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Covid -19 Associated Ischemic and Haemorrhagic stroke

Amira A.EL-Sebaey, Ahmed H.Abd-Elrahman, Ehab E.Afifi and Fatma A.AbdelFattah
Anesthesia , surgical intensive care and pain management Dept., Faculty of Medicine, Benha University
E-mail: rmvip2006@gmail.com

Abstract
Background: The The novel coronavirus from the Coronaviridae family, which was promptly named severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), was swiftly identified as the causative agent of this unusual sickness. The purpose of this research was to examine the typical stroke processes in order to assess the short-term outcomes of individuals who had a stroke linked with SARS-CoV-2. Approach: One hundred individuals hospitalized with acute stroke at Banha University Hospital were part in this observational research. One subset of the study's participants was the patients: A total of 38 patients with acute stroke and a positive SARS COV-2 PCR nasal sample were included in the study. The control group consisted of 62 patients with acute stroke who did not have a positive SARS COV-2 PCR sample. Findings: The patient group required considerably more low flow oxygen (p value < 0.001). There were notable disparities in the lymphocytic count (p value < 0.001), hemoglobin level (p value < 0.001), D dimer (p value < 0.001), and CRP (p value < 0.001) between the sick group and the control group. Researchers found that stroke patients with COVID-19 had lower total leukocytic counts, lymphocytic counts, and hemoglobin levels compared to non-covid patients, and higher mean values for platelet counts, D-dimer, and C-reactive protein. There was no statistically significant difference between the two groups for the demand of high flow oxygen, CPAP, or mechanical ventilation; however, the patient group had a considerably larger need for low flow oxygen, according to the supportive therapy.
Covid-19, arterial disease, hemorrhagic stroke, SARS-CoV-2 are all terms that need to be defined.

Keywords : CPAP, SARS COV-2, PCR

Introduction

A cluster of severe, unusual respiratory illnesses struck the Chinese city of Wuhan in December 2019 [1]

Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) was the name given to the novel coronavirus that caused this unusual sickness after its rapid identification as a member of the Coronaviridae family [2].

Researchers found that it was very similar to the coronavirus that caused the 2002–2003 respiratory epidemic, SARS. COVID-19 has spread quickly around the globe, and there is mounting evidence that SARS-CoV-2 may infect the brain and cause neurological symptoms [3].

A novel risk factor for acute ischemic stroke (AIS) may be COVID-19, which has been around for over 20 months and has been associated with SARS-CoV-2 infection and stroke [4].

The risk of stroke is 7.6 times higher in those infected with COVID-19 compared to those infected with influenza. Patients who already have cardiovascular risk factors and a severe SARS-CoV-2 infection are at a greater risk [5].

Hypertension, diabetes, and dyslipidemia occur together. The elderly are more likely to have moderate-severity AIS in COVID-19. The infection is most frequent in the anterior circulation, and up to 63.6% of cases are cryptogenic. Between 40.9% and 60% of cases, major artery blockage is present. The median time between the start of infection symptoms and stroke is 10 days (plus or minus 8 days) [6].

Nevertheless, persons experiencing symptoms, those under the age of 50, and those in the convalescent phase are all at increased risk. 3 AIS is known to increase mortality and the occurrence of acute respiratory distress syndrome (ARDS) in COVID-19 infections [7].

Neurologic symptoms were recorded in 36.4% of COVID-19 patients in 2014 according to the Wuhan search [8].

(Cascella et al., 2021) More and more research is showing that COVID-19 individuals may have neurological symptoms, which may range from moderate to severe, in addition to the usual respiratory symptoms including fever and dry cough. An independent risk factor for severe instances of COVID-19 infection has been indicated by previous investigations to be cerebrovascular illness [9].

As a result of the COVID-19 pandemic, stroke centers have been hit hard, and the number of

stroke or transient ischemic attack (TIA) patients seeking emergency care has dropped by more than 30% globally. This could have an effect on the prognosis of these patients [10].

Worse, if the patient remains in isolation during the epidemic and does not seek medical attention quickly enough, they run the danger of missing the therapeutic window. That is why, in light of the current COVID-19 epidemic, new protocols for the treatment of stroke victims are critically required [11].

Intraocular inflammatory infiltrates and edema manifest as ground-glass opacities on computed tomographic imaging. In the early stages of acute respiratory distress syndrome (ARDS), pulmonary edema fills the spaces between the alveoli and a hyaline membrane forms [12].

One possible component of the illness is bradykinin-induced lung angioedema. Disruption of the endothelial barrier, impaired oxygen transfer between alveoli and capillaries, and decreased oxygen diffusion capacity are characteristics of COVID-19 [13].

Coagulant activation and consumption of clotting factors happen in severe cases of COVID-19. A total of 183 COVID-19 infected individuals who passed away in Wuhan, China, had symptoms consistent with diffuse intravascular coagulation, according to a survey [14]. Thrombotic complications, including deep venous thrombosis, pulmonary embolism, and thrombotic arterial complications (such as limb ischemia, ischemic stroke, and myocardial infarction), are common in critically ill patients, and micro thrombi can form in inflamed lung tissue and pulmonary endothelial cells [9] Viral sepsis, characterized as potentially fatal organ dysfunction caused by an uncontrolled host reaction to infection, may worsen preexisting conditions such as multi-organ failure [1].

This research was aimed to assess the short-term results of individuals who had a stroke related to SARS-CoV-2 using the conventional stroke mechanisms.

Patients and methods

One hundred patients hospitalized to Banha University Hospital with acute stroke were part of this observational research. Research conducted between July 2022 and April 2024. NO, MS.15.6.2022, the ethics committee's consent was obtained before this was done.

All patients have to be at least 18 years old and had tested positive for the COVID-19 virus using PCR.

All patients were ruled out due to the presence of cardiogenic thrombosis, atherosclerosis, or vasculitis.

Grouping: The research groups were further split into: Included in the patient group were 38 individuals with acute stroke and a positive SARS COV-2 PCR nasal sample. In the control group, sixty-two patients with acute stroke and a negative SARS COV-2 PCR sample were included.

The results:

The major goal is to investigate the effects of COVID-19 on stroke patients, including the proportion of fatalities and the extent to which they increase mortality.

As a byproduct, we want to learn more about the characteristics of a full blood count, such as hemoglobin level, platelet count, total leukocyte count, and lymphocytic count. Smoking, age, sex, D-dimer, and prostate cancer risk No, I don't. Stroke history, History of TiA. The treatment-related oxygen requirements, include low-flow and high-flow oxygen, CPAP, and mechanical breathing pulmonary edema, infection, myocardial infarction, acute kidney injury, Possible complications include aspiration pneumonia, stroke, and fits. dyspnea, coronary thromboembolic history, disease of the small and big arteries....

What is the process for doing a COVID-19 pcr swap?

Collecting the sample: a medical professional inserted a swab into the nose to gather secretions from the respiratory system. The swab is a long, flexible stick with a delicate tip that is inserted into the nose. Nostrils and the nasopharynx were among the places from which the samples were taken. We sealed the tube after collecting the swab and submitted it to the lab.

After receiving the sample, the laboratory extracted the COVID-19 viral genome from the patient's respiratory secretions.

Polymerase Chain Reaction (PCR): A heat cycler, together with specialized reagents and enzymes, were used. In order to enhance the quantity of the targeted genetic material in the test tube, heating and cooling cycles were used. Multiple cycles later, the test tube had millions of copies of a little fragment of SARS-COV2 DNA. If SARS-CoV-2 was present in the sample, one of the compounds in the tube would create fluorescent light. After the SARS-CoV-2 genetic material has been amplified to a sufficient level, the system will be able to detect fluorescent signals. The signal was interpreted as a positive test result using specialized software.

Validation number:
Evaluation using statistical methods
Mean ± Standard Deviation (SD) and range were used to represent numerical data, whereas number and percentage were used for categorical data. The patient group's COVID-19 cases and the control group's non-COVID-19 cases were compared using the relevant statistical tests, including the Chi-square test (X2), the Fisher Exact Test (FET), the Student t-test (t), and the Mann Whitney test (z). A p-value less than 0.05 was deemed statistically significant. All statistical analysis was conducted using STATA/SE version 11.2 for Windows and MS Excel (STATA Corporation, College Station, Texas). It was deemed statistically significant if the two-tailed P value was less than 0.05.

Results

When looking at age, sex, and body mass index, there are no discernible variations between the two groups. When comparing the two groups for smoking, diabetes, hypertension, TIA, or stroke, no statistically significant differences were found. The patient group showed statistically significant differences from the control group in the following areas: total leukocytic count ($p = 0.0006$), lymphocytic count ($p < 0.001$), hemoglobin level ($p < 0.001$), platelet count ($p < 0.001$), D dimer ($p < 0.001$), and CRP ($p < 0.001$). In Table 2, it can be shown that the patient group had a considerably larger need for low flow oxygen (p value < 0.001), but when it came to high flow oxygen, CPAP, and mechanical ventilation, there was no statistically significant difference between the two groups. According to Table 3, the patient group had significantly higher rates of dyspnea (p value< 0.001), respiratory failure (p value= 0.02), and fits (p value = 0.006). However, there were no significant differences between the two groups in terms of aspiration pneumonia, sepsis, myocardial infarction, pulmonary embolism, cardioembolic complications, acute kidney injury, mortality, small or large arterial atherosclerosis, or cardioembolic complications.
Chapter 4

Discussion

Our analysis of 100 stroke cases reveals that there were 38 cases of COVID-19 and 62 cases of non-COVID-19.Recent COVID-19 events have shown how critical it is to assess the pandemic's effect on healthcare systems, and a number of studies have investigated the link between the virus and neurological symptoms [15].

More research on how the current epidemic is affecting stroke outcomes is required.The current prospective study aimed to assess the short-term effects of COVID-19 on stroke patients in Egypt and identify factors that could explain these effects. Stroke survivors fared worse than those without the virus, and the study found that hospital mortality rates were similar for the two groups, with a small increase in the COVID group. This could be because COVID patients are more likely to experience respiratory failure and hypoxia-induced ischemia as a result of severe pulmonary illness [11].Stroke consequences are worsened by this factor, leading to more neurological deficits and worse outcomes.The results were consistent with those of Mart Fabrigas's 2021 paper [8].

Our research found that COVID-19 patients had an increased risk of stroke-related complications, including fits, which are an indication of the severity of the stroke and lead to more negative outcomes. For their 2020 study, Roushdy and colleagues examined 93 individuals from Egypt who had suffered an acute ischemic stroke [17]. They discovered that some patients did not get timely stroke therapy due to healthcare resource shortages and fears of developing COVID-19 [18]. Concerning this matter, Aref and colleagues in 2021 found that during the COVID-19 period, the leading reasons of delayed arrival for stroke were concerns about infection and lockdown procedures [11].

It was noted that oxygen dependence was highly essential for both the COVID-19 group and the non-COVID-19 group when they examined the outcomes of patients who had acute strokes and were treated with the same treatment protocols (1).in the COVID-19 cohort, where low-flow oxygen is more essential.

We found that compared to the control group, the COVID-19 group experiencing a stroke had lower haemoglobin level, TLC, and lymphocytic count. However, when we compared the two groups, we found that the COVID-19 group had a higher contrast in D dimer [19].Alternatively, a greater percentage of cases in the UK multicentric case control study had D dimer measured, suggesting that the test criteria used to identify patients with higher D dimer may have been biased toward the control group's higher D dimer levels [20]. as opposed to those who stayed at home due to milder symptoms or treatment limitations. The overall incidence of stroke in COVID-19 patients may have been underestimated because of this.Furthermore, our retrospective analysis included data from a

registry that was established to identify cardiac and thromboembolic complications in COVID-19 patients.

Conclusion

Results from studies comparing stroke patients with and without covid showed that the former had much lower total leukocytic counts, lymphocytic counts, and hemoglobin levels, and much higher mean values for platelet counts, D Dimer, and CRP. There was no statistically significant difference between the two groups for the demand of high flow oxygen, CPAP, or mechanical ventilation; however, the patient group had a considerably larger need for low flow oxygen, according to the supportive therapy. Covid patients had a much greater incidence of dyspnea, respiratory failure, and fits compared to non-covid patients, according to complications.

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Contribution of the author

All writers had equal roles in writing the research.

Potential biases

There are no potential biases

Table (1) Sociodemographic and clinical data comparisons between COVID patients and non-COVID patients

Variable		**Non-COVID-19 (The control group) (n.=62)**		**COVID-19 (Thepatient group) (n.=38)**		**P**
		No.	**%**	**No.**	**%**	
Sex	Female	24	38.71	14	36.84	0.85
	Male	38	61.29	24	63.16	
Age (years)		68.74±7.22		67.53±5.80		0.38
BMI (kg/m^2)	Underweight (BMI<18.5)	15	24.19	8	21.05	0.46
	Normal range (BMI=18.5-24.9)	21	33.87	12	31.58	
	Overweight (BMI=25.0-29.9)	6	9.68	8	21.05	
	Obese (BMI=≥30.0)	20	32.26	10	26.32	
	Mean ± SD	24.84±7.50		24.1±5.94		0.86

Table(2) Lab data comparisons between individuals with COVID-19 and those without

Variable	**Non-COVID-19 (The control group) (n.=62)**	**COVID-19 (The patient group) (n.=38)**	**P**
TLC	11446.91±1508.9	9585.26±1878.45	0.0006*
Lymphocytic count	1444.72±243.76	1338.68±2069.89	<0.001*
Hb (mg/Ldl)	12613.58±1088.89	11463.16±1552.13	<0.001*
Platelet count	329172.8±301390.6	364210.5±28346.23	<0.001*
D-dimer	624.44±115.04	1397.89±459.03	<0.001*
CRP	38.86±41.1	82.84±10.27	<0.001*

P value <0.05 indicates statistical significance.

Comparisons between COVID-19 patients and those without the virus are shown in Table 3.

Variable	Non-COVID-19 (The control group) (n.=62)		COVID-19 (The patient group) (n.=38)		P
	No.	%	No.	%	
Room air	54	87.10	16	42.11	<0.001*
Low Fl. O2	3	4.84	12	31.58	<0.001*
High Fl. O2	2	3.23	2	5.26	0.63
CPAP	1	1.61	2	5.26	0.56
Mec. Ven.	2	3.23	4	10.53	0.20

P value <0.05 indicates statistical significance.

Table (4) shows the following comparisons between covid and non-covid patients with respect to complications:

Variable	Non-COVID-19 (Control group)		COVID-19 (Patient group)		Test	P
	No	%	No	%		
Hospital death	5	8.06	8	21.05	FET	0.07
Respiratory Failure	0	0.0	4	10.53	FET	**0.02**
Sepsis	1	1.61	0	(0.0	FET	1.00
Myocardial Infarction	1	1.61	0	0.0	FET	1.00
Pulmonary Embolism	1	1.61	2	5.26	FET	0.56
Acute Kidney Injury	0	0.0	2	5.26	FET	0.14
Aspiration Pneumonia	1	1.61	0	(0.0	FET	1.00
Stroke onset hours	5.06±1.2 3-7		5.5±1.33 3-8		t=1.71	0.09
Fits	3	3.23	8	21.05	FET	**0.006**
Small artery	15	24.19	10	26.32	0.06	0.81
Large artery	15	24.19	12	31.58	0.65	0.42
Cardioembolic event	1	1.61	4	10.53	FET	0.07
Dyspnea	0	0.0	38	100.0	100.00	**<0.001**

Statistically significant when the P value is less than 0.05.

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