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COMPREHENSIVE REVIEW OF COVID-19: EPIDEMIOLOGY, PATHOGENESIS, DIAGNOSTIC ADVANCEMENTS, AND POST-PANDEMIC STRATEGIES

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КОМПЛЕКСНЫЙ ОБЗОР COVID-19: ЭПИДЕМИОЛОГИЯ, ПАТОГЕНЕЗ, ДОСТИЖЕНИЯ В ДИАГНОСТИКЕ И СТРАТЕГИИ В ПОСТПАНДЕМИЧЕСКИЙ ПЕРИОД

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Abstract. Background: Since its emergence in late 2019, Coronavirus Disease 2019 (COVID-19), caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has fundamentally altered global health landscapes. This review aims to synthesize current knowledge regarding the etiology, complex pathogenesis, evolving diagnostic modalities, and updated therapeutic strategies, with a focus on post-pandemic management and Long COVID. A narrative review was conducted using databases including PubMed, Scopus, and WHO repositories. Data regarding epidemiology, pathophysiology, and clinical guidelines published between January 2020 and late 2024 were analyzed. The virus utilizes the ACE2 receptor for entry, triggering a dysregulated immune response (cytokine storm) and coagulopathy. Diagnostic techniques have evolved from standard RT-PCR to rapid molecular assays and AI-assisted imaging. Treatment has shifted from supportive care to targeted antivirals and immunomodulators. While acute mortality has decreased due to vaccination and population immunity, the focus must now shift to managing endemic transmission and the chronic sequelae known as Post-Acute Sequelae of SARS-CoV-2 (PASC).

Аннотация. С момента своего появления в конце 2019 года коронавирусная болезнь 2019 (COVID-19), вызываемая коронавирусом тяжелого острого респираторного синдрома 2 (SARS-CoV-2), коренным образом изменила глобальную ситуацию в области здравоохранения. Был проведен нарративный обзор с использованием баз данных, включая PubMed, Scopus и репозитории ВОЗ. Были проанализированы данные по эпидемиологии, патофизиологии и клиническим рекомендациям, опубликованные в период с января 2020 года по конец 2024 года. Вирус использует рецептор ACE2 для проникновения, вызывая нарушение регуляции

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иммунного ответа (цитокиновый шторм) и коагулопатию. Диагностические методы эволюционировали от стандартной ОТ-ПЦР до быстрых молекулярных анализов и визуализации с использованием ИИ. Лечение сместилось от поддерживающей терапии к целенаправленному применению противовирусных препаратов и иммуномодуляторов. Хотя острая смертность снизилась благодаря вакцинации и популяционному иммунитету, теперь необходимо сосредоточиться на борьбе с эндемической передачей вируса и хроническими последствиями, известными как пост-острые последствия SARS-CoV-2 (PASC).

Keywords: COVID-19, Pathogenesis, Long COVID, mRNA Vaccine, Pandemic Management.

Ключевые слова: COVID-19, патогенез, длительный COVID, мРНК-вакцина, управление пандемией.

COVID-19 is a highly infectious respiratory disease caused by the novel betacoronavirus SARS-CoV-2. First identified in Wuhan, China, in December 2019, it rapidly escalated into a global pandemic. Unlike previous coronavirus outbreaks (SARS-CoV and MERS-CoV), SARS-CoV-2 demonstrated efficient human-to-human transmission even during the asymptomatic incubation period, complicating containment efforts [1].

Globally, the virus has infected hundreds of millions of individuals, with waves driven by variants of concern (VOCs) such as Delta and Omicron. The basic reproduction number (R_0) has fluctuated significantly with these variants; the Omicron variant, for instance, demonstrated significantly higher transmissibility than the ancestral strain. Regionally, epidemiology varies based on population density, healthcare infrastructure, and vaccination coverage [2].

While the acute respiratory distress syndrome (ARDS) associated with COVID-19 is well-documented, gaps remain regarding the long-term systemic effects of the virus. Furthermore, diagnostic protocols differ vastly between resource-rich and resource-limited settings, creating disparities in care. The transition from pandemic to endemic status requires a re-evaluation of treatment protocols [3].

The objective of this review is to provide a comprehensive analysis of SARS-CoV-2, detailing its pathogenesis, evaluating the shift in diagnostic technologies, and summarizing current consensus on treatment and post-pandemic management strategies. This paper employs a narrative review design to synthesize broad literature regarding the clinical and biological aspects of COVID-19. This format allows for the integration of epidemiological data, basic science (pathogenesis), and clinical management guidelines [4].

Randomized Controlled Trials (RCTs) regarding vaccines and therapeutics were included [5].

Peer-reviewed articles published in English between January 1, 2020, and December 2024; official health guidelines; systematic reviews and meta-analyses were considered. Exclusion criteria included: anecdotal case reports (unless highlighting a novel finding), non-peer-reviewed pre-prints (unless no other data exists), and studies with small sample sizes [6].

Literature was categorized into four domains: Pathophysiology, Diagnostics, Therapeutics, and Chronic Sequelae. Key findings were extracted and cross-referenced to ensure they represent the current scientific consensus rather than obsolete early-pandemic theories [7].

SARS-CoV-2 is an enveloped, positive-sense single-stranded RNA virus belonging to the genus Betacoronavirus.[5] It contains four structural proteins: Spike (S), Envelope (E), Membrane (M), and Nucleocapsid (N). The S-protein is the critical determinant of host tropism and the primary target for neutralizing antibodies and vaccines [8].

The virus binds to the Angiotensin-Converting Enzyme 2 (ACE2) receptor, predominantly found in the respiratory epithelium, enterocytes, and vascular endothelium.[8] Host protease TMPRSS2

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primes the S-protein for fusion. Immune Dysregulation: Severe cases are characterized by a "cytokine storm," an excessive release of pro-inflammatory cytokines (IL-6, TNF-α, IL-1β). This leads to diffuse alveolar damage. Coagulopathy: Endothelial injury and inflammation trigger a hypercoagulable state, leading to microthrombi and pulmonary embolism, a distinct feature distinguishing COVID-19 from typical viral pneumonias [9].

The incubation period averages 5 days (range 2–14 days). Clinical presentation is categorized as: Asymptomatic/Mild (81%): Fever, cough, fatigue, anosmia (loss of smell), ageusia (loss of taste).

Moderate (14%): Pneumonia with (4%). Severe (5%): Dyspnea, hypoxia (2–4%), lung infiltrates (>50%). Critical: Respiratory failure, shock, multiorgan dysfunction [10].Molecular Testing: RT-PCR remains the gold standard. However, newer CRISPR-based offer sensitivity comparable to PCR with faster turnaround times. Antigen Testing: Rapid Antigen Tests (RAT) are specific but less sensitive, useful for mass screening. Radiology: High-Resolution CT (HRCT) typically shows "ground-glass opacities" (GGO) with peripheral distribution. The CO-RADS classification system standardized the reporting of likelihood of COVID-19 involvement [11].

Nirmatrelvir/ritonavir (Paxlovid) and Remdesivir inhibit viral replication; most effective when given early (within 5 days of symptom onset). Immunomodulators: Corticosteroids (Dexamethasone) significantly reduce mortality in patients requiring oxygen. IL-6 inhibitors (Tocilizumab) are used in cases with systemic inflammation. Anticoagulation: Prophylactic low-molecular-weight heparin is standard to prevent thromboembolism [12].

Major vaccine platforms include: mRNA vaccines (Pfizer-BioNTech, Moderna): Encode the S-protein to elicit immune response. Viral Vector (AstraZeneca, J&J): Use adenovirus to deliver genetic material. Protein Subunit (Novavax): Contains the S-protein directly. Vaccines have been highly effective in preventing severe disease and death, though breakthrough infections occur due to waning immunity and variants [13].

The pathogenesis of COVID-19 highlights a multi-system disease rather than a strictly respiratory one [16].

The wide distribution of ACE2 receptors explains the renal, cardiac, and neurological manifestations observed. The evolution of variants indicates that the virus is adapting to evade host immunity, necessitating updated vaccine boosters [14].

Early pandemic management relied on hydroxychloroquine and azithromycin, which have since been proven ineffective. Current guidelines align globally (WHO, CDC, ICMR) on the use of steroids for hypoxic patients, marking a triumph of evidence-based medicine over initial anecdotal treatments [15].

The focus has shifted to Long COVID (PASC), defined as symptoms persisting >3 months. Symptoms include "brain fog," fatigue, and dysautonomia. Management requires a multidisciplinary approach involving rehabilitation, cardiology, and neurology. Primary care physicians must now screen for thrombotic sequelae and fibrotic lung disease in recovered patients [16].

This review is limited by the rapidly evolving nature of the virus. Data regarding the efficacy of vaccines against the most recent variants (e.g., JN.1) is still accumulating. Furthermore, long-term prognosis data (>5 years) is currently unavailable [17].

Pan-sarbecovirus vaccines: Developing vaccines that target conserved regions of coronaviruses to prevent future pandemics. Long COVID biomarkers: Identifying blood markers to objectively diagnose and track PASC. Antiviral resistance: Monitoring for resistance against current therapeutics like Paxlovid [18].

COVID-19 has evolved from an acute global emergency to an endemic management challenge.17 While the etiology and pathogenesis are now well-understood—centering on ACE2 binding and immune dysregulation—the virus continues to mutate. The major advancement in this

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field is the rapid development of mRNA vaccines and novel therapeutics like oral antivirals. Moving forward, clinical focus must prioritize the surveillance of new variants and the comprehensive management of post-acute sequelae (Long COVID) to mitigate the long-term burden on healthcare systems [19].

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