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Review Article

The Unfinished Story of COVID-19: Long-Term Immune, Vascular, Rheumatological, Neurological and Cardiovascular Consequences

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Abstract: Post-COVID-19 condition, commonly called Long COVID, has shifted the clinical question from whether a patient survived the acute infection to whether normal health and function have returned. For some, recovery is straightforward; for others, symptoms persist, fluctuate or appear after an interval, affecting several organ systems at once. This narrative review examines rheumatological, hematological and vascular, immunological and infectious, neuropsychiatric, and cardiovascular sequelae, including sudden cardiac death (SCD). Proposed mechanisms include immune dysregulation, persistent antigenic stimulation in subsets of patients, endothelial injury, altered coagulation, autonomic dysfunction, neuroinflammation, residual tissue damage and the consequences of critical illness. These mechanisms are neither mutually exclusive nor universal. The practical challenge is to recognize meaningful patterns without assuming that every symptom is caused by COVID-19. Joint pain does not automatically indicate autoimmune disease; an elevated D-dimer does not diagnose thrombosis; urticaria does not establish generalized immune deficiency; and "brain fog" should prompt a thoughtful assessment rather than dismissal or premature attribution. Observational evidence suggests increased risks of selected cardiovascular outcomes after infection, but SCD is a separate endpoint and must be evaluated with specific evidence. A phenotype-led approach—targeted investigation, attention to alternative diagnoses, clear communication and individualized rehabilitation—offers the most defensible current strategy. Indian clinical publications add useful regional context, particularly for inflammatory markers, radiological outcomes, tuberculosis and post-COVID symptoms, but should be interpreted according to study design and limitations.

Keywords: Long COVID, Post-COVID-19 Condition, Rheumatology, Endothelial Dysfunction, Thrombosis, Immune Dysregulation, Urticaria, Tuberculosis, Cognition, Depression, Cardiovascular Risk, Sudden Cardiac Death.

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INTRODUCTION

The end of fever, cough or oxygen requirement does not always mark the end of illness. Many people recover fully after COVID-19, while others describe a less visible recovery: breathlessness on stairs, exhaustion after routine activity, joint pain, palpitations, sleep disturbance, difficulty concentrating or a sense that their previous health has not returned. These symptoms may occur together, vary from day to day and interfere with work, family responsibilities and quality of life. This longer trajectory has brought Long COVID, or post COVID-19 condition, into routine clinical practice. The World Health Organization describes post-COVID-19 condition as occurring in people with probable or confirmed SARS-CoV-2 infection, usually around three months from illness onset, with symptoms lasting at least two months and not explained by another diagnosis. Definitions used by studies and health systems differ, so prevalence estimates cannot always be compared directly. Results are influenced by the population studied, acute disease severity, vaccination, reinfection, variant period, pre-existing illness, follow-up duration and how symptoms are measured [1–5].

Long COVID is not one uniform disease with one diagnostic test. It is more useful to think of it as a group of overlapping clinical phenotypes. In one person, persistent respiratory impairment may dominate; in another, orthostatic symptoms, cognitive difficulty, inflammatory joint symptoms or cardiovascular disease may be more prominent. Several mechanisms have been proposed, including immune activation or dysregulation, persistent viral material or antigen in some patients,

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endothelial and coagulation abnormalities, autonomic disturbance, neuroinflammation, residual organ injury, sleep disruption and deconditioning. These mechanisms may coexist, but none should be assumed in every patient [6–29].

The clinician's task is therefore twofold: take persistent symptoms seriously and avoid attributing every post-infection problem to Long COVID. A temporal association is an important clue, not proof of causation. This review brings together evidence on rheumatological, hematological/vascular, immunological/infectious, neuropsychiatric and cardiovascular manifestations, with practical emphasis on assessment, red flags and evidence-based management. Indian clinical research is considered where relevant, particularly because prior tuberculosis, structural lung disease, comorbidities and barriers to timely investigation can shape post-COVID care [13–31].

Rheumatological Manifestations

Musculoskeletal symptoms are among the complaints that can make recovery feel incomplete. Patients may report diffuse myalgia, joint pain, morning stiffness, reduced grip, shoulder or hip discomfort and fatigue that limits daily activity. For most, pain alone does not establish inflammatory arthritis. In a smaller subset, clinicians may identify objective synovitis, myositis, vasculitis or a previously unrecognized autoimmune rheumatic disease. Published prevalence estimates vary widely because studies use different symptom definitions, follow-up periods and referral populations. Case reports describe reactive or post-viral arthritis, rheumatoid-arthritis-like presentations, polymyalgia-like syndromes, inflammatory muscle disease and vasculitic features. These reports are clinically useful signals, but they cannot establish incidence or causality. Population-based studies and meta-analyses have reported associations between infection and selected incident autoimmune diseases; residual confounding, surveillance bias, heterogeneity and low absolute event rates remain important considerations [32–37].

Several biological explanations are under investigation. Immune activation after infection may, in susceptible individuals, disturb immune tolerance or amplify an existing tendency to autoimmunity. Proposed pathways include molecular mimicry, bystander activation, B- and T-cell dysregulation and persistent antigenic stimulation. These mechanisms are plausible but are not proof that a particular patient's joint pain is autoimmune. Similarly, the proposed Th1/Th2 imbalance or "switch-failure" model should be described as a hypothesis rather than a universal explanation for Long COVID. Immune responses involve multiple interacting pathways, including regulatory T cells, Th17 cells, B cells and innate immune responses [24–46].

The key clinical distinction is between symptoms and signs. Arthralgia is pain; arthritis requires evidence of joint inflammation. Examination should look for synovitis, warmth, effusion, prolonged morning stiffness, objective muscle weakness, rash, Raynaud phenomenon, sicca symptoms, neuropathy or renal involvement. Tests should follow the clinical picture: CBC, ESR/CRP, renal function and urinalysis where appropriate; rheumatoid factor and anti-CCP if rheumatoid arthritis is suspected; ANA and disease-specific antibodies when connective-tissue disease is plausible; and creatine kinase for objective muscle weakness. Broad autoantibody panels in people with isolated fatigue or nonspecific pain can produce incidental positives, anxiety and unnecessary treatment. [figure 1].

A hot swollen joint with fever requires urgent exclusion of septic arthritis. Progressive weakness, purpura, renal abnormalities or signs of organ-threatening disease warrant prompt specialist assessment. Management should be diagnosis-specific. Pain without objective inflammation may call for sleep support, management of comorbidities, individualized symptom treatment and rehabilitation matched to tolerance. People with post-exertional symptom exacerbation should not be pushed through fixed activity increases that repeatedly worsen symptoms. Confirmed inflammatory disease should be treated according to established disease-specific guidance; glucocorticoids or immunosuppressants should not be started solely for unexplained post-COVID symptoms or speculative immune mechanisms. Indian publications describing rheumatological symptoms and immune hypotheses contribute to awareness, but their conclusions should be weighed alongside larger and better-controlled studies [32–47].

Hematological and Vascular Manifestations

COVID-19 brought the relationship between inflammation and clotting into sharp focus. During acute infection, some patients develop lymphopenia, platelet abnormalities, elevated inflammatory markers and activation of the coagulation system. Severe disease is associated with venous thromboembolism (VTE), arterial thrombosis and microvascular injury. This process—often described as immunothrombosis—reflects interactions among immune cells, platelets, endothelial cells, complement and coagulation pathways. Neutrophil extracellular traps may also contribute to clot formation. The intensity of these changes differs between acute severe disease and the post-acute period, so findings from intensive-care cohorts should not automatically be applied to people with persistent symptoms months later [48–75].

D-dimer is a useful example of why laboratory results require context. It reflects fibrin turnover and can help exclude VTE in patients with an appropriate pre-test probability, but it is nonspecific. Infection,

inflammation, age, cancer, pregnancy and many other conditions can raise it. A raised D-dimer is not itself a diagnosis of pulmonary embolism or deep-vein thrombosis, and serial measurement is not a validated general-purpose measure of Long-COVID activity. CRP, ferritin, LDH and IL-6 have also been investigated as markers of acute severity, ventilatory support requirements or residual radiological change. Their prognostic value during acute illness does not establish that they should be measured routinely during long-term follow-up. Proposed "microclot" and impaired-fibrinolysis explanations remain research areas; tests and definitions are not yet sufficiently standardized to guide routine clinical treatment [66—77].

The most important clinical question is not whether a biomarker is abnormal, but whether the patient has a syndrome requiring urgent action. New unilateral leg swelling, sudden or disproportionate breathlessness, pleuritic chest pain, hemoptysis, syncope or unexplained hypoxaemia should trigger prompt assessment for VTE. Diagnostic imaging should follow established clinical pathways. Persistent fatigue, chest discomfort, palpitations or exercise intolerance have a broad differential, including residual lung disease, cardiac disease, anemia, thyroid dysfunction, dysautonomia, sleep disturbance and deconditioning [figure 2].

Routine D-dimer testing or anticoagulation for every person with Long COVID is not supported and can cause harm, including bleeding and incidental diagnostic cascades. Anticoagulation is indicated for confirmed VTE and other established indications; extended prophylaxis after hospitalization requires an individualized assessment of thrombotic and bleeding risks. Long-term vascular prevention should also address smoking, blood pressure, diabetes, lipid levels, weight, mobility and pre-existing cardiovascular disease.

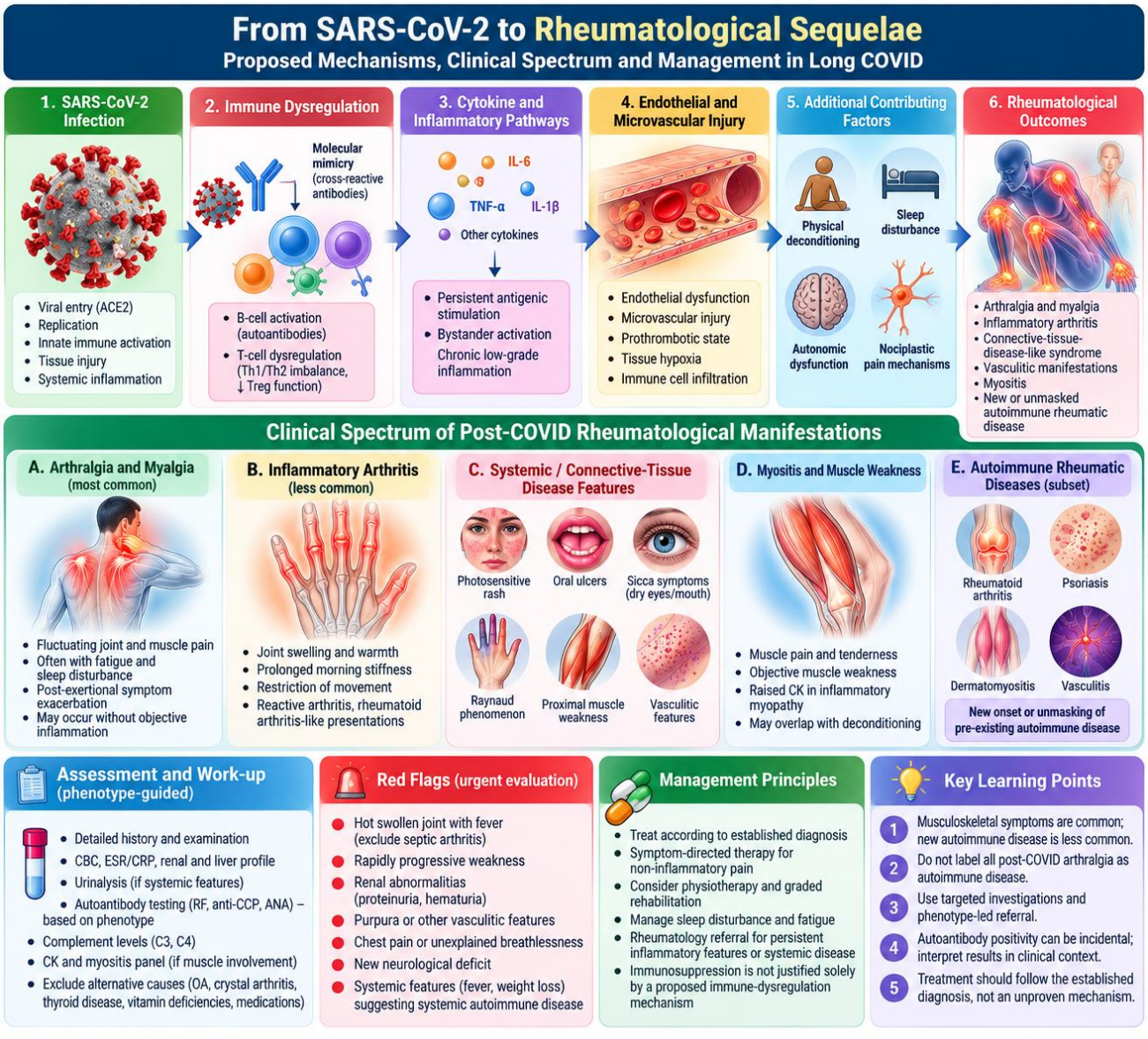


Figure 1: Rheumatological manifestations of long Covid

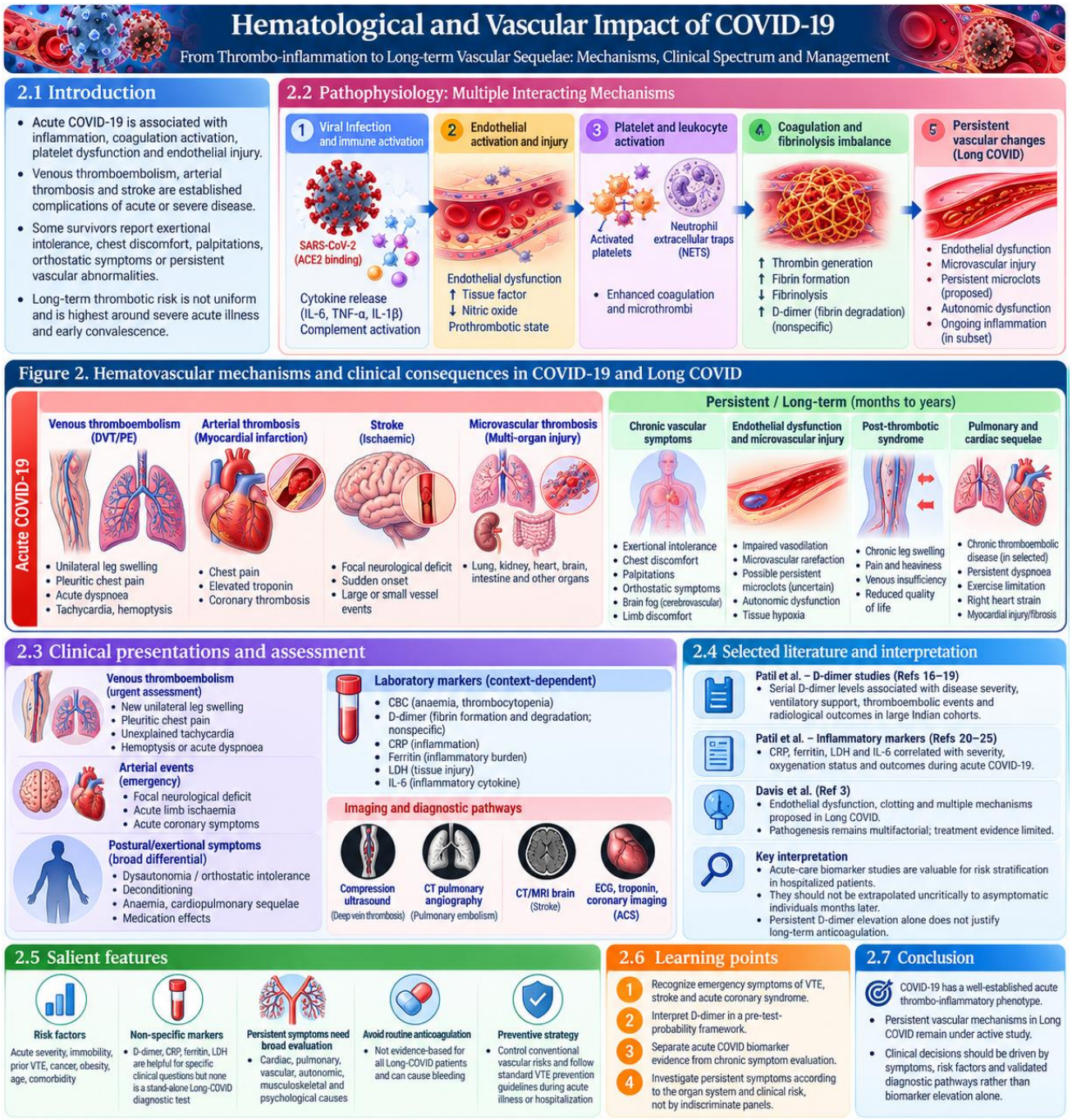


Figure 2: Hematological and vascular manifestations of long Covid

The supplied Indian publications on CRP, IL-6, ferritin, LDH and serial D-dimer primarily examine acute COVID-19 severity, ventilatory support or radiological outcomes. They provide regional data on acute inflammatory and coagulation markers, but should not be extrapolated to chronic disease without longitudinal validation [48–91].

Immune Dysregulation, Urticaria and Infection-Related Concerns

Some patients describe a changed pattern of health after COVID-19: repeated respiratory infections, persistent skin symptoms, unusual fatigue or worsening of an existing inflammatory condition. Such experiences deserve careful evaluation, but they do not establish that all Long-COVID patients have an immunodeficiency. Studies have reported changes in inflammatory signalling, lymphocyte subsets and autoantibody profiles, yet results are heterogeneous and influenced by disease stage, severity, age, comorbidities, medications, vaccination and reinfection. There is no single laboratory signature that diagnoses immune dysregulation in every patient, and a universal persistent Th2-predominant state has not been established. When infections recur, the first step is to define what is recurring. Repeated, microbiologically confirmed bacterial infections are different from frequent viral illnesses, persistent cough due to bronchiectasis, allergic symptoms or flares of a

chronic disease. A focused history should cover infection sites, severity, antibiotic courses, hospitalizations, vaccination, steroid or immunosuppressant exposure and prior structural lung disease. Depending on the pattern, clinicians may consider CBC, glucose testing, nutritional status, immunoglobulins or specialist immune evaluation. Testing should be proportionate to the history rather than based on the assumption that infection follows from Long COVID [24–95]. [Figure 3]

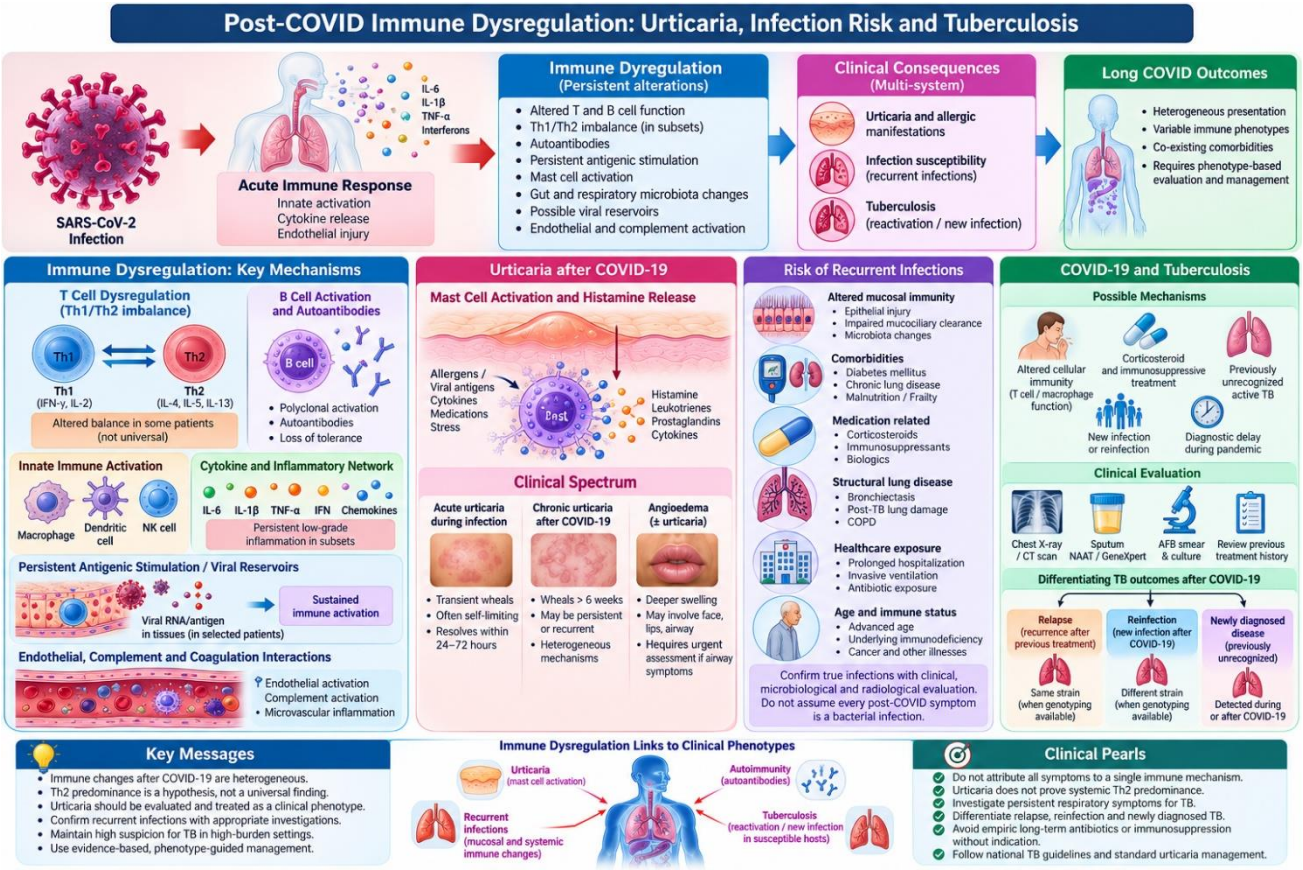


Figure 3: Immune-dysregulation related manifestations of long Covid

Urticaria is another symptom that can be misinterpreted mechanistically. Wheals and angioedema may occur during infection, after infection or in temporal association with vaccination; these are distinct exposures and should not be conflated. Chronic urticaria is primarily a clinical diagnosis, and its presence does not prove a generalized Th2-dominant or deficient immune state. Management should follow standard urticaria guidance and be escalated according to severity and response. Proposed immune-directed treatments require trial evidence before being regarded as routine care. Swelling involving the tongue or throat, breathing difficulty or circulatory symptoms require emergency assessment.

Tuberculosis (TB) deserves particular attention in India because TB, post-TB lung damage, post-COVID radiological abnormalities and other respiratory infections can produce overlapping symptoms. A cough or radiographic change after COVID-19 does not by itself establish TB reactivation. Clinicians should distinguish relapse, reinfection and previously unrecognized active disease using prior treatment records, symptom history, imaging and microbiological testing. Persistent cough, fever, night sweats, weight loss, hemoptysis or new radiological abnormalities warrant evaluation through the current National Tuberculosis Elimination Programme pathway, including rapid molecular testing when indicated. A negative initial result may not end the evaluation when clinical suspicion remains high [84–97].

The report of concurrent COVID-19 pneumonia and pulmonary TB illustrates diagnostic overlap, not proof that SARS-CoV-2 caused TB reactivation. Publications on post-COVID immune dysregulation have raised hypotheses that need prospective testing [6–99]. Repurposed drugs and supplements proposed in narrative or hypothesis-generating papers should not be presented as established treatment without convincing clinical-trial evidence [6–109].

Neuropsychiatric Manifestations

For some patients, the most disruptive part of Long COVID is not visible on a chest radiograph or routine blood test. They may describe losing their train of thought, struggling to find words, forgetting appointments, reading more slowly or becoming

exhausted by tasks that were previously automatic. Others experience anxiety, low mood, irritability, disturbed sleep or a loss of confidence after illness. These symptoms can affect employment, education, family life and independence. They may occur after mild or severe acute disease and can coexist with fatigue, pain, breathlessness, autonomic symptoms and post-exertional symptom exacerbation. Systematic reviews and large cohort studies support associations between COVID-19 and several neuropsychiatric outcomes, although estimates vary with population, measurement tools, baseline health, follow-up and comparator groups [30–115].

There is no single explanation for these experiences. Proposed contributors include immune and glial activation, endothelial or microvascular changes, autonomic disturbance, sleep disruption, metabolic effects, residual organ injury and the psychological consequences of critical illness, isolation, bereavement or financial stress. These factors may interact. Recognizing psychological distress does not mean symptoms are "all in the mind"; equally, attributing every cognitive complaint to direct viral injury may delay recognition of treatable conditions. [21–114]. [figure 4]

Assessment should explore onset, progression, day-to-day variability, functional impact, prior neurological or psychiatric illness, sleep, medications, substance use, pain, fatigue, orthostatic symptoms and exertional worsening. Validated tools such as PHQ-9 and GAD-7 can support screening but do not replace clinical assessment. Consider common and treatable contributors such as anemia, thyroid disease, sleep apnea, medication effects, depression and anxiety. Persistent or progressive cognitive decline, focal neurological signs, seizures, severe new headache, delirium or a marked change in daily functioning require targeted investigation [30–115].

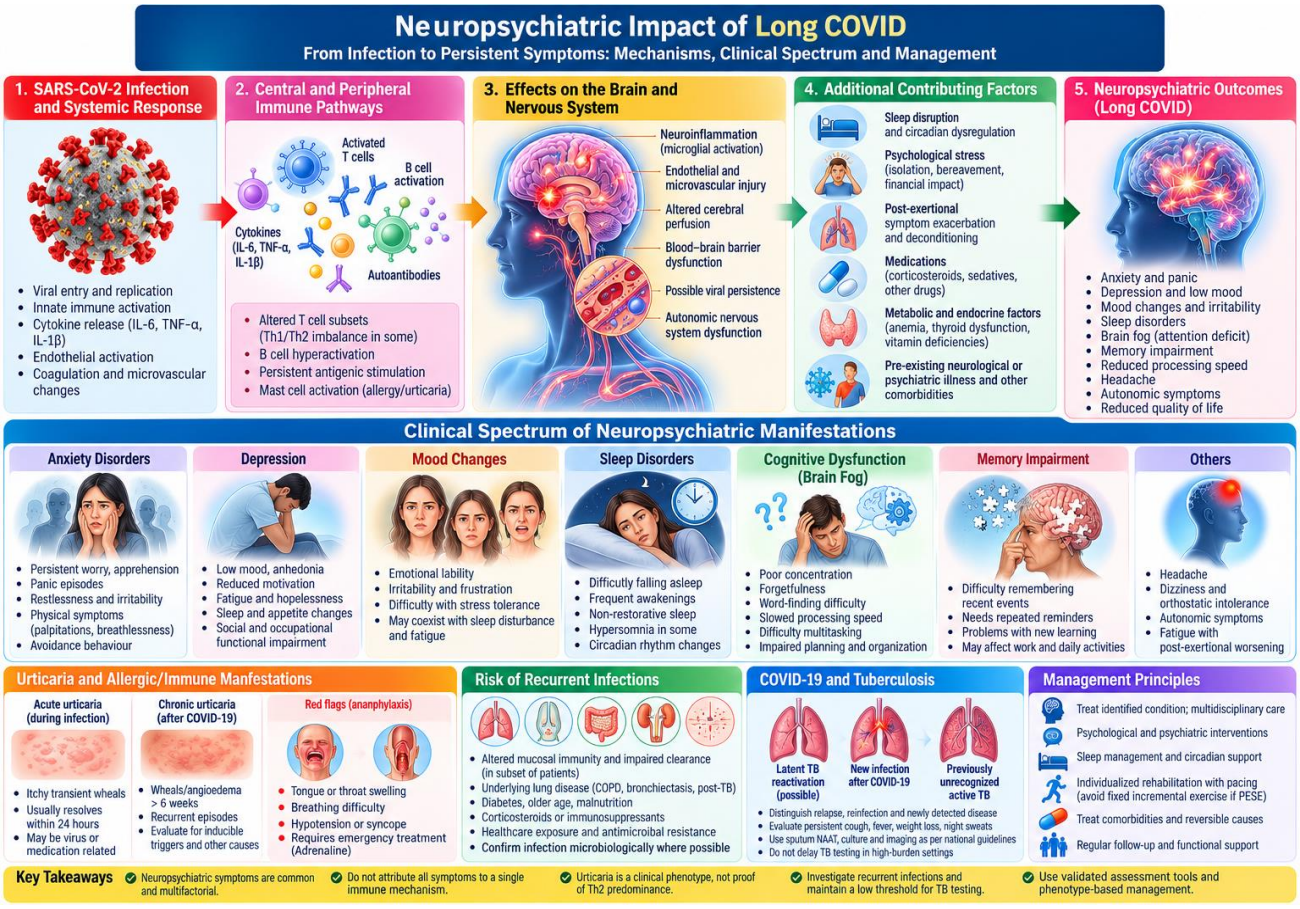


Figure 4: Neuropsychiatric manifestations of long Covid

Management should match the person's needs. Options may include education, sleep interventions, psychological therapies, treatment of a diagnosed mood disorder, workplace or educational adjustments and rehabilitation adapted to symptom tolerance. People who experience post-exertional symptom exacerbation may benefit from pacing and individualized energy management; rigid graded increases that repeatedly provoke relapses should be avoided. Suicidal thoughts, psychosis, severe confusion or acute neurological symptoms require urgent care. Indian publications describing Long-COVID symptom patterns and cardio-pulmonary/neurological pathways provide useful context, while prospective controlled studies are needed to determine mechanisms and treatment effects [6–115].

Cardiovascular Risk and Sudden Cardiac Death

Cardiovascular concerns can be especially frightening because symptoms such as chest pain, palpitations, breathlessness and fainting are difficult to interpret—and because sudden cardiac death (SCD) receives intense public attention. COVID-19 can affect the myocardium, coronary circulation, endothelium, coagulation system and cardiac conduction. Acute complications include myocardial injury, acute coronary syndrome, heart failure, myocarditis or pericarditis, arrhythmias and thromboembolic disease. Mechanisms include systemic inflammation, hypoxaemia, endothelial injury, thrombosis, stress-related myocardial dysfunction and, in selected cases, inflammatory myocardial injury. However, a racing heart after infection may also reflect fever recovery, anemia, dehydration, medication effects, orthostatic intolerance or deconditioning rather than a dangerous arrhythmia [116–123].

Large observational cohorts have reported increased post-acute risks of selected cardiovascular outcomes, including ischemic heart disease, arrhythmia, heart failure and thromboembolic disease. The landmark Veterans Affairs cohort and subsequent systematic reviews support the possibility of elevated relative risk after infection, including in some people who were not hospitalized; risks are influenced by acute severity and baseline health [116–123]. These results should be communicated carefully. Observational studies can be affected by residual confounding, changing variants, vaccination, reinfection and differences in healthcare use. Relative risk is not the same as absolute risk, and the risk for an individual depends on age, underlying cardiovascular disease and other clinical factors.

SCD is a specific outcome and must not be used as a synonym for all cardiovascular complications. It generally refers to an unexpected death presumed to be cardiac, occurring over a short interval after symptom onset. Possible pathways include acute coronary events, ventricular arrhythmia in the setting of myocardial disease, severe myocarditis, inherited electrical disorders, pulmonary embolism and decompensation of underlying heart disease. An increased rate of broad cardiovascular outcomes after COVID-19 does not prove that all survivors have a high risk of SCD. Nor does a death occurring after infection establish that infection caused it [116–123]. [figure 5]

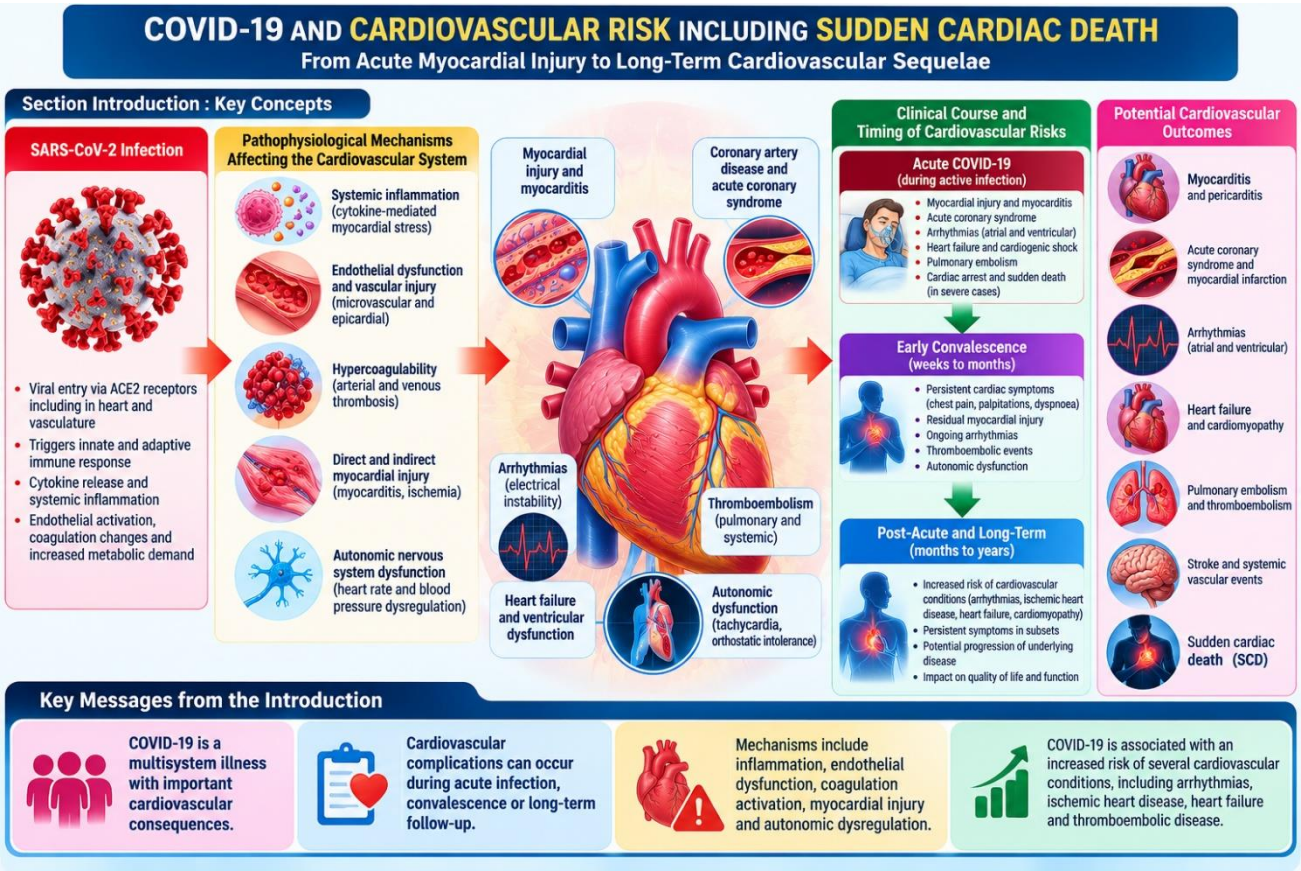


Figure 5: Cardiac events and SCD as manifestations of long Covid

Pandemic-era studies reported changes in out-of-hospital cardiac arrest in some settings, but these trends are difficult to interpret. Delayed emergency response, reduced access to care, reluctance to seek help, lockdown effects and direct infection-related complications may all have contributed. Evidence specifically quantifying SCD after recovery is less definitive than evidence for several broader

cardiovascular outcomes. Claims about sudden death among younger adults require age-specific, adjudicated data and careful consideration of baseline disease, undiagnosed inherited conditions, substance exposure, ascertainment bias and other causes [116–123].

Infection-related cardiovascular risk should also be distinguished from rare adverse events reported after vaccination. Myocarditis or pericarditis after some vaccine platforms has been recognized as uncommon and concentrated in particular demographic groups. That finding does not establish a generalized population-level increase in SCD. Population-based studies do not justify automatically attributing unexplained sudden deaths to vaccination; both infection and vaccine safety questions require appropriate comparison groups, validated outcomes and transparent risk communication [124–128].

A practical approach is to assess symptoms rather than screen everyone indiscriminately. Ask about chest pain, syncope, palpitations, exertional dyspnoea, prior cardiac disease, family history of premature sudden death, acute illness severity, conventional risk factors and medications. ECG, troponin, natriuretic peptides, echocardiography, ambulatory rhythm monitoring or cardiac MRI should be selected according to the presentation. New severe chest pain, unexplained syncope, sustained tachycardia with haemodynamic symptoms, acute dyspnoea or focal neurological deficits need urgent evaluation. Prevention remains grounded in blood-pressure control, diabetes and lipid management, smoking cessation, adherence to indicated medication and individualized return to activity. People with suspected myocarditis, syncope or significant arrhythmia should avoid strenuous exercise until evaluated [117127].

Clinical Priorities across Domains

A useful Long-COVID consultation begins with the patient's main concern: what changed, when it began, what makes it better or worse, and how it affects everyday function. The clinician should record the acute illness course, prior health, vaccination and reinfection history, medications and relevant comorbidities. Symptoms should be grouped into a working phenotype, while remaining alert to overlap between respiratory, cardiovascular, neurological, rheumatological and psychological presentations [1–130].

Testing should answer a clinical question. A broad panel of inflammatory markers, autoantibodies, coagulation tests, cardiac imaging or immune assays is unlikely to help every patient and can produce incidental abnormalities that lead to further testing without improving outcomes. Red flags should take priority over a generic Long-COVID label. Clear explanations help patients understand both that symptoms are real and that their cause may be multifactorial. Follow-up should track function, symptom burden, safety and response to treatment—not merely laboratory values [1–130].

For research and publication, the distinction between symptoms, objective findings, suspected mechanisms and confirmed diagnoses is equally important. Report absolute risks where possible, describe comparator groups and follow-up, and avoid implying causation from temporal association. Case reports can generate hypotheses but cannot estimate risk. Conference abstracts should be identified as such, and overlapping cohorts or duplicate analyses should be checked before citing them as independent evidence [1–130].

Research Priorities

Several gaps limit confident diagnosis and treatment. Prospective multicentre cohorts need standardized case definitions, appropriate comparison groups, serial assessment and adjustment for vaccination, reinfection, acute severity and baseline health. Outcomes should include function and quality of life as well as biomarkers. Rheumatology studies should separate nonspecific pain from objectively confirmed inflammatory disease and report absolute incidence. Hematovascular studies need validated assays and clinically adjudicated thrombotic events. Immunology studies should seek reproducible signatures rather than assume a universal Th1/Th2 failure. TB studies in high-burden settings should distinguish relapse, reinfection and newly detected disease using microbiology and treatment records. Neuropsychiatric studies need baseline measures, functional endpoints and longer follow-up. Cardiovascular research should clearly distinguish arrhythmia, cardiac arrest, adjudicated SCD and other cardiovascular mortality.

Indian research can make an important contribution because post-TB lung disease, structural airway disease, diabetes, access to diagnostic testing and patterns of healthcare use may influence both symptoms and outcomes. Stronger multicentre designs, transparent methods, registry linkage and independent replication will help establish which findings are generalizable and which are context-specific [13–130].

CONCLUSION

Long COVID is best approached as a heterogeneous post-infectious condition, not a single disease with a single test or mechanism. Rheumatological, hematological/vascular, immune, neuropsychiatric and cardiovascular manifestations can overlap, but each symptom still deserves a careful differential diagnosis. Joint pain does not automatically mean autoimmune disease; D-dimer elevation does not diagnose thrombosis; urticaria does not prove generalized immune deficiency; and cognitive or mood symptoms should be assessed rather than dismissed or prematurely explained. COVID-19 is associated with increased risks of selected cardiovascular outcomes, but

sudden cardiac death is a distinct endpoint requiring specific evidence. The most defensible approach is patient-centred and phenotype-led: recognize persistent symptoms, investigate red flags, test selectively, treat established conditions, communicate uncertainty honestly and tailor rehabilitation to individual tolerance [1-130].

Key Learning Points

1. **Long COVID is a Multisystem Condition:** Persistent or newly emerging symptoms after acute SARS-CoV-2 infection can affect multiple organs, even after apparent recovery from the initial illness.
2. **Rheumatological Manifestations May Persist:** Arthralgia, myalgia, fatigue and inflammatory musculoskeletal symptoms can contribute to prolonged functional impairment and may require evaluation for underlying inflammatory or autoimmune disorders.
3. **Haematological and Vascular Complications Deserve Attention:** Persistent coagulation abnormalities, endothelial dysfunction and thromboinflammatory mechanisms may contribute to vascular complications in selected patients. Abnormal biomarkers alone do not establish ongoing thrombosis.
4. **Immune Dysregulation May Have Prolonged Consequences:** Altered immune responses may contribute to persistent inflammation, changes in infection susceptibility and post-infectious manifestations. Recurrent infections and tuberculosis require appropriate clinical investigation rather than automatic attribution to Long COVID.
5. **Urticaria and Other Cutaneous Manifestations May Occur:** Persistent or recurrent skin symptoms can be part of the post-COVID clinical picture, although their mechanisms and relationship to immune dysregulation remain incompletely understood.
6. **Neuropsychiatric Sequelae Can Substantially Affect Quality of Life:** Anxiety, depression, sleep disturbances, cognitive difficulties and memory complaints may persist after infection and warrant systematic assessment.
7. **Cardiovascular Complications Extend Beyond Acute Myocardial Injury:** Long-term manifestations may include arrhythmias, exercise intolerance, autonomic dysfunction, chest discomfort and heart failure in some patients. Persistent symptoms require evaluation for alternative or coexisting causes.
8. **Sudden Cardiac Death Requires Particular Caution:** Potential arrhythmic and cardiovascular mechanisms warrant clinical attention, but the possibility of sudden cardiac death should not be confused with proof of an increased long-term risk in every person with Long COVID.
9. **Symptoms May Overlap Across Organ Systems:** Fatigue, breathlessness, palpitations, cognitive impairment and exercise intolerance can arise from several interacting mechanisms. A coordinated, symptom-directed assessment is therefore preferable to a single-organ approach.
10. **Biomarkers must be interpreted in Clinical Context:** Inflammatory markers, coagulation parameters, cardiac biomarkers and imaging findings can support evaluation when indicated, but no single test establishes the full spectrum of Long COVID.
11. **Management Should be Individualized and Multidisciplinary:** Care may involve primary care, pulmonology, cardiology, rheumatology, neurology, psychiatry and other specialties according to the patient's symptoms and objective findings.
12. **Important Research Gaps Remain:** Better-defined phenotypes, longitudinal follow-up, validated diagnostic approaches and robust studies of mechanisms, treatment and long-term outcomes are needed to improve care.

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