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SPECIAL ARTICLE

Toward Understanding COVID-19 Recovery: National Institutes of Health Workshop on Postacute COVID-19

Over the past year, the SARS-CoV-2 pandemic has swept the globe, resulting in an enormous worldwide burden of infection and mortality. However, the additional toll resulting from long-term consequences of the pandemic has yet to be tallied. Heterogeneous disease manifestations and syndromes are now recognized among some persons after their initial recovery from SARS-CoV-2 infection, representing in the broadest sense a failure to return to a baseline state of health after acute SARS-CoV-2 infection. On 3 to 4 December 2020, the National Institute of Allergy and Infectious Diseases, in collaboration with other Institutes and Centers of the National Institutes of Health, convened a virtual workshop to summarize existing knowledge on postacute COVID-19 and to identify key knowledge gaps regarding this condition.

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Over the past year, the SARS-CoV-2 pandemic has swept the globe, resulting in more than 113 million persons infected and 2.5 million deaths (1). However, the additional toll resulting from long-term consequences of the pandemic has yet to be tallied. Heterogeneous disease manifestations and syndromes are now recognized among some persons after their initial recovery from SARS-CoV-2 infection. Although a standardized case definition does not yet exist for these manifestations, in the broadest sense they represent a failure to return to a baseline state of health after acute SARS-CoV-2 infection. The various terms used to describe this condition have included postacute (or late) sequelae of COVID-19, post-COVID condition or syndrome, long COVID, and long-haul COVID. In this article, we use the general umbrella term of "postacute COVID-19" to refer to multiple disease processes that may have varying degrees of overlap (including but not limited to sequelae of critical illness and hospitalization in persons with COVID-19) and the entity of long COVID, which refers to prolonged health abnormalities in persons previously infected with SARS-CoV-2 who may or may not have required hospitalization. Of note, there is not yet a consensus on terminology, which will likely evolve with a better understanding of this condition.

Reported symptoms are wide-ranging and may involve nearly all organ systems, with fatigue, dyspnea, cognitive dysfunction, anxiety, and depression often described (2–5). Although abnormalities in imaging studies and functional testing have been reported, the long-term clinical significance of some of these findings is not yet clear (3,6,7). Postacute manifestations of COVID-19 have been seen in persons of all demographic groups and include reports of multisystem inflammatory syndrome in children (8,9). Although the epidemiology of the diverse manifestations of postacute COVID-19 is not yet known, the expansive global burden of SARS-CoV-2 infection suggests that the potential public health effects of postacute COVID-19 are significant if even a small proportion of persons with SARS-CoV-2 infection have prolonged recovery or do not return to their baseline health.

On 3 to 4 December 2020, the National Institute of Allergy and Infectious Diseases, in collaboration with other Institutes and Centers of the National Institutes of Health, convened a virtual workshop (available via videocast at https://videocast.nih.gov/watch=38878 and https://videocast.nih.gov/watch=38879) to summarize existing knowledge on postacute COVID-19 and to identify key knowledge gaps. The speakers and participants included epidemiologists, clinicians, clinical and basic scientists, and members of the affected community. The videocast was open to the general public and had more than 1200 registered participants.

Clinical Observations

In response to the increasing need to care for patients who do not return to their baseline state of health after COVID-19, specialty clinics have been established in the United States and globally. Observations from clinicians at the workshop provided valuable insights on the vast scope of symptoms, as well as how to begin to differentiate groups of patients by symptoms and structure potential treatment strategies. Clinicians and patients outlined the diverse needs and experiences of affected individuals, including multisystem symptoms (such as fatigue, mental health problems, and pain) and other signs and symptoms pointing toward specific organ systems (for example, renal, cardiac, pulmonary, and gastrointestinal). The diversity of clinical presentations clearly indicates the need for an integrated and multidisciplinary approach to treatment and care.

Postacute manifestations of COVID-19 have been reported among persons of all ages. The clinical presentation of SARS-CoV-2 infection in children differs from that in adults (10); of note, children typically have a mild presentation of acute disease. However, postacute manifestations of COVID-19 have also been reported in the pediatric population, including but not limited to multisystem inflammatory syndrome. Postacute manifestations of COVID-19 may be more challenging to diagnose in the elderly given the increased prevalence of preexisting cognitive dysfunction and other comorbid conditions. Thus, it will be necessary to study the disease processes in pediatric and geriatric populations, as well as in the general adult population.

Although an association between racial or ethnic background and postacute COVID-19 has not been established, racial and ethnic minority populations in the United States have been disproportionately affected by the pandemic. Furthermore, these populations are affected by structural and socioeconomic inequities that affect access to and delivery of health care for acute and postacute COVID-19. Presentations at the workshop showed that many persons affected by postacute COVID-19 have experienced stigma, and many struggle to be heard and believed by their family, friends, and health care providers about their health conditions. Awareness and understanding of the various manifestations of postacute COVID-19 are insufficient in many communities. Therefore, a comprehensive response to postacute COVID-19 must effectively address structural and socioeconomic inequities and increase communication with historically underserved and vulnerable communities.

Given the diversity of symptoms, patient populations, and scientific and clinical questions that need to be addressed—from pathologic mechanisms to structural barriers to care—postacute COVID-19 must be addressed in partnership with stakeholders, including clinicians, researchers, and patient advocacy groups. Individuals affected by postacute COVID-19 made invaluable contributions to the discussions at the workshop, providing crucial insights on the effects of COVID-19 on multiple organ systems and overall function and quality of life, as well as the stigma associated with SARS-CoV-2 infection and ongoing symptoms. Engagement of clinicians and community stakeholders is crucial for communication to improve public health, reduce stigma, and enhance health care. The National Institutes of Health is committed to building partnerships with academia, the medical establishment, and the patient community to share information and collaborate to better understand, prevent, and treat COVID-19 and postacute manifestations.

Key Gap: A Need to Define Epidemiology, Clinical Spectrum, and Natural History

One of the fundamental gaps identified during the workshop is the need to better understand the incidence and prevalence of the diverse manifestations of postacute COVID-19. Reports in the literature often describe only selected patient populations, such as those who were hospitalized, and measure outcomes at varied time points. Studies have examined periods ranging from 3 weeks to 7 months after either onset of symptoms or date of diagnosis (3,5,11–13). Workshop discussions highlighted the need for prospective studies that include representative populations of patients with acute COVID-19 to describe the epidemiology of the diverse array of postacute manifestations of COVID-19 at different time points after initial infection or disease onset, and across patient populations with varying characteristics and severity of acute disease. Studies using electronic health records may provide important data to answer certain epidemiologic questions and measure effects on health services; however, these studies may not capture the full spectrum of postacute COVID-19 and may miss important subgroups with postacute disease, such as those who lack access to care or who do not seek care because they believe that no treatment exists.

The workshop also highlighted the need to recognize the full clinical spectrum of postacute COVID-19, as well as the need to characterize the phenotypes of disease that are beginning to emerge and identify risk factors for their development. Weakness, cognitive dysfunction, and psychological disorders after hospitalization for severe COVID-19 may overlap with the well-described post–intensive care syndrome seen with other critical illnesses (14). Persistent fatigue, cognitive dysfunction, and other multisystemic symptoms after COVID-19 in persons who did not require hospitalization for their acute infection may characterize another distinct phenotype. Autonomic dysfunction and postural tachycardia may represent yet another. Comparisons have been drawn between postacute sequelae of COVID-19 and myalgic encephalomyelitis/chronic fatigue syndrome because many symptoms overlap. Care must be taken not to conflate the 2 conditions because neither is completely understood. Lessons learned from several decades of studying myalgic encephalomyelitis/chronic fatigue syndrome may be applied to studies of postacute COVID-19—and in fact, better understanding of both conditions may be mutually beneficial to patients in both groups.

The expected time course of COVID-19 recovery and what would constitute a "prolonged" or "postacute" manifestation of disease also have not yet been clearly defined. One proposed framework for symptomatic SARS-CoV-2 infection includes an acute stage from weeks 0 to 1 after symptom onset, a phase of postacute inflammatory illness from weeks 2 to 4, and late sequelae occurring beyond week 4 (15). Another framework defines "ongoing symptomatic COVID-19" as signs and symptoms continuing for 4 to 12 weeks and "post-COVID syndrome" as continuing for more than 12 weeks after initial infection (16). The natural histories, including duration, of various manifestations of postacute COVID-19 also remain uncharacterized.

A detailed understanding of the clinical spectrum and natural history of postacute COVID-19 will require systematic longitudinal assessment of individuals after SARS-CoV-2 infection. Workshop participants noted that studies will need to characterize disease phenotypes across multiple organ systems; collect biospecimens; assess cardiovascular, pulmonary, immune, metabolic, and neurologic function and mental health; and be tailored to evaluate pediatric and elderly populations. Enrollment of participants from diverse populations, including groups disproportionately affected by COVID-19, is critical. Identifying adequate control groups is vital to distinguishing the myriad of individual and contextual factors likely to play a role in COVID-19 recovery. Data and specimens obtained as close as possible after—or even before—infection or symptom manifestation would be extremely valuable in this regard. Cohorts from ongoing therapeutic and prevention trials could be

leveraged to answer relevant questions about the effects of these interventions on the development and natural history of postacute COVID-19.

Key Gap: A Need to Understand Pathophysiology

The pathophysiology that drives the diversity of postacute COVID-19 manifestations is poorly understood. Elucidating the pathophysiology not only will be critical for developing treatment of postacute COVID-19 but may also improve our understanding of the sequelae of other viral infections (for example, influenza, chikungunya, Ebola, and Epstein–Barr viruses) or conditions where viral infection may be suspected but not confirmed (such as some cases of myalgic encephalomyelitis/chronic fatigue syndrome). Workshop speakers and participants discussed potential disease mechanisms underlying postacute COVID-19.

A key question identified at the workshop is the extent to which postacute COVID-19 manifestations are direct effects of the virus as opposed to a host response to SARS-CoV-2 infection. The relationship of duration, magnitude, and site of the initial viral infection with timing and type of postacute sequelae needs further investigation. Because SARS-CoV-2 uses angiotensin-converting enzyme 2 (ACE2) as a receptor to enter host cells, understanding the expression and localization of ACE2 in the body may yield insight into postacute COVID-19. Numerous tissues and organ systems express ACE2, including in the respiratory, cardiovascular, gastrointestinal, and nervous systems (17). In discussing the pathogenesis of diseases caused by other coronaviruses, speakers described feline coronavirus, an enteric infection that is sometimes persistent. Occasionally, viral mutations during persistence will result in macrophage infection, leading to the severe disease of feline infectious peritonitis. It has been hypothesized that persistent or prolonged SARS-CoV-2 infection may contribute to the development of postacute sequelae. One recent study analyzed intestinal biopsies obtained from asymptomatic persons 4 months after COVID-19 onset, which revealed the persistence of SARS-CoV-2 nucleic acids and immunoreactivity in the small bowel of 7 of 14 participants (18).

The immune response may also play a critical role in the pathogenesis of certain postacute manifestations of COVID-19, as it likely does in the pathogenesis of acute disease. One analysis of B-cell responses in patients with COVID-19 found that those with severe disease showed B-cell features previously described in autoimmune processes, such as activation of the extrafollicular pathway (19). Another study described the detection of prothrombotic antiphospholipid autoantibodies in 52% of a cohort of 172 patients hospitalized with COVID-19 (20). Neutralizing autoantibodies against type I interferons have been described in some patients with severe COVID-19, an example of the adaptive immune response impairing the innate immune response (21). Workshop participants discussed whether certain types of autoantibodies could play a pathogenic role in or be a biomarker for postacute sequelae of COVID-19. The potential role of T cells in some postacute manifestations of COVID-19 was also discussed, given their role in pathophysiology of acute disease. A recent exploratory analysis of antigen-specific adaptive immune responses to SARS-CoV-2 showed that coordinated $CD4^+$ T-cell, $CD8^+$ T-cell, and antibody responses were associated with milder acute disease (22). Deep immune profiling of samples from children with multisystem inflammatory syndrome, which can present concurrently with or after SARS-CoV-2 infection, revealed activation of vascular-patrolling $CD8^+$ T cells and patterns of T-cell activation similar to those seen in adults with severe COVID-19 (23). Research is needed to elucidate how certain aspects of the host immune response may affect the natural history of recovery from and development of postacute sequelae of COVID-19.

The potential role of vasculitis and coagulopathy in the pathophysiology of postacute manifestations of COVID-19 was also raised by workshop participants as an area that needs to be explored. Endothelial dysfunction could contribute to postacute disease manifestations in many organs (24,25). Although limited by incomplete clinical information, an autopsy study of persons who died of COVID-19 found evidence of multifocal microvascular brain injury using magnetic resonance microscopy, histopathological evaluation, and immunohistochemical analysis, without detection of SARS-CoV-2 in the brain (26).

Animal and in vitro models have played a significant role in studying the pathology resulting from acute SARS-CoV-2 infection. Several animal models, including mice, hamsters, ferrets, and nonhuman primates, are being used; however, the extent to which these models mirror aspects of human disease varies. Workshop participants discussed several animal models that mimic acute lung injury (27), anosmia (28), and some neurologic complications of COVID-19 (29). Animal models that can recapitulate severe or chronic disease or display

Table. Key Gaps Identified at the Workshop on Postacute COVID-19

Common terminology and case definitions
Characterization of epidemiology (including incidence and prevalence of various phenotypes in the full spectrum of patients and in diverse communities)
Description of clinical spectrum and natural history of various phenotypes
Identification of risk factors, including comorbid conditions, severity of initial disease, viral characteristics, host genetics, and host immune response
Understanding of pathophysiology
Characterization of the influence of therapeutics for acute disease and vaccines in preventing or modulating postacute COVID-19
Identification of therapeutic and preventive strategies for postacute COVID-19

extrapulmonary manifestations, such as cardiac complications, should be explored.

In vitro studies are a valuable tool for understanding cellular responses to SARS-CoV-2 infection. In particular, workshop participants discussed the value of stem cell–derived human tissues and organoids. Benefits of using stem cell–derived human tissues include their ability to grow indefinitely in vitro, maintain a normal genetic makeup, and differentiate into a wide range of somatic tissues. Several stem cell–derived tissues and organoids have successfully been infected with SARS-CoV-2 (30,31). These in vitro studies complement investigations using animal models and can be used to answer basic research questions, including which cells are infected by SARS-CoV-2, how they recover after acute infection, and what happens after infection.

Conclusions

Workshop presentations and discussions identified fundamental gaps that must be addressed in knowledge about postacute COVID-19 (Table). An initial need is for clear, common terminology to describe the condition. Accepted case definitions are also essential to advancing the field, harmonizing research, conducting surveillance, and developing and providing relevant treatment and prevention strategies. Developing such terminology and definitions will require ongoing communication with all stakeholders, including clinicians, researchers, and advocacy and patient community groups.

In addition, we need to describe the epidemiology of postacute COVID-19, characterize the various phenotypes and their clinical spectrum, delineate natural histories, and identify risk factors for development. Studying the effects of vaccination, as well as therapeutics given during the acute phase of disease, on the development of postacute COVID-19 will also be informative. Delineation of the pathogenesis of various manifestations of postacute COVID-19 is another fundamental knowledge gap, and findings from these studies will inform the development and evaluation of safe and effective methods of treatment and prevention. In addition, the effects, if any, of SARS-CoV-2 variants on the development of postacute COVID-19 need to be explored.

As the number of reports of postacute COVID-19 continues to increase, fundamental questions must be answered. We urgently need a collaborative research approach that includes harmonized case definitions; delineation of epidemiology, natural history, and pathophysiology; and strategies to address longtime health disparities. This will enable the optimum implementation of evidence-based diagnostic, therapeutic, and preventive strategies essential to help mitigate the pain and suffering of individuals with postacute COVID-19.

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