Paraplegia **26** (1988) 56–61

Evolution of Post-Traumatic Cervical Syringomyelia: Case Report

I. Umbach, MD, A. Heilporn, MD
Center for Traumatology and Rehabilitation, Brugmann Hospital, pl. A. Van Gehuchten 4, 1020 Brussels, Belgium

Summary

A patient with complete post-traumatic paraplegia below T6 developed urinary problems and late secondary syringolmyelia. The concordance between the appearance of micturition difficulties and the first sensory symptoms leads us to discuss the rôle of important and repeated efforts to obtain reflex micturition, during the development of post-traumatic syringomyelia.
Key words: *Paraplegia; Post-traumatic syringomyelia; Delayed difficulty with micturition.*

A recent patient with post-traumatic paraplegia with secondary syringomyelia caused us to consider the factors which may intervene in the genesis of such a complication.

Case report

A male patient aged 30 years had a car accident in 1972. He was comatose for 3 days, and had a compression fracture of the 6th and 7th thoracic vertebrae with paraplegia, fractures of 3 ribs on the right side and a fracture of the right clavicle. The vertebral fractures were treated by an orthopaedic surgeon. There was no neurological recovery. After rehabilitation the patient was independent for daily life activities in a wheelchair; micturation was achieved by reflex stimulation.

After a few years, he developed increasing urinary problems: micturition became more and more difficult and very firm suprapubic pressure was necessary to empty the bladder. Recurrent urinary infections occurred, and later incontinence.

In 1981 he was admitted to hospital for these problems. At that time he had

sensory changes in the right upper limb: thermo-algesic anaesthesia with normal tactile sensation.

A C.T. examination after myelography showed slight enlargement of the cervical spinal cord. No particular treatment or follow up was done.

In February 1984 the patient complained of pain in his right arm, followed by progressive deformation of the right shoulder, without any evidence of trauma. Radiography showed neuropathic arthropathy of the right shoulder.

In December 1984 the patient was admitted to the C.T.R. for treatment of urinary, neurological and joint complications. Examination showed: a complete sensorimotor paraplegia below T6 with a moderate degree of spasticity, thermo-algesic anaesthesia and loss of vibratory sense from C3 to T6 on the right side, areflexia of both upper limbs, normal muscular strength, and joint effusion of the right shoulder with painless deformation and chronic subluxation. Bilateral equinism of the feet, and a necrotic wound of the right trochanteric region were also noted. Micturition was very difficult, needing prolonged percussion and heavy suprapubic pressure.

Urography showed bilateral hydronephrosis and a spastic distorted bladder.

The urinary complications were treated by transurethral resection of the bladder neck and by bilateral ureterovesical reimplantation.

X-Rays of the right shoulder showed destruction of the head of the humerus, enlargement of the glenoid cavity and joint dislocation.

A C.T. of the cervical spine with Metrizamide showed a large intramedullary cyst extending from C2 to the upper thoracic region (Fig. 1). Its diameter was

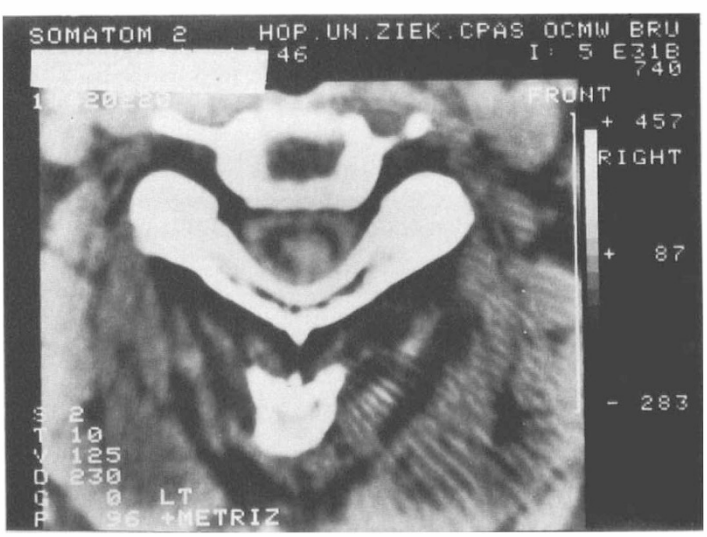

Figure 1 C.T. of the cervical spine: cyst filled with Metrizamide.

greatest at the C5 level where it was 7 mm. There was also some atrophy of the medullary tissue at the C2–C3 level.

A N.M.R. examination showed a large syringomyelic cavity whose upper limit

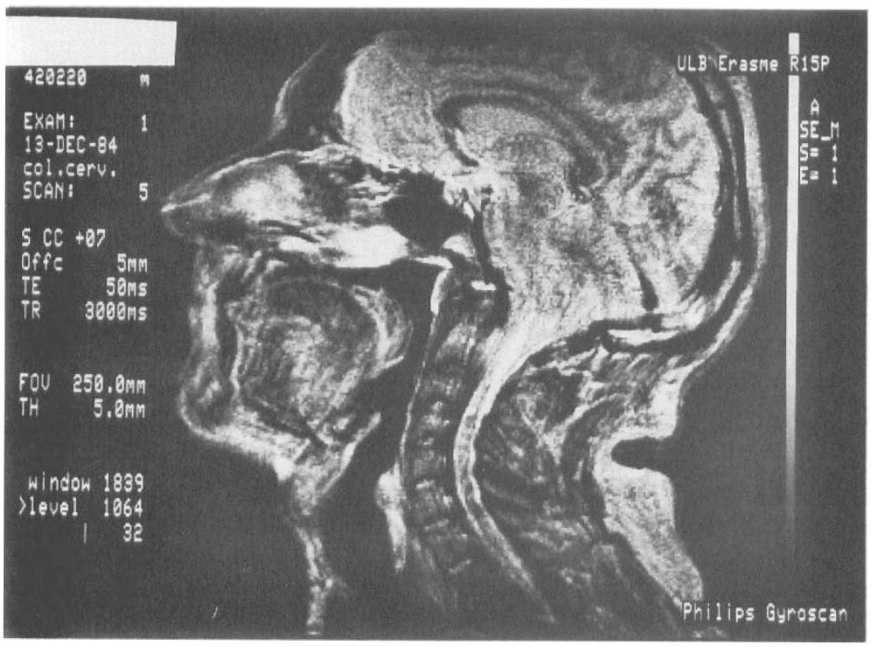

Figure 2 Pre-operative N.M.R. examination.

was at the level of the bulbomedullary junction. There was no associated Arnold-Chiari malformation (Fig. 2).

The patient was operated on in April 1985. After incision of the medullary cavity, a Holter catheter was inserted in a caudal direction for a length of 7 to 8 mm; it was cut and stitched to the arachnoid with a 6/0 silk suture. The catheter was cut short so that it was able to follow the movements of the spinal cord. There were no postoperative complications.

Neurological examination showed no deterioration in the ensuing months, but there was subjective improvement: the patient stated that he had more strength and function of his upper limbs.

A comparative N.M.R., 3 months later, showed shrinking of the cyst, whose cranial end was now at the C3 level while before it had extended to C1. The cavity was irregular, did no longer seem to be under pressure and was even locally collapsed (Fig. 3).

Post-traumatic syringomyelia: generalities

Late post-traumatic syringomyelia or cystic myelopathy is now a well recognised entity.

The incidence of this rather rare late complication of paraplegia is estimated in the literature to be about 1.3%, which is much higher than the incidence of primary syringomyelia: about 0.01% (Barnett, 1966; Vernon, 1982). In a recent publication an even higher incidence was noted: 3.2%, but in the case of complete paraplegia the figures reached those noted in the literature of 1.4% (Rossier, 1985).

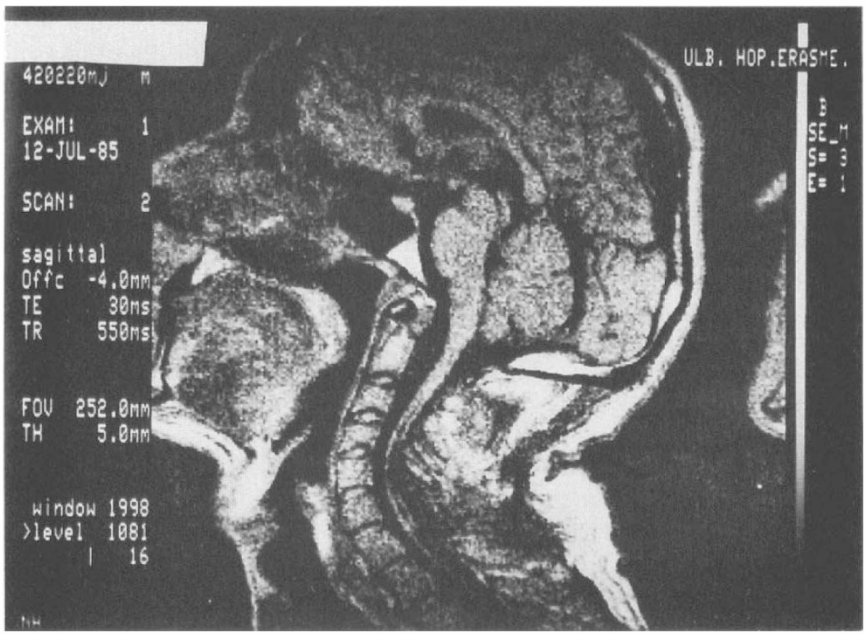

Figure 3 Post-operative N.M.R. examination.

The delay for the first symptoms to appear varies widely, from a few weeks to several years; a delay of more than 30 years has been found in several series (Vernon, 1982; Rossier, 1985).

The symptoms are similar to those of primary syringomyelia, with pain increasing on effort, dissociated sensory loss, muscular weakness, and depressed reflexes in the upper limbs.

In the spinal cord the lesion is often localised initially in the grey matter between the posterior horn and the posterior column, which is a zone of weak supportive tissue at the limit between anterior and posterior vascular supply.

The neurological deficit is either continuous, or suspended with a zone of normal sensation between the upper level of the paraplegia and the sensory loss due to syringomyelia. The symptoms are often unilateral or asymetric.

The evolution of the cyst if unforeseeable, varies between progressive extension of the lesion and stabilisation of symptoms for months or years; however no spontaneous remission has ever been reported to our knowledge.

Treatment is either conservative, or surgical with drainage of the cyst by various techniques.

Discussion

Although many studies have been reported on this subject, the etiopathogenesis of post-traumatic syringomyelia has not yet been determined with certainty. Different theories have been proposed as to the origin of the cavity and its extension.

The following mechanisms have been put forward to explain the initial cavitation:

1. A vascular ischaemic factor, with small zones of infarction leading to secondary cavitation (Williams, 1981) or cell lesions producing late necrotic degeneration (Schott, 1962). The localisation of the cyst at the junction of anterior and posterior vascular territories may—at least partially—support the ischaemic theory.
2. A possibly associated second traumatism in the cervical region, which may be infraclinic, has also been suggested. Syringomyelia has been described after minor cervical traumatism with slight neurological involvement (Lacert, 1977; Maury, 1964).
3. Mechanisms secondary to the initial traumatism have also been discussed:
   a) biochemical activity of the lysosomes at the site of the spinal cord lesion, which may lead to microcysts (Kao);
   b) late resorption of a haematoma after liquefaction of the clot, leaving a cavity (Williams, 1981).
4. Some publications have put forward the possible rôle of the medullary concussion with cellular destruction and secondary vasopathy (Schott, 1962), and also the possible rôle of post-traumatic oedema (Feigin, 1971).
5. Others refer to the rôle of tension and torsion applied to the cervical spinal cord by movements of the neck, which are much more frequent and important in the case of paraplegia. (Barnett, 1966; Lacert, 1966).
6. Arachnoiditis was also considered as a possible cause of cystic myelopathy, for example in non-traumatic cases such as tuberculosis.

The mechanisms of extension of the cavity seem to be mainly related to pressure changes in the venous system, by the action of intraspinal veins on CSF pressure. Any effort which increases the pressure in the abdomen and thorax leads to the distension of epidural veins which compress the dura and force the CSF upwards to the head. The upward movement of CSF which is short and energetic is followed by a slower movement downwards, with appearing of a temporary intraspinal pressure dissociation. Those pressure variations and movements are transmitted to any fluid containing cavity in the spinal cord, and contribute to its increase in volume (Williams, 1981).

Those mechanisms may be reinforced by partial or complete obstruction of the subarachnoid space at the level of the traumatism.

Whatever the cause of initial cavitation might be, it seems most probable that its expansion is secondary to pressure variations in the venous system and in the CSF, during efforts that increase the pressure in the abdomen and thorax. Such efforts are very frequent during current activities of paraplegics in wheelchair.

The rôle of increased mobilisation of the upper part of the body during rehabilitation and sport activities has already been discussed by many authors.

Another cause of repeated increase of venous pressure has perhaps not been emphasised enough: the rôle of the effort of micturition, especially if there are urological complications such as chronic infections, or hypertrophy of the bladder neck, which demand more forceful and longerlasting efforts.

Although no relationship between syringomyelia and urinary problems has been described so far, we believe that micturition problems may play a role in

the evolution of secondary syringomyelia, making it therefore important to diagnose and treat any urinary complication early.

Conclusion

The purpose of our presentation is to emphasise restraints imposed by paraplegia, which may possibly lead to the development of post-traumatic or secondary syringomyelia, and among these, urinary complications should certainly not be neglected.

Acknowledgement

The authors wish to thank Professor Brotchi (Neurosurgery) and Dr Baleriaux (Radiology) of the Erasmus Hospital of the U.L.B. Brussels and Dr Rakofsky of the Brugmann Hospital, Brussels, for their useful help.

References

BARNETT HJM, BOTTERELL EH, JOUSSE AT, *et al.* 1966 Progressive myelopathy as a sequel to traumatic paraplegia. *Brain* **89**:159–174.

FEIGIN I, OGATA J, BUDZILOVICH G 1971 Syringomyelia: the rôle of edema in its pathogenesis. *Journal of Neuropathology and Experimental Neurology*. **30**:216–232.

KAO CC, CHANG LW 1977 The mechanism of spinal cord cavitation following spinal cord transsection. Part I: A correlated histochemical study. *Journal of Neurosurgery*. **46**:197–209.

LE MOUEL MA, NORMAN J 1981 Syndromes syringomyéliques tardifs in MAURY *La Paraplégie*, Flammarion, Paris, p. 632–637.

MARTIN L, MAURY M 1964 Syndromes syringomyéliques après paraplégie traumatique. A propos de six cas de syndrome syringomyélique cervical survenant après une paraplégie dorsale ou lombaire. *Presse med.* **72**:2839.

ROSSIER AB, FOO D, SHILLITO J, *et al.* 1985 Post-traumatic cervical syringomyelia: incidence, clinical presentation, electrophysiological studies, syrinx protein and results of conservative and operative treatment. *Brain* **108 Part II**:439–462.

SCHOTT B, TRILLET M, VAUTERIN C *et* KOSHBIN 1962 Syndromes syringomyéliques tardifs suslésionnels après traumatisme médullaire (à propos de trois observations cliniques). *Revue Neurologique* **106**:751–755.

VERNON JD, SILVER JR, OHRY A 1982 Post-traumatic syringomyelia. *Paraplegia* **20**:339–364.

WILLIAMS B, TERRY AF, JONES F, *et al.* 1981 Syringomyelia as a sequel to traumatic paraplegia. *Paraplegia* **19**:67–80.