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ORIGINAL ARTICLE

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Comparison of different operative modalities in post-traumatic syringomyelia: preliminary report

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Abstract Post-traumatic syringomyelia (PTS) is a relatively rare, but potentially disastrous, complication of spinal cord injury. Operative treatment by shunting procedures often shows only a short-term improvement, and the rate of recurrence of syringomyelia is high, so different treatment modalities have been used in the last years. The various results are discussed in this analysis. A prospective clinical study was conducted of 30 patients with PTS treated by shunting procedures or with pseudomeningocele over a period of 9 years, and followed with regular clinical and magnetic resonance imaging examinations. Shunting procedures like syringosubarachnoid and syringopleural or -peritoneal shunting showed good results only at the first follow-ups. In our department, we perform an artificial liquor reservoir at the level of the lesion after opening the spinal pathways and arachnoid adhesions at that level. This procedure was performed in 12 patients. Five of these had been previously operated by shunting procedures; all of them had suffered a recurrence of syringomyelia because of internal occlusion. In the group of patients treated by shunting procedures, a neurological improvement was be recorded in five, and a steady state in eight. Five patients showed a further deterioration. The performance of an artificial liquor reservoir to guarantee a free flow of cerebrospinal fluid around the lesion resulted in a neurological improvement in ten patients, with two maintaining a steady state. Our experience is that shunting procedures often show a neurological improvement only in the short term; the rate of recurrence of typical shunting complications is high. The performance of a pseudomeningocele is an encouraging new step in the treatment of PTS. Further long-term follow-up studies are necessary to assess the benefits of this new method.

Key words Spinal cord injury · Post-traumatic · Syrinx · Syringomyelia · Paraplegia

Introduction

Longitudinal cavitations of the spinal cord may appear under several conditions: deformity of the craniocervical junction, which leads to downward migration of hindbrain and cerebellar tonsils or basilar impression; hydrocephalus; spinal infection; cord tumors or spinal trauma [2,11]. The established operative treatment of hindbrain-associated syringomyelia consists of enlargement of the foramen magnum combined with widening of spinal pathways. A spinal cavity associated with spinal tumor can be treated effectively by removal of the tumor.

In recent years, shunting procedures have been the treatment of choice for post-traumatic syringomyelia (PTS).The brevity of the periods of neurological improvement led to a variety of modifications, such as implantation of shunt devices in the subarachnoid, peritoneal or

pleural space. Critical appraisals showed that drainage procedures are not effective in treating a progressive PTS [10] in all cases. The treatment of choice for PTS in receent years has been implantation of syringosubarachnoid, syringoperitoneal or syringopleural shunts [6, 10, 15, 16, 18,19]. Umbach and Heilporn noted that no shunting procedure was superior to any other [14]. Sgouros and Williams found a 15.7% immediate complication rate after operation in their population; only half of the patients remained clinically stable after 10 years [10]. Late complications consisted of induction of fibrosis and gliosis around the catheter [10,18]; very often, pre-existing or new cavities will be excavated if the underlying pathology persists.

For these reasons, new approaches in treating PTS were first presented in 1992. Starting from the theory that the etiology of PTS is very closely associated with changes in and obstruction of spinal pathways due to several factors, such as arachnoiditis or kyphoscoliosis, the aim of operative treatment should be an attempt to re-establish the original spinal fluid flow conditions [21].

Materials and methods

We report 30 patients treated in our department over a 9-year period. Half of the patients had suffered traffic accidents. The period of time between trauma and clinical deterioration caused by syringomyelia was between 7 months and 20 years, with an average of 3.5 years. In all cases, operative treatment was performed shortly after diagnosis. In our population, we treated 21 male and 9 female patients, with an average age of 41 (range 29–70) years at the time of accident. The average age at which syrinx was detected was 44.5 years.

The first postoperative magnetic resonance imaging (MRI) study was performed 1 week after surgery. Clinical examinations with MRI were then conducted at 6 months and subsequently once a year.

We performed 40 operations in 30 patients. Fourteen patients were operated once by single shunting, four patients were shunted two times, five patients had to be operated by pseudomeningocele after the shunt systems proved insufficient (one patient who initially underwent implantation of a syringosubarachnoid, and later a syringoperitoneal shunt system before pseudomeningocele). Seven patients were operated by single pseudomeningocele (group III). Single or multiple shunting procedures are listed in group I, patients with shunting procedures before pseudomeningocele are listed in group II.

Statistical analysis was done by chi-square testing. We determined pre- and postoperative values for pain and for motor and sensory deficits, and compared them; we took the last regular examination for postoperative classification. The individual pain intensity before and after operation was determined according to a numeric rating scale from 0 to 10 points, motor deficits were scaled by muscle strength, grading from 0 (no contraction) to 5 (normal strength) and sensory deficits were classified according to the categories present/absent and improvement, deterioration or no change.

Operative treatment

The level of pseudomeningocele should be at the level of the lesion: the caudal part of the syrinx should be reached in cases of superior extension. If the syrinx extends downwards, the site of pseudomeningocele should be at the level of the lesion, in contact with the cranial part of the cavity. In selected cases of downward extension, especially when the syrinx is far from the site of the lesion, the level of pseudomeningocele should be chosen according to the presence of septations, neurological state or thickness of the cavity. At the beginning of the operation, a laminectomy over two segments is performed. The bony defect must be enlarged if the arachnoid adhesions extend further up- or downwards from the level of lesion. In our experience, a laminectomy at two levels provides sufficient bony decompression for the subsequent performance of an artificial meningocele.

After this procedure, the dura is microsurgically opened at the midline and the edges are sutured back to the muscle. The arachnoid adhesions are then untethered, and the subarachnoid spaces must be opened wide to allow a free flow of cerebro-spinal fluid (CSF) around the lesion. Another possibility is to suture the fixed and thickened arachnoid to the inner surface of the dura. The next step is the opening of the syrinx: we prefer the intermediolateral sulcus, as an intended side effect in pain reduction. The syrinx can be opened by laser or conventionally. The implantation of additional syringosubarachnoid shunt systems should be avoided because of possible induction of new adhesions by the synthetic material. At this point, the whole cavity is covered by a piece of artificial dura substitute, to create a pseudomeningocele around the lesion. A median stitch from substitute to the muscle should prevent a collapse of the cavity.

Case reports

Case 1

In September 1975, a 35-year old man had an agricultural accident resulting in incomplete paraplegia. He suffered from spasticity of both legs, with a left-sided predominance. Until July 1993, the sensory deficit was below T9.

When we saw him first, in December 1993, he reported increasing numbness of the lower extremities and pain sensations in the whole of both legs. In a lying position, the patient was not able to lift his left leg, extension movements of the knee were not possible. MRI showed PST extending from T4 to T10, with a maximum diameter at T8 (Fig. 1). We operated on the patient in December 1993, with a syringopleural shunt at T8. After leaving the

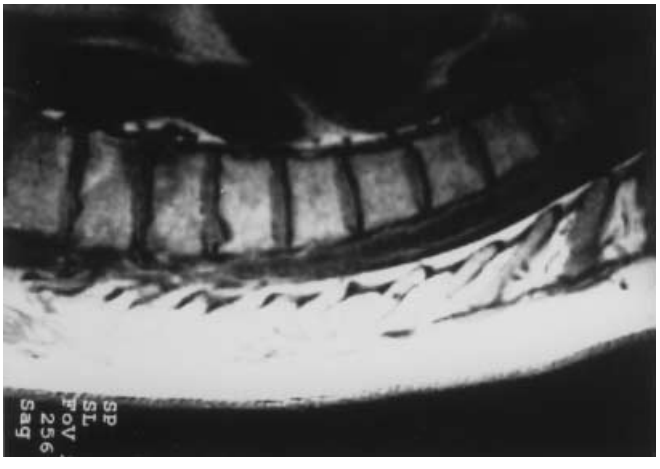

Fig. 1 Thoracic post-traumatic syringomyelia (PTS) 18 years after trauma

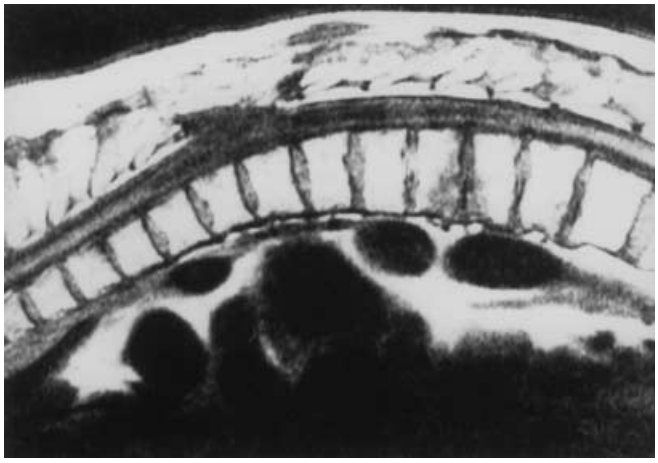

Fig. 2 Postoperative conditions after pseudomeningocele

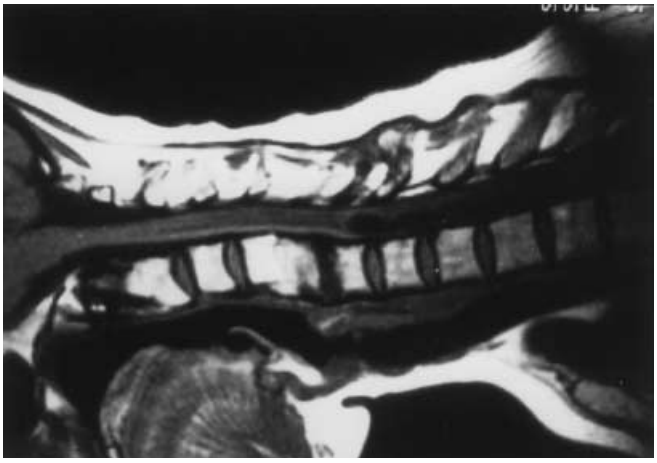

Fig. 3 Cervical PTS extending from T11 to C6

hospital, the patient described an improvement in numbness; sensory levels were nearly normal, except below the left knee, and no pain sensations remained. The power of the left leg had improved: the patient could lift his leg about 30°. An MRI examination in June 1994 showed a slight increase in the width of the syrinx, but the patient was in a neurologically steady state. No operative consequence followed. Over the next 4 months, he developed an increasingly severe burning pain, with left-sided predominance, below the mammilarian line, and numbness below the knee on both legs.

We performed repeat surgery in October 1994, with pseudomeningocele at the same level as the previous operation (Fig. 2). On external examination, the shunt was blocked.

When the patient left the clinic, he reported a definite improvement in pain sensations, while his neurological status remained unchanged. The patient has been in a neurologically steady state for 4 years.

Case 2

The patient suffered a car accident with complete paraplegia as a result of a fracture-dislocation at T11, in October 1976, at the age of 36 years. No initial stabilisation was performed, and a severe obstruction of the spinal pathways with scars and adhesions at the site of the lesion developed. In this case, the period between trauma and first signs of syringomyelia was 14.5 years. In 1989, a spinal disc prolapse at C4/5 led to an anterior stabilisation. In March 1991, additional neurological deficits above the lesion appeared. Loss of proximal power of the right arm and right-sided paresthesia at C6 developed, and MRI showed a PST extending from the site of the lesion to C6 (Fig. 3, Fig. 4). We operated on the patient in March 1991, with syringosubarachnoid shunting at the level of maximum diameter, at T8/9, and the neurological deficits disappeared. Postoperative tethering of the spinal cord followed by new neurological deficits could develop after secondary stabilisation, so an operative attempt to straighten the bony deformations was not undertaken. The first operation showed no significant post-traumatic scars around the lesion, so in our opinion the fluid flow seemed to be guaranteed. Under those circumstances, a syringosubarachnoid shunt system seemed to be the treatment of choice. The same deficits re-appeared in April 1994, so a syringoperitoneal shunt at T8/9 and a bony decompression of both C6 nerve roots were performed.

After a brief intermittent improvement, a loss of muscle strength in both arms with a rapid deterioration in sensory levels happened in February 1997.

Fig. 4 Fracture-dislocation at T11 in a 36-year-old patient

The MRI study showed a recurrence of syringomyelia, and a pseudomeningocele at T8/9 was performed. The postoperative control showed a decrease in diameter of the syrinx and an extension below C6. Unfortunately, a local infection led to severe adhesions inside the artificial liquor reservoir 4 months later, so another cavity at T2/3 was necessary. Muscle power and sensibility improved, and her preoperative state returned slowly.

Results

Twenty-four of our patients had neurologically complete lesions; six were incomplete. In ten cases, the level of the lesion was located at the inferior cervical spine; the superior thoracic spine was affected in eight cases. Twelve spinal traumas were located in the middle and lower thoracic spine. In 18 patients, the syringomyelia extended cranially and caudally from the level of lesion; single superior and inferior extensions were seen in 15 and 6 cases respectively.

Twenty-seven patients had sensory deficits, a dissociation between pain and proprioception was recorded in

25 patients. Motor deficits were always associated with pain or sensory loss. Pain was registered in 18 patients, brain stem symptoms were seen in four patients. There were no patients with hyperhydrosis or neuropathic joints.

Overall, we implanted 22 syringosubarachnoid and 4 syringoperitoneal shunts and 1 syringopleural shunt in 23 patients (groups I and II). There was no significant difference in outcome between the different shunting procedures; none was superior to any other. The median follow-up time for syringosubarachnoid shunts was 7.3 years, for syringoperitoneal 4.5 years, for syringopleural 6.1 years and for pseudomeningoceles 3.8 years. The immediate complication rate with hematomas, infections, and shunt dislocation and disconnection was 12%. A pseudomeningocele was performed in 12 cases; three patients had previously been operated by syringosubarachnoid shunting, one by syringopleural shunting and one patient had been operated first by syringosubarachnoid shunting, second by syringopleural shunting, and third by pseudomeningocele, when the syrinx appeared again. The patients operated by shunting procedures are summarized in group II. All of them had sensory deficits or paresis due to syringomyelia, one suffered pain. One patient with syringobulbia showed a postoperative improvement (see Table 2). In the first years, four patients were shunted twice because of recurrence of syringomyelia; these patients are listed in group I. For shunting procedures, a neurological improvement was obtained in five patients; eight patients are in a neurologically steady state. Fifteen patients had sensory deficits preoperatively, 13 suffered from motor deficits and 14 patients suffered pain. An improvement in all qualities (pain, sensory and motor deficits, syringobulbia) was registered in five cases, a postoperative deterioration in motor deficits and pain was recorded in four patients (see Table 1).

Table 1 Group I. Outcome of patients treated by shunting procedures (*n*=18, median follow-up: 6.7 years)

	Preoperative status	Postoperative improvement	Postoperative deterioration	No change
Sensory deficit	15	5	2	8
Motor deficit	13	5	4	4
Pain	14	5	4	5
Syringobulbia	2	1	1	–

Table 2 Group II. Outcome of patients treated by shunting procedures and pseudomeningocele (*n*=5, median follow up: 4.3 years)

	Preoperative status	Postoperative improvement	Postoperative deterioration	No change
Sensory deficit	5	4	1	–
Motor deficit	5	3	1	1
Pain	1	1	–	–
Syringobulbia	1	1	–	–

Table 3 Group III. Outcome of patients treated by pseudomeningocele (*n*=7, median follow up 3.8 years)

	Preoperative status	Postoperative improvement	Postoperative deterioration	No change
Sensory deficit	7	6	–	1
Motor deficit	6	2	1	3
Pain	3	3	–	–
Syringobulbia	1	1	–	–

We succeeded in achieving a neurological improvement in ten patients treated with a pseudomeningocele, four of whom had been previously operated by shunting procedures. Five patients showed no improvement or deterioration after operation. The mean follow-up time for these patients is now 4.3 (group II) and 3.8 (group III) years. Table 2 and Table 3 show the different courses of neurological deficits after operation. Sensory deficits were improved in 10 of 12 patients, motor deficits only in 5 of 11 patients (groups II and III). Of all patients, overall improvement was achieved in 15 patients, 10 are neurologically stable, further neurological deterioration happened in 4 patients, while 1 tetraplegic 74-year-old woman died of pneumonia 4 weeks after operation.

The statistical comparison between shunting procedures, shunting procedures with secondary performance of pseudomeningocele and pseudomeningocele alone without additional shunting procedures showed a discrete improvement in sensory deficits and postoperative pain in those treated with pseudomeningocele in comparison to shunting procedures. This was evident whether or not a shunt was implanted before the pseudomeningocele operation. Nevertheless, we conducted a separate statistical analysis of groups II and III, because some benefits, such as possible function of the catheter as a stent, or disadvantages of shunting, such as arachnoid adhesions induced by the artificial material, could influence the results of treatment with pseudomeningocele. The statistical analysis of the parameters pain, motor deficit and sensory deficit showed no significant differences between the three groups (comparison of I/II, I/III and II/III).

Discussion

PTS is a relatively uncommon disease. The incidence of PTS in the present literature varies between 0.3 and 3.2%. The interval between spinal trauma and syringomyelia is variable; periods between 2 months and 32 years have been described [2]. Nevertheless, it can have serious consequences for every para- or tetraplegic patient. Even small neurological deteriorations can lead to potentially significant impairment in daily life. Immediate operative treatment should be performed when there are signs of neurological deterioration, but there are still different opinions regarding methods of surgical treatment. Cord

transection is an effective way of treating complete lesions [10]. The most common therapy in the last years comprised implantation of shunt systems, but this method has its own complications [10]. Very often, an initial neurological improvement is followed by further deterioration, so it is unclear whether this improvement is a consequence of initial decompression of the surrounding spinal cord tissue at the site of lesion or of the shunt system itself.

The most common clinical feature in the literature is pain; provocation by straining, coughing, sneezing or other mechanical influence is possible [17]. The nature of the pain varies. The second most common feature is sensory deficits, especially dissociation between pain and proprioception. Other deficits involve vibration and touch sensation. Motor deficits are the third most common feature of PTS, normally associated with pain sensations or sensory deficits. Rare clinical signs are hyperhydrosis, brain stem deficits, Horner's syndrome, urinary involvement or neuropathic joints [2, 13,17].

Opinions still differ regarding the pathogenesis of PTS. Several theories have been proposed [7, 15, 20, 22], the most accepted one in recent years was presented by Bernard Williams in 1992 [21]. According to his theory of hydrodynamic forces, post-traumatic cavities in the spinal cord are initially determined by liquefaction of the cord tissue or hematoma at the site of the lesion [4,7]. Additional positive factors might consist in the activity of lysosomal and other cellular enzymes or microinfarcts [8,20].

In the second step, extension and enlargement of the syrinx might occur by transmission of energy pulses to the spinal cord due to intra-abdominal or intrapleural pressure elevations, with consequential fluid movements in the preformed cavity and in the subarachnoid space. Other studies support the importance of subarachnoid blockage by arachnoiditis in initiating the extension of a syringomyelia cavity [3,16].

The routine use of MRI follow-up studies showed new aspects in pathogenesis [5]: some authors found a synchronization between flow in the syrinx and cardiac cycle, rather than the respiratory cycle, and a caudal direction of CSF flow in the syrinx during systole and rostral CSF flow during diastole was reported [12]. In this study, the flow of CSF in the subarachnoid space was faster than in the syrinx.

An important question in operative treatment is whether the syrinx should be opened or not. Several authors are against opening a syrinx in treatments with a pseudomeningocele [21,22], because the filling mechanism will be interrupted by treating the underlying pathology, and the consequence is a collapse of the syrinx. In our opinion, neurological deficits disappear faster if the cavity is opened.

We do not agree with Williams' opinion that the dura should be left open to allow a large meningocele to develop beneath the muscles. The most important consideration in our opinion is the development of new scars and adhesions by blood or fibrinogenic material around the spinal cord if the dura is not closed. This point is supported by the often enormous vasculature of fractured bone, muscle and soft tissue at the level of a former spinal trauma. A collapse of the dura substitute can be prevented by median stitches from substitute to the surrounding muscles.

There are no concrete studies concerning the value of initial stabilisation of kyphoscoliosis after spinal trauma and PTS. In the past, a reserved attitude regarding initial stabilisation, especially in complete lesions, has been the rule. There is no doubt about the role of initial prevention of PTS, consisting in attempts to reduce residual deformity with postoperative spinal canal stenosis and post-traumatic arachnoiditis [1,9]. Our experiences in secondary stabilisations showed a neurological deterioration after operation in several cases.

Arachnoid adhesions and scars develop as a consequence of change in bony deformities. New tethering of the cord after initial establishment of changed spinal pathways is the consequence in rare cases. Further studies are needed in order to formulate concrete recommendations in this regard.

A more complete understanding of the etiology and pathomechanism of PTS is also key to establishing an adequate treatment. However, despite the lack of clarity over the pathology of PTS, there is common agreement on a close correlation between change in spinal fluid flow conditions and extension or expansion of a syrinx. The performance of a pseudomeningocele is, therefore, a justified method of treatment. However, a final assessment should only be made after long-term follow-up.

The interval between trauma and clinical signs of PTS can be longer than 30 years, so a final outcome can not be reported after a shorter follow-up. A long period of surveillance with annual follow-up controls by clinical examination and MRI every year is a must. In conclusion, performing a pseudomeningocele is an encouraging beginning in the treatment of PTS, but further long-term follow-ups are necessary to estimate the benefits of this method.

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