The Egyptian Journal of Hospital Medicine (October 2022) Vol. 89 (2), Page 7378- 7382

The Relation between Prehospital Usage of Aspirin to D-Dimer and Radiological Changes in COVD-19 Patients in Wasit Province

Hussein Adnan Mohammed, Ahmed Basheer, Usama A. Al-Sari

Department of Internal medicine / Wasit University, Iraq.

***Corresponding author**: Hussein Adnan Mohammed, **Mobile**: +9647810451024,

Email: haliqabi@uowasit.edu.iq, **ORCID**: https://orcid.org/0000-0001-9766-4919

ABSTRACT

Background: Coronaviruses are known to cause various diseases. One of them is SARS-CoV-2 which was first reported in 2019. The main risk factor for severe disease is the disorder of coagulation, which can usually happen during the course of the illness.

Patients and Method: Adult COVID-19 patients admitted to Al-Zahra teaching hospital in Wasit province between February and July 2020 were studied in different sections of the hospital in cross-sectional, observational cohort research. In the current study. 79 patients were involved, 36 patients previously used aspirin and 43 patients did not.

Results: There was a significant relationship between usage of aspirin and the CT scan changes in the lung (P = .005) in aspirin-taken group, while no significant correlation with the D-dimer level (P = >.05)

Conclusions: In hospitalized COVID-19 patients, using aspirin before infection may reduce severe outcomes. So a larger randomized controlled research is needed to establish if there is a link between past aspirin use and decreased lung damage and death in COVID-19 individuals.

Keywords: Aspirin, D-dimer, COVD-19, Wasit province**,** Radiological changes.

INTRODUCTION

Coronaviruses are critical human pathogens known to cause severe respiratory infections in children and adults. They're members of the Nidovirus group and are capable of replicating using a set of mRNAs. The incidence of upper respiratory infections as a result of this pathogen may reach one third of respiratory infections in the community [1, 2].

In addition to its health impact, as it infects humans and other mammals, it infects birds, livestock, and accompanying animals and poses an economic and veterinary danger [2]. The Corona viridae family belong to the Nidovirales order and the Corona virineae suborder of the Nidovirales. Alpha corona virus, beta corona virus, gamma corona virus, and delta corona virus are all members of the Ortho corona virinae subfamily, there is a discrepancy in the spectrum of infection with the types of corona viruses, where the alpha and beta corona viruses infect only mammals, while the gamma corona viruses and delta corona viruses infect a wider group of species. Coronavirus infections mostly cause respiratory and gastrointestinal diseases in people and animals [3]. In November 2002, SARS cases were first reported in Guangdong Province, China. 792 cases were recorded in this province between November 16, 2002, and February 28, 2003 [4, 5]. The outbreak seems to have disproportionately impacted health care personnel and their connections.

COVID-related mortality is mostly linked to hypercoagulability and an increased risk of venous thromboembolism (VTE) events, which can result in thrombo-inflammation in extreme cases [6].

The sickness showed numerous epidemiologic and clinical signs of infection, and early efforts to identify an associated culprit immediately focused on a novel coronavirus strain discovered in February and March 2003 [7-9].

Most illnesses occurred in adults, however, 12 years children or older had a clinical presentation similar to adults, whereas younger children had milder disease [10, 11].

The possibility of transmission of the Corona virus by droplets is possible, according to what has been shown from the transmission of the disease from the infected person to the healthy person through face-to-face contact, according to studies in Hong Kong and Canada [12, 13]. Due to the fast and widespread spread of SARS in Hong Kong, it has been speculated that alternative routes of transmission, such as faecal-oral or airborne, may be feasible [14, 15].

Peak virus shedding in respiratory secretions occurs 6 to 11 days after the onset of the infection, when severe respiratory symptoms are evident, as revealed by PCR [16, 17]. When compared to other respiratory viral illnesses, SARS has a peculiar late viral excretion peak. Symptoms associated with COVID-19 are very wide, as shown by examination of patients and the history of the disease, ranging from minor pain to serious complications. The period of appearance of the first symptom of the disease may appear within four days and may extend to 14 days [18. Symptoms of illness are characterized by fever (temperature >100.5°F [>38°C]) may last 3-7 days and may be accompanied by headache, malaise, and muscle aches [19].

Received: 15/7/2022
Accepted: 18/9/2022

Disease symptoms and diagnostic methods are used to identify the type of infection with the SARS-CoV-2 virus that causes COVID-19. Early diagnosis of the virus is often done by real-time PCR testing of respiratory samples, as work began in February 2020. A test that provides three reactions in one well was approved by the World Health Laboratories in July 2020 presented by the Center for Disease Control (CDC) and is called the SARS-CoV-2 (Flu SC2) Multiplex Influenza Assay [(20)].
The study aimed to evaluate whether previous aspirin usage is associated with a decrease in the severity, the serious complications of COVD-19, and admissions and in-hospital mortality.

PATIENTS AND METHOD

A total of 79 blood samples and CT scans from patients were collected from February 2020 to July 2020 in Al-Zahra teaching hospital.

D-dimer test is done as follows:

1. Transfer samples to tube containing detection buffer including blood, plasma, and control at 10 ml per sample using a pipette.
2. Mix the sample after covering the tube by shaking it well 10-15 times.
3. Transfer 75 µl of the sample mixture to the cartridge sample well by pipette.
4. Leave the loaded sample cartridge for 12 min incubation at room temperature to cool. Then scan this cartridge as quickly as possible.
5. To scan the sample-loaded cartridge for Ichroma' testing, place it in the instrument's cartridge holder. Make sure the cartridge is properly orientated before placing it into the cartridge holder. For this reason, an arrow is prominently shown on the cartridge.
6. To start the scanning process in the Ichroma device, press the start or select button.
7. Then the cartridge loaded with the sample is immediately erased by the Ichroma device.
8. The display screen will show the result were could read directly. Boditech Med Incorporated (43, Geodudanji 1-gil, Dongnae-myeon, Chuncheon-si, Gang-won-do, 24398, Republic of Korea) Ichroma (43, Geodudanji 1-gil, Dongnae-myeon, Chuncheon-si, Gang-won-do, 24398, Republic of Korea) (ichromaTM II, A small and easy-to-use fluorescence based POCT immunoassay analyzer).

Ethical Consideration:

The study was approved by the Ethics Board of Collage of Medicine/Wasit University and an informed written consent was taken from each participant in the study.

Statistical analysis

The relation between two groups of COVD-19 patients one used aspirin and the other not used aspirin where CT-scan findings and D-dimer levels were assessed using independent sample test, all results were compared between 2 groups using Descriptive Statistics study.
A 0.05 p-value was judged significant. SPSS 22 was used to analyze the statistical data.

RESULTS

A retrospective study for 79 patients with COVD-19 infection involved in this study, 36 patients previously used aspirin and 43 patients not used aspirin in Al-Zahra Teaching Hospital in the Wasit province from February 2020 to July 2020. Every patient underwent a CT-scan of the chest and D-dimer test with the demographic data including age and gender for each patients' groups as showing blow.

Table (1): Age distribution of patients group used aspirin and group not used aspirin Descriptive Statistics

	N	Minimum	Maximum	Mean		Std. Deviation
Age	Statistic	Statistic	Statistic	Statistic	Std. Error	Statistic
aspirin group	36	30.00	80.00	57.2222	2.55390	15.32339
non aspirin group	44	14	100	62.91	2.866	19.008

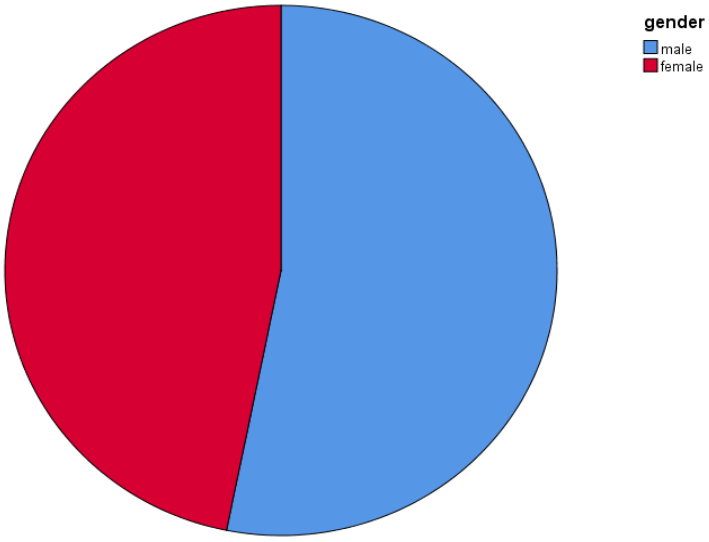

Figure (1): gender distribution for patients group used aspirin.

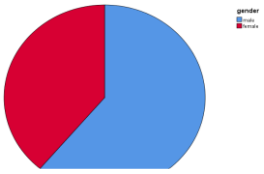

Figure (2): gender distribution for patients' groups not used aspirin.

In our study we found that patients who used aspirin significantly had fewer CT scan changes in the lung (P = 0.005) compared to those who did not use aspirin as shown in table (2).

Table (2): Statistical analysis of CT-scan of the lung for patients group used aspirin and group not used aspirin

CT-scan	Levene's Test for Equality of Variances		t-test for Equality of Means						
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
								Lower	Upper
Equal variances assumed	14.311	.000	-3.630	77	.001	-.41150	.11337	-.63724	-.18576
Equal variances not assumed			-3.559	66.597	.001	-.41150	.11563	-.64232	-.18068

However, no significant correlation between the usage of aspirin or not used and D-dimer level (P = >.05) as shown in table (3).

Table (3): Statistical analysis of D-dimer for patients group used aspirin and group not used aspirin

Independent Samples Test									
	Levene's Test for Equality of Variances		t-test for Equality of Means						
D. dimer	F	Sig.	T	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference Lower	Upper
Equal variances assumed	.078	.781	1.218	77	.227	758.93798	623.34561	-482.30170	2000.17767
Equal variances not assumed			1.219	74.955	.227	758.93798	622.50046	481.15878	1999.03475

DISCUSSION

COVD19 is a major public health problem receiving wide attention among researchers and research centres. The current study is a cross-sectional cohort study done in Al-Zahra Teaching Hospital in the Wasit province. The current study showed that the incidence of acute pneumonia and acute respiratory syndrome was significantly reduced among aspirin users before admission to hospital ($P < 0.005$), and this was confirmed by **Erlich *et al.*** [21] in his study of the association of prehospital use of antiplatelet therapy with a lower incidence of acute lung injury and acute respiratory syndrome. The results are also in agreement with **Chen *et al.*** [22]. Reduced danger of acute respiratory syndrome in this selected cohort study of critically ill patients was independently associated with Pre-hospital aspirin use, even after adjusting for the propensity of pre-hospital aspirin use. This finding also agrees with other studies in animals done by **Song *et al.*** [23] and **Chelucci *et al.*** [24]. Another research indicated that taking aspirin was related to a 44 % lower chance of being placed on a mechanical ventilator, a 43 % lower risk of ICU admission, and a 47 % lower risk of dying in the hospital when compared to those who did not take aspirin [25].

Our findings showed that there is no significant relationship between aspirin use and D-dimer levels ($P > 0.05$), implying that aspirin does not affect thrombogenesis markers. ***Lip et al.*** [26] showed that adding ultra-low-dose warfarin (1 mg) or aspirin 300 mg had no impact on D-dimer levels, a marker of intravascular thrombogenesis. This was also supported by **Suzanne *et al.*** [27], who showed that antiplatelet medications had no significant influence on D-dimer levels.

LIMITATIONS

The limitations of our study were the difficulty of CT-scan and the sampling of the blood for D-dimer from patients severely ill in addition to the risk of infectivity of the virus.

CONCLUSION

In hospitalized COVID-19 patients, using aspirin before infection may reduce severe outcomes. So a larger randomized controlled research is needed to establish if there is a link between past aspirin use and decreased lung damage and death in COVID-19 individuals.

Funding: This work was funded by the corresponding author.

Author Contribution: all authors were contributed equally.

Conflict of Interest: The authors declared no conflict of interest.

REFERENCES

1. **Carstens E (2009):** Ratification vote on taxonomic proposals to the International Committee on Taxonomy of Viruses. Arch Virol., 155: 133.
2. **Centers for Disease Control and Prevention (CDC) 2012:** Severe respiratory illness associated with a novel coronavirus--Saudi Arabia and Qatar. Morb Mortal Wkly Rep., 61: 820.
3. **Corman V, Muth D, Niemeyer D, Drosten C (2018):** Hosts and sources of endemic human coronaviruses. Adv. Virus Res., 100 (4): 163–188.
4. **Christian M, Poutanen S, Loutfy M *et al.* (2004):** Severe acute respiratory syndrome. Clin. Infect. Dis., 38: 1420.
5. **Peiris J, Yuen K, Osterhaus A, Stöhr K (2003)**: The severe acute respiratory syndrome. Engl. J. Med., 349: 2431.
6. **Bikdeli B, Madhavan M, Jimenez D *et al.* (2020):** COVID-19 and thrombotic or thromboembolic disease: implications for prevention, antithrombotic therapy, and follow-up: *JACC* state-of-the-art review. J. Am. Coll. Cardiol., 75 (23): 2950–2973.
7. **Drosten C, Günther S, Preiser W *et al.* (1967):** Identification of a novel coronavirus in patients with

severe acute respiratory syndrome. www.sciepub.com/reference/349376

8. **Ksiazek T, Erdman D, Goldsmith C *et al.* (2003):** A novel coronavirus associated with severe acute respiratory syndrome. Engl. J . Med. , 348: 1953.
9. **Peiris J, Lai S, Poon L *et al.* (2003):** Coronavirus as a possible cause of severe acute respiratory syndrome. Lancet, 361: 1319.
10. **Hon K, Leung C, Cheng W *et al. (2003):*** Clinical presentations and outcome of severe acute respiratory syndrome in children. Lancet, 361: 1701.
11. **Stockman L, Massoudi M, Helfand R *et al.* (2007):** Severe acute respiratory syndrome in children. Pediatr. Infect. Dis. J., 26: 68.
12. **Booth T, Kournikakis B, Bastien N *et al.* (2005):** Detection of airborne severe acute respiratory syndrome (SARS) coronavirus and environmental contamination in SARS outbreak units. J. Infect. Dis., 191: 1472.
13. **Donnelly C, Fisher M, Fraser C *et al.* (2004):** Epidemiological and genetic analysis of severe acute respiratory syndrome. Lancet. Infect. Dis., 4: 672.
14. **Poutanen S, Low D, Henry B *et al.* (2003):** Identification of severe acute respiratory syndrome in Canada. Engl. J. Med., 348: 1995.
15. **Olsen S, Chang H, Cheung T *et al.* (2003):** Transmission of the severe acute respiratory syndrome on aircraft. Engl. J. Med., 349: 2416.
16. **Cheng P, Wong D, Tong L *et al.* (2004):** Viral shedding patterns of coronavirus in patients with probable severe acute respiratory syndrome. Lancet, 363: 1699.
17. **Peiris J, Chu C, Cheng V *et al.* (2003):** Clinical progression and viral load in a community outbreak of coronavirus-associated SARS pneumonia: a prospective study. Lancet, 361: 1767.
18. **National Center for Immunization and Respiratory Diseases (2021):** Division of Viral Diseases . CDC., 24: 7.
19. **Centers for Disease Control , Prevention (2003)**: Preliminary clinical description of severe acute respiratory syndrome. Morb. Mortal. Wkly. Rep., 52: 255.
20. **National Center for Immunization and Respiratory Diseases (NCIRD). (2021)** :Division of Viral Diseases . CDC., 12: 8.
21. **Erlich J, Talmor D, Cartin-Ceba R, Gajic O, Kor D (2011):** Prehospitalization antiplatelet therapy is associated with a reduced incidence of acute lung injury: a population-based cohort study. Chest, 139: 289–295.
22. **Chen W, Janz D, Bastarache J *et al.* (2015):** Prehospital aspirin use is associated with reduced risk of acute respiratory distress syndrome in critically ill patients: a propensity- adjusted analysis. Crit. Care. Med., 43: 801–807.
23. **Song C, Suzuki S, Kubo H *et al.* (2004):** Effects of antiplatelet agents on pulmonary haemodynamic response to fMLP in endotoxin primed rats. Thorax, 59 (1): 39–44.
24. **Chelucci G, Boncinelli S, Marsili M *et al.* (1993):** Aspirin effect on early and late changes in acute lung injury in sheep. Intensive care medicine, 19 (1): 13-21.
25. **Jill M (2021):** Aspirin Use Reduces Risk of Death in Hospitalized COVID-19. Patients Life Sciences, 34 (9): 55-59.
26. **Lip G, Lip P,zarfis J *et al.* (1996):** Fibrin D-dimer and beta-thromboglobulin as markers of thrombogenesis and platelet activation in atrial fibrillation. Eur. J. Clin. Invest., 94 (3): 425-31.
27. **Suzanne S (2018):** Clinical effects of antiplatelet drugs and statins on D-dimer levels. Eur. J. Clin. Invest., 48 (7): 129.