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The relationship between infectious diseases and COVID-19: Co-infections, immune dysregulation, antimicrobial resistance, and clinical outcomes

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Abstract

SARS-CoV-2 has brought about an unprecedented change in the global landscape of infectious diseases. In addition to its own respiratory pathology, COVID-19 patients are vulnerable to a wide variety of secondary bacterial, fungal, and viral co-infections, with multiple mechanisms of convergence: massive lymphopenia; defect in macrophage and neutrophil function; disruption of the mucosal barrier; corticosteroid therapy; and use of invasive supportive therapies. This review comprehensively examines the bidirectional relationship between COVID-19 and infectious diseases, analyzing the epidemiology and microbiology of bacterial co-infections caused by *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, and *Acinetobacter baumannii*; fungal infections including COVID-19-associated pulmonary aspergillosis (CAPA), *Candida* spp., and the unprecedented mucormycosis epidemic; and viral co-infections including influenza and herpesvirus reactivation. Host immune dysregulation—the central facilitating mechanism—is examined at molecular and cellular levels. The review further addresses the antimicrobial resistance crisis catalyzed by the pandemic through empirical antibiotic overuse, disrupted stewardship, and nosocomial multidrug-resistant organism outbreaks. Diagnostic advances, therapeutic strategies, and public health implications are discussed with emphasis on low- and middle-income settings.

Keywords: COVID-19, Co-infections, Cytokine Storm, Antimicrobial Resistance, Infectious Diseases

Introduction

The COVID-19 pandemic, declared by the WHO in March 2020, resulted in over 700 million confirmed infections and more than 7 million deaths globally by 2024. While SARS-CoV-2 itself causes a spectrum of disease from asymptomatic infection to fatal acute respiratory distress syndrome (ARDS), the clinical course of severe COVID-19 is substantially shaped by secondary infections. This pattern was set by history during the 1918 Spanish flu pandemic when most deaths were due to secondary bacterial pneumonia, not the influenza virus. COVID-19 is a recapitulation and extension of it with further complexity.^[1, 2]

The link between COVID-19 and infectious diseases is two way and multi-dimensional. The mechanisms by which SARS-CoV-2 actively undermines innate and adaptive immune defenses include interferon antagonism, induction of lymphopenia and neutrophil dysfunction, which result in a permissive immunological environment for co-pathogens. At the same time, the global pandemic has led to unprecedented empirical antibiotic prescriptions, disturbed antimicrobial stewardship, extended hospitalizations and a healthcare system overwhelmed, all of which have had a significant impact on the global antimicrobial resistance (AMR) crisis.^[3, 4]

This review aims to provide a summary of the existing evidence on all aspects of co-infections associated with COVID-19 infection, including the immune basis, clinical consequences, diagnostic and therapeutic approaches, as well as public health implications, with special emphasis on the accompanying infections in Iraq and the Middle East region.^[5]

2. Epidemiology of Co-infections in COVID-19

2.1 Overall Burden

Previous meta-analyses showed co-infections rates between 6.9% and 27% in hospitalized COVID-19 patients and around 50% in those who had been admitted to the intensive care

unit. In a ground-breaking systematic review, Lansbury *et al* (2020) combined 30 studies that included 3,834 patients, and found that the prevalence of co-infection was 6.9% at the time of admission and 14.3% in patients admitted to the ICU. About 72% of the reported co-infections are bacterial, 19% fungal, and 9% viral, but these figures may be significantly under-estimates because of antibiotic pre-treatment and inter-variant differences in testing practice. [6, 7]

There is a high degree of geographical variation. Iraq, Iran,

and India were among the low- and middle-income countries that had significantly higher rates of fungal co-infections, especially mucormycosis, due to high prevalence of diabetes, overuse of steroids, and poor health care systems that control infections. Factors associated with risk of co-infections are mechanical ventilation, central venous catheterization, broad-spectrum antibiotics, diabetes mellitus (which causes a decrease in chemotaxis, phagocytosis and oxidative burst of neutrophils, and supplies the glucose for fungal growth), and corticosteroid administration. [8, 9]

Table 1: Summary of major co-infections in COVID-19: pathogen, prevalence, and clinical impact. [10-12]

Type	Key Pathogen(s)	Prevalence in COVID-19	Main Clinical Impact
BACTERIAL			
Bacterial	<i>Staphylococcus aureus</i> / MRSA	12-21% severe cases	Secondary pneumonia, MRSA bacteraemia, toxic shock
	<i>Pseudomonas aeruginosa</i>	6-18% ICU	VAP, XDR infections, prolonged ventilation
	<i>Klebsiella pneumoniae</i>	5-14%	Septicaemia, ESBL/CRE emergence, multi-organ failure
	<i>Acinetobacter baumannii</i>	4-10% ICU	Carbapenem-resistant VAP, outbreak clusters
FUNGAL			
Fungal	<i>Aspergillus fumigatus</i> (CAPA)	3-33% ICU/steroid Rx	Invasive pulmonary aspergillosis, ~47% mortality
	<i>Candida spp.</i> / <i>C. auris</i>	5-26% ICU	Candidaemia, nosocomial outbreaks, MDR <i>C. auris</i>
	<i>Mucor</i> / <i>Rhizopus spp.</i>	~0.1-1.8% (India epidemic)	Rhinooorbital-cerebral mucormycosis, 35-54% fatality
VIRAL			
Viral	<i>Influenza A/B</i>	3-20% early pandemic	Additive respiratory failure, higher mortality
	CMV / EBV reactivation	20-30% ICU patients	Interstitial pneumonitis, immunosuppression amplification

3. Bacterial Co-infections

3.1 *Staphylococcus aureus* and MRSA

In severe COVID-19, *Staphylococcus aureus* is the most common bacterial co-pathogen. In the ICUs, the incidence of bloodstream infection (BSI) due to methicillin-resistant *S. aureus* (MRSA) rose between 13% and 20% during periods of pandemic. COVID-19-induced lymphopenia depletes CD4⁺ T helper cells essential for staphylococcal opsonophagocytic killing; mechanical ventilation bypasses mucociliary clearance; and dexamethasone suppresses neutrophil antimicrobial function. Staphylococcal super antigens—TSST-1 and enterotoxins—may synergize with SARS-CoV-2 cytokine storm, producing multi-system inflammatory presentations phenotypically resembling toxic shock syndrome. [13, 14]

3.2 *Pseudomonas aeruginosa*

Pseudomonas aeruginosa is the leading cause of ventilator-associated pneumonia (VAP) in mechanically ventilated COVID-19 patients, accounting for 6-18% of ICU-acquired infections. Its type III secretion system effectors (*ExoS*, *ExoT*, *ExoU*), quorum-sensing-regulated biofilm, and extracellular proteases thrive in the secretion-rich, damaged COVID-19 lung environment. Extensively drug-resistant (XDR) *P. aeruginosa* VAP became disproportionately prevalent in COVID-19 ICUs, with rates of 30-40% reported from Middle Eastern centers, driven by heavy empirical carbapenem use creating strong resistance selection pressure. [15, 16]

3.3 *Klebsiella pneumoniae* and Other Gram-Negatives

Klebsiella pneumoniae is the second most common gram-negative co-pathogen, responsible for pneumonia, bacteraemia, and urinary tract infections in 5-14% of cases. ESBL-producing and carbapenem-resistant *K. pneumoniae* were documented with alarming frequency in COVID-19 ICUs. *Acinetobacter baumannii*, with its extraordinary environmental persistence and near-universal carbapenem resistance in some regions, caused ICU outbreak clusters

directly linked to pandemic overcrowding and contact precaution failures. *Streptococcus pneumoniae* co-infection complicates approximately 11% of COVID-19 pneumonia cases, presenting with lobar consolidation, bacteraemia, and empyema at higher rates than COVID-19 mono-infection. [17, 18]

4. Fungal Co-infections in COVID-19

4.1 COVID-19-Associated Pulmonary Aspergillosis (CAPA)

CAPA emerged rapidly as a major fungal complication, with prevalence ranging from 3.3-33% across ICU cohorts, and mortality of approximately 47% in meta-analyses. *Aspergillus fumigatus* accounts for over 90% of CAPA cases. Multiple SARS-CoV-2 mechanisms facilitate *Aspergillus* invasion: airway epithelial damage impairs conidial clearance; COVID-19-induced lymphopenia depletes CD4⁺ T cells orchestrating anti-*Aspergillus* immunity; dexamethasone suppresses neutrophil oxidative killing of germinating hyphae; and tocilizumab further blunts pro-inflammatory responses required to contain hyphal growth. [19, 20]

Diagnosis is complicated by atypical CT presentations—consolidation and ground-glass opacities indistinguishable from COVID-19 pneumonia—and low sensitivity of serum galactomannan. BAL galactomannan (index ≥ 1.0 , sensitivity ~65%, specificity ~95%) and *Aspergillus*-PCR from BAL provide superior diagnostic performance. Standardized ECMM/ISHAM consensus criteria facilitate consistent case ascertainment. Treatment follows established invasive aspergillosis guidelines: voriconazole or isavuconazole first-line, with liposomal amphotericin B for azole-refractory cases. Azole-resistant *A. fumigatus*—increasingly prevalent globally—significantly complicates CAPA therapy. [21, 22]

4.2 *Candida* Infections and *C. auris*

Candidaemia rates of 5-26% in COVID-19 ICUs far exceed pre-pandemic baselines of 2-4%. Risk factors converge in COVID-19: prolonged ICU stay, central venous catheters, broad-spectrum antibiotics disrupting the gut microbiome

with subsequent *Candida* translocation, total parenteral nutrition, and corticosteroid therapy. *Candida auris* nosocomial transmission in COVID-19 ICUs—confirmed by whole-genome sequencing across the United States, United Kingdom, India, and Spain—represents a quintessential pandemic-within-a-pandemic scenario. Its intrinsic fluconazole resistance, weeks-long environmental persistence, and frequent misidentification make it exceptionally dangerous in overwhelmed ICU settings. Echinocandins are first-line for candidaemia; *C. auris* infections require susceptibility-guided therapy. [23, 24]

4.3 COVID-19-Associated Mucormycosis (CAM)

The CAM epidemic in India during April-June 2021 produced over 47,000 cases—a 100-fold increase above baseline—with rhino-orbital-cerebral mucormycosis (ROCM) predominating and case fatality rates of 35-54%. The pathobiological convergence of multiple COVID-19-specific factors drives CAM: hyperglycaemia (both pre-existing and steroid-induced) creates an iron-rich, acidotic environment optimal for *Mucor* and *Rhizopus* germination; corticosteroid therapy suppresses macrophage conidial killing; COVID-19-induced sinonasal epithelial disruption provides a portal of entry; and COVID-19-associated hyperferritinaemia supplies fungal iron acquisition. Treatment requires urgent surgical debridement combined with liposomal amphotericin B, with isavuconazole as an alternative. [25, 26]

5. Viral Co-infections and Herpesvirus Reactivation

Influenza A/B co-infection with SARS-CoV-2—so-called 'flurona' was documented in 3-20% of early pandemic cohorts, with co-infected patients demonstrating greater respiratory failure severity and higher mechanical ventilation rates. Respiratory syncytial virus, parainfluenza, rhinovirus, and human metapneumovirus collectively complicate 1-8% of cases. Multiplex syndromic PCR panels simultaneously detecting SARS-CoV-2 alongside influenza and RSV from a single nasopharyngeal swab enable pathogen-directed therapy within 1-2 hours and reduce inappropriate antibiotic use. [27, 28]

Reactivation of latent herpesviruses—particularly EBV, CMV, and HSV—occurs in 20-30% of COVID-19 ICU patients, substantially exceeding rates in comparable non-COVID-19 ICU populations. COVID-19-induced lymphopenia and T-cell exhaustion (PD-1↑, TIM-3↑, LAG-3↑) remove the immunological restraint normally suppressing herpesvirus latency. CMV reactivation causes interstitial pneumonitis, hepatitis, and gastrointestinal disease in severe COVID-19 patients, contributing to the

immune dysfunction enabling bacterial and fungal superinfections. EBV reactivation, documented in 30-60% of severe cases, has been proposed as a contributing mechanism to long COVID symptomatology. [29, 30]

6. Host Immune Dysregulation: The Facilitating Mechanism

6.1 Innate Immune Suppression

SARS-CoV-2 encodes multiple interferon antagonists—NSP1, NSP3, NSP12, ORF3b, ORF6, ORF9b—that suppress type I and III interferon production and signaling at multiple levels, creating a window of innate immune vulnerability paradoxically profound in severe disease. COVID-19 simultaneously shifts alveolar macrophages from antimicrobial M1 toward anti-inflammatory M2 phenotype, markedly reducing phagocytic killing of bacteria and fungi and creating a permissive pulmonary microenvironment. [31, 32]

6.2 Lymphopenia and Adaptive Immune Failure

Lymphopenia (count $<1.0 \times 10^9/L$) occurs in 83% of hospitalized COVID-19 patients and is the strongest independent predictor of severity and mortality. SARS-CoV-2 depletes CD4+ T helper, CD8+ cytotoxic T, and NK cells through direct lymphocyte infection, apoptosis induction, and lymphoid sequestration. The resulting functional exhaustion—characterized by PD-1, TIM-3, and LAG-3 upregulation—resembles sepsis-induced immunosuppression and HIV-associated immunodeficiency, explaining susceptibility to opportunistic pathogens including *Pneumocystis jirovecii* and *Cryptococcus neoformans* typically associated with severe immunocompromised. [33]

6.3 Cytokine Storm and the Therapeutic Paradox

Massive IL-6, IL-1β, TNF-α, and IL-8 release in the COVID-19 cytokine storm damages respiratory epithelium and intestinal mucosa, creating microbial invasion portals, while driving the compensatory anti-inflammatory response that produces profound subsequent immunosuppression. Therapies designed to suppress cytokine storm—dexamethasone (WHO-recommended standard of care for severe COVID-19 requiring oxygen), tocilizumab (anti-IL-6R), baricitinib (JAK inhibitor)—while reducing COVID-19 mortality, simultaneously suppress immune effectors required to resist secondary pathogens. This therapeutic paradox demands individualized immunosuppression balancing cytokine storm prevention against infection susceptibility, with antifungal prophylaxis warranted in high-risk patients receiving prolonged corticosteroids. [34, 35]

Table 2. Key immune dysregulation mechanisms in COVID-19 facilitating secondary bacterial, fungal, and viral infections. [36, 37]

Mechanism	Host Immune Effect	Pathogens Exploiting This
IFN-I/III suppression (ORF6, NSP1, NSP3)	Impaired antiviral/antifungal innate response; viral replication window	CMV, EBV reactivation; Aspergillus, Candida establishment
Lymphopenia (CD4+/CD8+/NK depletion)	Loss of adaptive fungal/bacterial immunity; T-cell exhaustion (PD-1↑, TIM-3↑)	Aspergillus, Candida, Pneumocystis, Cryptococcus
Macrophage M2 polarisation	Reduced phagocytic killing; pro-fibrotic, anti-inflammatory phenotype	Candida, Cryptococcus, gram-negative bacilli
Neutrophil dysfunction / dysfunctional NETs	Impaired oxidative burst; reduced killing of bacteria and fungi	<i>P. aeruginosa</i> , <i>Klebsiella</i> , <i>Candida</i> spp.
Cytokine storm → barrier disruption	IL-6/TNF-α/IL-1β-driven epithelial/endothelial damage → microbial invasion portals	All secondary pathogens; driven by SARS-CoV-2 and co-pathogens synergistically
Corticosteroid therapy (dexamethasone)	Suppresses neutrophil oxidative burst, macrophage antifungal killing; promotes fungal growth	Primary driver of CAPA, mucormycosis, Candida infections

7. COVID-19 and the Antimicrobial Resistance Crisis

7.1 Pandemic-Driven Antibiotic Overuse

Rawson *et al.*'s systematic review found that 71.9% of COVID-19 patients received antibiotics despite a confirmed bacterial co-infection rate of only 6.9%, generating immense selective pressure. Azithromycin, initially proposed as a COVID-19 antiviral and prescribed to millions before being disproven, drove macrolide resistance in *S. pneumoniae* and gut Enterobacterales. Broad-spectrum empirical carbapenem, cephalosporin, and fluoroquinolone use in COVID-19 ICUs selected ESBL, carbapenemase-producing, and MDR phenotypes at unprecedented speed. [2, 38]

7.2 Disruption of Antimicrobial Stewardship

Antimicrobial stewardship programs (ASPs) that had achieved significant pre-pandemic AMR reductions were severely disrupted during surge periods: ASP personnel were redeployed; audit/feedback activities ceased; and pre-authorization requirements were relaxed. European and American centres documented 20-40% increases in ESBL, MRSA bloodstream infections, and *Clostridioides difficile* infection in the first pandemic year. *Candida auris* outbreaks confirmed by whole-genome sequencing—in COVID-19 ICUs, alongside XDR *K. pneumoniae*, *A. baumannii*, and VRE outbreaks, represent the AMR consequences of overwhelmed infection control infrastructure. [39]

Table 3: Impact of the COVID-19 pandemic on antimicrobial resistance: mechanisms, documented consequences, and evidence. [40, 41]

AMR Factor	Pandemic Effect
Antibiotic overuse	~71.9% of COVID-19 patients received empirical antibiotics despite only 6.9% confirmed bacterial co-infection
MRSA bloodstream infections	13-20% increase in ICU MRSA BSI during pandemic peaks; contact precaution fatigue major driver
ESBL/CRE emergence	Prolonged hospitalisations and heavy carbapenem use drove ESBL-Klebsiella and CRE rates upward in COVID-19 ICUs
<i>C. auris</i> nosocomial spread	<i>C. auris</i> outbreaks confirmed in COVID-19 ICUs globally; shared equipment and skin contact transmission
ASP programme disruption	ASP activities ceased or deprioritised during surge; audit/feedback, pre-authorisation relaxed under pandemic pressure

8. Diagnostics and Therapeutic Strategies

8.1 Diagnosis of Co-infections

Radiological overlap between COVID-19 pneumonia and bacterial or fungal pneumonias is profound; CT findings of consolidation and bilateral ground-glass opacities are non-specific. Procalcitonin (PCT) levels >0.5 ng/mL have moderate discriminatory value for bacterial co-infection (sensitivity 56%, specificity 68%); PCT-guided de-escalation in COVID-19 reduces antibiotic exposure without worsening outcomes. Multiplex syndromic PCR panels simultaneously detecting SARS-CoV-2, influenza, RSV, and selected bacterial targets enable pathogen-directed therapy within hours. MALDI-TOF mass spectrometry provides rapid organism identification from positive blood cultures. For fungal diagnosis, BAL galactomannan and *Aspergillus*-PCR are preferred over serum biomarkers for CAPA; serum β -D-glucan provides pan-fungal screening for *Candida* and *Pneumocystis* infections. [41, 42]

8.2 Antibacterial and Antifungal Management

Empirical antibiotic therapy should be based on local resistance patterns and severity of the infection, and should aim to cover *S. pneumoniae* and *S. aureus* in community-acquired pneumonia and gram-negative organisms, such as *P. aeruginosa*, in ICU-onset infections, and then be de-escalated upon culture data. Novel beta-lactam/inhibitor combinations (ceftolozane-tazobactam, ceftazidime-avibactam) provide extended MDR gram-negative coverage; MRSA VAP should be treated with vancomycin, using AUC monitoring, or linezolid. Voriconazole or isavuconazole are being used as first-line antifungals in COVID-19 patients with high-risk features in ICUs who are on prolonged corticosteroid use, where antifungal prophylaxis may be indicated; echinocandins are being used in candidaemia; and liposomal amphotericin B is indicated in mucormycosis, where surgical debridement is urgently required. [43]

9. Conclusions and Public Health Implications

COVID-19 and infectious diseases have a complicated relationship that is bi-directional and clinically relevant. SARS-CoV-2 generates a complex immune deficiency: interferon suppression, lymphopenia, barrier damage by cytokine storm, and iatrogenic immunosuppression (allergic and autoimmune deficiencies), leading to an incredible spectrum of secondary bacterial, fungal, and viral infections. The pandemic led to an exponential increase in the global AMR crisis, with huge numbers of empirical antibiotic prescriptions given, disruption to stewardship, and nosocomial outbreaks of MDR in overcrowded ICUs. [44]

These challenges are especially significant for LMCs such as Iraq, where baseline AMR is high, and the prevalence of fungal disease and diabetes is high; as well as in which access to novel antifungals (isavuconazole, liposomal amphotericin B), advanced diagnostics, and second-line antibiotics is limited. AMR surveillance strengthening, ASP and IPC sustaining during public health emergencies, essential antimicrobial supply chains, and investing in infrastructure for swift diagnostics must be part of pandemic preparedness planning. [45]

Lessons from pandemic co-infection should guide future preparedness for health emergencies: Microbiological sampling before starting empirical therapy, pathogen-driven de-escalation, ongoing ASP activity, strong IPC even during a surge, and equitable global access to diagnostics and therapeutics are non-negotiables of an integrated response to the intersection of COVID-19 and the global burden of infectious diseases. [46]

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