SURGICAL TREATMENT OF A PATIENT WITH POSTTRAUMATIC SYRINGOMYELIA ASSOCIATED WITH COMPLEX POSTTRAUMATIC DEFORMITY OF THE SPINE

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A clinical case of surgical treatment of a female patient with post-traumatic syringomyelia which led to tetraparesis and dissociated sensory loss in the trunk and upper and lower limbs is presented. Clinical manifestations of these spinal cord changes occurred 21 years after complicated fracture of the L1 vertebra associated with complex posttraumatic spinal deformity.
Multistage surgical intervention made it possible to restore liquorodynamics, perform the necessary correction of severe kyphotic deformity of the spine, and reduce the risk of torso imbalance. As a result, the syrinx practically disappeared at all levels of the study.
Key Words: syringomyelia, posttraumatic syringomyelia, posttraumatic deformity.

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Posttraumatic syringomyelia is a chronic, slowly progressive disease, which is based on the formation of cavities in the affected area of the spinal cord containing fluid that can subsequently spread to other segments. Symptoms may appear several months or decades after an injury, when a patient considers himself already recovered. Progression of the disease can lead to increase in neurological deficits in the long-term period after the trauma.

Widespread introduction of MRI to visualize the spinal cord resulted in significant increase in the number of detected cases, and consequently the number of patients who underwent surgical treatment for posttraumatic syringomyelia. According to studies conducted on a large number of patients with severe spinal cord trauma using spinal injury registries in different countries, the likelihood of development of clinically significant syringomyelia is 2.0–4.7 %, and that of clinically insignificant syringomyelia, up to 28.0 % [1, 5, 8, 12, 14, 19, 24].

Posttraumatic arachnopathy at the level of damage and formation of bone block are believed to disrupt the cerebrospinal fluid flow and act as triggers for development of syringomyelia [4, 14, 15, 19]. In case of progression of neurological deficit, which occurs in approximately 10 % of patients with posttraumatic syringomyelia, surgical treatment is indicated. In 1980, it was proposed to use syringoperitoneal and syringopleural shunts for drainage of fluid from a cyst [21]. Later, when doctors' understanding of the causes of syringomyelia changed, it has become more common to use interventions aimed at restoring the circulation of cerebrospinal fluid in the subarachnoid space. A number of authors [5, 7, 12, 15, 20, 21, 23] believe that it is necessary to eliminate all factors that may lead to development of posttraumatic syringomyelia (intra-extradural compression, arachnopathy). We adhere to the same views when treating this category of patients.

We present early outcomes of surgical treatment of a patient with posttraumatic syringomyelia of all parts of the spinal cord in the long-term period after a spine and spinal cord injury accompanied by persisting deformity of the spine.

In 1994, at the age of 12, female patient I. sustained a complicated compression fracture of the body of L1 vertebra in a traffic accident; it led to weakness and decreased sensitivity in both legs. L1–L2 level laminectomy and posterior decompression of the spinal cord and its roots were performed on the day of the injury. The patient still suffered from paresis of the legs and sensitivity disorders in the postoperative period. Two years after the injury, she started to experience back pain and a plate was fixed using the spinous processes of the T12–L3 vertebrae. In the postoperative period, the patient noted a decrease in the pain intensity in the lumbar region, increased strength and improved sensitivity in both legs; she adapted to the upright position and was able to move

Fig. 1
Patient I.'s hand: pronounced muscle atrophy and clawed paw type deformity, characteristic of the syringomyelia of the cervical spinal cord

independently with the help of crutches. In December 2013, she noted weakness in both arms, a sensitivity disorder in the left half of the trunk and double vision. Conservative treatment led to regression of double vision, but the loss of sensitivity and weakness in the arms and legs progressed. The patient repeatedly turned to specialists, however, given the level of the spinal trauma, these changes were regarded as manifestations of depression and mental disorder. Despite ongoing conservative therapy, the progression of clinical manifestations continued. By 2015, paresis in legs and hands increased significantly, hand muscles atrophied, and the hands started to look as a clawed paw (Fig. 1). There was pain and temperature sensitivity disorder in the upper and lower extremities, in the lower third of the chest and in the abdomen on the left. The patient lost the ability to move around independently in a wheelchair and to work.

Upon admission to the N.I. Pirogov National Medical and Surgical Center on February 6, 2014, she complained of pain in the lumbar region, weakness and decreased sensitivity in both arms and legs, pain and temperature sensitivity disorders in the arms and legs, in the lower third of the chest and in the abdomen on the left, difficulty urinating due to retention.

Objectively: somatic status without any special features. Karnofsky score of 60 points.

Neurological status: consciousness is clear, oriented, adequate. Pupils OS = OD, live photoreaction and corneal reflexes. There are no oculomotor disorders and nystagmus. The face is symmetrical, the tongue is on the middle line. Tendon reflexes of the hands are drastically lower on the right, live on the left; knee reflexes of the legs are symmetric, lowered, Achilles reflexes are absent. Right arm paresis: 4–5 points in the proximal sections, 2–3 points in the hand; left arm paresis: 2–3 points in the hand. Paresis in the right leg: up to 4–5 points in the proximal sections and up to 1 point in the foot; left leg paresis: up to 2–3 points in the foot. Atrophy of the muscles of hands, hips, shins and feet (more pronounced on the right). Dissociated sensitivity disorders of 'gloves and socks' type, hypostasis at the level of T6–L2 on the left. Babinski's sign is negative on both sides. Finger-to-nose and heel-to-sheen tests are performed with intention tremor and past-pointing. The function of the pelvic organs is impaired due to retention.

Neuro-orthopedic status: the patient moves in a wheelchair. Kyphotic deformity of the spinal column with the apex at T12–L2 level. Postoperative scar in the thoracolumbar region without signs of inflammation. The long back muscles are tense at the level of T11–L3 vertebrae. Palpation and percussion of paravertebral points and spinous processes are painless.

MRI of the thoracolumbar spine revealed signs of syringomyelia of all parts of the spinal cord (C1–L1), wedge deformity of the L1 vertebra, posttraumatic kyphotic deformity of the spine, polyfactorial stenosis of the spinal canal (Fig. 2).

CT of the thoracolumbar spine revealed a consolidated fracture of the L1 vertebra with the formation of a gross posttraumatic and kyphotic deformity (Fig. 3).

Given the presence of syringomyelia and the progression of neurological disorders, the patient underwent a multistage surgery on 08.12.2015 (Fig. 4):

1) stage 1: resection of the L1 vertebral body, anterior decompression of the spinal cord and its roots, mobilization of the spine;

2) stage 2: removal of metal structures, installation of screws, posterior internal correction and fixation of the spine with a transpedicular system, followed up by intraoperative sonography, which determined the zone of syringomyelia and the level of cicatricial changes;

3) stage 3: removal of the spinal cord fixation at the level of T11–L2 vertebrae, plastic surgery of dura mater at this level (Fig. 5);

4) stage 4: anterior interbody fusion using a mesh cylindrical implant with autobone.

Control examination 6 months after the surgery showed increased strength in the arms up to 4–5 points, in the proximal sections of the right leg, up to 4 points, of the left leg, up to 3 points.

MRI of cervical, thoracic and lumbar spine 4 months after the surgery showed significant reduction in the size of the syringomyelitic cyst throughout the entire spinal cord (Fig. 6).

CT of the lumbar spine showed that the position of the metal structures and the sagittal profile of the spine are satis-

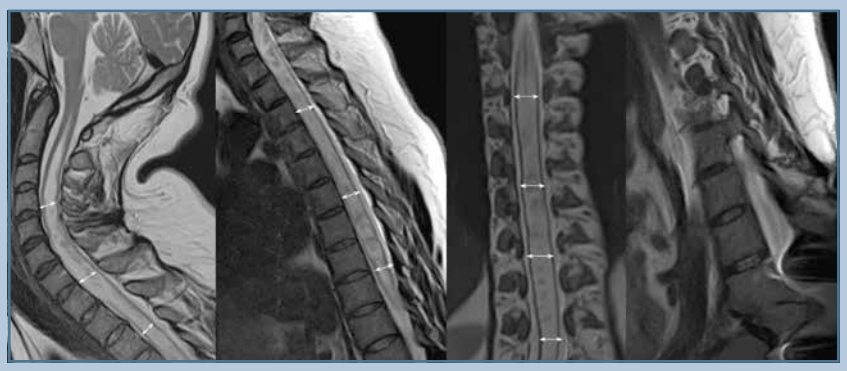

Fig. 2
T2-weighted MRI images of the cervical and thoracic spine of the patient I.: signs of syringomyelia at all examined levels (C1–L1); diameter of cysts 8 to 11 mm in different parts of the spinal cord; maximum Vakuero index 84.6 % in the cervical spine

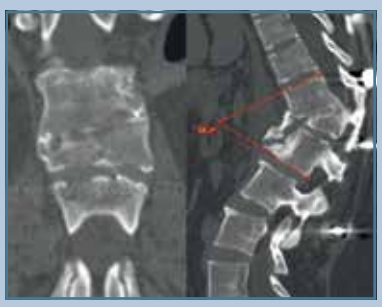

Рис. 3
CT of the lower thoracic and lumbar spine of the patient I.: formed bone block at the level of L1–L3 vertebrae, wedge deformity of L1 vertebra, posttraumatic kyphotic deformity of spine (local kyphosis 58°), posttraumatic spinal stenosis, condition after the T11–L3 fusion with interspinous metal implants

factory. There are no signs of narrowing of the vertebral canal (Fig. 7).

Discussion

The result of surgical treatment confirms the effectiveness of pathogenetic therapy aimed at the recovery of liquorodynamics and elimination of all compression factors in the treatment of patients with syringomyelia.

Most researchers adhere to the theory that syringomyelia is caused by partial or complete disruption of the cerebrospinal fluid flow in the subarachnoid space due to posttraumatic arachnopathy at the level of injury and (or) direct intra- or extradural compression [2, 3, 9, 15, 17, 20, 22, 23]. In this case, the development of syringomyelia increases the intramedullary pressure, which leads to further trauma to the spinal cord and the progression of the neurological deficit.

Therefore, surgical interventions aimed at eliminating the causes of the cyst formation have an advantage over those aimed at draining the existing cyst. For example, an assessment of shunting operations outcomes conducted five or more years after the surgery showed that 80 % of patients required repeated surgical interventions, and more than 40 % of patients experienced progression of neurological disorders. Only in 3 % of cases did the effect persist five years after surgery [7, 15, 19, 24]. Reconstruction of the subarachnoid space allows achieving better clinical results and reduces the number of repeated operations. At the same time, a number of authors [5, 6, 15, 23] note the inadequacy of removal of the spinal cord fixation and plastic surgery of the dura mater in case of persistent narrowing of the vertebral canal or pronounced kyphotic deformity of the spine. Such patients require a comprehensive approach, which involves restoration of the sagittal profile, elimination of factors leading to the narrowing of the spinal canal and recovery of the cerebrospinal fluid circulation. Unfortunately, there are very few publications devoted to posttraumatic syringomyelia, and they are limited to a small number of observations (less than 10).

The techniques of the proposed surgeries differ significantly from each other.

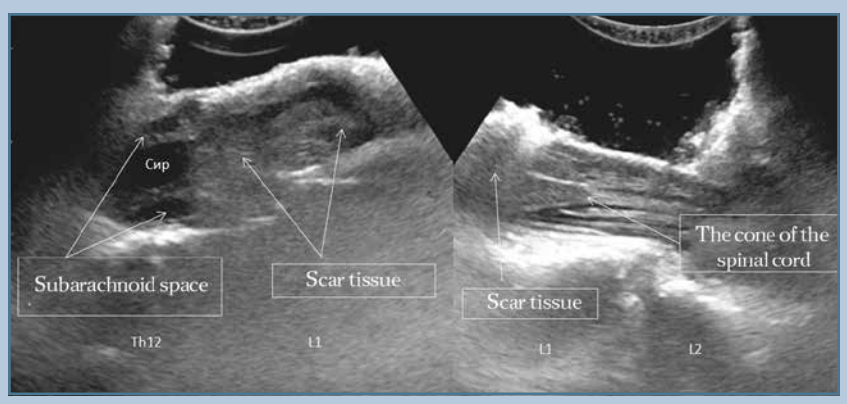

Fig. 4
Data of intraoperative ultrasound examinations of the patient I.

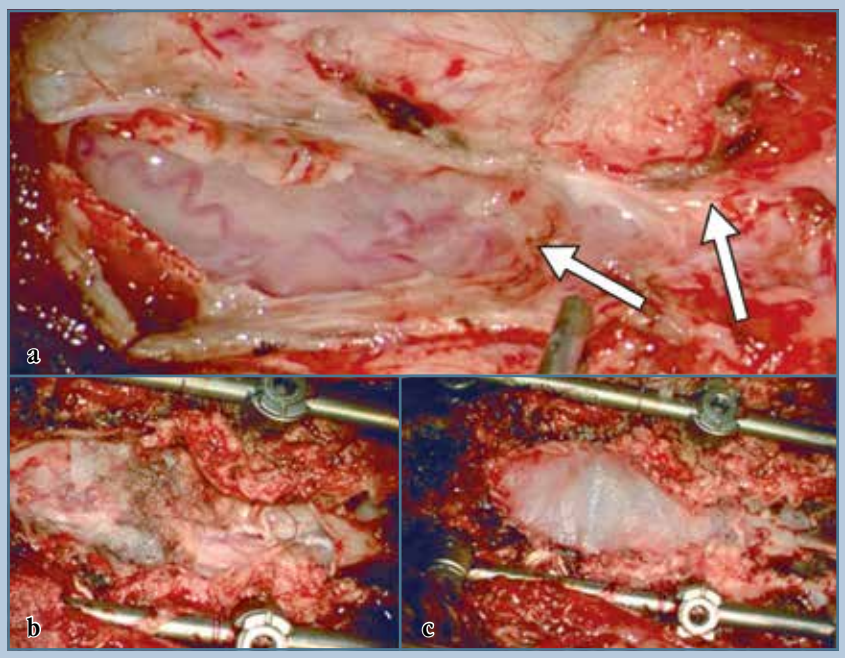

Fig. 5
Intraoperative photos of the patient I.: **a** – pronounced arachnopathy (Klekamp type 2) at the level of T12–L2 vertebrae; **b** – fixation of the spinal cord at T12–L2 level was removed; **c** – expanding plastic surgery of the dura mater with an artificial sheath; arrows indicate arachnopathy zone

Fig. 6
T2-weighted MRI images of the cervical, thoracic and lumbar spine of the patient I. 4 months after the surgery: syringomyelitic cavity practically disappeared at all examined levels, there is clear flow of the cerebrospinal fluid in the region of the pre-operative fixation of the spinal cord; the maximum Vaquero index is 14.8 % in the cervical spine

Some authors suggest performing shunting operations [7, 10, 22], others propose only elimination of bone compression [10, 16, 18, 23,], while the third group proposes restoration of liquorodynamics at the level of trauma [5, 11, 12, 13, 21]. In our opinion, the most promising technique for treating such patients is the simultaneous elimination of all factors that may be the cause of syringomyelia. This clinical example is a specific demonstration of this approach in the surgical treatment of a patient with posttraumatic syringomyelia associated with a complex posttraumatic deformity of the spine.

The study did not have sponsorship. The authors state that there is no conflict of interest.

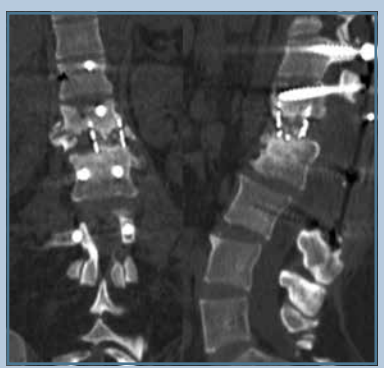

Fig. 7
CT of the lumbar spine of the patient I. after the surgery: the amount of posttraumatic kyphotic deformity of the spine decreased significantly on the sagittal reconstruction, the transpedicular screws pass through the pedicles of the arch, a mesh cylindrical implant with autobone replacing the body of the L1 vertebra was installed between the T12–L2 vertebral bodies

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