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SMALL FIBER NEUROPATHY IN THE PATHOGENESIS OF POST-COVID SYNDROME

© Alena A. Basina[1], Assiya Akhmetova[1], Natalia Yu. Gavrilova[1, 2], Lidia A. Soprun[1, 2], Victoriia A. Volovnikova[1], Vladimir I. Utekhin[1, 4], Leonid P. Churilov[1, 3]

[1] Saint Petersburg State University. Universitetskaya embankment, 7-9, Saint Petersburg, Russian Federation, 199034
[2] Saint Petersburg University's (SPU) N.I. Pirogov Clinic of High Medical Technologies. Embankment Fontanka River, 154, Saint Petersburg, Russian Federation, 190020
[3] Ministry of Health of Russian Federation, Federal State Budgetary Institution "St. Petersburg Research Institute of Phthisiopulmonology". Ligovsky Ave., 2/4, Saint Petersburg, Russian Federation, 191036
[4] Saint Petersburg State Pediatric Medical University. Lithuania 2, Saint Petersburg, Russian Federation, 194100

Contact information: Alena A. Basina — 6th year student of the Faculty of Medicine. E-mail: a.a.lamova@mail.ru ORCID: https://orcid.org/0009-0000-0087-8807

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Abstract. ***Introduction.*** Small fiber neuropathy (SNF) is a condition that occurs due to hereditary, metabolic, autoimmune, infectious and other diseases. Data on the possible role of SNF in the pathogenesis of post-Covid syndrome (PCS) are rare. ***Aim:*** To review literature on small fiber neuropathy in the pathogenesis of post-Covid syndrome. Summarize the authors' many years of experience working with patients with post-viral immunological complications. ***Results.*** There are several stages in PCS pathogenesis including antigenic mimicry of viral particles with human proteins, activation of coagulation and neuroglia, and the long-term presence of residual viral particles in certain areas of the central nervous system. Increased production of nonspecific antibodies allows us to consider PCD as an immunological process. The lack of a gold standard for instrumental diagnostics, given the variety of clinical manifestations of PCS, makes diagnosis difficult. Neuropathic pain and autonomic dysfunction in PCS patients combined with normal electroneuromyography (ENMG) indicators can be explained by the presence of SNF in the structure of the pathogenesis of PCS. This hypothesis is confirmed by data from confocal microscopy and skin biopsy with determination of the density of intradermal nerve endings in patients suffering from PCS, as well as by the clinical observations of the authors of the article. ***Conclusion.*** Consideration of small fiber neuropathy as an important stage in the pathogenesis of post-Covid syndrome opens new horizons for the diagnosis of post-Covid syndrome.

Keywords: post-Covid syndrome, small fiber neuropathy, new coronavirus infection, COVID-19, SARS-CoV-2

НЕЙРОПАТИЯ МАЛЫХ ВОЛОКОН В ПАТОГЕНЕЗЕ ПОСТКОВИДНОГО СИНДРОМА

© Алёна Алексеевна Басина[1], Асия Ахметова[1], Наталия Юрьевна Гаврилова[1, 2], Лидия Александровна Сопрун[1, 2], Виктория Александровна Воловникова[1], Владимир Иосифович Утехин[1, 4], Леонид Павлович Чурилов[1, 3]

[1] Санкт-Петербургский государственный университет. 199034, Российская Федерация, г. Санкт-Петербург, Университетская наб., 7–9
[2] Клиника высоких медицинских технологий им. Н.И. Пирогова Санкт-Петербургского государственного университета. 190020, Российская Федерация, Санкт-Петербург, наб. реки Фонтанки, 154
[3] Федеральное государственное учреждение «Санкт-Петербургский научно-исследовательский институт фтизиопульмонологии» Федерального агентства по высокотехнологичной медицинской помощи. 191036, Российская Федерация, г. Санкт-Петербург, Лиговский пр., 2/4
[4] Санкт-Петербургский государственный педиатрический медицинский университет. 194100, Российская Федерация, г. Санкт-Петербург, ул. Литовская, 2

Контактная информация: Алёна Алексеевна Басина — студентка 6-го курса медицинского факультета. E-mail: a.a.lamova@mail.ru ORCID: https://orcid.org/0009-0000-0087-8807

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Резюме. ***Введение.*** Нейропатия малых волокон (НМВ) — состояние, возникающее при наследственных, метаболических, аутоиммунных, инфекционных и других заболеваниях. Данные о возможной роли НМВ в патогенезе постковидного синдрома (ПКС) единичны. ***Цель*** — обзор литературы о нейропатии малых волокон в патогенезе постковидного синдрома и обобщение многолетнего опыта работы авторов с пациентами, имеющими поствирусные иммунологические осложнения. ***Результаты.*** Среди звеньев патогенеза ПКС выделяют антигенную мимикрию вирусных частиц с белками человека, активацию коагуляции и нейроглии, длительное присутствие резидуальных вирусных частиц в отдельных областях центральной нервной системы. Повышение выработки неспецифических антител позволяет рассматривать ПКС как иммунологический процесс. Отсутствие «золотого стандарта» инструментальной диагностики при разнообразии клинических проявлений ПКС затрудняет постановку диагноза. Нейропатическая боль и вегетативная дисфункция при ПКС на фоне нормальных показателей электронейромиографии (ЭНМГ) могут быть объяснены наличием НМВ в структуре патогенеза ПКС. Эта гипотеза подтверждается данными конфокальной микроскопии и биопсии кожи с определением плотности интрадермальных нервных окончаний у пациентов, страдающих ПКС, а также клиническими наблюдениями авторов статьи. ***Заключение.*** Рассмотрение нейропатии малых волокон в качестве важного звена патогенеза постковидного синдрома открывает новые горизонты для диагностики постковидного синдрома.

Ключевые слова: постковидный синдром, нейропатия малых волокон, новая коронавирусная инфекция, COVID-19, SARS-CoV-2

INTRODUCTION

On the 30th of January 2020, the World Health Organization (WHO) declared the risk of a new coronavirus infection (NCI), which subsequently killed at least 7 million people, with a cumulative incidence of more than 771 million [55]. On the 5th of May 2023, the official end of the pandemic was declared, but doctors are still struggling with the consequences of the disease: immunological, neurological, respiratory, cardiovascular and cognitive impairments that can significantly affect the quality of life and daily activity of patients [40].

According to WHO experts, 10-20% of NCI patients may develop a condition called post-COVID-19 syndrome (PCS, post-COVID-19 condition, post-COVID-19 syndrome, long-COVID) [56]. Practicing physicians experience considerable difficulties in the management of such patients. Due to the peculiarities of the course of new strains of NCI (asymptomatic course or course like acute respiratory viral infections (ARVI) in a mild form, without loss of sence of smell), it is difficult to establish the main criterion of PCS, i.e. the connection with the disease. Clinical manifestations of this syndrome are nonspecific and include manifestations characteristic of psychiatric, neurological, endocrine and some other diseases, so it often becomes a diagnosis of exclusion. The search for a “gold standard” of PCS diagnosis is still underway, as the links in the pathogenesis of this condition are not fully understood. One hypothesis that could explain the onset of most symptoms is small fiber neuropathy (SFN). This review summarizes our experience of many years of work with patients with postviral, including postviral immunologic complications, who were first studied at the Laboratory of Autoimmunity Mosaics of St. Petersburg State University and then at the Center of the Study of Autoimmune Diseases and Consequences of New Coronavirus Infection of N.I. Pirogovs Clinic of High Medical Technologies in St. Petersburg.

POSTOVOID SYNDROME: CLINICAL DESCRIPTION

According to the WHO Delphi Consensus, PCS develops in patients with confirmed or suspected NCI, most often within 3 months of the disease. The characteristic symptoms presented in the Consensus must persist for at least 2 months and cannot be explained by another diagnosis [16].

The prevalence of post-covid syndrome is independent of the severity and duration of NCI and is found in asymptomatic course, in young patients, and even in children. The difficulty in diagnosing PCS is obvious in the case of asymptomatic NCI — it is impossible to confirm the fact of the disease after virus elimination from the body and to associate with it the symptoms that appear within 2–3 months after the disease [54]. It is estimated that 10 to 60% of patients with mild to moderate NCI experience PCS-like symptoms for 12 weeks or more [10]. According to our experience, it is the asymptomatic or low-symptomatic course of NCI that leads to the development of PCS. This can be explained by the fact that patients genetically prone to severe course of viral and bacterial infections develop severe classical viral pneumonia in case of NCI.

If the outcome of this process is favorable, PCS or other immunological complications are not formed. There is another group of patients, where there is an immunologic predisposition to a pronounced immune response. Such patients tolerate NCI easily, but may develop PCS, which lasts for weeks, months and even years. Among the patients receiving immunological treatment for PCS at the postcovid center of the N.I. Pirogovs Clinic of High Medical Technologies. For more than two years of work, no patient who was in intensive care or needed active respiratory support in the acute period of NCI has been identified, which can indirectly confirm this notion.

The pathogenesis of PCS still requires comprehensive study. Let us briefly review those links of PCS pathogenesis that seem to us the most significant.

Some experts attribute the progression of PCS to the direct action of the virus, while others note the predominant role of immunologic complications. The first hypothesis may explain anosmia — the prolonged presence of residual viral particles in the olfactory epithelium may cause inflammation and loss of sense of smell, as *in vivo* experiments demonstrated [15]. The development of meningitis or encephalitis is also possible against the background of persistence of viral infection [26]. The second point of view suggests activation of the immune response, most likely by the mechanism of antigenic mimicry with human proteins [13], or direct damage to the structures of the organism against the background of long-term systemic action of inflammatory mediators [11]. In this case, such complications as small fiber neuropathy, acute and chronic demyelinating polyneuropathies are formed [36, 48]. There is evidence of neuroglia activation, which provokes persistent inflammation in nervous tissue even after virus elimination [49]. Coagulation activation, microthrombosis and vasculitis [5, 37], which may contribute to the development of cognitive and mental disorders, are no less important aspects of NCI. Indirectly, these processes are confirmed by a decrease in brain metabolic activity and dysregulation of GABAergic chains found in patients with complaints of anosmia, “brain fog”, and chronic fatigue [51].

The WHO document presents a list of clinical manifestations typical for PCS, but our experience allows us to distinguish three main groups of symptoms: flu-like symptoms, small fiber neuropathy, and CNS symptoms (Fig. 1).

The first group of clinical observations includes such immunologic manifestations as temperature fluctuations ranging from 34.0 to 37.5 °C, constant or wave-like influenza-like condition, arthralgias and myalgias, tendinitis, chills, and weakness. The second group of clinical manifestations may be due to the lesion of small nerve fibers developing against the background of systemic action of inflammatory mediators and includes sensory (pain) and vegetative manifestations. Patients may describe classic complaints typical for polyneuropathy — burning or crawling goosebumps in the hands and feet, mainly at night or after exercise. Autonomic manifestations are diverse and include orthostatic reactions, syncopal states, dyspnea, gastrointestinal dysfunction, mycosal dryness, and hyperhidrosis [44]. It cannot be excluded that anosmia and agenesis may also be a manifestation of neuropathy, but this issue requires further research. Finally, the third group consists of neuropsychiatric disorders — persistent fatigue, depression, and anxiety. There is a special type of cognitive dysfunction described as “brain fog” — decreased attention, concentration, speed of information processing, and impaired executive functions [1].

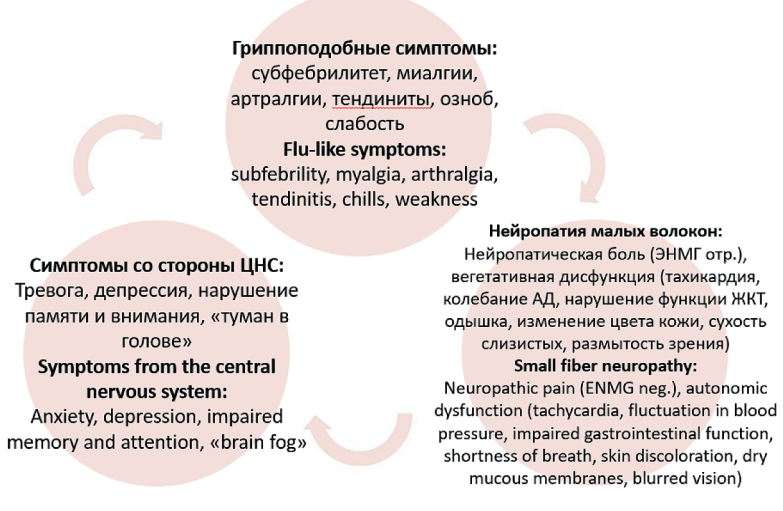

Fig. 1. Three main groups of clinical manifestations of post-Covid syndrome
Рис. 1. Три основные группы клинических проявлений постковидного синдрома

WHO declared a pandemic of NCI on the 30th of January 2020, i.e. the duration of the study of postcovirus syndrome is less than 4 years at the time of writing, so there is no reliable information on the duration of this condition. PCS-like complications have also been observed in patients with MERS and SARS-CoV-1 infections, which are also members of the highly pathogenic coronavirus family: myalgia, fatigue, and neuropsychiatric abnormalities persist in patients to date. The authors observed postherpetic patients with similar symptoms whose disease duration was 8 years or more. These observations suggest that PCS may last from a few months to several years [54].

SMALL FIBER NEUROPATHY AS A MANIFESTATION OF POSTCOVID SYNDROME

Small fiber neuropathy (SFN) is a selective lesion of myelinated A-delta fibers and unmyelinated C-fibers, which together account for up to 80–90% of all peripheral nerves and are responsible for the transmission of pain, temperature stimuli, and autonomic nervous system function.

According to a 2013 study in the Netherlands, the incidence of SFN is 52 per 100,000 and the incidence of new cases is 12 per 100,000 per year [35]. The prevalence is expected to increase with increasing awareness of this condition. SFN is somewhat more common in women [34]. Single clinical observations of SFN in children have been published [19, 29, 43]. The incidence of small fiber neuropathy in PCS is unknown due to the poorly studied nature of both conditions.

Metabolic disorders, especially diabetes mellitus [24], but also vitamin B12 deficiency and iron deficiency [7, 14] are frequent causes of SFN. Hereditary diseases can lead to this condition: Fabry disease [14, 23], Wilson disease [47], familial amyloidosis [3]. The relationship of SFN with HIV infection [17], hepatitis C [31], systemic tick borreliosis (Lyme disease) [32] has also been revealed. Toxic factors in the development of neuropathy include alcohol [25], neurotoxic drugs, and chemotherapy [9, 50]. SFN is often found in autoimmune diseases and immunologic conditions, which include fibromyalgia, systemic lupus erythematosus, sarcoidosis, and other systemic connective tissue diseases [20, 39]. In about 50% of cases, the cause remains unidentified, in which case idiopathic SFN is referred to [21].

With the growing interest in SFN, other causes for this condition are being identified, and novel coronavirus infection is one of them. There are observations that severe or moderate NCI can provoke neuropathy one month after the onset of the disease [33]. Other researchers claim that it may manifest during the course of the disease [2]. It cannot be ruled out that NCI vaccination may provoke the development of short-term autoimmune complications as well as SFN [22, 53].

The pathogenesis of SFN is still unclear, although some of its links are known. For example, antisulfatide antibodies, immunoglobulins M against trisulfated disaccharide heparan, and immunoglobulins G against fibroblast growth factor are found in patients [27]. A possible link between SFN and ion channel damage has been traced [45]. Antibodies against interferon-induced guanosine triphosphate (GTP)-binding protein MX1 may interact with a specific type of calcium channels that are found in the brain, astrocytes, pyramidal cells, neurons, and cerebral arteries [12]. Patients with intervertebral disc degeneration and chronic back pain have been found to have elevated levels of antibodies against interferon-induced GTP-binding protein MX1, which may cause pain through interaction with calcium channels, but this issue requires further study [41]. Antibodies against cytokeratin 8, a link in the pathogenesis of chronic demyelinating neuropathy, and drebrin-like protein, which plays an important role in synapse formation, endocytosis, and neuronal cytoskeleton function, have also been found in SFN patients [12]. It has been suggested that autoimmune damage may be mediated through tumor necrosis factor α (TNFα) and interleukins (ILs) -2, -6, and -8 [6, 52]. It is known that different genetic variants of peripheral potential-dependent ion channels play a role in the formation of neuropathic pain, and the peculiarities of their functioning may explain the occurrence of SFN [45]. Autopsy of postcovicular patients revealed neuritis with perivascular macrophage infiltrate, but there were no viral particles in the tissues. Thus, an inflammatory immune response persisted even after complete elimination of the virus. There is also evidence that up to a quarter of dorsal root neurons, which are the first neurons in the sensory pathways, express mRNA encoding receptors for SARS-CoV-2 and ACE2-protein. The development of a cross-reactive immune response activates the production of antibodies that can damage neural tissue [42]. In summary, it should be noted that despite the probable immunologic pathogenesis of SFN both in PCS and other nosologies, no unique and specific autoantibodies have been identified, which does not allow us to call SFN a classical autoimmune process. It would be more correct to call it an immunologic manifestation, and in the case of PCS — post-viral immunologic syndrome.

Within the clinical manifestations of PCS, clinical manifestations of SFN present as sensory disturbances and autonomic dysfunction. Patients describe tingling, burning or shooting pain, numbness, sensation of tactile stimuli as painful (allodynia), paresthesias, hyper- or hypoalgesia, temperature sensitivity disorder and other symptoms [38]. Lesions of the distal parts of the extremities in the type of “socks” and “gloves” are typical, but manifestations of gangliopathy in the

form of localized unstretched sensory disturbances in various parts of the body have also been described [6]. Autonomic nervous system damage in SFN can cause orthostatic hypotension, dry eyes and oral mucous membranes, disorders of the urogenital system (impotence, vaginal dryness, dysuria, incontinence), gastrointestinal tract (fecal incontinence, diarrhea or constipation, pseudoobstruction of the intestine), "hot flashes", accommodation disorders, palpitations [34]. New studies have shown that neurogenic rosacea may be one of the manifestations of SFN [28].

Diagnosis of SFN is difficult for several reasons. Firstly, clinical manifestations vary widely, as neuropathy affects both autonomic and sensitive fibers. Secondly, diagnostic criteria have been developed only for "sock" and "glove" type neuropathy of distal limbs, but not for localized lesions [46]. The third, reliable diagnosis is possible only with a combination of noninvasive methods and skin biopsy, which is a labor-intensive invasive procedure but is considered the "gold standard" [18].

Diagnostic measures possible in SFN [38, 46]:

1) quantification of small fibers:
   - skin biopsy;
   - confocal microscopy;
2) functional assessment of small fibers:
   - quantitative sensory testing;
   - microneurography;
   - evoked nociceptive potentials;
3) autonomic nervous system function testing:
   - thermoregulation study;
   - quantitative sensory testing;
   - quantitative study of axonal reflexes responsible for sweating;
   - study of cutaneous sympathetic reactions;
   - study of electrochemical potentials of the skin;
   - neuroindicator test (Neuropad©);
   - heart rate variability.

There are the following diagnostic criteria:

- *possible* SFN — there are symptoms and clinical manifestations of small fiber injury;
- *suspected* SFN — there is normal conduction of the calf nerve in combination with symptoms and clinical manifestations of small fiber damage;
- *confirmed* SFN — there is normal conduction of the calf nerve in combination with symptoms and clinical manifestations of small fiber damage, as well as decreased intraepithelial nerve fiber density and/or abnormal temperature thresholds on quantitative sensory testing (QST) [38].

The use of several diagnostic tests at once significantly increases the probability of a correct diagnosis. For example, it has been proposed to use a combination of 4 methods: quantitative sensory testing, skin biopsy, electrochemical skin potentials, and laser stimulation-induced potentials for the most reliable diagnosis [18].

We would like to emphasize two methods of SFN diagnosis in PCS for which there is international standardization.

The first one is skin biopsy with intraepidermal nerve fiber density examination, which is considered to be the "gold standard" for SFN diagnosis. Its positive aspects include a good level of evidence, the possibility of verifying the results by several histologists, the simplicity of calculations. The disadvantages include the need for an invasive procedure and a specialized histology laboratory. Also, the patient may not have nerve fiber lesions at the biopsy site, leading to a false-negative result. According to unpublished data from the authors, analysis of skin biopsies from patients with PCS showed that more than 60% of patients had decreased small nerve fiber density below age- and gender-specific norms.

The second, corneal confocal microscopy (CCM), is an opportunity for noninvasive assessment of SFN. It has been shown that NCI shows a decrease in the number of nerve fibers and an increase in the number of mature and immature dendritic cells compared to controls. It cannot be excluded that coronavirus has the ability to activate glial cells, provoking an immune system attack on neuronal tissues [8].

CCM data are consistent with individual clinical observations: autopsy of a patient who died of NCI revealed hyperactivation of neuroglia and neurophagia in some brain regions [4]. In a larger study of 184 patients, microglia activation was observed in 42.9% of cases, with microglial nodule formation in 33.3%, and astrogliosis was found in 27.7% of cases [30].

CONCLUSION

The relatively recent emergence of postcovid syndrome leaves many things unclear: prevalence, influence of the course of NCI on subsequent severity of symptoms, optimal diagnostic methods... The pathogenesis of postcovid syndrome also remains incompletely understood. Several possible variants of SARS-CoV-2 virus impact on the human nervous system are considered, including autoimmune damage, microglia activation, coagulation disorder, and prolonged presence of residual viral particles in certain regions of the CNS. All of the mentioned variants of virus exposure are capable of forming small fiber neuropathy, which, in our opinion, underlies such symptoms of postcoccygeal syndrome as dysautonomia and neuropathic pain. Understanding the pathogenesis of the pathological processes

underlying postcovical syndrome opens up the possibility of developing new methods of diagnosis and treatment that improve the quality of life of patients.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

Competing interests. The authors declare that they have no competing interests.

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