STAGED SURGICAL TREATMENT OF BRACHIOPLEXOPATHY IN AN ADOLESCENT WITH KLIPPEL – FEIL SYNDROME: A RARE CLINICAL CASE AND LITERATURE REVIEW

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Klippel — Feil syndrome is a congenital malformation, the leading component of which is a violation of segmentation of the cervical vertebral bodies. The syndrome can be combined with other skeletal anomalies: skull asymmetry, scoliosis, high shoulder blades, and cervical ribs. Treatment of the syndrome is usually symptomatic; indications for surgical treatment are progressive neurological disorders and persistent pain syndrome, which usually develop due to instability of unblocked segments, or neurogenic pain. A clinical case of treatment of a 17-year-old patient with Klippel — Feil syndrome who developed a picture of severe upper limb monoparesis during three years due to compression of the brachial plexus associated with cervical ribs is presented. Decompression of the brachial plexus was performed, which led to rapid relief of pain syndrome and gradual partial regression of motor disorders. Due to incomplete restoration of the gripping function, tendon-muscle plasty of the right hand was performed, which significantly improved the possibility of self-care. The results of radiation and staged neurophysiological studies are described, as well as a review of the literature on the Klippel — Feil syndrome.

Key Words: Klippel-Feil syndrome, brachial plexus, plexopathy, cervical ribs, surgical treatment.

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Klippel – Feil syndrome (KFS) is a congenital spine malformation whose leading component is improper segmentation of the cervical vertebral bodies. This syndrome is characterized by the typical triad of clinical signs: short neck, restricted neck movement (first of all, rotation), and low hairline at the back of the head (the so-called pterygium-like effect or webbed neck) [1]. Another typical feature of KFS involves a variety of concomitant abnormalities of the skeleton (the viscerocranium, scapulae, thoracic and lumbar vertebrae, and ribs), as well as a combination of aesthetic, orthopedic, and neurological complications. The reasons for neurological disturbances in patients with KFS usually include the concomitant congenital pathology of the spinal cord (syringomyelia, syringobulbia, split cord malformation, and cysts) and spinal stenosis at the level of the craniovertebral junction or an interblock segment with preserved mobility [2–5].

Some patients with KFS (10–15 %) have cervical ribs, which may also cause neurological symptoms caused by the brachial plexus involvement [6]. The unique nature of clinical manifestations and treatment used in these cases allowed us to present our case report.

A male patient S. was first admitted to clinical center at the age of 17 years. He complained of restricted mobility of the cervical and thoracic spine, spinal deformity, hearing loss in the right ear, and muscle weakness in the right hand gradually progressing over the past three years.

When being a newborn, the patient was diagnosed with uranostaphyloschisis and underwent an uraniscoplasty surgery at the age of 6 years. Child's motor and speech development was normal. He had no head injuries, seizures, or syncopal episodes. The patient was diagnosed with KFS at the age of 4 years according to typical clinical signs; the diagnosis was verified radiologically.

At the age of 13 years, the patient developed hearing loss. An audiogram showed grade 3 mixed hearing loss in the right ear. At the same age, the patient started experiencing gradually progressing muscle weakness in the right hand and alteration in its muscle contour. Neither episodes of numbness in the hand nor color changes were observed. Orthopedic examination revealed no indications for surgical treatment; the symptoms were considered to be caused by the concomitant spinal cord pathology (see below).

Examination of the patient at the age of 17 years showed that his head was tilted in the antero-leftward direction. The range of motion in the cervical spine in the sagittal, frontal, and horizontal planes was no more than 5°. The patient had significant facial asymmetry and low hairline at the back of his head; chest excursion and mobility of the thoracic spine were limited; the patient's trunk was shifted to the left in the frontal plane

(Fig. 1a, b). The patient had S-shaped cervicothoracic scoliosis, as well as elevated right shoulder girdle and scapula. The upper extremities had an equal length; no abnormalities were revealed for the left upper extremity. The lower extremities had an equal length; full range of passive and active motion in the hip and ankle joints was preserved. The neutral heel position was preserved; no abnormal plantar reflexes were identified. No pelvic organ dysfunction was revealed.

Status localis. Active and passive abduction of the shoulder joint, 120°; range of motion for shoulder flexion, 180°; range of motion for the elbow joint was physiologically normal. Neurological examination revealed flaccid monoparesis affecting the right side manifesting itself as moderate forearm hypotrophy, severe hypotrophy of interosseous muscles of the hand and the thenar eminence; active extension in the wrist joint was limited; ape hand deformity of the right hand was observed (Fig. 1c, d); active extension of fingers on the right hand was minimal, being possible only during wrist extension; function of the opposable thumb was absent; active flexion was limited; bilateral hand grip was absent; interdigital, cylindrical, and hook grasp was preserved; score for muscle strength in the proximal regions and forearm was 5; score for opposition and flexion of the thumb was 1–2; score for finger extension was 1; and score for finger flexion was 3. Deep, surface, and pain sensibility was preserved.

Palpation revealed an osseous structure in the supraclavicular area; the palpation attempt caused sharp pain radiating to the right hand.

Additional examination was carried out. Stimulation electroneuromyography (ENMG) of the upper limbs was performed using a NeuroMVP-4 device. Excitability and conduction in sensory and motor fibers of the upper limbs, as well as conduction in spinal roots, were studied using the f-wave approach. Needle electromyography (EMG) was also performed.

Stimulation of sensory fibers of the median, ulnar, and radial nerves in the right hand elicited no sensory response. Right-sided stimulation of the lateral cutaneous nerve of forearm reduced potential amplitude eightfold compared to that for the left hand (2 μV for the right hand vs. 16 μV for the left hand) and the speed of nerve impulse conduction down to 30 m/s. Stimulation of the shoulder girdle at Erb's point elicited a normal M response from *m. deltoideus* and *m. biceps brahi*i on the right side. No M response from *m. abductor pollicis* brevis was detected after stimulation of motor fibers of the median nerve; stimulation of the ulnar nerve resulted in 82% reduction of the M response from *m abductor digiti minini* compared to that for the left hand; and stimulation of the radial nerve resulted in 91 % decline in M response from the *m. extensor indicis proprii.*

Needle EMG of *m. trapezius* (innervation of the C2–C4 segments) and *m. paraspinalis* (C7) revealed no abnormalities on the right side. There were signs of complete denervation of *m. abductor pollicis brevis* (segmental innervation of C8–T1), existing severe denervation changes with signs of minimally preserved residual innervation of *m. abductor digiti minini* (C8–T1) and *m. ext. digitorum communis* (C7–C8) on the right side, and mild chronic denervation/reinnervation changes in *m. biceps brachii* and *m. deltoideus* (C5–C6) on the right side. The EMG data were indicative of severe disturbance of the middle trunk (C7) and, to a greater extent, the inferior trunk (C8–T1) of the brachial plexus (Fig. 2d) on the right side, with an emphasis on the disturbances in the innervation zone of the T1 nerve root and signs of existing denervation changes in muscles of the right forearm and hand, complete denervation of *m. abductor pollicis brevis*; severe damage to sensory fibers, and mild extent of damage to motor fibers of the superior trunk (C5–C6) of the brachial plexus on the right side, without loss of functional activity of *m. biceps brachii* and *m. deltoideus* on the right side. ENMG revealed no signs of damage to motor neurons of the spinal cord and motor nerve roots at the C2–C4 level or

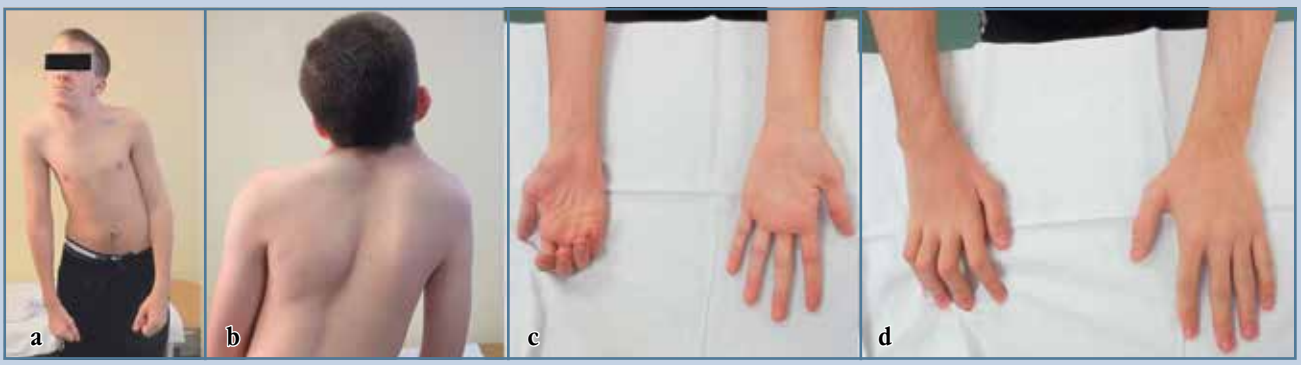

Fig. 1
The 17-year-old patient S. having Klippel – Feil syndrome (see explanation in the text)

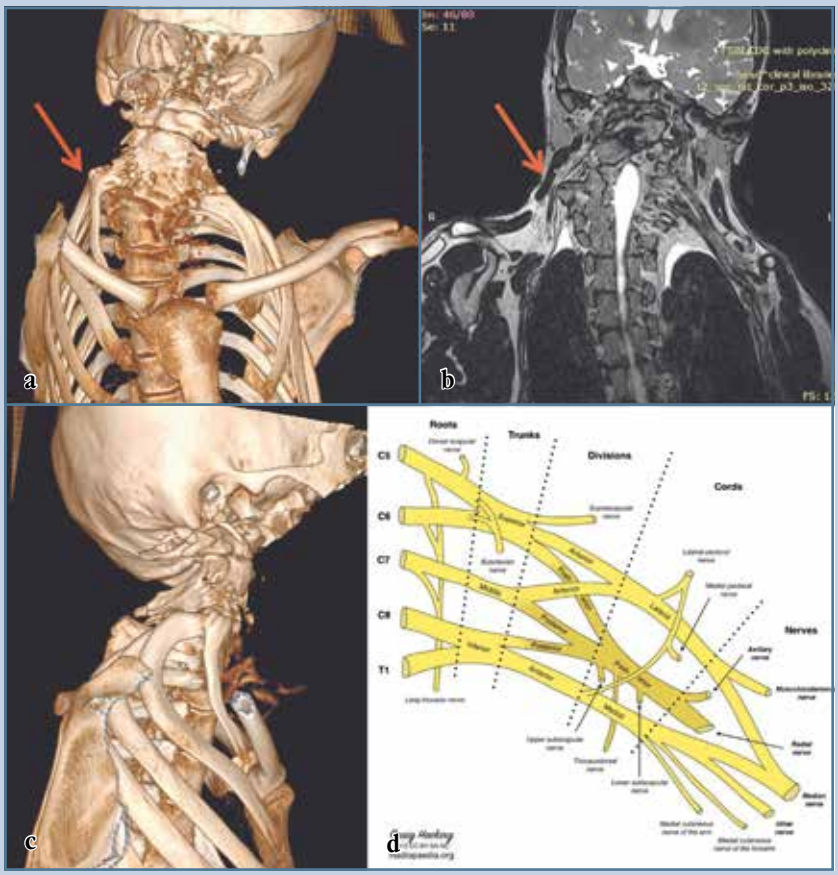

Fig. 2
A 3D CT scan **(a, c)** and coronal MR image **(b)** of the cervicothoracic spine of the 17-year-old patient S. (see the explanation of the features of malformation anatomy in the text): the costotransverse joint in the area of the false C7 cervical rib on the right side is shown with arrows; the bending C5–C6 spinal roots can be seen in the MR image; the scheme **(d)** clarifies the features of the formation of cervical plexus nerve bundles

right-sided cervical intumescence at the C5–T1 level.

Duplex ultrasound of the major arteries and veins of the upper limbs detected no abnormal blood flow patterns.

The following abnormalities were found by comparing the CT and MRI data of the brain and cervical spine:

– the suboccipital zone: the abnormality consisted of a combination of aplasia of the posterior half of the C1 vertebral arch and the C2 vertebral arch, enlargement of the posterior subarachnoid space and a cerebrospinal fluid-filled cyst at this level (Fig. 3a–d); there was no bony union between the C2 vertebral body and the odontoid process, but the C2 body formed a fibrous block with the odontoid process and the anterior half of the atlas, without any signs of segmental instability (Fig. 2a);

– incomplete diplomyelia at the C1–C3 level with the central canal opening posteriad, into the cavity of the cerebrospinal fluid-filled cyst (Fig. 3c, d);

– hypoplasia of the right vertebral artery at the level of the craniovertebral junction;

– C2, the subaxial cervical spine, and the thoracic spine (numbering of vertebrae was approximate because of the abnormal number of ribs on the right side (12), while there were 11 ribs on the left side): starting with C2 vertebra, in the caudal direction, the vertebral bodies and lateral masses of five vertebrae were blocked leading to straightening of normal cervical lordosis and formation of the right-sided scoliosis curve (19°; Fig. 2a).

The intervertebral foramina were narrowed bilaterally within the entire blocked area. The transversal processes of the C3–C5 vertebrae were rudimentary; those of the C6–C7 vertebrae were hypertrophied bilaterally (on the right side, the process of the subjacent vertebra was 2 cm long and its outer end passed into the rib whose bony section ended near the posterior surface of the right clavicle) (Fig. 2c). The right brachial plexus got bent at the level of this rib (Fig. 3b, e–g; Fig. 2b); its components came close together. Muscles of the brachial plexus were atrophied. The chest was deformed.

Taking into account the changes revealed using the CT, MRI, and ENMG data, the pathology was considered to be the neural component of the compression (tunnel) syndrome caused by compression of the brachial plexus nerve roots (C5 and C6) at the level of the C7 cervical rib. Because of the aggravating neurological symptoms, a decision was made to perform neural bundle decompression.

The surgery was performed through the subclavicular approach. The brachial plexus elements closely adjoined the anterior surface of the rib and were hard to displace. Neurolysis of the interior trunk of the brachial plexus was performed. There was no need to transect the transverse cervical artery, since after performing traction of the brachial plexus and isolating the anterior section of the rib it was successfully resected using an ultrasonic bone shaver and bone forceps almost up to the transverse processes. After rib resection, transposition of the nerve trunks to the place formerly occupied by the resected osseous structures was carried out; the bundle was hanging freely in the wound, and the subclavian artery was visualized deep inside the wound.

The postoperative period was uncomplicated. Almost immediately after the

surgery, the patient reported that pain intensity in the hand was reduced and minimal finger mobility appeared. The patient was discharged to further receive outpatient treatment and attend the scheduled follow-up visit.

Eight months after the surgery, an examination revealed signs of profound hypotrophy of thenar muscles in the right hand. Opposition of the thumb and the bilateral hand grip function were absent. Meanwhile, muscle strength of finger flexors was increased to score 4 (score 3 at baseline), finger extensors, up to score 2 (score 1 at baseline), and flexor pollicis longus, to score 3 (score 1–2 at baseline).

ENMG revealed reduced severity of denervation changes in the forearm muscles on the right side, emergence of the signs of reinnervation in the flexor muscles of the right hand and sensory-evoked potentials in response to stimulation of the ulnar and radial nerve on the right side, and intensified M response from *m. abd. digiti minini* on the right side. Stimulation of the median nerve did not elicit functional activity of *m. abd. poll brev* on the right side, being indicative of its atrophic changes. Taking into account the long interval after the time when these signs had emerged, the patient underwent tendon and muscle repair surgery involving transposition of the ulnar flexor of the wrist to extensors of the second through fifth fingers (the surgery being opposite to the Steindler procedure) in order to improve the function of the right hand. Active finger and wrist extension, as well as the grip function were improved after the surgery (Fig. 4).

Discussion

The emergence of the Klippel – Feil syndrome is attributed to improper segmentation of mesodermal somites at week 3 of pregnancy [7, 8]. Its prevalence rate is estimated at 1 case per 40,000–42,000 newborns; girls are more likely to have this syndrome [8]. This disorder can have the autosomal dominant and autosomal recessive inheritance patterns; however, sporadic cases are more frequent. In the case of autosomal dominant inheritance pattern, there is a mutation in the GDF6 (chromosome 8q22.1) and GDF3 genes (chromosome 12p13.31). If both gene alleles carry this defect, the

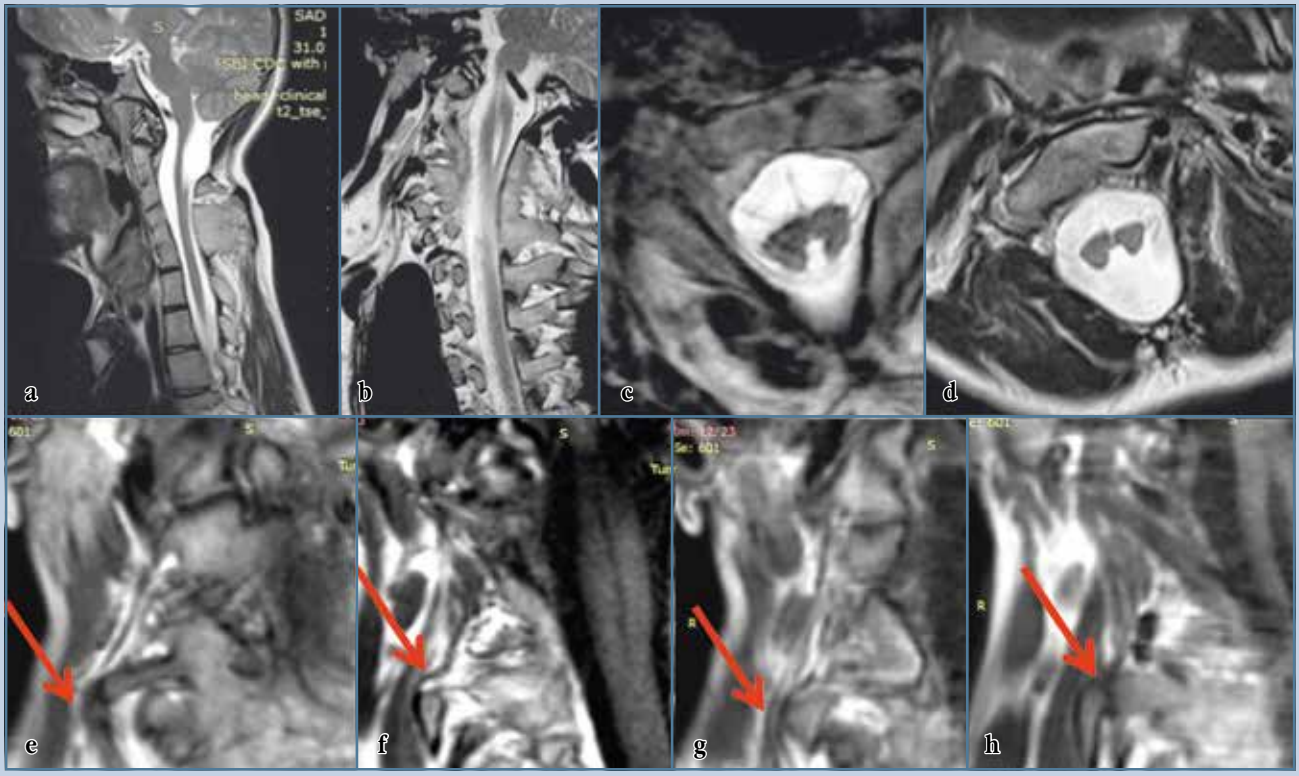

Fig. 3
An MR image of the 17-year-old patient S: the sagittal **(a)**, coronal **(b)**, and axial **(c, d)** T-weighted MR images of the spinal cord: the cerebrospinal fluid-filled cyst in the posterior section of the craniovertebral segment; incomplete diplomyelia (splitting of the posterior spinal cord columns) leading to formation of a window opening towards the cavity of the cerebrospinal fluid-filled cyst; **e–h** – T1-weighted coronal MR images of the costotransverse joint with C7 on the right side (an arrow), with bending at this level of nerve trunks (**d** – the most ventral view; **h** – the most dorsal view)

syndrome has the autosomal recessive inheritance pattern (mutation MEOX1 in chromosome 17q21.31), while the parents who are carriers of this pathology have only a single chromosomal copy carrying the mutation [9].

The principles of anatomic classification of the syndrome were outlined by Klippel and Feil who had described it [1]. Klippel and Feil distinguished three types of the disorder: improper segmentation of the C2–C3 vertebrae and possible atlanto-occipital fusion (type 1), extensive blocking of subaxial vertebrae and an abnormality at the craniovertebral level (type 2), and the presence of two immobile zones with a cervical segment whose mobility was preserved (type 3). They also mentioned that patients with KFS may have fewer cervical vertebrae or the disorder can be associated with abnormalities of other spine sections [1].

Later clinical and radiological classifications focused on detalization of the disorders. The most strategically convenient suggestion was that made by Samartzis et al. [10, 11] as it both refined the anatomical variants of segmentation disorders (type I: isolated congenital bone blocks (25 % of cases); type II: multiple non-adjacent congenital bone blocks (50 %), the C2–C3 and C5–C6 segments being affected most frequently; type III: multiple adjacent bone blocks (25%)) and distinguished the symptoms associated with the cervical pathology, including those developing throughout life. It was mentioned that because of the limited range of motion in the cervical spine, patients with type I disorder are more likely to complain of headache and cervical pain, whereas the risk of neurological complications is higher in patients with types II and III disorder. The mean age for developing myelopathy, cervical pain, and radiculopathy is 10, 13, and 18 years, respectively [2, 5]. It is characteristic that only 50 % of patients have the classic clinical triad of symptoms [4]; in some cases, the syndrome remains asymptomatic for a long period and is diagnosed only when the patients become adults.

In 30 % of cases, the associated symptoms involve sensorineural or mixed hearing loss, facial palsy, ptosis, esotropia, Duane's syndrome, rib fusion abnormality, upper limb hypoplasia, Sprengel deformity, and instability of the non-affected cervical segments [11–13]. Up to 50 % of patients have scoliosis or other severe spinal deformities [3, 12, 14].

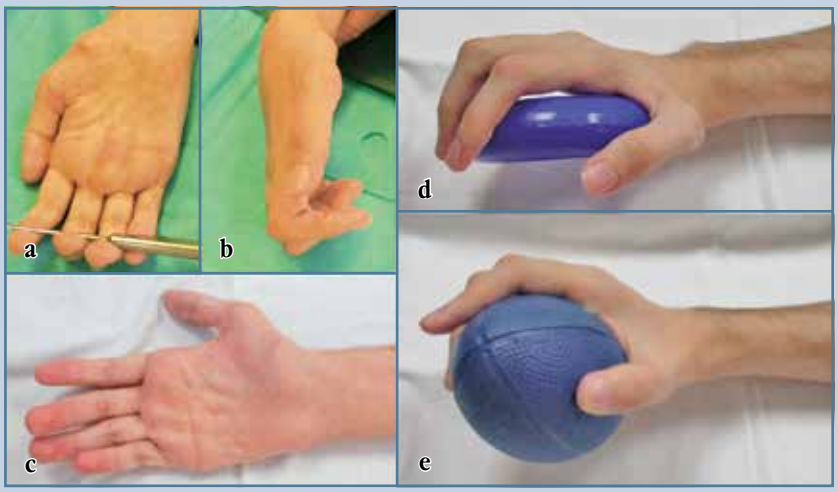

Fig. 4
The functional status of the right hand of the 17-year-old patient S. before **(a, b)** and 1 month after **(c–e)** tendon and muscle repair surgery (transplantation of the *flexor carpi ulnaris* to the extensor muscle of the second through fifth fingers being an opposite surgery to the Steindler procedure)

Some cases when KFS was associated with Chiari malformation and basilar invagination, with the respective clinical presentation of CSF circulation disorder and the posterior cranial fossa compression syndrome, have been reported [7].

Patients having concomitant spinal cord disorders (syringomyelia, syringobulbia, split cord malformation, spinal stenosis, and cysts) especially come under notice [12, 16]. According to the type of neurological impairments, Nagib et al. [2] singled out three clinico-anatomical groups of patients:

– patients with acute and chronic spinal cord injury, two fused vertebral blocks, as well as concomitant myelodysplasia (syringomyelia, syringobulbia, split cord malformation, spinal stenosis, and cysts);

– patients with occlusion of the great occipital foramen, atlanto-occipital fusion, basilar invagination and increased mobility of the craniocervical segment; association with Chiari malformation and syringomyelia is possible;

– patients with spinal stenosis and vertebral fusion in the craniovertebral, cervical, and thoracic spine.

Flexion and extension of the cervical spine is often maintained even if only two adjacent cervical vertebrae remain non-fused. It was reported that instability of the upper cervical spine is a factor predisposing to neurological impairment, while instability of the lower cervical spine is a factor predisposing to cervical osteochondrosis [2, 4, 7].

In patients having a combination of KFS and split cord malformation, conventional principles are used for classifying the latter disorder: type 1 – diastematomyelia with duplicated dural sac and type 2 – diplomyelia with a single dural sac. For these patients, surgical treatment is recommended only if clinical symptoms get worse [17]. In turn, the concomitant vascular abnormalities accidentally detected in patients with KFS when performing ultrasonography and/or MRI that are asymptomatic in case of both dynamic and permanent brain ischemia do not require surgical correction because children are well adapted to impaired main blood flow [4].

A total of 10–15 % of patients with KFS have cervical ribs, which can cause neurological symptoms [6]. In the presented case report, the 17-year-old patient with a complex malformation of the cervical and upper thoracic spine and spinal cord (incomplete diplomyelia) and scoliosis as a part of KFS associated with sensorineural hearing loss was having progressive upper extremity monoparesis for three years because of the compression of the right brachial plexus elements, which led to irreversible changes because the initial pathology was underestimated. Brachial plexus decompression by resecting the uppermost rib (cervical C7 vertebra) improved the function of forearm and hand muscles, as well as a number of neurophysiological parameters; however, the hand function was not completely restored. Patient's self-care capacity was significantly improved (mostly due to improvement of the grip function) by hand tendon reconstruction surgery.

Conclusions

Taking into account the progressive course of the disease, even if patients with KFS have no neurological symptoms, they must be continuously followed up by an orthopedic spine surgeon and a neurologist on a regular basis so that neurological complications developing during their life could be timely detected. One should remember that the causes for developing these complications may be not only the manifestations of primary myelodysplasia, but also secondary changes caused by the concomitant abnormality (cervical ribs). In order to objectivize the data, refine the affected spine level, and determine the optimal strategy for patient management in these cases, the patients need to undergo complete examination using CT scanning, MRI, and ENMG. Timely treatment can allow one to prevent the emergence of irreversible complications, while the interdisciplinary approach using the modern stepwise treatment methods will make it possible to improve patients' quality of life.

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