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Veterinary Medicine in Human Pandemics: Feline Infectious Peritonitis Compared to Long Covid

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Both diseases are poorly understood and multisystemic with a variety of similar symptoms

Feline Infectious Peritonitis (FIP)

Coronavirinae

α-Coronavirus

FCoV

High mutation rate[1]
Same family as Human common cold virus HCoV-229E known to interfere with Reactive Oxygen Species[2]

Latency period
Feline Enteric Coronavirus (FECV) mutates spontaneously to the virulent Feline Infectious Virus (FIPV). Changing in cell tropism (7-14 %) from enterocytes to monocytes/macrophages

Respiratory burst

FCoV replication after infection in mature apical columnar epithelial cells of the small intestine, persistence in the colonic columnar epithelial cells. Persistence in monocytes/macrophages is required for the outbreak of FIP (distribution all over the body)[1]. Following primary infection, three scenarios have been documented: fairly resistant (5 %) temporary low-level shedding (70-80 %), long-term persistent shedding (10-15 %) of high viral loads[1].

Characteristics of FIP

FIP in young animals from a crowded environment (catteries).
The mutation from FECV to FIPV is stress-related and depends on genetics[1].

Antibodies: No diagnostic value in FIP
(In end-stage FIP antibodies can drop below limit of detection)

Non protective antibodies and formation of immune complexes[3]
Granulomatous vasculitis in the follow-up of Antibody-dependent Enhancement (ADE)[4,5]

B-Cell/T-Cell populations modified[6,7]

Increased serum amyloid A[8]

Progression with waxing and waning

30 % show neurological symptoms. Fatal outcome[1]

New infection with mutated virus always possible[1]

Diagnosis, Treatment and Vaccination

Diagnostic criteria: Not specific
Treatment : 3-months administration of GS-441524 or Remdesivir[9]
Vaccination: Attenuated virus is known to induce a fatal outcome[10]
A successful vaccine for FCoV free cats is Primucell® FIP, the virus is modified and can only multiply at the temperature of nasal mucosa.

Long Covid (LC)

Coronavirinae

β-Coronavirus

SARS-CoV-2

High mutation rate
Zoonosis transferred likely from bats to the human species

No latency period
Primary target of SARS-CoV-2 in the acute phase Covid-19 is the ACE-2 receptor

Respiratory burst

Characteristics of LC

Viral remains/viral persistence in multiple organs[11], including the gastro-intestinal tract[12]

73 % women (mean 46 years old)[13]
Modified monocytes when the virus remains[14]
MCAS (Mast Cell Activation Syndrome)[15]

Antibodies: No diagnostic value in LC[16,17]

Autoimmune antibodies[16,18,19]

Imbalance of T-Cell population[20]

Amyloid fibrin microclots[21] can explain the onset of autoimmune disease, including those of the brain.

Frequently detected autoimmune diseases in patients with onset during Covid-19 and LC: lupus, rheumatoid arthritis, Hashimoto's thyroiditis, Bell's palsy, atypical myasthenia gravis, type 1 diabetes, tinnitus (Autoimmune Inner Ear Disease), ...
How many more?

Progression related to crashes, fluctuating, depending on the clinical case (improvement, stabilization, worsening of symptoms)

High proportion of neurological symptoms

New infection possible

Diagnosis, Treatment and Vaccination

Diagnostic criteria: Not specific
Treatment : None (spontaneous recovery in about 10 % of the patients, healing sawtooth-like ➔ patients don't know, if they are in a crash or healing ➔ psychologically devastating situation
Vaccination : Several vaccines available

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SCAN ME
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Matou – Died at 7 months from FIP

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LONG COVID BELGIUM
Scannez-moi!