R3298

Do Stealth Adapted Viruses Predispose to More Severe and Prolonged Covid-19 Illness?

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Cat. 4165-ASIP Viral Pathogenesis

Abstract

Infection with the SARS-CoV-2 virus can lead to variable clinical outcomes. Approximately 40% of those infected will remain asymptomatic; with a similar percentage developing a mild to moderate illness, essentially localized to the respiratory system. A markedly more severe and extensive illness involving multiple organs occurs in the remaining approximately 20% of those infected. A further intriguing consequence of Covid-19 disease is the continuing illness in about 10% of those with either the mild to moderate or the more severe acute Covid-19 disease. This Long Covid-19 syndrome has many features in common with the chronic fatigue syndrome (CFS).

The concept of coinfecting virus pathogens is central to understanding several human diseases. Another useful concept is that of stealth adapted viruses. These viruses differ from the viruses from which they are derived in not typically evoking inflammation. This is because of deletion or mutation of the genes coding for the relatively few virus components that are normally targeted by the cellular immune system. It is a generic process that can potentially apply to all viruses, including the SARS-CoV-2 virus.

Stealth adaptation has occurred with the cytomegaloviruses of rhesus and African green monkeys. Kidney cells from African green monkeys are still being used to produce poliovirus vaccines. DNA sequencing studies on these monkey-derived stealth adapted viruses show that in addition to deletions or mutations of some of the originating virus genes, stealth adaptation can involve the incorporation of additional genetic sequences from cells, other viruses, and bacteria. These “renegade” sequences become components within the reformed, replicating and infectious stealth adapted viruses.

The variable and diverse genetic compositions of different stealth adapted viruses are likely to at least occasionally include some elements that are interactive with genes of the SARS-CoV-2 virus. This could lead to the potentiation of either or both viruses with added pathogenicity for the infected individual. Moreover, although the residual virus components may not be ordinarily immunogenic, some may become so if the virus levels are greatly increased and/or the immune system is sufficiently stimulated.

A strong case can be made for a role of stealth adapted viruses in patients with the Long Covid-19 syndrome. These patients can be divided into those who had similar but milder symptoms prior to Covid-19 and those who had previously been asymptomatic. The former patients were probably infected earlier with a stealth adapted virus, which has now become potentiated through its interaction with the SARS-CoV-2 virus. The later patients are likely to be newly infected with a stealth adapted SARS-CoV-2 virus.

The next step in exploring this hypothesis is to culture the blood of patients for evidence of infection with stealth adapted viruses, followed by genetic sequencing of the positive cultures.

The testing should include patients who have experienced severe acute Covid-19 illness and patients with the Long Covid-19 syndrome. Identifying severe acute Covid-19 illness as a co-infection and the realization that the Long Covid-19 syndrome is also an infectious and transmissible disease will have important Public Health implications with regards to an expanding spectrum of human illnesses.

Known Factors Accounting for Variable Outcomes in Covid-19 Infections

- Fragility of old age requiring nursing-home care
- Medical risk factors: Obesity, hypertension, diabetes, COPD, asthma, chronic kidney disease, liver disease, thrombotic disorders, etc.
- Immunosuppression due to HIV or chemotherapy. Yet, severity is also seen with over-reactive immune response leading to cytokine storms
- More virulent virus variants

Proposed Additional Factors

- Other viruses which can positively or negatively interact with the SARS-CoV-2 virus. These include “stealth adapted viruses” (Topic of this Poster)
- Inadequate alternative cellular energy (ACE) pathway. This pathway provides effective non-immunological resistance to infections. It also restricts the triggering of excessive immune reactivity. Aging and certain brain-related disorders can specifically impair the ACE pathway. (Discussed elsewhere)

Stealth Adapted Viruses: Potential Co-Pathogens in Covid-19 Infections

- Stealth adaptation is an immune evasion mechanism in which there is deletion or mutation of the genes coding for the relatively few virus components that are normally targeted by the cellular immune system
- Incorporation of additional genetic sequences from cellular genome and from other microbes to restore infectivity and transmissibility
- Genetic instability of the remaining genes of the originating virus and of the incorporated “renegade” cellular and microbial genes
- Generic process potentially applicable to all viruses
- Stealth adapted African green monkey simian cytomegaloviruses (SCMV) resulting from using CMV infected monkeys to produce polio vaccines

Prototype SCMV-Derived Stealth Adapted Virus Stealth virus-1 on GenBank

- Initially cultured in 1991 from a patient with chronic fatigue syndrome
- Many subsequent positive blood cultures from the same patient
- One of three PCR amplified sequences showed partial homology to human cytomegalovirus. *Am J Path* 145: 440-451, 1994.
- Subsequently this and some other cloned sequences were shown to be nearly identical with regions of SCMV. *Clin Diag Virol* 4: 93-103, 1995
- Some sequences matched to human cellular sequences, while other matched to certain bacterial sequences
- Pathogenic in cats leading to severe, widespread infection. Yet an absence of inflammation. Neurological illness with clinical recovery.

DNA Sequence Analysis of Clones from Stealth Virus-1 (Data on GenBank)

- 200 clones match to approximately half of the SCMV genome. Uneven distribution of the matching clones with several parts of SCMV genome being repeatedly represented and no matching to other regions of SCMV
- 14 clones match to non-coding regions of the human genome. Some of these cellular sequences have presumably replaced previously present African green monkey cellular sequences by homologous recombination
- 18 clones match to Ochrobactrum; 10 to mycoplasma, 6 to undefined bacteria
- Closely related SCMV sequences in viruses from several other patients e.g., stealth virus-2 from a comatose patient with history of a bipolar psychosis

Blast Analysis of a PCR Amplified Cellular Sequence in Stealth Virus-3

Species	Identical Nucleotides	% Identity	Gaps
Rhesus	504/510	98.8	3
African green	478/508	94.1	4
Human	447/508	88.5	16

- The DNA matching data indicate that the sequence originated from the rhesus monkey genome. Closely related sequence were identified in PCR amplified products from stealth adapted virus cultures from two of six other chronic fatigue syndrome patients

Pandemic of Stealth Adapted Viruses

- Stealth adapted viruses can be cultured from many individuals, not all of whom have noticeable clinical illness
- High prevalence of these viruses in patients with chronic fatigue syndrome, Gulf War syndrome, multiple myeloma, and autism
- Stealth adapted viruses can potentially act as carriers of any cellular or microbial “renegade” genetic sequences

Potential Interactions Between SARS-CoV-2 & Stealth Viruses

- SARS-CoV-2 gene products interacting with stealth adapted virus
- Stealth adapted virus gene products interacting with SARS-CoV-2
- Immune stimulation by SARS-CoV-2 inducing immunity/inflammation against some residual components on the stealth adapted virus that are not normally recognized by the cellular immune system
- Certain of the illnesses, which predispose to severe Covid-19, are likely due to infections with stealth adapted viruses

Conclusions

- Public Health authorities should acknowledge the existence of vaccine-derived stealth adapted viruses and test for their potential interactions with SARS-CoV-2 virus and with Covid-19 vaccines.
- SARS-CoV-2 may become stealth adapted, especially because the vaccine is restricted to the spike protein and is given intramuscularly
- Potential incorporation of vaccine mRNA into the genome of a stealth adapted virus, producing excess ACE2 receptor binding spike protein
- Potential restoring of replication of the adenovirus vector used in certain vaccines because of preexisting stealth adapted adenoviruses