Critter COVID-19- Zoonotic Contagion

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Preface

Coronavirus (CoVs) corresponds to the family Coronaviridae which comprises of a cluster of enveloped, positive-sense, single-stranded ribonucleic acid (RNA) viruses. RNA viruses harbouring a genome of 26 kilo-base to 32 kilo-base with a crown-like morphology upon electron microscopy are denominated as “Coronaviruses”. Coronaviruses perpetually invade the species barriers and emerge as significant human pathogens. Severe acute respiratory syndrome arising due to coronavirus 2 (SARS-CoV-2) manifests as causative agent of the contemporary pandemic labelled as coronavirus disease 2019 (COVID-19).

Of the diverse human coronaviruses, preponderantly pathogenic SARS-CoV-2 engenders severe lower respiratory tract infection which may evolve into possible acute respiratory distress syndrome (ARDS) with diverse extra-pulmonary manifestations. Nevertheless, transmission, pathogenicity and sustainable viral dissemination following human infection with SARS-CoV-2 influences ultimate outcome of ongoing pandemic of COVID-19. Notwithstanding, the fact that SARS-CoV-2 is fabricated deliberately or accidentally remains unproven and unsustainable.

Physiology and pathogenesis

World Health Organization (WHO) defines zoonosis as “any infection that is naturally transmissible from vertebrate animals to humans”. Thus, it can be corroborated that the infection is maintained within an animal population or a reservoir and continues to be a perpetual source of human infection [1,2].

Besides, infections which are acquired by humans through direct contact with animals or infections transmitted through routes of indirect exposure such as vector-borne pathogens or environmental and food system pathogens are comprehensively integrated [1,2].

World organization for animal health (OIE) defines wildlife as:

- Wild animals or phylogenetically diverse, free ranging wild animal species.
- Feral, domesticated or free-ranging animals.
- Non-domestic animals confined in captivity or farming [1,2].

However, zoonosis is contemplated to be an amalgamation of diseases which originate in animals and independently persist in infected humans along with diseases which necessitate a non-human animal host for survival and persistence of diverse pathogenic organisms [1,2].

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High impact “zoonotic diseases” are endemic zoonosis such as rabies, brucellosis and cysticercosis, diseases which demonstrate definitive transmission from domestic animals. However, divergence in zoonotic viruses is intensely concurrent with abundance and diversity of mammalian species, especially domesticated animals. Additionally, enhanced viral abundance is observed within evolving domesticated animal species appearing in proximity to humans, as encountered with livestock food protocol altered due to elevated urban demand for animal products [1,2].

Human coronaviruses (HCoVs) depict a zoonotic origin wherein the viral infection is contracted from bats, mice or domestic animals. It is posited that evolutionary origin of human coronaviruses from bats is comprised of well adapted, non-pathogenic and genetically diverse organisms [3,4].

However, non-pathogenic, parental viruses of human coronaviruses confined to natural reservoir hosts convert into pathogenic organisms following interspecies transmission within a new host. Also, evolution of human coronaviruses exhibit enhanced transmissibility in association with decimated viral pathogenicity [3,4].

Emergence of SARS-CoV-2 infection appears identical to infection with severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV). A bat coronavirus (CoV) demonstrating around 96% nucleotide homology with SARS-CoV-2 can be discerned. SARS-CoV-2 is a moderately pathogenic, transmissible variant with nucleotide sequence homologous to SARS-CoV. However, immediate intermediate hosts of SARS-CoV-2 infection remain obscure [3,4].

Pangolin beta-CoVs is intensely homologous to SARS-CoV-2, a feature which indicates that pangolin may function as an intermediate host to SARS-CoV-2 infection or pangolin beta CoVs may contribute genetic fragments towards the ultimate configuration of SARS-CoV-2 [3,4].

SARS-CoV-2 demonstrates a reproduction number (R0) of between 2.0 to 2.8. Incubation period varies from three days to fourteen days with a mean incubation period of 4.75 days to 7 days [3,4].

Viral load is enhanced following onset of cogent clinical symptoms wherein viral load is examined in sputum, throat swab, urine or stool. Also, viral load appears elevated within the nasal passages, in contrast to the throat. Also, respiratory specimens depict an enhanced viral load, in contrast to stool samples. Additionally, SARS-CoV-2 can be isolated from saliva or faecal samples although urine may be devoid of the virus [3,4].

Generally, asymptomatic individuals depict a viral load akin to symptomatic subjects. Viral transmission may emerge during the incubation period or from asymptomatic or mildly infections individuals. Generally, viral loads spikes within 5 days to 6 days following onset of clinical symptoms. Significant viral load manifests as $10^4$ copies/millilitre to $10^{11}$ copies/millilitre, as discerned by reverse transcriptase-polymerase chain reaction (RT-PCR). Undiagnosed instances usually portend significant viral dissemination [3,4].

In addition to pertinent animal hosts, viral factors contributing to viral navigation across species can be enumerated. CoV is associated with a significant, moderate to high proportion of mutation along with RNA replication, in contrast to diverse single-stranded RNA viruses [3,4].

An average substitution rate of $\sim 10^{-4}$ substitutions per year per site is encountered, contingent to phasic adaptation of CoV to novel hosts. Deletion of proof-reading exonuclease of CoV engenders exceedingly enhanced mutability to nearly a million times as compared to the host, attenuated viruses or non-viable viruses. Also, proportionate viral mutation of CoVs is enhanced where viruses are inadequately adapted to host [5,6].

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In contrast to SARS-CoV, proportionate mutation of SARS-CoV-2 is reduced which indicates enhanced viral adaptation to incriminated human subjects. Possibly, SARS-CoV-2 is previously adapted to a mammal host, akin to humans [5,6].

It is posited that genetic variation of SARS-CoV-2 may not be competent in rendering SARS-CoV-2 vaccines and antivirals rapidly ineffectual. The enlarged RNA genome of CoVs exudes additional plasticity along with genome modification required for genetic mutations and recombination. Thus, possible interspecies co-evolution is enhanced which is beneficial in the emergence of novel CoVs under appropriate conditions [5,6].

Coronaviruses (CoVs) randomly and frequently switch templates during RNA replication through a unique “copy-choice” mechanism. Strand switching is frequent during CoV RNA transcription within the host. Additionally, homologous, full-length and sub-genomic RNAs may recombine to generate novel coronaviruses (CoVs) [5,6].

Advent and conveyance

Morphologically, coronavirus (CoV) demonstrates non-segmented genomes with a similar organization. Contingent to diverse protein sequences, coronavirus (CoV) is classified into distinct genera denominated as alpha-CoV, beta-CoV, gamma-CoV and delta-CoV [4,6].

Gene source of beta-CoV genera is provided by bats and rodents wherein the beta-CoV variant is constituted of majority of human CoVs which are further categorized into lineages designated as A, B, C and D.

Human infection with SARS-CoV-2 primarily occurs due to slaughter and consumption of game meat. Several mammals and domestic animals are susceptible to infection with SARS-CoV-2 [4,6].

Also, recombination and adaptation of SARS-CoV-2 may emerge within an animal species which interacts with bats and pangolins. Exploration of animal origins of SARS-CoV-2 remains a continual process [5,7].

Human to human transmission of SARS-CoV-2 occurs comprehensively within families, communities and individuals, particularly health care workers. Predominant transmission occurs through droplets, inanimate objects or aerosols wherein the respiratory tract is commonly infected. Besides, an oro-faecal mode of disease transmission appears with SARS-CoV-2 and MERS-CoV, thereby infecting the gastrointestinal tract [5,7].

Severe instances of SARS-CoV-2 infection may be misinterpreted as SARS-CoV infection [5,7].

Transmission of SARS-CoV-2 displays patterns akin to disease transmission of community-acquired human coronaviruses (HCoVs) and SARS-CoV. Besides, disease dissemination of SARS-CoV-2 appears identical to community-acquired HCoVs. However, it remains to be ascertained if disease transmissibility of SARS-CoV-2 declines following human infection, as encountered with SARS-CoV and MERS-CoV [5,7].

Viral interaction with host receptor contributes significantly to interspecies viral transmission [5,7].

SARS-CoV-2 depicts identical cellular receptors as SARS-CoV with around 30% distinction between SARS-CoV-2 and SARS-CoV receptors, especially within S1 unit of S protein. Aforesaid demarcation implicates altered or reduced binding affinity of S protein of SARS-CoV-2 with human angiotensin converting enzyme 2 (ACE2) receptors [6,7].
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Additionally, co-receptors may be required for SARS-CoV-2 transmission such as different segment of S, aminopeptidase N or 9-O-acetylated sialic acid, as demonstrated in diverse human coronaviruses (HCoVs). Aforesaid co-receptors facilitate adaptation of CoVs within humans following interspecies transmission from pertinent animal hosts [6,7].

Apart from cellular receptors, interspecies transmission of human coronaviruses (HCoVs) is contingent to host dependency and restriction factors. Divergent host proteins between humans and natural reservoir hosts of HCoVs such as bats, dromedary camels or rodents may impede interspecies transmission [6,7].

HCoVs are required to circumvent host dependency factors and suppress host restriction factors for appropriate interspecies viral transmission. Nevertheless, molecular determinants pertaining to virus-host interaction are yet to be identified and categorized. Genome-wide screening with clustered regularly interspaced short palindromic repeats (CRISPR) for assessment of host dependency and restriction factors applicable to SARS-CoV-2 may be beneficial [7,8].

**Symptoms and significance**

Severe lower respiratory tract infection is a component of COVID-19 and is engendered by SARS-CoV-2. The respiratory infection usually represents with pyrexia, cough and dyspnoea. Severe instances manifest with pneumonia with rapid emergence of acute respiratory distress syndrome (ARDS). Diarrhoea occurs in individuals with gastrointestinal involvement [7,8].

Asymptomatic subjects infected with SARS-CoV-2 contribute significantly to viral dissemination to pandemic proportions. SARS-CoV-2 infection demonstrates an incubation period and duration of human coronavirus disease identical to diverse genera of human coronaviruses.

Novel coronaviruses (CoVs) are non-pathogenic or induce mild clinical symptoms within reservoir hosts such as bats and camels. Viral replication is intense and is devoid of accompanying sturdy host immune response. Thus, asymptomatic carriers are engendered which display an immune response in disjunction to coronavirus (CoV) replication [7,8].

Severe instances of human coronavirus infection exhibit severe clinical symptoms which predominantly arise due to hyper-activation of immune response and associated cytokine storm. An intense immune response is associated with severe damage to pulmonary parenchyma [7,8].

Severity of clinical symptoms associated with COVID-19 arising due to SARS-CoV-2 is intermediate to infection with severe acute respiratory syndrome with coronavirus (SARS-CoV) and community-acquired human coronaviruses (HCoVs) as HCoV-229E, HCoV-OC43, HCoV-HKU1 and HCoV-NL63 [7,8].

Infection with SARS-CoV-2 may exhibit features akin to infection arising due to community-acquired HCoVs such as non-specific, mild or asymptomatic disease representation. Also, infection with SARS-CoV-2 may exemplify seasonal variation as delineated in community-acquired human coronavirus (HCoVs) infections [7,8].

**Therapeutic strategies**

A delinked immune response to coronavirus (CoV) replication may be beneficial for adequate outcome to anti-SARS-CoV-2 therapy. Interferon reaction is intense within natural hosts such as bats. Therefore, adoption of type I interferon in order to treat preliminary phase of SARS-CoV-2 infection in humans is beneficial. Also, as NLR family pyrin domain containing 3 (NLRP3) inflammasome activation in bats is defective, selective inhibition of NLRP3 inflammasome with MCC950 may be advantageous in the management of COVID-19 [7,8].

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**Figure 1:** Zoonosis associated with SARS-CoV-2 demonstrated by exposure to diverse animal categories and species [9].

**Figure 2:** Zoonosis associated with SARS-CoV-2 exhibiting sources of various human coronaviruses and predominant animal species accompanying respiratory infections [10].

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Figure 3: Zoonosis associated with SARS-COV-2 enunciating various modes of viral transmission from different animal species [11].

Figure 4: Zoonosis associated with SARS-CoV-2 delineating viral transmission route with reservoir, intermediary and ultimate hosts [12].

Bibliography


10. Image 2 Courtesy: MDPI.


12. Image 4 Courtesy: Science in the news.

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