Original article

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Early and delayed surgical management of the pronator teres syndrome. Selective reinnervation of the anterior interosseous nerve aimed to restore pinch grip among patients with late clinical presentation

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Background. The incidence of pronator teres syndrome (PTS) is low. The misdiagnosis leads to delay in surgical treatment and irreversible changes not only within the median nerve (MN) itself, but within the sensory and muscular apparatus as well.

Objective: to compare the outcomes of early and delayed surgical management of PTS; to compare the restoration of the pinch grip (PG) after decompression and reinnervation (nerve transfer, NT) of the anterior interosseous nerve (AIN) vs. decompression of MN alone in late terms of the disease (PTS).

Materials and Methods. Six patients with verified PTS were included into the study. Three patients with the history of the disease (HoD) less than 3 mos. received surgical decompression (SD) of MN under standartized methodology alone. Another three patients with the HoD more than 3 mos. received SD of MN, with two of them received simultaneous NT of the branches of the radial or MN to AIN. In all patients sensory and motor deficit (function of "extrinsic"and "intrinsic"muscles), intensity of the neuropathic pain, both pre- and post-surgery have been evaluated according to MRC Scale and VAS, respectively. An ability to reproduce PG, or "OK" sign, with help of the thumb (flexor pollicis longus muscle – FPL) and index finger (deep flexor muscle – FDP2) were evaluated.

Results. All patients showed complete relief of the neuropathic pain (VAS0) regardless of the terms of the disease. Three patients with HoD less than 3 mos. showed good recovery of FPL, FDP2 (M4-5) – all patients were able to reproduce "OK" sign. One patient with HoD more than 3 mos. after SD of MN alone showed no recovery of FPL, FDP2 (M0-1). Another two patients with the HoD more than 3 mos. showed good recovery of FPL and FDP2 (M3-4) after NT to AIN. No patient with HoD more than 3 mos. was able to reproduce "OK" sign.

Conclusions. Early decompression of MN in PTS cases results in complete relief of the sensory and motor neurologic deficit; late decompression of MN in PTS cases does not lead to relief of the sensory deficit within NCP autonomous area, while the prognosis of the recovery of the median nerve innervated "extrinsic" and "intrinsic" muscles is rather unfavorable; In case of late PTS presentation, NT to AIN allows restoring only a single component ("extrinsics") of the motor functions of the hand which are required for the succesfull reproduction of the pinch grip; In case of late PTS presentation, poor recovery of OP should be expected, hence the succesfull reproduction of the pinch grip due to the thumb hyperadduction would be impossible; carefull interpretation of the clinical, radiological and electophysiological data on the pre-surgical stage could potentially help avoiding the misdiagnosis and improve the outcomes of the surgical treatment in all cases of a single or multilevel MN entrapment.

Key words: *median nerve; carpal tunnel syndrome; pronator teres syndrome; nerve compression syndrome; surgical treatment; selective nerve transfer*

Introduction

Currently, neuropathies of the median nerve (MN) of compressive etiology are no longer a clinical "mystery" for most clinicians, both general practitioners and surgeons. Over the last decade, the clinical diagnosis of compression neuropathy of the MN at the level of the carpal tunnel ("carpal tunnel syndrome" (CTN)) has gained mass acceptance [1]. The widespread implementation of electrophysiological diagnostic methods [2–4] allowed the verification of clinical findings and a qualitative differential diagnosis [1] with other chronic pain syndromes caused by pathology of

the articular-ligamentous apparatus of the hand [1]. The surgical management of MN compression in CTN has become the prerogative not only of neurosurgical specialists, but also orthopedic traumatologists and even general surgeons [5].

Other potential compression points of MN on its way along the upper extremity segments has received little attention. This is primarily due to the relatively low prevalence of the pathology [6] and, in most cases, a complex process of both clinical [1] and differential diagnosis [1]. If we exclude the cases of "exotic" compression neuropathies of the MN in the distal segment of the shoulder (according to studies [7], Struther's ligament is present in only 1-2% of the population), then the most common potential point of MN compression is the structures of the anterior compartment of the forearm, connected to the pronator teres syndrome (PTS). The etiological cause of compression neuropathy in the area of the forearm segment (PTS) can be the forearm's actual pronator teres (in 33–76% of cases), *lacertus fibtosus* - (<42%), the tendon component of the belly of the flexor digitorum superficialis (in 14–36%) [8–11] and other muscle, tendon, vascular and bone structures [1].

In addition to the spontaneous development of intense pain syndrome [1] and sensory disorders of various kinds [1], the initial clinical manifestations of PTS are rarely accompanied by muscle paresis, the innervation of which is provided primarily by the anterior interosseous nerve (AIN) (flexor pollicis longus (FPL) and deep flexor muscle 2 (FDP2)) [12]. Most researchers attribute the priority of sensory deficits to the lower resistance of these fibers compared to motor fibers to the influence of an external compressive factor [13]. In the later stages of MN compression, paresis of the forearm and "intrinsic" muscles of the hand necessarily accompanies the clinical and electrophysiological picture of PTS [13].

The main problem of reinnervation of striated muscles denervated due to various reasons of traumatic and non-traumatic genesis, is an increased risk of developing irreversible degenerative changes according to the duration of the denervation process [14]. Prediction of the potential for spontaneous recovery of compressed nerve function is based on the duration of the sensory and motor apparatus dysfunction if the compression factor itself is removed. A large number of invasive [15] and non-invasive methods [15] make it possible to assess the degree of effectors degradation [15].

Among the latest surgical techniques aimed at maintaining the viability of the muscular apparatus, only two main ones can be distinguished: 1) invasive chronic electrical stimulation of both the effector muscle and the damaged nerve, mainly in experimental and rarely in clinical models [16], 2) performing reinnervation of a damaged nerve using the "nerve baby-sitting procedure" [17,18] or "supercharge nerve transfer" [19] method. Compared to a more aggressive approach used in traumatic peripheral nerve injuries (nerve transfer), the implementation of which requires a complete anatomical crossing of a non-functioning nerve [20], "supercharge nerve transfer" in the case of compression-induced neutopathy allows a certain extent of potential of the damaged nerve for spontaneous regeneration by maintaining the effector muscle viable [19] during a long period of recovery (for example, remyelination [19]). On the other hand, selective nerve transfer as a more aggressive surgical technique allows the entire potential of the donor nerve to be involved (in particular, by ensuring the appropriate motor fiber ratio between the donor nerve and the recipient nerve (axon count ratio) [21]) and does not depend on the degree of probability of spontaneous regeneration, performing the required function in the expected time.

Objective: to compare the outcomes of surgical treatment of the pronator teres syndrome in the early and late stages of the disease, as well as the results of pinch grip restoration when combining median nerve decompression with selective nerve transfer of the anterior interosseous nerve and after only median nerve decompression in patients in the late stage of the disease.

Materials and methods

Study design. A retrospective analysis of a series of 6 consecutive cases of surgical treatment of MN neuropathy in the area of the forearm segment (PTS) in 2017–2021 was carried out.

Inclusion and exclusion criteria. The main inclusion and exclusion criteria for patients are shown in ***Table. 1***. No patient in a series of consecutive cases was diagnosed with "double crash" syndrome [1]. All patients were additionally subjected to standard clinical, electrophysiological and radiological diagnostic procedures [22, 23] before being included in the study to rule out pathologies of competing peripheral and central nervous system structures of traumatic and non-traumatic genesis ("thoracic outlet" syndrome, polyneuropathies of various origin, "upper" motoneuron disease, radiculopathy of various origin, history of the upper limb or cervical spine injury, etc.).

Characteristics of the patients involved in the study (***Table 2***). The study included 6 patients (3 women and 3 men) aged 38 to 59 years (mean age - 46 years). The period from the onset of the disease to inclusion in the study ranged from 1 to 8 months (4.3 months on average). The main distal clinical and neurological manifestations of MN neuropathy in the area of the forearm segment (PTS) were: impaired sensitivity in the zone of autonomic innervation *n. cutaneus palmaris n. mediani* – S0-1 according to the MRC Scale [26], in the zone of autonomic innervation of the MN – S1-2 according to the MRC Scale, peripheral paresis of FPL and FDP2 from M0-1 to M3-4 according to the MRC Scale, muscles of 1st finger elevation (in particular *m. opponens pollicis*) - from M0 to M4 according to the MRC Scale and pain syndrome of a neuropathic nature from 2 to 8 points according to the Visual Analog Scale (VAS) [27].

This article contains some figures that are displayed in color online but in black and white in the print edition.

Table 1. Criteria for inclusion and exclusion of patients from the study based on clinical and neurological examination data at the pre-surgical stage

Diagnostic criteria	Inclusion criteria	Exclusion criteria
Pains	Anterior surface of the forearm, increased by muscles tension of the anterior compartment of the forearm; night and daytime	Hand and fingers with irradiation in proximal parts of the upper limb; mostly at night
Impaired sensation	Impaired sensation on the palmar surface of the hand above the thenar muscles (except for the autonomous zone of innervation of the median nerve)	Sensory impairment only in the autonomous zone of innervation of the median nerve
Impaired motor function	Impaired function of "extrinsic " muscles, particularly FPL and FDP2	Impaired function of "intrinsic" muscles, in particular *m. opponens pollicis*
PCS[1]	Positive	Negative
Provocative tests[2]	Positive	Negative
Tinel's symptom	Positive in projection of median nerve in the upper and middle third of forearm's anterior surface	Positive, in median nerve projection proximal and distal to transverse carpal ligament
Test Phalen [24]	Negative	Positive
Test Durkan [25]	Negative	Positive

Note: FPL – flexor pollicis longus ; FDP2 – deep flexor muscle 2; 1 – Pronator compression test [1]; 2 – dynamic provocative tests involving the forearm muscles to identify potential compressive muscular and tendon-aponeurotic factors within the forearm segment in the structure of complex "pronator teres" syndrome [1].

Table 2. Characteristics of the patients involved in the study

№ i/o	Sex	Age, years	DD	Surgical treatment method		Clinical picture				
				Neurolysis*	Neurolysis+N	*S*	*M*			*VAS, points*
								FDP2	*Th.*	
1	M	46	2	+		0-1	3-4	3-4	5	8
2	F	41	6	+		0-1	0-1	0-1	2	2
3	F	55	3	+		1	3-4	3-4	4	5
4	F	38	6		+	0-1	0-1	0-1	2	3
5	M	38	1	+		1	3-4	3-4	4	4
6	M	59	8		+	1	0	0	0	2

Note: DD - disease duration; N – neurotization of the anterior interosseous nerve; S - assessment of preservation of sensory function in the zone of autonomous innervation n. cutaneus palmaris n. mediani according to the MRC Scale; M - assessment of muscle motor function according to the MRC Scale; FPL - flexor pollicis longus muscle; FDP2 – deep flexor muscle of the 2nd finger; Th. - m. opponens pollicis; VAS - assessment of the neuropathic pain syndrome intensity rating by Visual Analog Scale.

** – Neurolysis – performing median nerve decompression accompanied by myotomy and tenotomy of the deep head of pronator teres forearm, as well as Z-shaped lengthening of its distal tendon.*

Among patients with disease duration >3 months, the neurological deficit was mainly represented by severe paresis of FPL, FDP2, and *m. opponens pollicis* (M0-1 according to the MRC Scale) ***(Fig. 1)***, whereas in patients with disease duration <3 months – M3-4 according to the MRC Scale (see ***Table 2***). In patients with disease duration >3 months, the intensity of neuropathic pain syndrome was 2-3 VAS points, in patients with disease duration <3 months it was 4–8 VAS points (see ***Table 2***).

An extreme manifestation of trophic dysfunction in the zone of autonomous innervation of *n. cutaneus palmaris n. mediani* in patients with disease duration >3 months was the formation of a chronic trophic ulcer ***(Fig. 1, A and B)*** (in two patients above the 1st finger elevation muscles). The size and depth of trophic skin disorders, according to anamnestic data, varied during the course of the disease, but as the wound was cleaned, no tendency to its epithelization was observed in any case.

Characteristics of surgical interventions. In all patients involved in the study, during the surgical release of the MN within the upper and middle third of the anterior surface of the forearm according to the standard technique [28], direct macroscopic signs of compression of the MN itself, caused by the muscle and tendon components of the deep head of the pronator teres of the forearm, were revealed ***(Fig. 2)***. The macroscopic picture was characterized by the formation of a circular invagination on the trunk of the MN and AIN, which

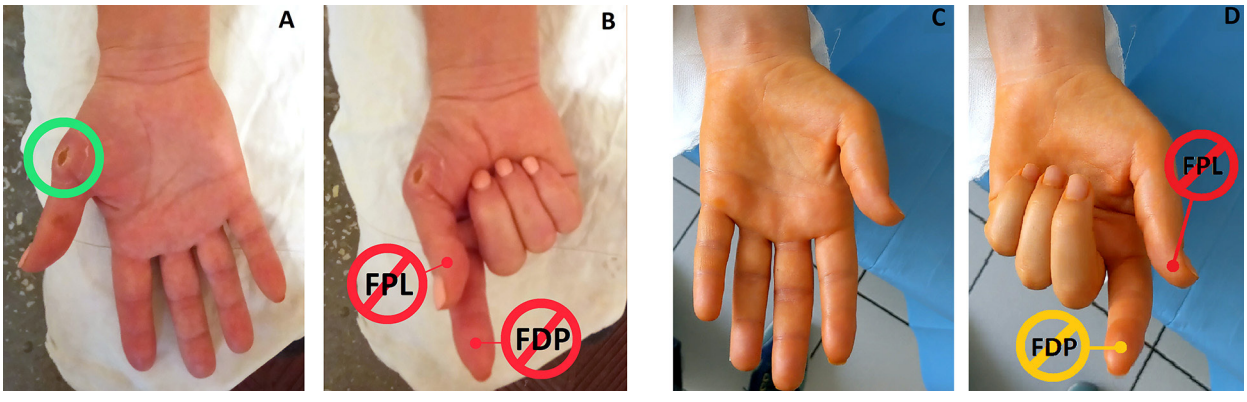

Fig. 1. Clinical manifestations of long-term compression (>3 months) of the median nerve in the area of the forearm segment ("pronator teres" syndrome):
FPL - flexor pollicis longus muscle;
FDP2 – deep flexor muscle of the 2nd finger;
green circle – chronic trophic ulcer above the 1st finger elevation muscles and in the projection of the zone of autonomous innervation of n.cutaneus palmaris n. mediani;
red circle – peripheral plegia of the effector muscle;
orange circle – severe peripheral paresis of the effector muscle

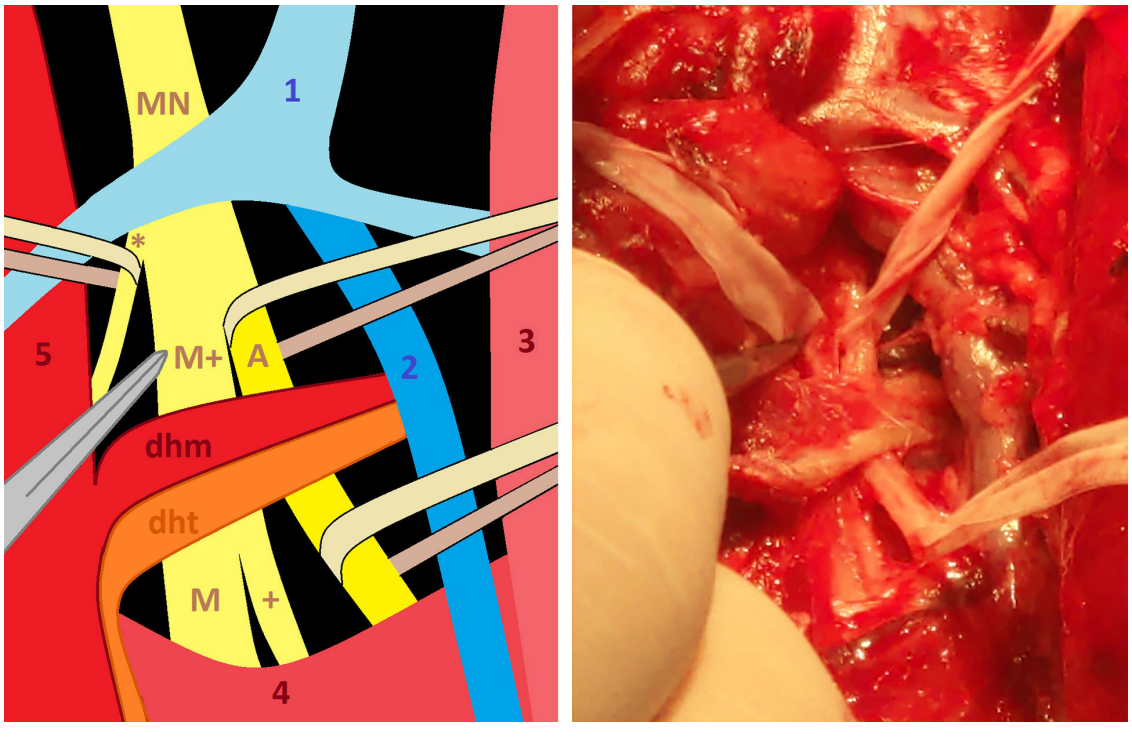

Fig. 2. Macroscopic intraoperative and schematic representation of the point of median nerve compression in the area of the forearm segment ("pronator teres" syndrome), caused by the muscle and tendon components of the deep head of pronator teres of the forearm:
1 – median cubital vein;
2 – radial artery and vein;
3 – belly of the brachioradialis muscle;
4 – belly of the flexor digitorum superficialis;
5 – belly of the muscle of round forearm pronator;
dhm – muscle component of the deep branch of pronator teres of the forearm;
dht – tendon component of the deep branch of the pronator teres of the forearm;
MN – median nerve;
M+ – common median nerve trunk after branching of the anterior interosseous nerve;
A – anterior interosseous nerve;
M – common median nerve trunk after branching off the branch to the belly of the superficial flexors of the 2nd and 3rd fingers;
+ – branches to the radial belly of the superficial flexors of the 2nd and 3rd fingers;
* – distal branch to the pronator teres muscle of the forearm

corresponded in projection to the position of the muscle and tendon components of the deep head of pronator teres of the forearm at rest and during contraction (Hourglass Constriction (HGC)) [14].

Four patients (see ***Table 2***) with PTS underwent surgical release of MN according to the standard technique [28]. In these patients, the myotomy and tenotomy of the deep head of pronator teres of the forearm was combined by the transection of the distal parts of the *lacertus fibrosus*, as well as the Z-shaped lengthening of the distal tendon of the pronator teres muscle of the forearm.

In two patients with a disease duration of more than 6 months and severe peripheral paresis of FPL and FDP2 **(**see **Table 2)**, surgical release of MN was supplemented by selective nerve transfer due to the radial and median nerve branches **(*Fig. 3 and 5*)**.

After surgical release of the MN within the upper and middle third of the forearm's anterior surface,

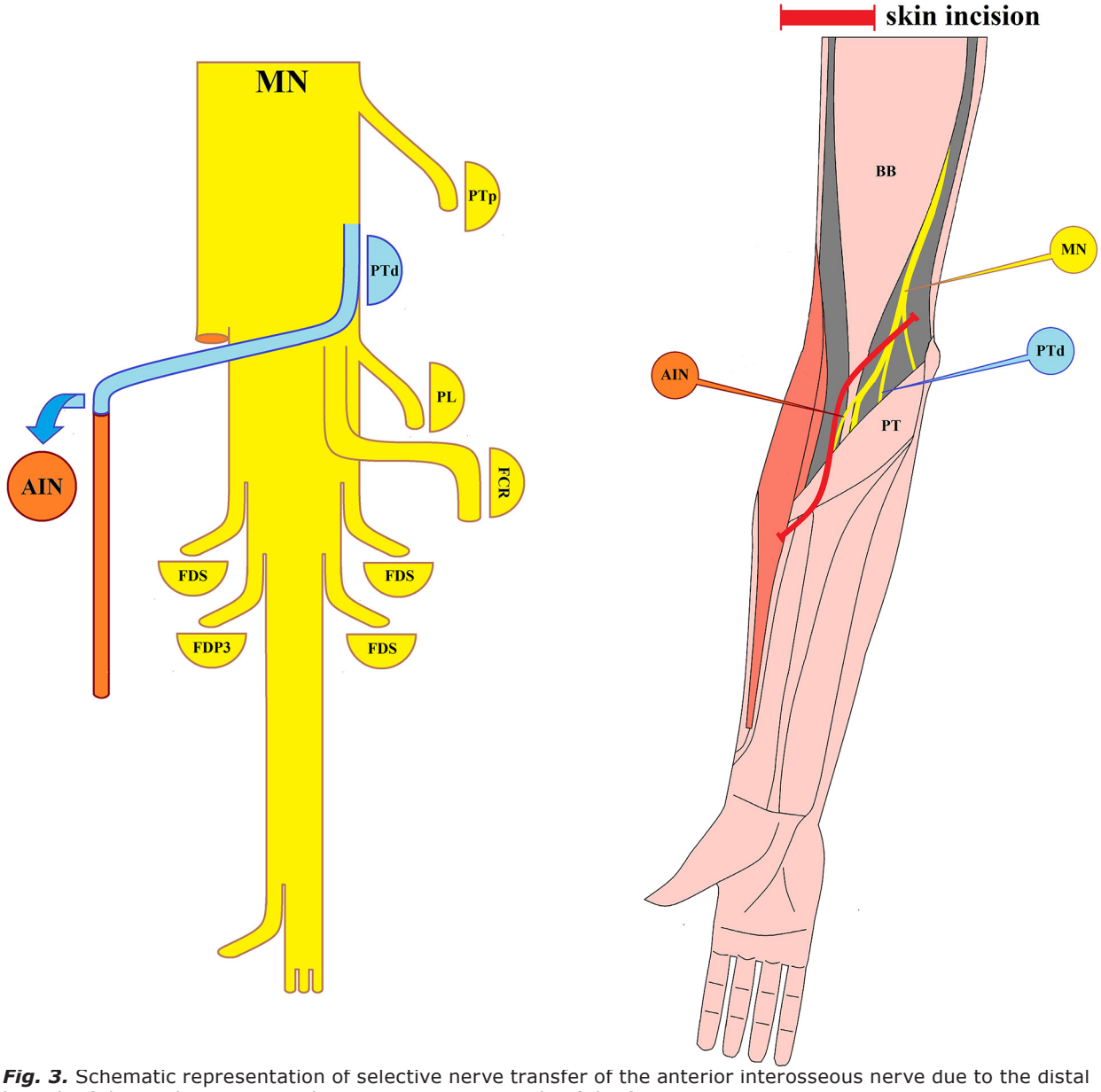

Fig. 3. Schematic representation of selective nerve transfer of the anterior interosseous nerve due to the distal branch of the median nerve to the pronator teres muscle of the forearm:
MN – median nerve;
PTp - proximal branch of the median nerve to the pronator teres muscle of the forearm;
PTd – distal branch of the median nerve to the pronator teres muscle of the forearm;
PL – a branch of the median nerve to the palmaris longus muscle straining the palmar aponeurosis;
FCR – a branch of the median nerve to the muscle of the flexor carpi radialis;
FDS – branches of the median nerve to the radial and ulnar heads of the belly of the flexor digitorum superficialis;
FDP3 – a branch of the median nerve to the muscle of the flexor digitorum profundus of the 3rd finger (sometimes as a part of AIN);
AIN – anterior interosseous nerve;
BB – belly of the biceps brachii muscle;
PT - belly of pronator teres muscle of the forearm

according to the standard technique [28], the AIN trunk was identified, based on standard anatomical landmarks. After identifying the AIN, a longitudinal epineurotomy of the common MN trunk in the proximal direction was performed. In the common trunk of the MN, the AIN fiber was traced and transected at the border with the cubital fossa (nerve-acceptor) ***(Fig. 4, A)***. At the border of the middle and upper third of the ulnar margin of the anterior surface of the forearm, the distal branch to the pronator teres of the forearm (PTd) was identified, based on standard anatomical landmarks ***(Fig. 4, A)***. The functional capacity of PTd was confirmed by stimulation of the fiber with an electric current of 0.1-0.2 mA, which was accompanied by a contraction of the pronator teres of the forearm. The PTd was traced to the entry of effector muscle thickness and was transected (donor nerve). Coaptation of the donor and acceptor nerves was performed using fascicular sutures with atraumatic non-resorbable 9/0 suture material using x 5–8 microscopic optical magnification ***(Fig. 4, B)***. Microanastomosis was performed in a "tension-free-manner" with 180° extension in the elbow and radiocarpal joints ***(Fig. 4, C)***. The surgical wound was closed tightly in layers without creating a passive or active drainage system ***(Fig. 4, D)***.

After surgical release of the MN within the upper and middle third of the forearm's anterior surface according to the standard technique [28], the trunk of the AIN was identified, based on standard anatomical landmarks. After identifying the AIN, a longitudinal epineurotomy of the common MN trunk in the proximal direction was performed. In the common trunk of the MN, the AIN fiber was traced and transected at the border with the cubital fossa (nerve-acceptor) ***(Fig. 6, A)***. The superficial branch of the radial nerve (RSRN) ***(Fig. 6, A)***, which was traced proximally, was identified on the border of the middle and upper third of the radial anterior surface of the forearm. At the border of the cubital fossa and the upper third of the forearm, the posterior interosseous nerve and fibers to the extensor carpi radialis brevis (ECRB) were identified, based on standard anatomical landmarks ***(Fig. 6, A)***. The functional capacity of the

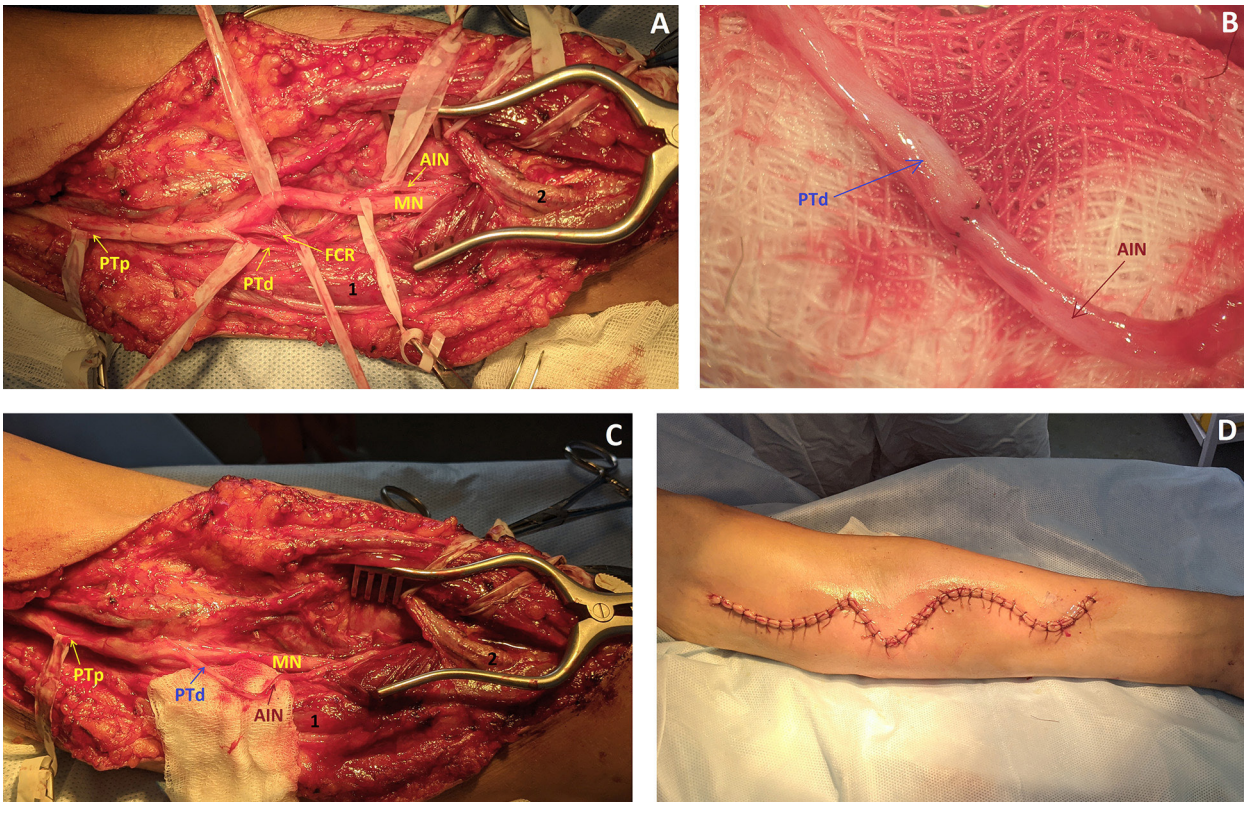

Fig. 4. Intraoperative macroscopic and microscopic surgical anatomy of the stages of selective nerve tranfer of the anterior interosseous nerve due to the distal branch of the median nerve to the pronator teres of the forearm:
A – macroscopic anatomy of branches of the median nerve in the segment of the upper and middle third of the forearm;
B – microscopic view of anastomosis between the donor nerve and the acceptor nerve;
C – macroscopic intraoperative view of anastomosis between the donor nerve and the acceptor nerve;
D - skin access after closure of the surgical wound;
MN – median nerve;
PTp - proximal branch of the median nerve to the pronator teres muscle of the forearm;
PTd – distal branch of the median nerve to the pronator teres muscle of the forearm;
AIN – anterior interosseous nerve;
1 – the belly of the pronator teres muscle of the forearm;
2 - a. et v. radialis

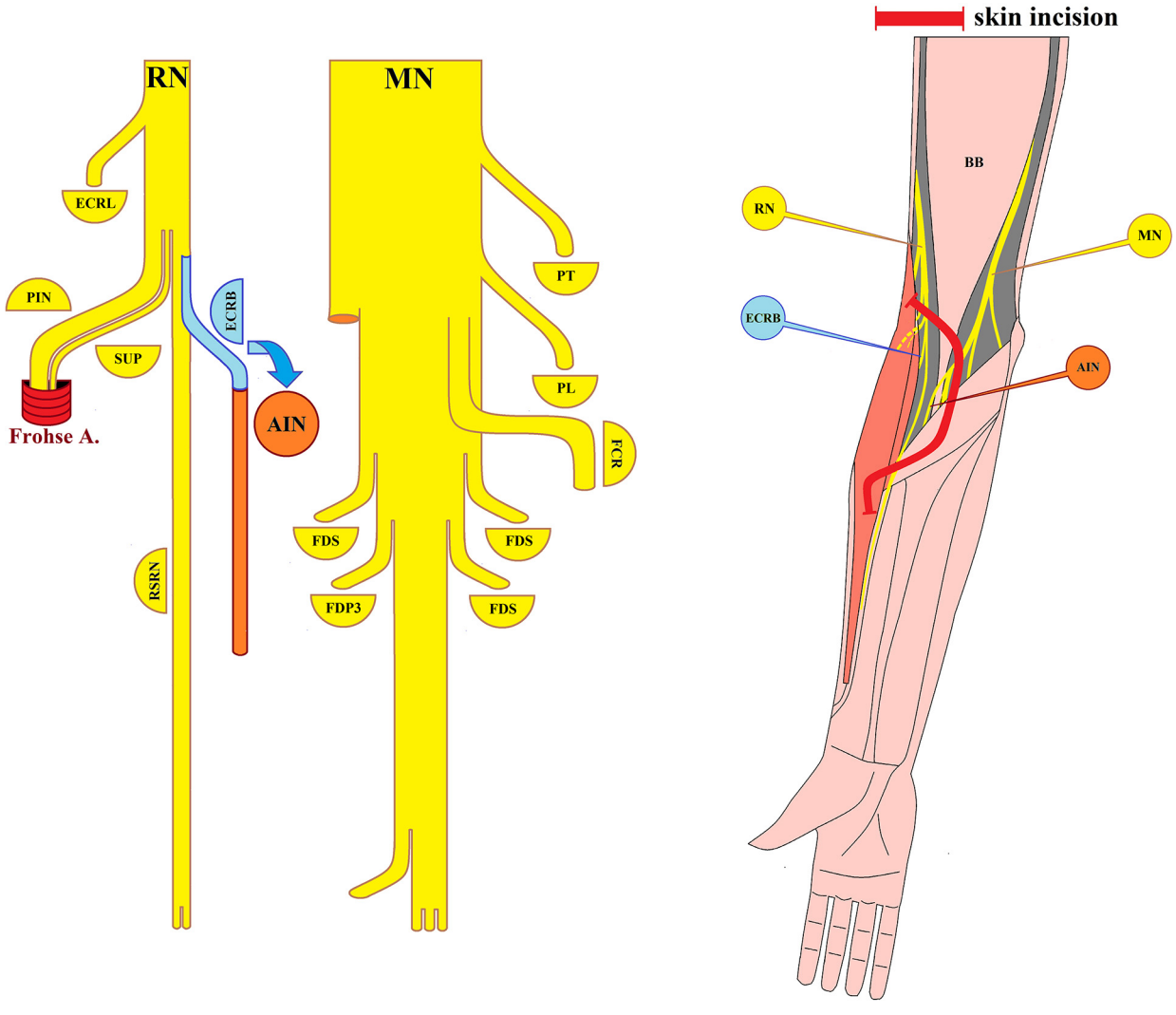

Fig. 5. Schematic representation of selective nerve transfer of the anterior interosseous nerve due to the radial nerve branches to the extensor carpi radialis brevis:
MN – median nerve;
PT – a branch of the median nerve to the pronator teres muscle of the forearm;
PL – a branch of the median nerve to the palmaris longus muscle straining the palmar aponeurosis;
FCR – a branch of the median nerve to the muscle of the flexor carpi radialis;
FDS – branches of the median nerve to the radial and ulnar heads of the belly of the flexor digitorum superficialis;
FDP3 – a branch of the median nerve to the muscle of the flexor digitorum profundus of the 3rd finger (sometimes as a part of AIN);
AIN – anterior interosseous nerve;
RN - radial nerve;
ECRL - a branch of the radial nerve to the muscle of extensor carpi radialis longus;
ECRB – a branch of the radial nerve to the muscle of extensor carpi radialis brevis;
PIN – posterior interosseous nerve;
SUP - a branch of the radial nerve to the supinator muscle of the forearm;
RSRN - superficial branch of the radial nerve;
Frohse A. – vascular arch of Frohse [29];
BB - belly of the biceps brachii muscle

ECRB was confirmed by stimulation of the fiber with a 0.1-0.2 mA electric current, which was accompanied by contraction of the extensor carpi radialis brevis and extension in the radiocarpal joint. The ECRB was traced distally to the entry of effector muscle thickness and was transected (donor nerve). Coaptation of the donor and acceptor nerves was performed using fascicular sutures with atraumatic non-resorbable 9/0 suture material using x 5–8 microscopic optical magnification ***(Fig. 6, B)***. Microanastomosis was performed in a "tension-free-manner" with 180° extension in the elbow and radiocarpal joints ***(Fig. 6, C)***. The surgical wound was closed tightly in layers creating a passive drainage system ***(Fig. 6, D)***.

Drug therapy and surgical wound management in all patients in the postoperative period were carried out in accordance with national and industry standards [30].

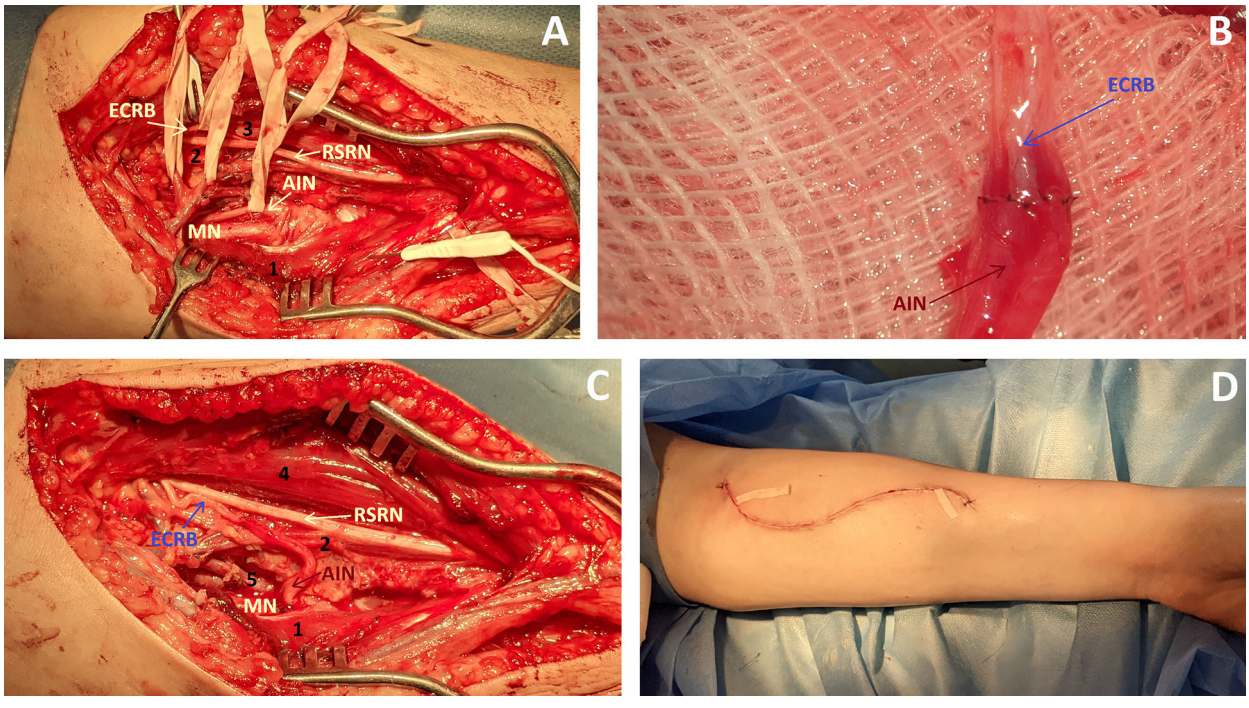

Fig. 6. Intraoperative macroscopic and microscopic surgical anatomy of the stages of selective nerve transfer of the anterior interosseous nerve using the radial nerve branches to the extensor carpi radialis brevis:
A – macroscopic anatomy of median nerve branches in the upper and middle third segment of the forearm;
B – microscopic view of anastomosis between the donor nerve and the acceptor nerve;
C – macroscopic intraoperative view of anastomosis between the donor nerve and the acceptor nerve;
D - skin access after closure of the surgical wound;
MN – median nerve;
AIN – anterior interosseous nerve;
ECRB – branches of the radial nerve to the muscle of extensor carpi radialis brevis;
RSRN - superficial branch of the radial nerve;
1 –belly of the pronator teres muscle of the forearm;
2 - a. et v. radialis;
3 –belly of extensor carpi radialis brevis;
4 –belly of the extensor carpi radialis longus;
5 – anterior interosseous artery

Evaluation of outcomes of surgical treatment of "pronator teres" syndrome. The outcomes of surgical treatment were evaluated based on the presence or absence of changes in three neurological clinical criteria: 1) sensation disorders in the zone of the autonomic innervation of the *n.cutaneus palmaris n. mediani*, 2) FPL and FDP2 function disorders, 3) intensity of neuropathic pain syndrome. In order to objectify and assign numerical values (quantitative changes) to changes in the neurological profile, the MRC Scale [26] and VAS [27] were used. The qualitative component of the change in the neurological profile was assessed by the ability or inability of reproducing the pinch grip (PG) - the object is held between the nail phalanges of the first and second fingers of the hand [31]. Assessment of quantitative changes in the clinical neurological status was necessarily performed during the entire 14-day period of the patient's stay in the hospital. Quantitative and qualitative components of changes in the neurological profile were necessarily assessed in the 3rd month after surgery. All further patient examinations were performed on a proactive basis without explicit reference to the time factor, except for patients who underwent selective AIN nerve transfer. Two patients, after AIN neurotization, had mandatory clinical neurological examination at 6 and 9 months, which corresponded to the timing of predicted reinnervation of the effector muscles. All further examinations of patients were performed on a proactive basis with no explicit reference to the time factor. Electrophysiological research methods (needle, stimulation electroneuromyography according to the standard technique in accordance with approved national and industry standards [30]) were used in patients with delayed or absent regeneration, the findings were not taken into account in final outcome analysis and did not affect the therapeutic treatment strategy. The final outcome of the change in the qualitative component of the neurological profile (ability or inability of reproducing PG) was considered to be the data obtained during the patient's last examination.

Limitations of the study: a small number of study participants, hence the inability to allocate the participants and form groups according to similar characteristics did not allow a reliable statistical analysis to be carried out.

Results

The minimum follow-up period for the patient after surgery was 5 months, the maximum was 24 months, and the average was 14 months ***(Table 3)***. The average follow-up period for 4 patients after performing only surgical release of MN within the upper and middle third of the forearm's anterior surface using the standard technique [28] was 14.0 months, for 2 patients after selective nerve transfer of AIN - 13.5 months, for 3 patients with disease duration <3 months – 10.5 months, for 3 patients with disease duration >3 months – 17.0 months.

Patients with a history of disease <3 months showed better regression of the initial neurological deficit ***(***see ***Table 3)***. All patients with disease duration <3 months showed return of sensation in the zone of autonomous innervation of the *n. cutaneus palmaris n. mediani* (on average – +0.7 points on the MRC Scale) and strength characteristics of FPL and FDP2 (on average – +1.7 points on the MRC Scale). One patient with a disease duration <3 months had no effective recovery of *m. opponens pollicis*. All patients with a disease duration <3 months were able to reproduce PG at the end of the follow-up period. In one patient with a disease duration <3 months without effective recovery of the *m. opponens pollicis* function the reproduction of other basic pinch grips of the hand [31] was significantly impaired.

In none of the patients with disease duration >3 months the regression of sensory neurological deficit in the zone of autonomous innervation of *n. cutaneus palmaris n. mediani* was recorded ***(***see ***Table 3)***, but healing and epithelization of a chronic trophic ulcer above the 1st finger elevation muscles was observed in 2 patients with trophic function disorders in the zone of autonomic innervation of the *n.cutaneus palmaris n. mediani*. No relapse of the trophic ulcer was registered in these patients during the entire follow-up period, despite the absence of regression of sensory neurological deficit. In 1 patient with disease duration >3 months no recovery of strength characteristics of FPL and FDP2 and *m. opponens pollicis* was observed during the 24-month follow-up after performing only surgical release of the MN within the upper and middle third of the forearm's anterior surface using the standard technique [28] ***(***see ***Table 3)***. In 2 patients with disease duration >3 months, 11 and 16 months after selective nerve transfer of AIN, effective recovery of strength characteristics of FPL and FDP2 (+3 points on the MRC Scale) was recorded ***(***see ***Table 3)***. None of the patients with disease duration >3 months showed a recovery in the strength characteristics of *m. opponens pollicis* ***(***see ***Table 3)***.

Although the effective recovery of FPL and FDP2 strength characteristics in 2 patients with disease duration >3 months after selective reinnervation of AIN, effective reproduction of PG in everyday life was not possible ***(Fig. 7)***. The lack of restoration of *m. opponens pollicis* function ***(Fig. 7, C)*** due to the long-term compression of the MN caused by PTS created prerequisites for the hyperadductive position of the 1st finger ***(Fig. 7, D)*** due to the preserved function of the transverse and oblique heads of *m. adductor pollicis*, while the reduction of the hyperadductive position of the 1st finger ***(Fig. 7, A)*** with the active involvement of the restored FPL and FDP2 contributed to the reproduction of the PG ***(Fig. 7, B)***. Two patients with a specified "biomechanical problem" were suggested to compensate for the lack of function of *m. opponens pollicis* at the expense of an external orthosis, the use of which in everyday life allowed the most effective involvement of the distal segments of the injured upper limb (hand and fingers) to reproduce not only the PG, but also other types of basic grips of the hand [31].

The only clinical neurological criterion that underwent significant changes in all patients, regardless of the time of the disease, was complete regression of neuropathic pain ***(***see ***Table 3)***: on average, pain intensity according to VAS decreased by 4 points (from 2 to 8 points).

Table 3. Clinical outcomes of surgical treatment of patients with median nerve compression in the area of the forearm segment ("pronator teres" syndrome)

№ i/o	Disease duration		Surgical treatment method	Follow-up, months	Clinical picture*				
					S	M			VAS, points
	<3 months	>3 months				FPL	FDP2	Th.	
1	2		Neurolysis	18	+2	+5	+5	+5	+0
2		6	Neurolysis	24	w/ch	w/ch	w/ch	w/ch	+0
3	3		Neurolysis	9	+1	+4	+4	w/ch	+0
4		6	Neurolysis +N	11	w/ch	+4	+4	w/ch	+0
5	1		Neurolysis	5	+1	+5	+5	+5	+0
6		8	Neurolysis +N	16	w/ch	+3	+3	w/ch	+0

Note: N – neurotization of the anterior interosseous nerve; S - is an assessment of preservation of sensory function in the zone of autonomous innervation *n. cutaneus palmaris n. mediani* according to the MRC Scale; M - assessment of muscle motor function according to the MRC Scale; FPL - flexor pollicis longus muscle; FDP2 – deep flexor muscle of the 2nd finger; Th. - *m. opponens pollicis* + – positive dynamics or regression of sensory and motor deficits when assessing function according to the MRC Scale and reduction of the neuropathic pain syndrome intensity according to VAS; w/ch – without changes compared to the original data; pale green color – moderate regression of neurological deficit; green color – significant regression of neurological deficit;

* - any figure indicates the final (the latest data according to the duration of the patient's follow-up) indicator of the assessment of sensory and motor functions, as well as the intensity of the neuropathic pain syndrome according to VAS

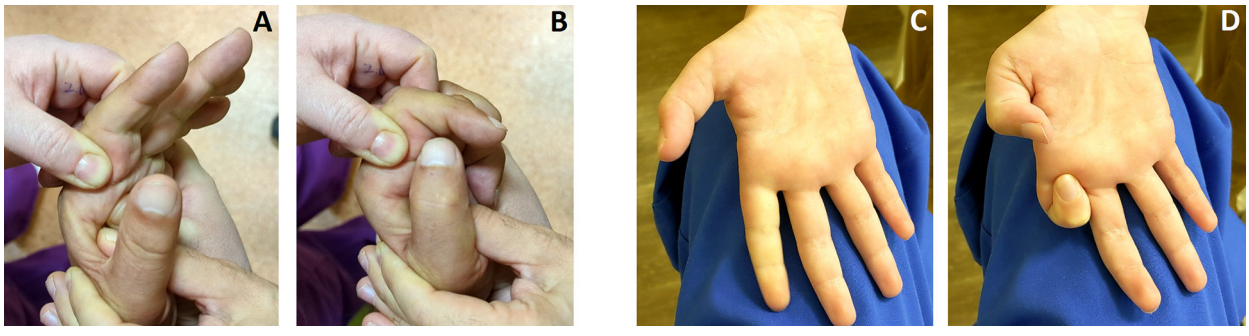

Fig. 7. Results of selective nerve transfer of the anterior interosseous nerve in patients with disease duration >3 months - reproduction of the pinch (final) grip:
A and B - 16 months after selective nerve transfer of the anterior interosseous nerve due to the distal branch of the median nerve to the pronator teres of the forearm;
C and D - 11 months after selective nerve transfer of the anterior interosseous nerve due to the radial nerve branches to the the extensor carpi radialis brevis;
A – manual reduction of the hyperadduction position of the 1st finger;
B – effective reproduction of pinch (final) grip after manual reduction of the hyperadduction position of the 1st finger;
C - no tone of m.opponens pollicis, "monkey" hand;
D – hyperadduction position of the 1st finger and ineffective reproduction of the pinch (final) grip

Discussion

Timely diagnosis [1] of the most common compression neuropathies of MN in the area of the forearm and hand segment is crucial for a positive outcome, regardless of treatment methods. There is no set time limit for the duration of multimodal therapeutic treatment [1], but in most cases it can provide regression of the primary neurological deficit [1], particularly of the neuropathic pain syndrome, on most cases, leaving enough time for verification of the diagnosis using modern radiological [15] and electrophysiological techniques [2–4]. Despite the possibilities of auxiliary diagnostic methods, most of the initially established diagnoses of MN neuropathies are based only on pathognomonic clinical and neurological signs [1].

The differential diagnosis of compression MN neuropathies of the forearm and hand has not been sufficiently addressed by the therapeutic, as well as the surgical, community. Even in developed countries, there is a problem of misdiagnosis or false-point diagnosis of MN compression, resulting in ineffective therapeutic and incorrect surgical tactics [9,32]. Thus, according to W.K. Olehnik et al. [9] and C.G. Hagert et al. [32], 32 and 49% of patients, respectively, underwent primary surgical decompression of the MN in the area of the carpal tunnel due to CTN before the final diagnosis of PTS was established [1,9,32]. There is no unified algorithm for the differential diagnosis of compression neuropathies of MN in the area of the forearm and hand segment [1]. The diagnostic algorithms proposed [1], in particular for the detection of the "double crash" syndrome [8] - the combination of CTN and PTS have a more recommendatory nature. None of the proposed algorithms is included in the list of approved national and industry standards [30].

It is generally accepted that the development of a pronounced motor and/or sensory deficit, regardless of the rate of onset, with established MN neuropathy of compression origin at any point within the segment of the shoulder, forearm or hand requires the termination of any therapeutic measures due to their ineffectiveness and is an absolute indication for surgical release [33]. Analysis of the results of surgical treatment of 6 consecutive cases of neuropathy caused by verified compression of the MN by the muscular and tendon components of the deep head of the pronator teres of the forearm (PTS) has led to the identification of the main factor contributing to or excluding the achievement of a positive outcome (regression of the existing neurological deficit at the time of involvement in research) after surgical decompression of the MN, – the duration of MN compression.

None of the patients with early presentation (<3 months) to specialized neurosurgical care developed a significant motor deficit - a decrease in the strength characteristics of FPL and FDP2 **(**see ***Table 2)***. Appropriate application of a complex of clinical-neurological and electrophysiological methods of diagnosis made it possible to determine the point of compression in a timely manner and eliminate the compression of the MN by a surgical method before irreversible denervation changes in the effector muscles ***(***see ***Table 3)***. In one patient with early treatment, in addition to significant regression of sensory and motor deficits and regression of neuropathic pain syndrome ***(***see ***Table 3, № 3)***, there was no recovery of function of *m. opponens pollicis*. Having analysed the initial clinical, neurological and electrophysiological findings in this patient and compared them with the elements of the algorithm [8], we do not exclude the possibility of misinterpretation of the available data at the pre-surgical stage and, accordingly, errors in the diagnosis of the "double crash" syndrome.

In all patients with late presentation (>3 months) for specialized neurosurgical care, chronic MN compression led to progressive, gradual and slow motor deficits - a significant reduction in the FPL and FDP2 strength characteristics ***(***see ***Table 2)***. The application of a complex of clinical, neurological and electrophysiological diagnostic techniques made it possible to determine the point of compression and eliminate the compression of

the MN surgically. Carrying out only surgical release of MN in one patient with late presentation did not contribute to the recovery of strength characteristics of FPL and FDP2 ***(***see ***Table 3, №2)*** for the next 24 months. At the same time, performing MN decompression alone according to the standard technique [28] allowed a significant regression of sensory deficit and neuropathic pain syndrome ***(***see ***Table 3, №2)***.

Given the negative experience obtained in a patient with long-term MN compression (late presentation), the lack of predictable potential for spontaneous recovery of the function after performing MN decompression alone [28], two patients in a consecutive case series with disease duration of 6 and 8 months, respectively, underwent selective reinnervation of AIN to restore the FPL and FDP2 function ***(***see ***Table 2, №4 and №6)***. In these patients, after 11 and 16 months, effective recovery of strength characteristics of FPL and FDP2 was observed ***(***see ***Table 3)***. In none of these patients, despite the effective recovery of strength characteristics of FPL and FDP2, the possibility of reproduction of the PG was recorded. In our opinion, the distinct dysfunction of *m. opponens pollicis* prevented maximum involvement of the restored FPL and FDP2 function in the reproduction of PG. Although the dysfunction of *m. opponens pollicis* is not a typical clinical manifestation of PTS, in our opinion, long-term proximal (relative to the muscles of 1st finger elevation) compression of the MN could lead to the development of such symptoms. Given the fact that the initial clinical and neurological symptoms in patients with late presentation already corresponded to the typical clinical picture of PTS, and the differential diagnosis with "AIN" syndrome [34] did not pose any difficulties, the absence of vigilance of researchers and, accordingly, the absence of electrophysiological data concerning the presence of concomitant pathology (CTN) does not allow to deny the false interpretation of data and errors in the diagnosis of the "double crash" syndrome at the pre-surgical stage.

The results of this study show that it is the awareness of primary neurological care professionals about clinical diagnosis and treatment that allows early and, most importantly, timely presentation for specialized neurosurgical care. The surgical community's awareness of the existence of potential MN compression points in the segment of the hand and forearm [1] allows not only to clearly define and substantiate the clinical task for specialists in auxiliary diagnostic methods, but also to realize the appropriate time limits for productive use of surgical methods of influencing the course (early presentation) [1] and residual clinical manifestations (late presentation) of the disease [35,36].

Conclusions

1. Performing surgical release of the median nerve in the early period of the disease (<3 months) achieves a complete regression of pain neuropathic syndrome, as well as sensory and motor neurological deficits in patients with " pronator teres" syndrome.

2. Performing surgical release of the median nerve in the late period of the disease (>3 months) makes it possible to achieve complete regression of the neuropathic pain syndrome alone.

3. Performing surgical release of the median nerve in the late period of the disease (>3 months) does not contribute to the regression of sensory disorders.

4. The prognosis for the recovery of the "extrinsic" muscles of the hand, which are innervated by the anterior interosseous nerve (flexor digitorum longus 1 and deep flexor muscle of 2nd finger), is doubtful and, most likely, unfavorable.

5. Performing selective nerve transfer of the anterior interosseous nerve in patients with long-term compression of the median nerve caused by the "pronator teres" syndrome effectively restores only one of the motor components (the function of the "extrinsic" muscles of the hand) necessary for the reproduction of pinch (final) grip

6. Surgical release of the median nerve in patients with long-term compression caused by the "pronator teres" syndrome does not contribute to the recovery of any of the motor components ("intrinsic" muscles of the hand, in particular *m. opponens pollicis*) necessary for the reproduction of pinch (final) grip.

7. Reproduction of pinch (final) grip in patients with long-term compression of the median nerve caused by the "pronator teres" syndrome was impossible and did not depend on the type of surgical intervention performed.

8. Complete clinical-neurological, radiological and electrophysiological examination at the pre-surgical stage and correct interpretation of findings can significantly improve the functional outcomes of surgical treatment of isolated and multilevel compression neuropathies of the median nerve.

Information disclosure

Conflict of interest

The authors declare no conflict of interest.

Ethical approval

All procedures performed on patients comply with the ethical standards of institutional and national ethics committees, the 1964 Declaration of Helsinki and its amendments or similar ethical standards.

Informed consent

Informed and voluntary written consent to participate in the study was obtained from each patient.

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Authors' contribution:

Olexander O. Gatskiy: the concept and design of the study, collection and processing of materials, analysis of the data obtained, writing the text.

Ihor B. Tretyak: analysis of the data obtained, writing the text.

Vitalii I. Tsymbaliuk: analysis of the data obtained, writing the text.

Iaroslav V. Tsymbaliuk: collection and processing of materials, analysis of the data obtained, writing the text.

Olexander S. Lemeshov: collection and processing of materials, analysis of the data obtained, writing the text.

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