leg, a neuritic lesion was suspected. On March 20, 4 weeks after the onset of symptoms, the patient suddenly developed flaccid paraplegia of both legs and complete loss of all sensory modalities from T-12 downward, associated with saddle anesthesia. The child complained of paresthesia in both legs; in addition, he had neurogenic bladder dysfunction.

Lumbar cerebrospinal examination showed normal pressure, a protein content of 55 mg%, 33 cells (lymphocytes), a chloride value of 124 mEq, and a glucose content of 65 mg%. On the same day the child was transferred to the neurosurgical depart-

Received for publication May 12, 1969.
ment for a suspected space-occupying intra-
spinal process.

Suboccipital Pantopaque myelography re-
vealed a complete block at the level of T-12
(Fig. 1 left). The neurological symptoms
were those of a transverse lesion at the level
of L-1 and L-2. Several hours after the onset
of the paresis and the sensory deficits, some
sensibility returned in the area of the hips
and thighs. Loss of urethral and rectal
sphincter function persisted; reflexes in the
lower extremities were absent, but pyramidal
signs could not be elicited.

*Operation.* The patient was operated on
March 30, 1968. After laminectomy from
T-11 to L-1, the spinal cord in the region of
the conus and epiconus was found to be dis-
tended cylindrically, of tight and elastic con-
sistency, and a pale color. Tortuous arteries
of large caliber and congested veins were
seen on the dorsal aspect of the spinal cord.
In the region of the conus a right paramed-
ian incision of 10 mm was made in the spi-
nal cord. At a depth of 2 mm, a cavity 15
mm long \(\times\) 6 mm wide \(\times\) 6 mm deep was
found, from which brownish blood clots es-
caped upon pressure. The walls of the cavity
showed brownish discoloration. There was
no evidence of neoplastic tissue; biopsy spe-
cimens were taken from the wall of the cav-
ity. As evacuation of the hemorrhagic mate-
rial from the conus medullaris failed to re-
lieve the distension of the spinal cord in the
region of the epiconus, a second right par-
amedian incision was made 20 mm higher,
and another cavity 18 mm long \(\times\) 6 mm
wide \(\times\) 6 or 7 mm deep was exposed. Again
old blood clots were present, and the walls

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**Fig. 1. Left:** Pantopaque myelogram showing obstruction at the level of L-1. **Right:** Schematic
drawing of the operative field. The cavities of the hemorrhages in the conus and epiconus region have
been opened and some blood clots removed.