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Clinical and laboratory features of COVID–19 patients infected with SARS–CoV–2 variant B.1.1.7 versus those infected with other SARS–CoV–2 strains: A retrospective observational study

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ABSTRACT

Objective: To investigate the clinical symptoms of coronavirus disease 2019 (COVID-19) patients with and without B.1.1.7 mutation.

Methods: This retrospective observational study included COVID-19 patients who were divided into two groups, the mutation and the non-mutation group. Demographics characteristics, clinical characteristics, laboratory parameters, and mortality rates were recorded and compared between the two groups.

Results: A total of 196 patients were included in the study. The relationship between the mutant virus status and sex, age, comorbidity, survival status, and disease severity was not significant ($P>0.05$). No significant differences were found in duration of hospitalization between the mutation and the non-mutation group ($P>0.05$). However, there was a statistically significant difference between patients with and without mutant viruses in hemoglobin, mean platelet volume, procalcitonin, low density lipoprotein, iron-binding capacity, potassium, calcium, C-reactive protein, folate, creatine kinase myocardial band, D-dimer, and international normalized ratio ($P<0.05$).

Conclusions: No significant difference is found in mortality rate, disease severity or duration of hospitalization between the patients with and without variant B.1.1.7. Careful monitoring of COVID-19 patients is required for all variants.

KEYWORDS: COVID-19; B.1.1.7; Variant; Mutation; Clinical parameters; Laboratory parameters

1. Introduction

Coronavirus disease 2019 (COVID-19) emerged in December 2019 and has caused an epidemic globally and an unprecedented disruption in human society. Since then, the virus has infected more than 169 million people and has killed more than 3.5 million people[1]. Previous studies have clearly shown that epidemic and pandemic spread of the RNA virus may cause mutations that alter

Significance

Many studies have been conducted on the B.1.1.7 variant, mostly focusing on length of hospital stay, disease severity, and mortality of this variant. This study aims to compare patients with and without variant B.1.1.7 in terms of clinical and laboratory parameters, retrospectively. It provides physicians with enlightenment in terms of different variants of SARS-CoV-2.

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the pathogenicity, virulence, transmissibility, or a combination of these in the RNA virus[2]. These mutations among coronaviruses in animals and humans have recently been studied. Early variants of the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) show limited genetic diversity because the virus may be from a single source[3].

Early animal variants of the SARS-CoV-2 virus, introduced in 2003, affected the receptor-binding domain binding to the spike protein, thereby increasing the binding and entry of the virus through the human angiotensin-converting enzyme 2 receptor. However, despite coronavirus RNA-correcting activity supporting replication, genetic epidemiological studies have identified an emerging D614G mutation that affects the glycoprotein protein of SARS-CoV-2 strains from southern Europe. This variant has since spread rapidly and has become the most common genotype worldwide[4].

The B.1.1.7 variant was discovered in the UK, and it is declared that it is at least 50% more likely to be transmitted than the wild-type variant. The announcement was based on epidemiological data showing that the virus spread rapidly across the UK[1]. Lineage B.1.1.7 contains 17 mutations, including several identified in the spike protein, one of which, N501Y, contributes to the strong binding of the virus to the angiotensin-converting enzyme 2 cell receptor[5].

In the genome studies of SARS-CoV-2, the most frequent mutation rate was observed on genes encoding thorn (S) protein, RNA polymerase, RNA primase (ORF1ab), and nucleoprotein (N)[6]. Furthermore, studies have shown that these mutations in the virus change the clinical course of the disease and develop resistance to antiviral therapy[7]. There have been many studies on the B.1.1.7 variant, most of which focused on the duration of hospitalization, disease severity, and mortality of this variant. This study aimed to retrospectively compare clinical and laboratory parameters of patients with and without variant B1.1.7.

2. Patients and methods

2.1. Study design

This retrospective and observational study was conducted in Ayancik Government Hospital between April and July 2021.

2.2. Patients

Patients who were positive for COVID-19 were divided into two groups: the mutation and non-mutation group. Inclusion criteria was as follows: Over the age of 18, having COVID-19 PCR +, being reported as SARS-CoV-2 mutant and non-mutant as a result of PCR, being treated as an inpatient, having complete laboratory and clinical records. Patients who were under the age of 18, received a COVID-19 PCR result, were not hospitalized, and had missing laboratory and clinical records were excluded from the study. File

records were scanned, including age, sex, length of hospital stay, hemogram, biochemical parameters, and mortality rates, etc.

2.3. Ethical approval

Ethics approval of this study was obtained from the Non-Interventional Clinical Research Ethics Committee of Samsun Health Sciences University Samsun Training and Research Hospital with the protocol code GOKA/2021/8/6.

2.4. Statistical analysis

The sample size was not used in the study, and all patients who met the study criteria were included in the study.

The data analysis was performed with the SPSS 26 program. The Shapiro-Wilk normality test was used to determine the normal distribution. Parametric or non-parametric methods were used according to the normal distribution of the measurement.

Statistics were given as frequency (n , %) for categorical (qualitative) variables, and mean \pm standard deviation (mean \pm SD) or median (Q1, Q3) for numerical (quantitative) variables. The Chi-square test was used to analyze categorical variables. Mann-Whitney test or t -test was used for quantitative variables. Finally, the Spearman correlation test was used to analyze the correlation. The significant level was set at $\alpha=0.05$.

3. Results

The study groups consisted of 98 COVID-19 patients with the mutant virus and 98 patients without the mutant virus.

3.1. Demographic and clinical characteristics

There was a statistically significant difference in smoking, hypertension, cough, body pain between patients with and without mutation ($P<0.05$). There were more patients with coughing (63.3%), and pain (56.1%) in those with mutant viruses (Table 1).

3.2. Hospital stay, laboratory parameters, and mortality rates of mutant – and mutant + patients

The hospital stay, laboratory parameters and mortality rates are shown in the Table 2. No significant difference was found in the duration of hospitalization between the mutation and the non-mutation group ($P>0.05$). However, there was a statistically significant difference in hemoglobin, red cell distribution width-coefficient of variation, mean platelet volume, plateletcrit, iron binding capacity, potassium, calcium, C-reactive protein, folate, creatine kinase myocardial band, D-dimer, and international normalized ratio measurements ($P<0.05$). Hemoglobin, plateletcrit,

creatine kinase myocardial band, and international normalized ratio were significantly lower in those without the mutant virus. Red cell distribution width-coefficient of variation, mean platelet volume, iron binding capacity, C-reactive protein, folate, D-dimer were higher in patients with virus mutation.

3.3. Multivariate analysis of mutation-related factors

Multivariate analysis was performed based on the univariate analysis result. The established model was statistically significant ($\chi^2=38.192$; $P<0.05$; Nagelkerke $R^2=0.236$). According to the results

Table 1. Demographic and clinical characteristics (n, %).

Variables	Mutant – (n=98)	Mutant + (n=98)	Total (n=196)	χ^2	P
Sex					
Male	47 (47.9)	40 (40.8)	87 (44.4)	1.013	0.314
Female	51 (52.1)	58 (59.2)	109 (55.6)		
Age (year)					
<65	32 (32.7)	43 (43.9)	75 (38.3)	2.613	0.106
≥65	66 (67.3)	55 (56.1)	121 (61.7)		
Smoking					
No	98 (100)	89 (90.8)	187 (95.4)	7.453	0.006
Yes [#]	0 (0)	9 (9.2)	9 (4.6)		
Additional disease					
No	47 (47.9)	56 (57.1)	103 (52.6)	1.657	0.198
Yes	51 (52.1)	42 (42.9)	93 (47.4)		
Diabetes					
No	82 (83.7)	83 (84.7)	165 (84.2)	0.001	0.999
Yes	16 (16.3)	15 (15.3)	31 (15.8)		
Heart diseases					
No	85 (86.7)	93 (94.9)	178 (90.8)	2.998	0.083
Yes	13 (13.3)	5 (5.1)	18 (9.2)		
Asthma					
No	91 (92.9)	95 (96.9)	186 (94.9)	0.948	0.330
Yes	7 (7.1)	3 (3.1)	10 (5.1)		
Hypertension					
No	57 (58.2)	78 (79.6)	135 (68.9)	10.496	0.001
Yes	41 (41.8)	20 (20.4)	61 (31.1)		
COPD					
No	91 (92.9)	95 (96.9)	186 (94.9)	0.948	0.330
Yes	7 (7.1)	3 (3.1)	10 (5.1)		
Others diseases					
No	65 (66.3)	54 (55.1)	119 (60.7)	2.588	0.108
Yes	33 (33.7)	44 (44.9)	77 (39.3)		
Cough					
No	55 (56.1)	36 (36.7)	91 (46.4)	7.405	0.007
Yes	43 (43.9)	62 (63.3)	105 (53.6)		
Fever					
No	72 (73.5)	76 (77.6)	148 (75.5)	0.248	0.618
Yes	26 (26.5)	22 (22.4)	48 (24.5)		
Throat ache					
No	89 (90.8)	81 (82.7)	170 (86.7)	2.173	0.140
Yes	9 (9.2)	17 (17.3)	26 (13.3)		
Shortness of breath					
No	69 (70.4)	69 (70.4)	138 (70.4)	0.001	0.999
Yes	29 (29.6)	29 (29.6)	58 (29.6)		
Body pain					
No	62 (63.3)	43 (43.9)	105 (53.6)	7.405	0.007
Yes	36 (36.7)	55 (56.1)	91 (46.4)		
Weakness					
No	85 (86.7)	93 (94.9)	178 (90.8)	2.998	0.083
Yes	13 (13.3)	5 (5.1)	18 (9.2)		
Others symptoms					
No	51 (52.0)	77 (78.6)	128 (65.3)	15.222	<0.001
Yes	47 (48.0)	21 (21.4)	68 (34.7)		

Data were analyzed by Chi-square test. [#]Patients who smoke at least 1 cigarette a day and who have been smoking for at least one months. COPD: chronic obstructive pulmonary disease.

of the analysis, hypertension, body pain, and other complaints were found to be statistically significant risk factors of the mutant (-/+) status ($P<0.05$). Body pain was 1.887 times more common in patients with mutant (+). It was seen less frequently in mutant (+) patients with 74.9% of patients with hypertension and 65.7% in those with other complaints. Other factors were not statistically significant ($P>0.05$) (Table 3).

3.4. Correlation between different parameters and duration of hospitalization

As shown in Table 4, in the non-mutation group, there was a significant positive relationship between the duration of hospitalization and high density lipoprotein ($P<0.05$). In the mutation group, there was a significant positive relationship between the duration of hospitalization and the lymphocyte, glucose, phosphorus, and sedimentation ($P<0.05$).

Table 2. Comparison of measurements by mutant virus status.

Variables	Mutant- (n=98)	Mutant+ (n=98)	U/t/ χ^2	P
Duration of hospitalization (d, mean±SD)	8.91±4.60	7.82±3.46	4271.5	0.180
Death (n, %)				
No	89 (90.81)	93 (94.89)	0.692	0.405
Yes	9 (9.19)	5 (5.11)		
Disease severity (n, %)				
Mild	61 (62.24)	54 (55.10)	1.031	0.310
Severe	37 (37.76)	44 (44.90)		
WBC ($10^3/\mu\text{L}$, mean±SD)	5.66±2.05	6.05±2.29	4345.0	0.250
HGB (g/dL, mean±SD)	12.85±1.87	12.25±1.66	3789.5	0.011
PLT ($10^3/\mu\text{L}$, mean±SD)	195.88±63.77	193.89±69.84	4550.5	0.526
RDW-CV (% , mean±SD)	13.66±1.23	14.08±4.82	3760.5	0.009
LYM ($10^3/\mu\text{L}$, mean±SD)	1.12±0.51	1.16±0.61	4677.5	0.753
MCV (fL, mean±SD)	83.64±9.26	83.35±7.48	4306.5	0.212
MPV (fL, mean±SD)	10.04±1.20	10.42±1.00	-2.368	0.019
PCT (% , mean±SD)	0.31±0.16	0.20±0.08	2473.0	<0.001
ALP (U/L, mean±SD)	71.86±27.98	69.56±28.83	4489.0	0.430
GGT (U/L, median, Q1, Q3)	27.50 (18.00, 43.00)	26.00 (17.00, 42.00)	4681.0	0.760
LDH (U/L, mean±SD)	291.42±127.59	292.67±140.45	4738.5	0.873
AST (U/L, mean±SD)	30.47±13.54	33.37±18.28	4628.0	0.661
ALT (U/L, median, Q1, Q3)	20.00 (14.00, 28.00)	18.00 (14.00, 28.00)	4696.0	0.789
Iron ($\mu\text{g/dL}$, mean±SD)	40.07±24.48	33.07±17.94	4084.5	0.071
Iron binding capacity ($\mu\text{g/dL}$, mean±SD)	236.93±51.51	254.22±65.28	-2.059	0.041
Sodium (mmol/L, mean±SD)	136.26±4.34	135.95±3.89	4479.5	0.415
Potassium (mmol/L, mean±SD)	4.34±0.51	4.15±0.52	3791.5	0.011
Calcium (mg/dL, mean±SD)	8.63±0.83	8.09±0.64	2487.5	<0.001
Phosphorus (mg/dL, mean±SD)	3.43±0.60	3.39±0.62	0.423	0.673
ASO (IU/mL, median, Q1, Q3)	69.00 (40.0, 106.0)	76.50 (37.0, 112.0)	4645.0	0.693
CRP (mg/dL, median, Q1, Q3)	22.45 (6.40, 66.30)	53.05 (14.00, 98.80)	3852.5	0.017
RF (IU/mL, median, Q1, Q3)	20.00 (20.00, 20.00)	20.00 (20.00, 20.00)	4593.0	0.205
HbA1C (% , mean±SD)	6.47±1.35	6.84±1.80	4026.5	0.051
Free T4 (pmol/L, mean±SD)	1.17±0.21	1.11±0.21	4132.0	0.091
TSH (mIU/L, median, Q1, Q3)	1.29 (0.79, 2.08)	1.14 (0.67, 1.97)	4445.0	0.369
Ferritin ($\mu\text{g/dL}$, median, Q1, Q3)	135.30 (58.70, 229.80)	142.10 (72.50, 319.00)	4426.0	0.344
Vitamin B-12 (ng/dL, median, Q1, Q3)	404.00 (303.00, 530.00)	370.50 (287.00, 530.00)	4327.0	0.232
Folate ($\mu\text{g/dL}$, mean±SD)	12.26±4.35	15.62±5.16	2943.0	<0.001
Sedimentation (min, mean±SD)	43.96±26.94	46.36±23.15	4116.0	0.296
Troponin I (ng/L, median, Q1, Q3)	0.01 (0.00, 0.02)	0.01 (0.00, 0.02)	4320.0	0.524
CK-MB (U/L, median, Q1, Q3)	1.27 (0.84, 2.05)	0.82 (0.39, 2.21)	3537.5	0.008
D-dimer ($\mu\text{g/L}$ FEU, Median, Q1, Q3)	0.67 (0.51, 1.31)	0.88 (0.60, 1.68)	3959.5	0.044
INR (s, mean±SD)	1.11±0.20	1.08±0.25	3951.0	0.042
Vitamin D ($\mu\text{g/dL}$, median, Q1, Q3)	18.36 (11.45, 25.93)	14.52 (11.06, 22.62)	4052.0	0.095
Insulin (mIU/L, median, Q1, Q3)	11.55 (7.07, 21.24)	9.24 (5.82, 19.20)	3474.5	0.083
C-peptide ($\mu\text{g/dL}$, median, Q1, Q3)	4.36 (2.96, 5.93)	3.83 (2.40, 6.39)	4475.0	0.558

WBC: white blood cell; Hgb: hemoglobin; PLT: platelet; RDW-CV: red cell distribution width-coefficient of variation; LYM: lymphocyte; MCV: mean corpuscular volume; MPV: mean platelet volume; PCT: plateletcrit; ALP: alkaline phosphatase; GGT: gamma glutamyl transferase; LDH: lactate dehydrogenase; AST: aspartate aminotransferase; ALT: alanine aminotransferase; ASO: antistreptolysin O; CRP: C-reactive protein; RF: Rheumatoid factor; TSH: thyroid stimulating hormone; CK-MB: creatine kinase myocardial band; INR: international normalized ratio.

Table 3. Multivariate analysis of mutation-related factors.

Variables	B	sh.	Odds ratio	χ^2	P
Sex (Female)	0.231	0.322	1.260	0.265	0.473
Age (≥ 65 years)	-0.280	0.358	0.756	0.372	0.435
Smoke (Yes)	-0.881	0.707	0.414	2.414	0.213
Disease severity (Severe)	0.590	0.367	1.803	6.661	0.108
Additional disease (Yes)	0.627	0.465	1.871	3.284	0.178
Hypertension (Yes)	-1.381	0.512	0.251	52.727	0.007
Cough (Yes)	0.087	0.366	1.091	0.003	0.812
Body pain (Yes)	0.635	0.324	1.887	14.783	0.049
Other complaints (Yes)	-1.071	0.383	0.343	61.498	0.005
Length of stay in hospital	-0.056	0.042	0.946	3.122	0.184

Table 4. Correlation between parameters and duration of hospitalization.

Parameters	Mutation-		Mutation+	
	r	P	r	P
WBC	0.010	0.924	0.044	0.670
HGB	-0.039	0.703	-0.091	0.372
PLT	-0.079	0.438	0.105	0.305
RDW-CV	0.002	0.981	0.022	0.833
LYM	-0.022	0.827	0.227	0.025*
Gran/Neut	0.017	0.868	-0.017	0.867
MCV	0.001	0.995	-0.164	0.107
MPV	0.133	0.192	-0.144	0.156
PCT	-0.080	0.432	0.051	0.619
Monocyte	0.065	0.525	0.123	0.229
Fasting glucose	0.190	0.060	0.253	0.012*
Urea	-0.069	0.500	0.019	0.855
Creatine	-0.093	0.365	0.038	0.711
ALP	0.151	0.138	0.127	0.214
GGT	-0.092	0.370	0.076	0.459
LDH	0.017	0.866	-0.098	0.338
AST	-0.106	0.299	-0.066	0.519
ALT	-0.146	0.151	0.034	0.736
Albumin	0.098	0.336	0.151	0.138
Protein	0.129	0.205	0.136	0.183
HDL	0.273	0.006*	0.089	0.384
LDL	0.069	0.497	-0.040	0.699
Triglyceride	-0.172	0.090	0.081	0.429
Iron	-0.141	0.166	-0.070	0.493
Iron binding capacity	0.097	0.342	0.095	0.351
Sodium	-0.099	0.334	-0.034	0.741
Potassium	0.112	0.273	0.087	0.392
Calcium	0.045	0.663	0.150	0.141
Phosphorus	0.011	0.911	0.270	0.007*
ASO	0.032	0.757	0.061	0.551
CRP	-0.061	0.553	0.011	0.912
RF	-0.090	0.377	-0.052	0.613
HbA1C	0.062	0.546	0.170	0.095
Free T4	-0.049	0.630	0.068	0.506
TSH	-0.065	0.523	-0.032	0.757
Ferritin	-0.045	0.657	-0.101	0.322
Vitamin B-12	-0.073	0.474	0.000	0.999
Folate	-0.012	0.909	-0.101	0.322
ESR	0.065	0.531	0.297	0.004*
Troponin I	-0.089	0.397	-0.079	0.440
CK-MB	0.056	0.592	0.104	0.307
D-Dimer	-0.100	0.331	-0.057	0.578
INR	-0.083	0.417	0.161	0.114
Vitamin D	0.089	0.386	-0.091	0.376
Insulin	0.091	0.407	0.130	0.210
C-peptide	-0.095	0.352	0.066	0.523

HDL: high density lipoprotein; LDL: low density lipoprotein; ESR: erythrocyte sedimentation rate. * $P < 0.05$.

4. Discussion

One of the most important SARS-CoV-2 variants is B.1.1.7, first identified in October 2020 in the UK. However, the origin of this variant may be in another country[8]. Variant B.1.1.7 is important because it creates many mutations at the same time. Leung *et al.* speculate that the mutations may have originated in an immunocompromised patient who has been infected for a long time[9]. Volz *et al.* found that it is likely that only a small fraction of these changes would have reached the evolutionary benefits of these variants[10].

The present study investigates the clinical symptoms of COVID-19 patients with and without B.1.1.7 mutation. The results did not find any significant differences in mortality rate, disease severity, or duration of hospitalization between the two study groups.

Concerns about increased mortality of B.1.1.7 variant were first announced when the British Government's New and Emerging Respiratory Virus Threats Advisory Group showed an increase in mortality compared to wild-type strains[4]. In January 2021, Rambaut *et al.* reported evidence that type B.1.1.7 may be associated with an increased risk of death and transferability than other types[11]. Initial reports did not provide evidence that the mutation affected vaccine efficiency[12] and suggest that it is probably more deadly, leading to more hospitalizations than wild-type strains of the virus[13]. Davies *et al.* adapted data from studies by several institutes and found an increased severity of COVID-19 cases compared to other types[14]. Another study found that B.1.1.7 cases were 30% to 70% more lethal than the original wild-type[15].

Several other studies have examined the effect of type B.1.1.7 on hospitalization. In particular, a study used the S-gene target failure as a marker to identify B.1.1.7 variants of SARS-CoV-2. They noted that the risk of hospitalization is higher among cases of gene target failure compared with positive cases of the gene[16]. In addition, data from the Intensive Care National Audit and Research Centre also revealed a higher risk of ICU acceptance for this type of virus than for other types[17].

In this study, we did not see any difference in mortality rates between the B.1.1.7 mutation and non-mutation groups. However, some studies have different results from the findings of our study. For example, a review of 12 studies in the United Kingdom on this variant showed that mortality in patients with this variant is reported to be 36% to 71% higher[4]. The difference could be due to the small sample size, as mortality due to this variant has been analyzed in only 8% of all COVID-19 deaths in the UK[10].

A case-control study in the United Kingdom with 54 906 participants from October 1, 2020, to January 29, 2021 reported that 28 days after the test, the risk ratio for mortality in patients with variant B.1.1.7 was 1.64 compared to patients with other variants (95% confidence interval: 1.32-2.04)[12]. In addition, a study in Denmark showed that the hospitalization rate for people with this variant was 64% higher[18].

A study by Brookman *et al.* comparing the first and second waves of COVID-19 in the United Kingdom, coincided with the outbreak of variant B.1.1.7, showed that in the second wave, the number of children and young people admitted to the hospital increased[8]. Therefore, it could be due to the prevalence of variant B.1.1.7 in the second wave. However, there was no evidence of more severe disease in children and adolescents in the second wave, and the authors ultimately did not suggest a different treatment from the first wave for children in the second wave.

As a result, the transmission rate and the mortality of variant B.1.1.7 are shown to be higher than the non-mutated variants. However, not all studies agree that this type is prone to be more severe and an increased risk of death. For example, the COVID-19 Clinical Information Network data showed no correlation between different variants and higher hospital mortality[2]. Our result is consistent with these findings.

The Office for National Statistics analysis noted that while the risk ratio indicates a higher risk of all-cause mortality, they found that the number of deaths is too low for reliable inference, and there are potential limitations to all datasets used. However, these analyses suggest that the B.1.1.7 variant may be associated with an increased risk of hospitalization and death than infection with other variants[19]. Some studies believe that B.1.1.7 has not been conclusively proven to be more deadly[20,21]. It is unknown whether this increased infectivity is due to N501Y alone or a combination of other mutations in the spike protein[22]. Others believe that despite initial concerns, there is no real reason that this type of infection is more common in children than in the original variant[23].

One of the limitations of this study is the small sample size. Small sample size can be one of the reasons for conflicting results in studies regarding mortality rate, duration of hospitalization, and disease severity. Therefore, future studies should be performed with larger sample sizes and data from multiple treatment centers so that results can be inferred with greater confidence.

In conclusion, the present study compared the clinical symptoms between COVID-19 patients with and without B.1.1.7 mutation and did not find any significant difference in mortality rate, disease severity, or duration of hospitalization between the two groups. However, some biochemical parameters were significantly different between the two groups, which can be used as preliminary findings for future studies.

Conflict of interest statement

The authors declare no conflict of interest.

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Authors' contributions

HE and ZE collected and curated the data; HE, ZE, AÖ, GK, ÖKO, and Öİ contributed to writing the manuscript. All authors read and approved the final manuscript.

References

- [1] Davies NG, Abbott S, Barnard RC, Jarvis CI, Kucharski AJ, Munday JD, et al. Estimated transmissibility and impact of SARS-CoV-2 lineage B.1.1.7 in England. *Science* 2021; **372**(6538): eabg3055.
- [2] Ontario Agency for Health Protection and Promotion (Public Health Ontario). *COVID-19 B. 1.1. 7 (501Y. V1) variant of concern—What we know so far*. Toronto, ON: Queen's Printer for Ontario; 2021.
- [3] Ferguson N. *Non-parametric analysis of fatal outcomes associated with B1.1.7*. Imperial College London; 2021. [Online] Available from: https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/955239/NERVTAG_paper_on_variant_of_concern_VOC_B.1.1.7.pdf. [Access on 30 August 2022].
- [4] Horby P, Huntley C, Davies N, Edmunds J, Ferguson N, Medley G, et al. *NERVTAG paper on COVID-19 variant of concern B.1.1.7*. London: Crown copyright; 2021. [Online] Available from: <https://www.gov.uk/government/publications/nervtag-paper-on-covid-19-variant-of-concern-b117> [Access on 30 August 2022].
- [5] Galloway SE, Paul P, MacCannell DR, Johansson MA, Brooks JT, MacNeil A, et al. Emergence of SARS-CoV-2 B.1.1.7 Lineage - United States, December 29, 2020-January 12, 2021. *Morb Mortal Wkly Rep* 2021; **70**(3): 95-99.
- [6] Graham MS, Sudre CH, May A, Antonelli M, Murray B, Varsavsky T, et al. Changes in symptomatology, reinfection, and transmissibility associated with the SARS-CoV-2 variant B.1.1.7: An ecological study. *Lancet Public Health* 2021; **6**(5): e335-e345.
- [7] Jewell BL. Monitoring differences between the SARS-CoV-2 B.1.1.7 variant and other lineages. *Lancet Public Health* 2021; **6**(5): e267-e268.
- [8] Brookman S, Cook J, Zucherman M, Broughton S, Harman K, Gupta A. Effect of the new SARS-CoV-2 variant B.1.1.7 on children and young people. *Lancet Child Adolesc Health* 2021; **5**(4): e9-e10.
- [9] Leung K, Shum MH, Leung GM, Lam TT, Wu JT. Early transmissibility assessment of the N501Y mutant strains of SARS-CoV-2 in the United Kingdom, October to November 2020. *Euro Surveill* 2021; **26**(1): 2002106.
- [10] Volz E, Mishra S, Chand M, Barrett JC, Johnson R, Geidelberg L, et al. Assessing transmissibility of SARS-CoV-2 lineage B.1.1.7 in England. *Nature* 2021; **593**(7858): 266-269.
- [11] Rambaut A, Loman N, Pybus O, Barclay W, Barrett J, Carabelli A, et al. Preliminary genomic characterisation of an emergent SARS-CoV-2 lineage in the UK defined by a novel set of spike mutations, 18 December 2020. [Online] Available from: <https://virological.org/t/preliminary-genomic-characterisation-of-an-emergentsars-cov-2-lineage-in-the-uk-defined-by-a-novel-set-of-spike-mutations/563> [Access on 7 February 2021].

- [12]Challen R, Brooks-Pollock E, Read JM, Dyson L, Tsaneva-Atanasova K, Danon L. Risk of mortality in patients infected with SARS-CoV-2 variant of concern 202012/1: Matched cohort study. *BMJ* 2021; **372**: n579.
- [13]Iacobucci G. Covid-19: New UK variant may be linked to increased death rate, early data indicate. *BMJ* 2021; **372**: n230.
- [14]Davies N, Diaz-Ordaz K, Keogh R. Relative fatality hazard in Pillar 2 tested individuals with VOC. LSHTM-unpublished analysis. 2021. [Online] Available from: <https://www.gov.uk/government/publications/nervtag-update-note-on-b117-severity-11-february-2021/nervtag-update-note-on-b117-severity-11-february-2021> [Access on 30 August 2022].
- [15]Weissman D, Alameh MG, de Silva T, Collini P, Hornsby H, Brown R. et al. D614G spike mutation increases SARS CoV-2 susceptibility to neutralization. *Cell Host Microbe* 2021; **29**(1): 23-31.e4.
- [16]Brown KA, Gubbay J, Hopkins J, Patel S, Buchan SA, Daneman N. et al. S-gene target failure as a marker of variant B.1.1.7 among SARS-CoV-2 isolates in the greater Toronto area, December 2020 to March 2021. *JAMA* 2021; **325**(20): 2115-2116.
- [17]Care I. National Audit and Research Centre (ICNARC): ICNARC report on COVID-19 in critical care: April 24 2020. 2020. [Online] Available from: <https://ripetomato2uk.files.wordpress.com/2020/03/icnarc-covid-19-report-2020-04-24.pdf> [Access on 30 August 2022].
- [18]Bager P, Wohlfahrt J, Fonager J, Rasmussen M, Albertsen M, Michaelsen TY. et al. Risk of hospitalisation associated with infection with SARS-CoV-2 lineage B.1.1.7 in Denmark: An observational cohort study. *Lancet Infect Dis* 2021; **21**(11): 1507-1517.
- [19]Frampton D, Rampling T, Cross A, Bailey H, Heaney J, Byott M. et al. Genomic characteristics and clinical effect of the emergent SARS-CoV-2 B.1.1.7 lineage in London, UK: A whole-genome sequencing and hospital-based cohort study. *Lancet Infect Dis* 2021; **21**(9): 1246-1256.
- [20]Ackland JA, Ackland GJ, Wallace DJ. Evolution of case fatality rates in the second wave of coronavirus in England: Effects of false positives, a Variant of Concern and vaccination. *medRxiv* 2021.
- [21]Grint DJ, Wing K, Williamson E, McDonald HI, Bhaskaran K, Evans D, et al. Case fatality risk of the SARS-CoV-2 variant of concern B.1.1.7 in England, 16 November to 5 February. *Euro Surveill* 2021; **26**(11): 2100256.
- [22]Public Health England (PHE). Investigation of novel SARS-CoV-2 variant: Variant of concern(England)- Technical briefing 19, 23 July 2021. [Online] Available from: <https://www.gov.uk/government/publications/phe-investigation-of-novel-sars-cov-2-variants-of-concern-england-technical-briefing-19-23-july-2021> [Access on 30 August 2022].
- [23]Docherty A, Harrison E, Semple C, CO-CIN. Hospital case fatality and emergence of variant of concern B. 1.1. 7, rapid CO-CIN report to NERVTAG and SAGE. Unpublished analysis. 2021. [Online] Available from: chrome-extension://efaidnbmnnnibpcajpcglclefindmkaj/https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/961044/s1043-co-cin-voc-b117.pdf [Access on 30 August 2022].

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