The Course of Pit Syndrome in Comorbid Patients and Predictors of Recovery: Clinical Observations

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Introduction

Annotation

Intensive Care Outcome Syndrome (ICU) is currently a problem affecting all intensive care strategies. Knowledge of the mechanisms of development of PIT syndrome contributes to the prevention of the disease in patients with disorders of respiratory function, neuromuscular transmission and cognitive processes that require long-term protection of vital functions. A significant influence on the formation of PIT syndrome and its severity is exerted by the degree of severity of the systemic inflammatory response as an individual reaction of the body, which determines the degree of neurological and psychological deficit. The presence of underlying somatic diseases, such as diabetes mellitus (including as part of the metabolic syndrome), significantly aggravates the course of PIT syndrome and helps to slow down the recovery process. The earliest possible start of rehabilitation and restoration measures of a passive and active nature on the basis of the intensive care unit with the subsequent expansion of the individual rehabilitation program on the basis of a specialized department contributes to the most favorable prognosis in the restoration of not only impaired vital functions, but also emerging disorders of the cognitive, motor, affective spheres, reducing thereby lengthening the patient's stay in hospital and improving his quality of life after discharge. The article describes clinical cases of the consequences of intensive care in patients of different age categories. Observation of the dynamics of the development of the syndrome and analysis of laboratory data in the presented patients allowed us to conclude that the course and severity of the inflammatory process affect the restoration of impaired functions.

Relevance

The provision of medical care in the field of anesthesiology and resuscitation has changed significantly over the past 15 years due to the development of innovative technologies, standardization of circulatory support and respiratory equipment in intensive care units (ICU), as well as standardization and improvement of educational programs. With the development of these areas, short-term results of treatment of ICU patients, including mortality and 28-day survival, have significantly improved, however, there is no data on the significant impact of the applied techniques in the long term, including improvement in the quality of life of patients [1–3]. In the 21st century, The Society of Critical Care Medicine (SCCM) held a stakeholder conference to address subacute/chronic physical and psychological problems after ICU discharge, at which the term “post-intensive care syndrome” (PICS) was proposed. [1, 3-4]. Currently, PIT syndrome is understood as a set of somatic, neurological and socio-psychological consequences of staying in an ICU that limit the patient’s daily life [1, 4-5]. In various combinations, they increase the length of stay in intensive care units and reduce the quality of life of patients years after returning to normal life. The most acute period of a critical condition, requiring treatment in an ICU, is, in a complex of protective and adaptive mechanisms aimed at restoring impaired self-regulation, the lowest point of the patient’s personal independence, since intensive care technologies in the form of hardware monitoring, partial or complete replacement of vital functions, along with the course of the underlying disease, create for the patient a status of limited or complete loss of autonomous existence [1]. With a successful outcome of the intensive stage of treatment, the patient expects restoration of independence in everyday life, which can reach premorbid levels or decrease depending on individual reparative processes [5]. The paradox of the complex of life-saving intensive care technologies is that it simultaneously aggravates the patient’s condition, creating a status of limited or complete loss of autonomy due to hardware monitoring with the formation of PIT

syndrome, which is the reason for the decrease in the quality of life of patients after discharge from the hospital [1, 3 , 6]. According to statistics, PIT syndrome develops in 20–40% of patients discharged from the ICU [7]. Patients may be disabled for a long time and need help from others, and some patients acquire permanent disability [2-3].

The high social significance of PIT syndrome dictates the need to find ways of its prevention and rehabilitation treatment. In order to ensure the quality of life of the patient, starting from his stay in the ICU, specialists from the Federation of Anesthesiologists-Resuscitators of the Russian Federation, the Association of Neuroanesthesiologists and Neuroresuscitators, as well as the Union of Rehabilitation Doctors of Russia have developed clinical recommendations "Rehabilitation in Intensive Care" (ReabIT, 2021). The accumulated experience set out in clinical guidelines and scientific evidence prove that early initiation of preventive treatment, despite depression of the level of consciousness, is more likely to restore the patient's quality of life, improving short-term prognoses (weaning from mechanical ventilation, early transfer of the patient from ICU to specialized department), as well as long-term prognoses (discharge of the patient and the patient's quality of life after hospitalization). In 1985, for the first time, the formation of PIT syndrome in patients staying for a long time in ICU conditions was described by K. Girard and TA Raffin, who called them "chronically critically ill".

In 1991, BJ Daly proposed organizing specialized care units for this category of patients. In 1998, G. Vanden Berghe proposed the terms "prolonged critical illness" and "protracted critical illness," which meant "extended," "long-term," or "protracted" critical condition" [8].

At the beginning of the 21st century, the term "PIT syndrome" was first voiced in the materials of a conference on "multidisciplinary interaction between resuscitators and rehabilitation specialists" [3, 5].

Currently, the concept of PIT syndrome has been formed by Doctor of Medical Sciences, Professor of the Department of Nervous Diseases and Anesthesiology-Reanimatology of the Ural State Medical Academy, Chairman of the Supervisory Board of the Autonomous Non-Profit Organization A.A. Belkin.

Formation of PIT syndrome, according to A.A. Belkin, includes several stages. The first stage is characterized by the influence of factors such as sedation, artificial ventilation, myoplegia, dysproteinemia, which, together with the severity of the patient's condition requiring a stay in the ICU, leads to prolonged immobilization of the patient. Within 3–7 days (the second stage of the formation of PIT syndrome), the patient develops physical disorders, such as critical illness polyneuromyopathy, gravitational gradient disorders and circadian rhythms. With prolonged immobilization and the absence of early rehabilitation measures, after 8–30 days the patient develops PIT syndrome with the development of a neurological deficit.

The main factor in the formation of PIT syndrome is bed rest, i.e. a method of positioning a patient during an ICU stay [1, 6-7]. described for the first time in the 19th century by the English surgeon J. Hilton as the main therapeutic strategy for treating seriously ill patients [6, 9]. In the 20th century, this method received the term "immobilization syndrome" or "nonuse phenomenon" due to the limitation of the patient's motor and cognitive activity [1, 4, 6]. According to the literature, the incidence of immobilization syndrome in patients staying in the ICU for more than 48 hours reaches 55–98% [6]. Immobilization syndrome is divided [4] into primary immobilization (as a result of forced motor restriction due to disease) and secondary, or therapeutic, due to medical influences (bed rest, sedation, muscle relaxation, artificial ventilation, etc.).

The causes of immobilization syndrome are:

• acute cerebral insufficiency (stroke, traumatic brain and spinal cord injuries, infections and intoxications of the central nervous system, etc.) - 65–80% [7];

• acute damage to the neuromuscular system (polyradiculoneuropathies, myopathies, myasthenic crisis) - 60–75% [10].

• complications of medical effects (bed rest, sedation, muscle relaxation, artificial ventilation, etc.) - 45–50% [11].

Clinically, PIT syndrome manifests itself as disturbances in four areas: physical, autonomic, cognitive and mental [12]. Physical disorders include the following neuronal and muscular disorders.

1. Polymyoneuropathy of critical conditions: an acquired syndrome of neuromuscular disorders such as polyneuropathy and/or myopathy, clinically manifested by muscle weakness due to a decrease in muscle mass and strength, resulting in motor disorders - paresis, symmetrical in nature, with a predominance in both proximal , and in the distal parts of the limbs. Flexion of the feet may develop [3, 13-14].

2. Respiratory neuropathy as a subtype of critical illness polyneuromyopathy, as a result of which respiratory failure develops with a prolongation of the patient's transition to spontaneous breathing. Respiratory neuropathy, according to the literature, occurs in 59% of all cases [7].

3. Dysphagia of inactivity (dysphagia acquired in the ICU): a clinical symptom of impaired swallowing function, which makes it difficult to transfer to full enteral nutrition and decannulation due to the risk of aspiration complications [5-6]. Mechanisms of development of dysphagia of inactivity: (a) post-intubation dysphagia as a complication of prolonged standing of the endotracheal tube; (b) dysphagia due to the disappearance of subglottic pressure with prolonged cannulation; (c) dysphagia as a manifestation of the syndrome of weakness acquired in the ICU [1, 3–5].

The following disorders are classified as vegetative factors.

1. Orthostatic insufficiency: a sharp decrease in blood pressure (up to the occurrence of fainting) when the body position changes from horizontal to vertical, leading to a decrease in the gravitational gradient. Gravity gradient is the maximum angle of elevation of the patient, corresponding to 90°, which prevents the development of signs of orthostatic insufficiency and helps maintain vital functions, such as blood pressure, heart rate, stable [13].
2. Circadian rhythm disorder: a type of sleep disorder in which there is an imbalance between the internal sleep-wake cycle and the external day-night cycle. Circadianity is a physiological process responsible for the production of hormones, alternating the sleep-wake cycle. The conditions of a patient's stay in the ICU lead to their changes and the development of long-term consequences of post-resuscitation dyssomnia [14-15].

Cognitive impairments include:

a) cognitive-afferent dissonance: a human condition with an artificially reduced influx of sensory impulses from the sensory organs and peripheral organs against the background of sedation. It can cause pain, disorders of perception of parts of one's own body and other disorders of proprioception. The essence of this phenomenon is that due to the absence of familiar sensations (smells, touches, sounds of voices of loved ones), in place of which there are other stimuli that are often negative and even aggressive in nature (anxiety of monitors, groans of patients, rude tone of staff, etc.). d.), upon awakening, the patient experiences a disruption in the associative sphere and develops a state of derealization and depersonalization. The most severe manifestation of cognitive-afferent dissonance is delirium [1].

b) affective disorders:

• depression (a disorder accompanied by persistent depressed mood, negative thinking and slow reactions);
• anxiety (emotional reaction to factors that are associated with external circumstances or internal pathological processes);
• post-traumatic stress disorder (a severe condition of the patient resulting from a single or repeated event that has a negative impact on him) [16].

These clinical manifestations occur against a background of repeated exposure to physical pain and discomfort, as well as episodes of disorientation and confusion during prolonged sedation. Factors that increase the likelihood of developing PIT syndrome are: • time spent in the ICU; • presence of foreign bodies in the body (drains, catheters, probes); • stress from staying in the ICU (constant noise, room lighting); • aggravated premorbid condition (presence of chronic somatic and mental diseases, addictions); • elderly age.

We present an analysis of a clinical case of a patient with established PIT syndrome.

Description Of Clinical Cases:

Clinical case 1:

About the patient. Patient A., 29 years old, was admitted to the pulmonology department of a medical institution on January 23, 2022; due to the severity of her condition, she was placed in the department of anesthesiology, resuscitation and intensive care (ICU) No. 2. From the anamnesis: she became acutely ill on January 13, 2022, when general weakness and hyperthermia appeared up to 39.6°C, cough, aches, chills, headache. She treated herself. On January 20, 2022, due to increasing weakness and shortness of breath, the ambulance team transported her to the medical facility at the place of attachment. Upon admission, the condition was severe, due to severe respiratory failure (desaturation 75%, tachypnea up to 24/min). Research upon admission. According to laboratory monitoring, there is an increase in the level of inflammatory markers: C-reactive protein (CRP) up to 24.4 mg/l, procalcitonin concentration up to 1.2 ng/ml; in the general blood test, leukocytosis was up to 14.5 × 109/l with a neutrophil shift of up to 85.0%. According to multislice computed tomography (MSCT) of the chest: subtotal consolidation of both lungs, probably as part of respiratory distress syndrome; against this background, inflammatory infiltration may occur. The diagnosis was made: "Consequences of COVID-19. Bilateral subtotal viral-bacterial pneumonia of severe course." History: diabetes mellitus, type 2; target level of glycated hemoglobin <7.0%.

Taking into account the need for long-term protection of respiratory function using artificial lung ventilation, percutaneous dilatation-puncture tracheostomy was performed (on the 4th day). Dynamics of

the state. On 01/23/2022, due to increasing signs of respiratory failure (hypoxemia, PaO2, 48.7 mm Hg; desaturation, SaO2, 58%; tachypnea up to 35/min), she was transferred to extracorporeal membrane oxygenation, which she was on until 03/05/2022 (within 43 days).

Against the background of intensive therapy and forced long-term immobilization, on the 21st day the patient's neurologist verified PIT syndrome with the development of polyneuromyopathy in the form of flaccid tetraparesis with a decrease in muscle strength up to 4 points in the upper extremities and up to 2 points in the lower extremities (Rivermead mobility index - 1; Berg balance scale - 0; modified Rankin scale mRS - 4; Barthel index - 40; Rivermead activity of daily living scale - 41; functional independence scale, FIM - 76; rehabilitation routing scale, RRM - 5); dysphagia of inactivity (positive three-swallow test, laryngoscopy data), cognitive (Montreal Cognitive Assessment Scale, MoCA - 23 points) and affective (Beck scale - 22 points, Spielberger - 48 points) disorders. Electroneuromyography data revealed a decrease in the amplitude of the M-response from both motor and sensory fibers and widespread muscle denervation. Main diagnosis. Polyneuromyopathy of mixed origin (critical conditions, dysmetabolic). Tetraparesis to severe in the lower extremities. Background disease. Consequences of COVID-19: severe bilateral polysegmental viral-bacterial pneumonia. KT4. Extracorporeal membrane oxygenation from 01/23/2022 to 03/05/2022. Type 2 diabetes mellitus, target glycated hemoglobin level target glycated hemoglobin level <7.0%

On the 14th day, the patient showed positive dynamics in the form of a decrease in inflammatory markers: CRP to 9.3 mg/l, fibrinogen to 2.727 g/l, procalcitonin to 0.19 ng/ml; decrease in leukocytosis level to 6.7 × 109/l with a decrease in neutrophil concentration to 75.2%. According to MSCT of the chest: resolution of some of the inflammatory changes in the lungs. On the 28th day from the onset of the disease, despite massive antibacterial, nutritional, metabolic therapy, the patient had a fever with a repeated increase in inflammatory markers: CRP up to 67.7 mg/l, fibrinogen up to 0.813 g/l, procalcitonin up to 2, 9 ng/ml, leukocytosis up to 4.6×109/l with a neutrophil shift up to 85.5%. According to repeated MSCT of the chest, negative dynamics were revealed: in the upper lobe on the left, areas of ground glass-type lung tissue infiltration appeared over a large area; in the lower left lobe, areas of ground glass-type lung tissue infiltration appeared in combination with areas of consolidation, while the lower the lobe is completely affected; in the lower lobe on the right, the previously observed zone of consolidation has increased, against this background a small air cavity up to 5 mm in size can be traced, possibly an area of destruction; in the upper and middle lobes on the right, areas of ground glass-type infiltration appeared, and areas of consolidation previously observed here without dynamics. The patient was re-examined specialized specialists, including a clinical pharmacologist; therapy adjusted. On the 43rd day of stay in the ICU, when regression of the inflammatory process has been achieved (according to laboratory control: CRP 7.6 mg/l, fibrinogen 4.854 g/l, leukocytes 3.0×109/l; neutrophils 39.2%), After partial regression of respiratory failure, the patient was transferred to an auxiliary ventilation mode. On the 54th day of stay, the patient was transferred to spontaneous breathing.

To stabilize the condition, in addition to complex drug therapy, rehabilitation and recovery measures were started - intermittent compression of the lower extremities, magnetic therapy on the lower extremities, passive verticalization up to 30° with subsequent expansion of the motor regime. On the 61st day of illness, the symptoms of respiratory failure resolved amid ongoing rehabilitation and recovery measures.

For further rehabilitation treatment, the patient was transferred to a specialized department (neurological department for the treatment of patients with acute cerebrovascular accident). Rehabilitation and recovery measures. The patient was examined by members of a multidisciplinary rehabilitation team, and an individual program of rehabilitation and recovery measures was developed aimed at correcting disorders in the physical, cognitive and affective spheres. On the 64th day of stay in a specialized department, motor activity expanded: the patient was activated within the bed, and began to sit down with unilateral support. Taking into account vegetative tests, placement in a chair with legs lowered and feet supported began, and individual classes in active physical therapy were expanded. In order to train and restore independent swallowing, classes to strengthen the pharyngeal muscles were conducted under the supervision of a speech therapist-aphasiologist. She was decanalised on the 68th day of her stay. In order to correct cognitive impairment, individual sessions with a neuropsychologist were conducted, against the background of which, on the 68th day of illness, positive dynamics were noted: 27 points when re-screening cognitive impairment according to MoCA. On the 71st day, the patient's motor activity expanded: she walked up to 20 m with minimal support.

Exodus. On the 81st day of illness, the patient was in a state of moderate severity, closer to relatively satisfactory, and was discharged for outpatient follow-up treatment. In the neurological status, there was an increase in strength in the extremities, proximal muscle groups of the upper extremities up to 4.5 points, in

the proximal muscle groups of the lower extremities up to 4 points, in the distal: on the right in the dorsal flexors up to 3 points, in extensors up to 4 points; left: up to 4 points (Rivermead mobility index - 7; disability on the Rankin scale - 4; Barthel index - 55; Rivermead activity of daily living scale - 52; FIM - 86; ShRM - 4). Expanded physical activity: the patient moved up to 50 m 4–5 times a day within the department with minimal support.

Discussion:

The presented cases describe the formation of a syndrome of consequences of intensive care with the development of polyneuromyopathy, dysphagia, cognitive and affective disorders in patients with both primary somatic (pulmonological) and central neurological disorders.

According to the literature, there are three mechanisms for the development of polyneuromyopathy [7, 13].

• increased glycemic levels, which naturally occur in critically ill patients condition;

• formation of a systemic inflammatory response;

• the use of corticosteroids and drugs that block neuromuscular transmission, widely used in intensive care.

There are also 3 factors in the formation of muscle weakness, which are based on disorders of microcirculation, metabolism, reversible channelopathies and bioenergetic dysfunction:

• atrophy and necrosis of muscle fibers (loss of actin and myosin), loss of ability of muscle fibers to reduction, which is characteristic of the chronic phase muscle weakness;

• loss of the ability to generate action potentials, characteristic of the acute phase of muscle weakness [3, 7, 13].

• degeneration of sensory and motor axons.

Major changes at the microvascular level are associated with increased E-selectin expression, increased capillary permeability, decreased blood oxygen levels, increased cytokine production, ion channel dysfunction, increased reactive oxygen species, mitochondrial dysfunction, activation of proteolytic enzymes, and apoptotic mechanisms. Proteolytic and lysosomal enzymes (transforming growth factor β and mitogen-activated protein kinase) are the main factors of inflammation and stress, which further leads to the breakdown of actin and myosin. Activation of the proteolysis mechanism promotes protein loss due to myosin heavy chains, which reduces the volume of muscle mass of a seriously ill person by an average of 1.6–2% per day. Proteolysis of structural proteins explains the long course of polyneuromyopathy and slow recovery in chronic critical condition of the patient. The pathogenesis of mitochondrial dysfunction causes primary axonal degeneration, mainly in the distal parts of the nerve, where highly energy-dependent systems of axonal transport of structural proteins are realized, which explains the rapid axonal regeneration and early recovery of some patients with distal nerve damage [17, 18]. Summarizing the data from the described clinical cases, we identified the following unfavorable factors that aggravate the course of PIT syndrome and slow recovery of impaired functions (Table 1).

Conclusion:

Thus, according to the results of the analysis of the presented clinical cases, we have established the following direct correlation: the formation of PIT syndrome with pronounced neurological and psychological deficits, difficulties in weaning from artificial ventilation and restoration of full spontaneous breathing is more expected with a pronounced systemic inflammatory response and reaction of the body to a provoking pathogen against the background of prolonged forced immobilization. The earliest possible start of rehabilitation and restoration measures of a passive and active nature on the basis of the intensive care unit with the subsequent expansion of the individual rehabilitation program in a specialized department contributes to the most favorable prognosis for the restoration of impaired vital functions, as well as developing disorders of the cognitive, motor, and affective spheres, thereby reducing the length of stay of the patient in the hospital and improves the patient's quality of life after discharge from the hospital. The presence of underlying somatic diseases, such as diabetes mellitus (including as part of the metabolic syndrome), significantly aggravates the course of PIT syndrome and helps to slow down the recovery process. Of course, the topic raised requires further in-depth study in order to identify the gradation of biological markers in comparison with the clinical manifestations of the severity of PIT syndrome and the formation of predictors of restoration of impaired functions in the long term.

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Table 1. Dynamics of the main clinical and laboratory parameters in comparison with the severity of the manifestation of PIT syndrome

Basic indicators	On admission			7th day			28th day			43 days			50th day		60th day
	Cl 1	Cl 2	Cl 3	Cl 1	Cl 2	Cl 3	Cl 1	Cl 2	Cl 3	Cl 1	Cl 2	Cl 3	Cl1	Cl 2	Cl 1
SRP, mg/l	24.4	91.6	42.6	7.6	136.7	23.2	67.7	42.66	0.10	7.6	53.34	0.09	140.4	24.67	18.1
Fibrinogen,g/lg/l	1.87	4.239	3.358	1.17	4,671	4.9	0.813	2.7	1.7	4,854	2.9	1.6	3.6	2.7	4,854
Procalcitonin,ng/mlng/mL	1.2	<0.12	<0.12	0.56	<0.12	<0.12	2.9	<0.12	55.7	<0.12	<0.12	<0.12	<0.12	<0.12	<0.12
Leukocytes, 109/l	14.5	5.1	8.7	13.3	6.7	6.1	4.6	2.52	5.5	3.0	6.88	3.4	6.7	6.8	6.70
Neutrophils, %	85.0	71.9	81.0	77.2	87.6	64.0	85.5	55.6	63.4	39.2	70.9	47.7	78.3	75.3	49.5
Prothrombin index, secПТИ (sec)	12.7	16.8	10.7	13.5	10.4	13	14.5	12.4	12.3	13.6	14.2	12.3	eleven	12.3	13.6
Tetraparesis: A - 5 points B - 4 points C - 3 points D - 2 points E - 1 point	v/k - B n/k — D	h/c — D n/k — E	v/k - B n/k — B	v/k - B n/k — D	h/c — D n/k — E	v/k - B n/k — B	v/k - B n/k — D	h/c — D n/k — E	v/k - B n/k — B	v/k - B n/a: pr - B d (s) - C (p) - C	v/k - B n/a: pr — C d - B	v/k - B n/k — B	v/k - B n/a: pr - B d (s) - C (p) - B	v/k - B n/a: pr — C d - B	v/k - B n/a: pr - B d (s) - C (p) - B
Dysphagia of inactivity	+	+	+	+	+	+	+	+	-	+	-	-	+	-	-
Affective disorders (Berg scale; Spielberger scale)	+	+	+	+	+	+	+	+	-	+	-	-	+	-	-
Cognitive impairment	<23	<23	<23	<23	<23	<23	<23	>23	>23	<23	>23	>23	<23	>23	>23

(MoCA): ~~<23~~ ~~>23~~															

*Note.*Cl 1–3 – clinical cases 1–3; CRP—C-reactive protein; i/c - upper limb; n/k - lower limb; pr - proximal d - distal; d (s) - distal/flexors; (p) - extensors.

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