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HÆMATOMYELIA AFTER REDRESSION OF SCIATIC SCOLIOSIS UNDER ANÆSTHESIA

In orthopedics redression of a fixed sciatic scoliosis is a method of treatment that is employed not infrequently. As far as I have been able to find out no complication resulting from this treatment has been reported in the literature before Mutschler in 1937 described a case of hæmatomyelia that appeared in connection with redression of sciatic scoliosis under anæsthesia (Zeitschr. f. Orthopädie, vol. 67, 1937). Also I have had occasion to observe such a case. So special complications might arise from correction of sciatic scoliosis under anæsthesia. As this phenomenon has been merely touched upon in the literature, I think it may be of interest to give a more thorough account of the case I have observed.

The patient was a man, born January 7, 1898. In 1923 he suffered a particularly heavy direct blow over the loins, and afterwards he had repeatedly periodical attacks of pain over the lumbar part of the vertebral column and down in the legs, on which account he has been hospitalized several times and treated with rest in bed and physical treatment. The last troublesome period of this kind commenced in the last part of December 1935.

He applied to the Stockholm V. F. A. on January 28, 1936.

The physical examination showed: The lumbar lordosis is planed, and in erect posture the patient is inclining slightly forwards, with about 20° deviation of the lumbar spine to the right. The lumbar musculature is strongly contracted, and the lumbar part of the vertebral column is practically quite stiff. Lasegue's sign is positive on both sides (30°).

On February 25, 1936, correction of the sciatic scoliosis was performed under anæsthesia. Without any particular resistance the back was brought into a position of moderate overcorrection, and a plaster cast was put on both legs and the lower part of the trunk.

After this operation the patient was not able to void, and the bowels moved involuntarily. He has no feeling of necessity of defæcation, nor could he feel when the catheter was introduced through the urethra. He complained of a sensation of numbness in the crotch and on the anterior surface of the thighs.

On April 4th it says in the case record: The patient is still unable to void and has to be catheterized. He complains of numbness of the feet and of the anal and perineal regions, besides decreased sensibility of the legs. The sensibility for temperature, touch and pain is lowered or completely abolished over an area comprising both lower extremities and the lower part of the abdomen (up to a little below the umbilical transverse). Romberg positive. No complete paralysis but the muscular power of both legs is strongly reduced. Knee jerk normal. Babinski uncertain. Abdominal reflexes absent. Spinal fluid (from lumbar puncture): clear and colourless; Wassermann test negative.

April 14th: No changes in the roentgenogram of the upper lumbar and lower thoracic parts of the vertebral column.

On April 22, 1936, the patient was transferred to the Medical Department of the Serafimer Hospital. At that time his condition had been improving gradually. Now the urination was normal, and the defæcation was under control—at least partly. Neurological examination showed: Flexor power of the knees lowered, extensor normal. Extensor power of the feet lowered, flexor normal. Gradually increasing reduction in the sensibility of all kinds on the lateral surface of the crura. Distinct riding-breeches hypæsthesia. Romberg positive, with varying tendency to falling. Otherwise no definite signs of ataxia. Abdominal reflexes absent. Babinski uncertain. Other tested reflexes normal. Spinal fluid normal. Wassermann test on the blood negative.

January 2, 1937: The improvement has been progressing slowly. Urination normal; defæcation almost normal. Sensibility not so decreased as before; strong muscular power of the legs still impaired. The patient walks quite unsteadily, wabbling.

On June 3, 1937, it says in his record that he has resumed light work.

This, then, is the case of a patient with fixed sciatic scoliosis, in whom redression of the scoliosis with moderate overcorrection under anæsthesia was followed by complete paralysis of the bladder and anal functions, decrease in the sensibility for touch, temperature and pain in the lower extremities and lower part of the abdomen (up to just below the umbilical transverse), slight partial flaccid paralysis of the lower extremities, and abolition of abdominal reflexes. The spinal fluid was normal. The Wassermann reaction was negative in the blood as well as in the spinal fluid. Roentgenography of the thoracolumbar part of the vertebral column negative. The paralysis and decrease of sensibility gradually subsided almost completely; and the functions of urination and defæcation became normal again. The clinical features and course of the lesion are in keeping with those of hæmatomyelia and this hæmatomyelia had originated as a result of redression of a sciatic scoliosis under anæsthesia.

Usually distinction is made between the so-called spontaneous hæmatomyelia and traumatic hæmatomyelia. A greater majority of the cases of hæmatomyelia—about 90 %, it is generally stated—belong to the group of traumatic hæmatomyelia. In general the traumatic injury is of relatively mild character, so that usually it does not involve any fracture or dislocation of the vertebral column. According to Zwirner, traumatic hæmatomyelia is in most cases due to a direct traumatic injury to the back; the next greatest quota is made up of cases in which hæmatomyelia originates through twisting or flexion of the trunk or neck, while hæmatomyelia less frequently is seen in patients who have fallen from some height and landed on the feet or head. In several cases lifting of a heavy load has been given as the cause of hæmatomyelia. In a couple of cases hæma-

tomyelia has been attributed to stretching of the sciatic nerve (Rumpf; Petrén).

The cases which in this connection are of most interest to us are those in which hæmatomyelia originated through flexion or twisting of the vertebral column, as the mechanism for establishment of hæmatomyelia here ought to be similar to that which gives rise to hæmatomyelia on redression of sciatic scoliosis. In flexion of the vertebral column beyond the normal limit the hæmatomyelia is supposed to arise through stretching of the spinal cord with rupture of a blood vessel. In their histological examination of the spinal cord in cases of hæmatomyelia, Wagner and Stolper found the features of nerve fibers that had been torn, and they think it most probable that the pulling at the roots of the nerves that comes with excessive flexion of the vertebral column has led to tearing, not only of blood vessels but also of nerve fibers in the spinal cord. Hegar has measured the elongation of the spinal cord produced by maximal flexion of the vertebral column, and he calculates it to be 4—8 % of the length of the entire spinal cord. That excessive stretching of the spinal cord may be the cause of hæmatomyelia is also suggested by the circumstance that hæmatomyelia occurs most often in the cervical and lumbar parts of the back, that is, the most mobile portions of the vertebral column.

In the redression of sciatic scoliosis in the case described above there was not performed any flexion of the vertebral column beyond the normal range, so that flexion of the back cannot have been the sole cause of the hæmatomyelia in this case.

In sciatic scoliosis of long standing it seems reasonable to imagine that, as in other joint contractures, there is a shrinkage of the soft parts on the concave side and, with this, also some shortening of blood vessels and nerves. Under such conditions, it seems possible, redression of the scoliosis may become associated with tearing of blood vessels and overstretching of the nervous tissue even though the limits for the normal mobility of the vertebral column are not exceeded. But in such cases the shrinkage of muscles and ligaments ought to imply a noticeable hindrance to the redression. In this case the redression was

carried out easily, however, without any particular force, so that the hæmatomyelia cannot readily be explained in this way.

But also other cases of hæmatomyelia have been described in the literature, in which this lesion has originated in connection with a traumatic injury that often was relatively slight, and in which one would hardly think the trunk had been flexed beyond the normal limit. Zwirner and Bolten too think that in these cases the traumatic injury is not the only cause of the hæmatomyelia, and that various other factors also play a rôle in the origin of the hæmatomyelia. Thus, they think that the rise in blood pressure which commonly appears as a result of rapid and strong muscular tension is of some significance to the origin of hæmatomyelia. If we apply this reasoning to cases in which the hæmatomyelia appeared in connection with redression of sciatic scoliosis under anæsthesia, an all too superficial anæsthesia at the performance of the redression—causing general muscular tension and, thus, a rise in blood pressure—might conceivably be a contributory cause of the hæmatomyelia. But this is merely a hypothesis that is not supported by any definite evidence.

As long as we do not know more about the pathogenesis of hæmatomyelia in these cases, we are not able to give any particular precautional measures that may be taken in order to prevent the appearance of hæmatomyelia in redression of sciatic scoliosis. In many places this redression is performed with considerable overcorrection of the faulty position and with employment of considerable force, and yet the occurrence of hæmatomyelia is undoubtedly rare.

Even if we have to reckon with the possibility that hæmatomyelia may arise after redression of sciatic scoliosis under anæsthesia, this method of treatment in certain cases of sciatic scoliosis is all too valuable to be given up on account of the rare cases complicated by the appearance of hæmatomyelia.