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Introduction

The first scientific study describing Coronavirus (CoV - human Coronavirus) as a disease promoter human was released in the 1960s, although, the virus was initially founded in Gallinaceous birds in 1930 [1]. Currently, coronaviruses are classified in the order Nidovirales, subfamily Orthocoronavirinae and subdivided in four genera: *Alpha* (CoV), *Beta* (CoV) and some members belonging to *Gamma* (CoV) are pathogens to mammalian, although some strains belonging to genus *Gamma* and *Delta* (CoV) have adapted to cause disease in avian. Among the viral strains classified as CoV, the following stand out: -HCV_{229E}, -HCV_{NL63}, -HCV_{OC43}, -HCV_{HKU1}, -MERS-CoV, -SARS-CoV and -SARS-CoV₂.

The strains -HCV_{229E} and -HCV_{OC43} first identified in the mid-1960s [1-3], are closely related with the common cold syndrome and pneumonia [4-8], uncommonly causing severe diseases. It was also reported that both strains can cause LRTI (Lower Respiratory Tract Infections) and otitis media [3]. Often this virus is detected in co-infections with other strains of viruses and bacteria resulting respiratory infections [4,9-11]. In addition, this virus also is widely distribution worldwide with seasonal outbreaks [12]. CoV infections have been described for decades, occurring in 2- to 3-year cycles [13], with seasons of high infections rates caused by strains -HCV_{229E} or -HCV_{OC43} and sporadic infections caused by strains belonging to the others CoV groups [12].

The strain -HCV_{NL63} was reported for the first time in 2004 in a baby from Netherlands suffering from bronchiolitis [11,14]. However, this strain seems to affect mostly immunocompromised children inducing symptoms as cough, fever, and rhinorrhea or severe LRTI,

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may be due to the cytopathic effect mediated by local replication of the SARS-C V; or indirectly as a result of systemic responses to respiratory failure or the harmful immune response induced by viral infection.

In 2012, the outbreak of a new C V capable of causing Middle East respiratory syndrome (MERS-C V) was reported, being the first case occurred in Kingdom of Saudi Arabia (KSA), recording at least 2468 cases and 851 deaths worldwide [19]. Another well-documented case of MERS-C V occurred in May 2015, according to information from Korea Centers for Disease Control and Prevention (2015), an infected individual returning from the Middle East started the outbreak of MERS-C V in South Korea, which spread over 16 hospitals and 186 patients. On the next year, were reported 1,728 confirmed new cases of MERS-C V, and 624 deaths were notified in 27 countries [20]. In 2018 a new outbreak of MERS-C V was recorded in the Saudi Arabia [21], with 96 new cases. After these outbreaks of MERS-C V, very year's new cases are documented, usually associated with infected travels; often, these imported MERS cases resulted in nosocomial transmission [19]. Symptoms of MERS-C V are fever, cough and upper respiratory tract (URT) signs and symptoms usually occur first, followed within a week by progressive LRT distress and lymphopenia[22].

The current pandemic of COVID₁₉ (COroNaVIrusDisease and 19 refers to year 2019, was first reported at Wuhan, China. The clinical manifestations are very similar to SARS-C V infection (for that reason it was described as SARS-C V₂). Elderly population stands out as the group with the highest risk, especially those that feature previous comorbidities [23]. In addition, Jordan and collaborators (2020), reported that the restrictions imposed as a control measure during SARS-C V₂ pandemic, may lead elderly people to experience loneliness, isolation and loss of mental and physical stimulation, which could increase the severity of infection.


Genetic Recombination of CoV

C Vs are featured by a positive single-stranded RNA. At the molecular level, C Vs usually feature high mutation rate due to the propensity of errors during their replication [23-25]. It has been shown that these viruses naturally present 20% homologous RNAs recombination mixed infections with different viral strains the same group [23].

Genetic recombination and point mutations in the genome of C Vs can be considered as mechanisms of adaptation between strains, since it is speculated that HC V_{NL63} has arisen from the evolution of HC V_{229E} [26]. In 2018, Chinese scientists reported a new genotype of strain HC V_{OC43}, named H by Zhu and collaborators (2018). It is possible that the H strain emerged as a result after genetic recombination involving seven other strains of HC V_{OC43} [27], previously identified as HC V_{OC43}^a - HC V_{OC43}^b. In 2006 was described the circulation of HC V_{HKU1} strain throughout the United States, however, with genetic differences from the strain originally identified in Hong Kong, revealing the easy adaptation of C V strains to different environmental and climatic conditions [28].

Scientific approaches involving genetic simulation have been useful to address many hypotheses. In 2006, a prospective study developed by [29], using genetic predictions, analyzed 25 variants of HC V_{229E} disseminated in Australia, described during the years of 1979 to 2004. In the study, gene product S and N (spike protein and nucleoprotein) were selected, considering their primary role of facilitates the virus binding to the host cell. In this research the authors concluded that S protein has not mutated over the years, therefore, a type of positive selection mechanism exists, and probably are one of the mechanisms

responsible for the emergence of new viral strains [29]. These results were corroborated by Li and collaborators (2011) considering them

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Test	Results	Method	Reference value
Triglycerides	263 mg/dL	Enzymatic	< 150 mg/dL
Total cholesterol	218 mg/dL	Direct enzymatic	< 200 mg/dL
HDL	48.5 mg/dl	Colorimetric without precipitation	23-92 mg/dl
LDL	106.0 mg/dL	Direct enzymatic	< 130 mg/dL
VLDL	52.6 mg/dL	Calculation	< 40 mg/dL
Glucose	88 mg/dL	Enzymatic	70 – 99 mg/dL
Vitamin D	27.7 ng/mL	Chemiluminescence – LIAISON DiaSorin	
Vitamin B12	566 pg/mL	immunoassay – Immulite 2000 XPI – Siemens	193 – 982 pg/mL
Calcium	7.8 mg/dL	Colorimetric	8.6 – 10.3 mg/dL
Magnesium	2.2 mg/dL	Colorimetric	1.9 – 2.7 mg/dL
Urea	31 mg/dL	Kinetics	15 – 43 mg/dL
Creatinine	0.9 mg/dL	Colorimetric	0.6 – 1.3 mg/dL
Alkaline phosphatase	59 U/L	Enzymatic	34 – 104 U/L
GGT	62 U/L	Colorimetric	9 – 64 U/L
TGO	22 U/L	Enzymatic	13 – 39 U/L
TGP	30 U/L	Enzymatic	7 – 52 U/L
Serum phosphorus	3.5 mg/dL	Colorimetric	2.5 – 5 mg/dL
Chlorine	106 mEq/L	Colorimetric	98 – 107 mEq/L
Sodium	139 mEq/L	Colorimetric	136 – 145 mEq/L
Potassium	4.4 mEq/L	Colorimetric	3.5 – 5.1 mEq/L
TSH	1.030 µIU/mL	Microparticle chemiluminescent immunoassay – Immulite – Siemens	1.01 ng/dL
Free thyroxine (T4)	1.01 ng/dL	Microparticle chemiluminescent immunoassay – Immulite – Siemens	2000 XPI 0.89 – 1.79 ng/dL

Table 1: Laboratory tests requested for Biochemical analysis, method used for diagnosis and reference values adopted at the Hospital Geral Dr. César Cals, Fortaleza-CE.

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