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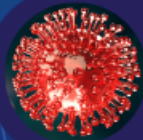
CLINICAL INSIGHTS INTO LONG COVID SUBTYPES

Nafysa Parpia, ND

Introduction, cont.



- Long Covid is a multi-systemic chronic illness following SARS-CoV-2 infection, with persistent symptoms affecting millions globally, resembling other chronic conditions like ME/CFS and fibromyalgia.
- Objective: To categorize Long Covid into two subtypes based on etiology and presentation (Type 1: virus and spike protein effects; Type 2: virus, spike protein, other infections, and environmental factors), exploring onset, pre-exposure patient context, and underlying mechanisms.
- Importance: Understanding mechanisms and subtypes is critical for effective, individualized management of Long Covid and related chronic illnesses.



SARS-COV-2



LONG COVID
SUBTYPE 1



LONG COVID
SUBTYPE 2



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Long COVID Subtypes in Research

- Research Subtypes: Diverse classifications in scientific literature reflect Long Covid's complexity - based on body systems:
 - Post-Viral Fatigue: Persistent exhaustion, similar to ME/CFS.
 - Neurological: Brain fog, cognitive impairment, dysautonomia, POTS.
 - Cardiopulmonary: Shortness of breath, myocarditis, pulmonary issues.
 - Multi-System Inflammatory: Systemic inflammation across organs.
 - Autoimmune: Immune dysregulation with autoantibody production.
- Challenges: Symptom overlap and lack of standardized diagnostics hinder clear subtype delineation.

Long COVID Type 1 & 2 Framework: Etiology vs. Presentation

Understanding Long COVID

Symptomatic Presentation of Long COVID

- **Onset Types:**
 - Immediate: Life-changing post-COVID or post-vaccine
 - Gradual: Slow progression over 1+ year
- **Systems Affected:**
 - **Nervous:** POTS, fatigue, brain fog, sleep/mood issues, dysautonomia
 - **GI:** IBS, IBD, pain, gas, bloating, diarrhea, constipation
 - **Musculoskeletal:** Joint/muscle pain
 - **ENT:** Sinusitis, eye pressure, sore throat
 - **Endocrine:** Sex hormone/thyroid imbalances
- **Similarity:** Resembles ME/CFS (fatigue, post-exertional malaise, cognitive impairment)

Viral and Spike Protein Reservoirs

SARS-CoV-2 and Spike Protein Reservoirs

- **Definition:** Tissues hosting viral genetic material or replication (virus itself or Spike Protein)
- **Locations:** Brain, nerves, GI tract, lymph, lungs, breast tissue
- **Persistence:** Viral RNA/proteins up to 2 years; spike protein in plasma >1 year
- **Evidence:** Elevated SARS-CoV-2-specific T-cells in LC patients
- **Note:** Negative nasopharyngeal/blood tests despite reservoirs
- Low-Level Replication can take place in these sites:
 - Evades complete clearance by immune system

Mechanisms of Reservoirs in Long COVID

Mechanisms of Reservoirs in Long COVID

- **Direct Tissue Damage:** SP binds to ACE2 receptors
 - Vascular endothelial Damage: microvascular injury
 - Epithelial Injury: brain, gut, sinuses, lungs
 - Organ dysfunction
 - Mitochondrial dysfunction
- **Coagulation:** Microclots trap viral proteins; abnormal clot formation
- **Latent Infections:** Reactivation of EBV, CMV, Mycoplasma, Lyme
- **Microbiome Disruption:** Dysbiosis and barrier breakdown drive inflammation
- **Vagus Nerve:** Altered signaling from reservoirs in gut/lungs
- **Disrupted systems:** Metabolism, gene expression, immune response

Mechanisms of Reservoirs in Long COVID, cont.

Mechanisms of Reservoirs in Long COVID

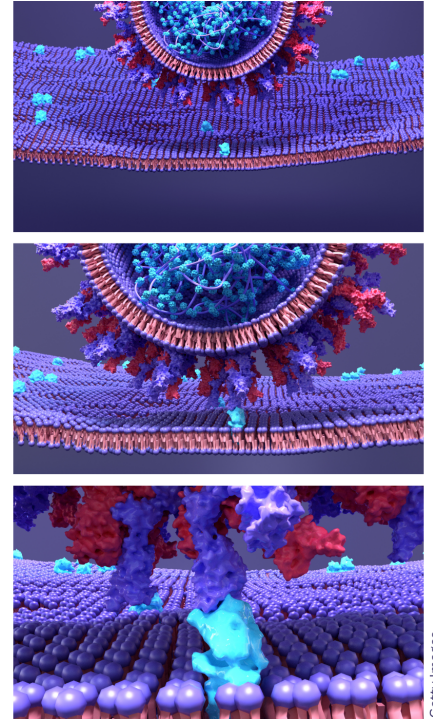
Immune:

- **Chronic Inflammation:** Prolonged immune activation and cytokine production leading to: epigenetic modulation
- **Dysregulated Innate Immunity:** Overactivation of innate pathways leading to: tissue damage and system wide symptoms
- **Immune Exhaustion:** T & B cell response to persistent proteins
 - Impaired ability to clear viral remnants; increasing susceptibility to other infections
- **Autoimmunity:** Cross-reactivity with self-antigens, molecular mimicry (heart, lung, kidney, brain)

Clinical Observations: Long COVID Subtype 1

Type 1: Effects of the Virus and Spike Protein

- Definition: Long COVID driven primarily by SARS-CoV-2 and its Spike Protein.
- Mechanisms:
 - Spike protein binding to ACE2 receptors in organs (e.g., gut, lungs, kidneys, brain), causing endothelial inflammation and **microclots**.
 - Viral and Spike Protein **reservoirs** in tissues (e.g., brain, GI tract, lymph) with active replication or persistent genetic material.
 - **Immune dysregulation**: cytokine storms, and T/B cell exhaustion, autoimmunity, MCAS.

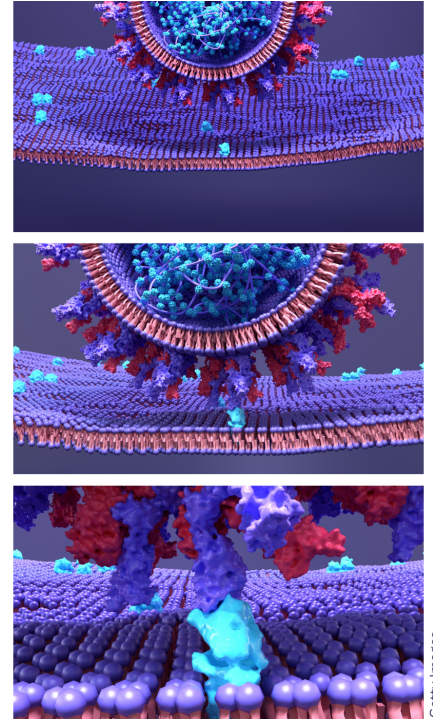


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Clinical Observations: Long COVID Subtype 1

Type 1: Effects of the Virus, Spike Protein, Microclots

- Presentation:
 - Symptoms: Chronic fatigue, brain fog, shortness of breath, cognitive dysfunction
 - Onset: Immediate post-infection or post-vaccine, life-changing OR over many months to years symptoms are slow and insidious



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Long COVID Subtype 1: Patient Considerations

- Patient Context:
 - Pre-exposure health: Relatively healthy or mild comorbidities.
 - Acute infection: Often severe and sometimes not!
 - Diagnostic Considerations:
 - Spike Protein presence
 - T-Cell tests IFN-gamma and IL-2 (Spike or Viral reservoir)
 - Microclots
 - Inflammatory Panels
 - **Often responds to protocol-driven treatments (e.g., Dr. Bruce Patterson's protocol within 6-12 weeks**
- Key Questions:
 - How does the spike protein drive chronic symptoms uniquely?
 - Why do some patients clear the virus while others develop persistent reservoirs?

Clinical Observations: Long COVID Subtype 2

- Type 2: Virus, Spike Protein, Other Infections, and Environmental Factors
 - Definition: Long Covid triggered by SARS-CoV-2 but amplified by concurrent infections, environmental stressors, and pre-existing conditions.
 - Mechanisms:
 - Reactivation of latent infections (e.g., EBV, CMV, Mycoplasma pneumoniae, Chlamydia pneumoniae, Lyme and Co-infections)
 - Concurrent infections (e.g., fungal, mold, parasitic) and microbiome dysregulation (dysbiosis, SIBO, leaky gut); occult dental infections; sinus colonizations
 - Environmental factors: Toxins (e.g., heavy metals, microplastics, glyphosate, mycotoxins), chronic stress, trauma
 - Stress, trauma

DOWNSTREAM EFFECTS

- Vagus nerve dysfunction altering autonomic and immune regulation, exacerbating inflammation.
- Autoimmunity and mast cell activation syndrome (MCAS) due to immune dysregulation
- Hormonal imbalances
- Multiple systems inflammatory process - multiple symptoms across multiple systems

Clinical Observations: Long COVID Subtype 2

- Presentation:
 - Symptoms: Multi-system (chronic fatigue, joint and muscle pain, GI issues, neurological symptoms, sinusitis, hormonal imbalances, post exertional malaise)
 - Onset: Often gradual, with slow progression over months or years.
 - Resembles ME/CFS, fibromyalgia, or MCAS in complexity and chronicity
- Patient Context:
 - Pre-exposure health: Pre-existing chronic conditions, undiagnosed infections, environmental exposures, or hormonal imbalances
 - Often: pre-existing conditions were manageable!
 - Acute infection: May be mild but triggers a cascade of systemic symptoms

Clinical Observations: Long COVID Subtype 2

- Diagnostic Considerations in addition to those for Subtype 1:
 - Tests for concurrent infections (e.g., EBV, Mycoplasma, Tick Borne Diseases, Mold)
 - Test microbiome
 - Test sinuses
 - Assess dental history
 - Test for environmental toxins (metals, microplastics, mycotoxins, pesticides, herbicides, glyphosate)
 - Test hormones

Clinical Observations: Long COVID Subtype 2

- Fails protocol-driven treatments due to complexity
- Treatment time can be from 1 year+
- Key Considerations:
 - How do pre-existing infections, environmental exposures, or vagus nerve dysfunction shape Long Covid severity?
 - What role does the patient's pre Long Covid immune, hormonal and microbiome status play?

Individualized Treatment Approaches

- **Principles:** Highly individualized due to unique illness expressions
- **Strategies:**
 - Modulate immune system/reduce chronic inflammation
 - Nervous system regulation
 - Balance hormones

Continue the above the 3 while:

- Heal GI tract
- Address environmental toxins
- Assess active issues that are SP or Covid driven
- Treat reactivated and concurrent infections
- Mitochondrial repair

Conclusion

- Summary: Long Covid is a complex, multi-systemic chronic illness with two primary subtypes: Type 1 (virus and spike protein-driven only) and Type 2 (virus, concurrent infections, and environmental factors)
- Understanding pre-exposure context at onset of chronic illness, and mechanisms like reservoirs, microclots, and vagus nerve dysfunction is key to effective management
- Silver Lining: Increased recognition of Long Covid drives awareness of post-infectious syndromes, benefiting chronic illness research broadly
- Call to Action: Prioritize personalized, patient-centered research and treatment to address what's really at play, improving outcomes for Long Covid and related conditions.

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About Nafysa Parpia, ND



Director of Naturopathic Medicine: Gordon Medical Associates | Mold/Mycotoxins | Tick-borne Illness & Lyme Disease | MCAS

Dr. Nafysa Parpia is a board-certified naturopathic doctor and the Director of Naturopathic Medicine at Gordon Medical. Throughout her career in holistic medicine, she has focused on treating patients with complex chronic illnesses. She specializes in tick-borne illness/ Lyme disease, environmentally acquired illness, mold/mycotoxin illness, autoimmunity, fibromyalgia, long haul COVID, ME/CFS (chronic fatigue syndrome) and MCAS (mast cell activation syndrome). Dr. Parpia's extensive knowledge has helped people worldwide overcome difficult-to-treat medical conditions. She uses cutting-edge laboratory tests and deep intuition applied to the full range of scientific data to create comprehensive treatment plans that are highly personalized. Her targeted system of care includes a synergistic blend of allopathic and functional medicine diagnoses paired with treatment that includes regenerative medicine, micronutrient therapies, peptide therapies, bioidentical hormone therapy, botanical medicine, pharmaceuticals and psychoemotional support.