Original Article

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A Retrospective Observational Study of the Occurrence of Thromboembolic Complications in the Post COVID-19 Phase and its Association with CT Severity Score

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Abstract

Background: To present thromboembolic complications after recovery from covid-19 and determine its Association with CT severity score. **Subjects and Methods:** An observational study was conducted retrospectively over a period of 1 month during which all cases who presented with thromboembolic complications after being discharged from covid-19 were enrolled. The data pertaining to the socio-demography, haematological parameters and inflammatory markers such as LDH, IL-6, ferritin, CRP and D-Dimer were recorded at the time of admission with COVID-19. Initial chest HRCT scans were done at presentation. For determining the area of embolism, CT angiography was done in the region of head and neck, coronary and lower limb as per the presenting symptoms. The patients were thrombolysed/ revascularized and followed up until recovery/death. **Results :** HRCT scoring was normal in 59.18% cases, mild in 8.16% and severe in 32.65% cases. Thromboembolic complications were seen in all patients, common being peripheral vascular disease (53.06%) followed by coronary artery disease (38.78%) and lastly cerebrovascular accidents (8.16%). Mean value of post covid days of study subjects was 12.33 ± 5.34 with median (25th-75th percentile) of 11(8-16). There was only a single mortality in the present study. HRCT score showed a significant association with the presence of hypertension (p=0.011,) and leukocytosis (p=0.004). However there was no significant association of HRCT with the region of thromboembolism. **Conclusions:** Thromboembolic complications are common in the initial month of post covid phase. Comorbidities like hypertension needs to be controlled as it shows a significant association with HRCT severity. Inflammatory levels of IL-6 and CRP may be implicated in the occurrence of thromboembolism. An early identification, treatment and thromboprophylaxis is required in such cases for a better outcome.

Keywords: Ferritin, severe, mortality, COVID-19

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Introduction

The first case of "severe acute respiratory syndrome coronavirus 2 (SARS-COV-2)" appeared in December 2019. Then World Health Organization, by March 2020, had declared it as a pandemic.[1,2] Still, not much is known about the virus, and its implications. However, one thing about the manifestation of the disease is clear that it can take two courses:

1) Life-threatening severe acute respiratory syndrome.

2) Mild respiratory illness or completely asymptomatic.

The main clinical problem is respiratory symptoms. Still, emerging evidence links the disease with coagulation dysfunction (puts the patient at risk of arterial and venous thromboembolism (TE)), due to which the risk of death increases.[3]

In literature, the occurrence rate of TE varies from 20-30% to 40-70%.[4–9] Certain complications like thromboembolism and hypercoagulation increase the severity of the condition causing intensive care unit (ICU) admissions and even death. Some of the studies linked TE in COVID-19 patients to a higher risk of death, but other studies did not resonate with the same result.[5,10]

The present study was conducted with an aim to determine the thromboembolic complications and outcomes after recovery from covid-19 and determine its association with CT severity score.

Subjects and Methods

A retrospective observational study was done in the department of medicine over a period of one month during which all cases who presented with thromboembolic complications after being discharged from covid-19 were enrolled. Patients were explained about the study and a written informed consent was taken. Institutional ethical clearance was obtained before beginning the study.

Inclusion criteria

- Consecutive SARS-CoV2 patients admitted & discharged in this hospital w.e.f. 01 April 2021 till 30th July 2021 who presented with complaints of chest pain, dyspnoea, sweating (suggestive of myocardial infarction); limb pain, numbness, paresthesia with absent distal pulse (suggestive of peripheral artery disease) and weakness of one side half of body (suggestive of cerebrovascular accidents) within one month of discharge.
- Both males and females
- Age more than 18 years

Exclusion criteria

- Patients with known pre-existing congenital bleeding or thrombotic disorders and/or pre-existing acquired coagulopathies,
- Active cancer patients and/or on chemotherapy,
- Pregnancy
- Patients with ongoing anticoagulant therapy.

Sample size

The study of Malas MB et al observed that rates of specific arterial thromboembolic events were as follows, MI=0.5%, CVA=1%, and ALI=0.4%.[11] Taking this value as reference, the minimum required sample size with 3% margin of error and 5% level of significance is 43 patients. To reduce margin of error, total sample size taken is 49.

The data pertaining to the socio-demography of the patients (Age, gender and co-morbidities), hematological parameters: hemoglobin (Hb), total leucocyte count (TLC), Differential leucocyte count (DLC), Platelet counts, urea, creatinine and inflammatory markers such as LDH, IL-6, ferritin, CRP) and D-Dimer were recorded at the time of admission with COVID-19.

Initial chest HRCT scans were done at presentation, the findings of which were recorded. The reporting was as per 25-point CT severity score by Chang et al. where 7 or less indicates Mild; 8–17 indicates Moderate and 18 or more indicates Severe disease.[12]

For determining the area of thromboembolism, CT angiography/conventional angiography was done in the region of head and neck, coronary and lower limb as per the presenting symptoms. Some of the representative images are shown in [Figure 1 and 2].

The patients were thrombolysed and revascularized by means of angioplasty/Embolectomy and followed up until recovery/death. The outcome measures were region of thrombosis and mortality.

Statistical analysis

The presentation of the Categorical variables was done in the form of number and percentage (%). On the other hand, the quantitative data with normal distribution were presented as the means ± SD. The data normality was checked by using Kolmogorov-Smirnov test. The cases in which the data was not normal, we used non parametric tests. The following statistical tests were applied for the results:

1. The association of age which was quantitative and normally distributed in nature were analysed using ANOVA.
2. Fisher's exact test was used for association of gender, diabetes mellitus, hypertension, blood investigations, diagnosis and outcome with HRCT score.

The data entry was done in the Microsoft EXCEL spreadsheet and the final analysis was done with the use of Statistical Package for Social Sciences (SPSS) software, IBM manufacturer, Chicago, USA, ver 21.0. For statistical significance, p value of less than 0.05 was considered statistically significant.

Results

The median age of the study patients were 56 years with predominant male population (73.47%). Among comorbidities, hypertension and diabetes mellitus, were found in 42.86% and 38.78% patients [Table 1].

Among lab investigation, total WBC(10^9/litre), neutrophil/lymphocyte ratio, blood urea (mg/dL), Creatinine (mg/dL), SGOT (U/L), SGPT (U/L), LDH (U/L), D dimer (ng/mL), CRP (mg/dL) and Ferritin (ng/mL) were significantly higher in critical disease followed by severe disease as compared to moderate and mild disease. [Table 2 and Figure 2]

Lab findings showed that anemia was present in 24.49% cases, leukocytosis(>11,000 cells/mL) was seen in 59.18% cases, thrombocytopenia was seen in 8.16% cases, high urea(>40 mg/dL) in 22.45% cases and high creatinine(>0.8 mg/dL) in 38.78% cases. Among inflammatory markers, CRP, LDH, D dimer(ng/mL), IL-6 and Ferritin(ng/mL) were increased in 100%, 63.27%, 89.8%, 100% and 71.43% cases respectively as shown in [Table 2].

HRCT scoring was normal in 59.18% cases, mild in 8.16% and severe in 32.65% cases. [Table 3]

Thromboembolic complications were seen in all patients, common being peripheral vascular disease (53.06%) followed by coronary artery disease (38.78%) and lastly cerebrovascular accidents (8.16%). [Figure 3]

Among the 19 cases of coronary artery disease, 17 has STEMI and 2 cases had NSTEMI. Among the 17 cases of STEMI, twelve cases had inferior wall MI and 5 cases had anterior wall MI. Among the 26 cases of peripheral vascular disease ,14 had thrombosis in the upper Limb, 10 in the lower Limb and two at the level of aorta.

Mean value of post covid days of study subjects was 12.33 ± 5.34 with median (25th-75th percentile) of 11 (8-16). There was only a single mortality in the present study.

HRCT score showed a significant association with the presence of hypertension (p=0.011, Table 4) and leukocytosis (p=0.004, Table 5). However there was no significant association of HRCT with the region of thromboembolism as shown in [Table 6].

Table 1: Distributionof socio-demographic characteristics of study subjects.

Socio-demographic characteristics	Frequen	Percentage
Age(years)		
Mean ± SD	57.04 ± 11.8	
Median(25th-75th percentile)	56(48-65)	
Range	35-85	
Gender		
Female	13	26.53%
Male	36	73.47%
Co-morbidities		
Diabetes mellitus -II	19	38.78%
Hypertension	21	42.86%

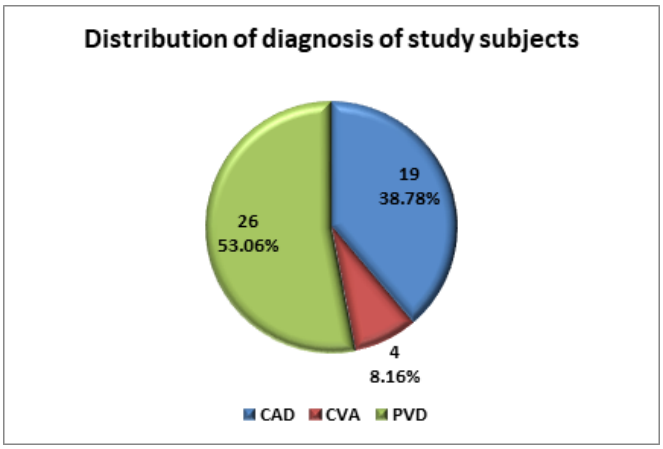

Figure 1: Distribution of diagnosis of study subjects.

Discussion

To our knowledge this is the first study with a large Indian data on the occurrence of post covid thromboembolic complications.

R1

Currently, the data is limited on the risk of TE. Klok et al. reported that out of the patients admitted in their hospital (in ICU) in the Netherlands with COVID-19 infections, 31% had thrombotic complications (27% had a pulmonary embolism or venous thromboembolism and 3.7% had arterial thrombotic complications).[13] In an extensive health system like that of New York City, thrombotic event (13.6% venous and 18.6% arterial) was seen in 29.4% of ICU patients with COVID-19, whereas 11.5% of non-ICU patients suffered from TE (3.6% venous and 8.4% arterial).[14] In Wuhan, China, 143 hospitalized patients with COVID-19 were studied, and it was found that 46% got deep venous thrombosis in the lower limbs, which was much higher than venous thromboembolism found in 30% of critically ill patients with SARS-CoV in a study by Umapathi et al.[10,15]

Several mechanisms are involved in the increased risk of thromboembolism in COVID-19 such as platelet activation, excessive inflammation, endothelial dysfunction, and stasis cause thrombosis in venous and arterial systems.[16]

R2

Proinflammatory cytokines level go up in COVID-19 patients, causing increased systemic inflammation and endothelial injury.[17] This mechanism starts when the virus attaches to the angiotensin-2 receptor of the endothelial cells and starts to replicate, causing prothrombotic endothelial dysfunction.[18,19]Corroborating with the mechanisms, study results show that all patients had high IL-6 and CRP with majority showing high levels of ferritin, LDH and D-dimer; platelet reduction was seldom observed.

Besides, in COVID-19 other factors that add to the prothrombotic state are the use of central venous catheters, and mechanical ventilation. However none of which were used in the present study since it was done in the post-COVID phase.

As per some previous reports, TE development and coagulopathy increase the risk of death in COVID-19 patients.[3,17] We observed only one mortality which may be because of immediate management and a small sample size.

Table 2: Distributionof blood investigations of study subjects.

Blood investigations	Frequency	Percentage
Haemoglobin(g/dL)		
Low	12	24.49%
Normal	33	67.35%
High	4	8.16%
Mean ± SD	12.86 ± 1.62	
Median (25th-75th percentile)	13(12-13.8)	
Range	7.5-16.6	
Total leucocuyte count(cells/mm^3)		
Normal	20	40.82%
High	29	59.18%
Mean ± SD	13.51 ± 6.17	
Median(25th-75th percentile)	13.1(9-15.5)	
Range	5-40	
Platelet count(cells/mm^3)		
Low	4	8.16%
Normal	44	89.80%
High	1	2.04%
Mean ± SD	243.12 ± 77.41	
Median(25th-75th percentile)	222(186-292)	
Range	105-484	
Blood urea(mg/dL)		
Normal	38	77.55%
High	11	22.45%
Mean ± SD	37.55 ± 18.66	
Median(25th-75th percentile)	34(25.3-41)	
Range	15.7-123.1	
Serum creatinine(mg/dL)		
Normal	30	61.22%
High	19	38.78%
Mean ± SD	1.18 ± 0.61	
Median(25th-75th percentile)	1.03(0.86-1.34)	
Range	0.4-4.06	
C-reactive protein test(mg/L)		
High risk	49	100.00%
Mean ± SD	53.99 ± 42.48	
Median(25th-75th percentile)	46(19-71)	
Range	4-203.89	

LDH(U/L)		
Low	1	2.04%
Normal	17	34.69%
High	31	63.27%
Mean ± SD	522.51 ± 246.65	
Median(25th-75th percentile)	446(355-628)	
Range	186-1293	
Serum ferritin(ng/mL)		
Normal	14	28.57%
High	35	71.43%
Mean ± SD	247.32 ± 138.97	
Median(25th-75th percentile)	222(145-328.1)	
Range	24.53-615.7	
IL-6		
>7	49	100.00%
Mean ± SD	207.17 ± 257.64	
Median(25th-75th percentile)	106.1(61-284)	
Range	10-1479	
D dimer(μgm/mL)		
Normal	5	10.20%
High	44	89.80%
Mean ± SD	5.74 ± 8.71	
Median(25th-75th percentile)	2.35(0.8-8.2)	
Range	0.22-46.8	

Table 3: Distribution of HRCT score of study subjects.

HRCT score	Frequency	Percentage
Mild	4	8.16%
Normal	29	59.18%
Severe	16	32.65%
Mean ± SD	13.49 ± 3.75	
Median(25th-75th percentile)	14(11-16)	
Range	5-22	

To understand the prothrombotic state in COVID-19 in a better way, autopsy studies were performed.[20,21] It was seen in a recent study that thrombosis was present in every organ.[22] Sometimes even in the early phase of the disease, significant microvascular and macrovascular thrombosis was seen in multiple organs, regardless of anticoagulation status. Some studies attributed increased hypercoagulopathy to factors like D-dimer levels, hospitalization and underlying medical conditions.[23,24] We here found a significant association of hypertension with HRCT severiti in patients with thromboembolic complications implicating the impact of raised blood pressure on the vascular walls. However diabetes showed no significant association.

Our study explains the range of events of thromboembolism affecting the neurovascular, cardiovascular and peripheral vascular regions. Special attention needs to be given to radiologic evaluation in COVID-19 patients, especially for the pulmonary arterial vascular system, as only pulmonary emboli can be a logical explanation for persistent hypoxemia in the presence of steroid use and oxygen supplementation. Besides, a focal nonadherent thrombus can occur in the aorta, right and left ventricles, branch vessels, and iliac vessels that may lodge and cause thromboembolism.[25]

Table 4: Association of socio-demographic characteristics with HRCT score.

Socio-demographic characteristics	Mild (n=4)	Normal (n=29)	Severe (n=16)	Total	P value
Age(years)	59 ± 11.4	56.24 ± 12.66	58 ± 10.85	57.04 ± 11.81	0.846†
Gender					
Female	0 (0%)	7 (53.85%)	6 (46.15%)	13 (100%)	0.373*
Male	4 (11.11%)	22 (61.11%)	10 (27.78%)	36 (100%)	
Diabetes mellitus -II					
No	3 (10%)	20 (66.67%)	7 (23.33%)	30 (100%)	0.24*
Yes	1 (5.26%)	9 (47.37%)	9 (47.37%)	19 (100%)	
Hypertension					
No	4 (14.29%)	19 (67.86%)	5 (17.86%)	28 (100%)	0.011*
Yes	0 (0%)	10 (47.62%)	11 (52.38%)	21 (100%)	

* **Fisher's exact test,** † **ANOVA**

The management in our study was not preventive rather we treated the patients immediately with thrombolysis leading to optimal outcomes. But understanding of the concurring events after COVID-19, preventive measures seek importance.

The "International Society of Thrombosis and Hemostasis (ISTH)" provided a set of temporary guidelines on identifying and controlling coagulopathy in COVID-19 so that prothrombotic state could be avoided in COVID-19 patients. It states that a prophylactic dose of "low molecular weight heparin (LMWH)" should be given to all patients (hospitalized patients including non-critical cases) in the absence of contraindications.[25] The "American Society of Hematology" also suggested that pharmacologic thromboprophylaxis with fondaparinux or LMWH should be given to all hospitalized patients due to COVID-19; the only contraindication as per them is patients at increased bleeding risk.[26] There are many more guidelines suggesting the use of thromboprophylaxis for COVID-19 patients (particularly for hospitalized patients).[16,27,28]

Limitations of the Study

The study suffers from few limitations. First a small sample size considering the ongoing pandemic of covid-19. Second, no controls were enrolled such as covid-19 cases who got discharged but did not present with thromboembolic complications.

In view of these limitations, for better identification of risk factors for systemic thrombosis, more studies are needed. Sample sizes need to be more significant for quantifying thromboembolic complications incidence in all the cases of COVID-19. To better calculate the risk of coagulation in COVID-19 patients, useful serum markers (D-dimer, CRP, IL-6 and fibrinogen levels) need to be identified and monitored. This sort of identification of serum markers will also help identify specific cohorts who can benefit from anticoagulation therapy.[25,29]

Conclusion

Thromboembolic complications are common in the initial month of post covid phase. Comorbidities like hypertension needs to be controlled as it shows a significant association with HRCT severity. Inflammatory levels of IL-6 and CRP may be implicated in the occurrence of thromboembolism. An early identification, treatment and thromboprophylaxis is required in such cases for a better outcome.

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Table 5: Association of blood investigations with HRCT score.

Blood investigations	Mild (n=4)	Normal (n=29)	Severe (n=16)	Total	P value
Haemoglobin(g/dL)					
Low	0 (0%)	9 (75%)	3 (25%)	12 (100%)	0.067*
Normal	2 (6.06%)	18 (54.55%)	13 (39.39%)	33 (100%)	
High	2 (50%)	2 (50%)	0 (0%)	4 (100%)	
Total leucocuyte count(cells/mm^3)					
Normal	1 (5%)	17 (85%)	2 (10%)	20 (100%)	0.004*
High	3 (10.34%)	12 (41.38%)	14 (48.28%)	29 (100%)	
Platelet count(cells/mm^3)					
Low	0 (0%)	4 (100%)	0 (0%)	4 (100%)	0.466*
Normal	4 (9.09%)	24 (54.55%)	16 (36.36%)	44 (100%)	
High	0 (0%)	1 (100%)	0 (0%)	1 (100%)	
Blood urea(mg/dL)					
Normal	4 (10.53%)	23 (60.53%)	11 (28.95%)	38 (100%)	0.581*
High	0 (0%)	6 (54.55%)	5 (45.45%)	11 (100%)	
Serum creatinine(mg/dL)					
Normal	3 (10%)	19 (63.33%)	8 (26.67%)	30 (100%)	0.532*
High	1 (5.26%)	10 (52.63%)	8 (42.11%)	19 (100%)	
C-reactive protein test(mg/L)					
High risk	4 (8.16%)	29 (59.18%)	16 (32.65%)	49 (100%)	No p value
LDH(U/L)					
Low	0 (0%)	1 (100%)	0 (0%)	1 (100%)	0.686*
Normal	1 (5.88%)	12 (70.59%)	4 (23.53%)	17 (100%)	
High	3 (9.68%)	16 (51.61%)	12 (38.71%)	31 (100%)	
Serum ferritin(ng/mL)					
Normal	2 (14.29%)	8 (57.14%)	4 (28.57%)	14 (100%)	0.707*
High	2 (5.71%)	21 (60%)	12 (34.29%)	35 (100%)	
IL-6					
>7	4 (8.16%)	29 (59.18%)	16 (32.65%)	49 (100%)	No p value
D dimer(μgm/mL)					
Normal	1 (20%)	3 (60%)	1 (20%)	5 (100%)	0.571*
High	3 (6.82%)	26 (59.09%)	15 (34.09%)	44 (100%)	

* **Fisher's exact test**

Table 6: Association of diagnosis with HRCT score.

Diagnosis	Mild (n=4)	Normal (n=29)	Severe (n=16)	Total	P value
CAD	2 (50%)	13 (44.83%)	4 (25%)	19 (38.78%)	0.136*
CVA	1 (25%)	3 (10.34%)	0 (0%)	4 (8.16%)	
PVD	1 (25%)	13 (44.83%)	12 (75%)	26 (53.06%)	
Total	4 (100%)	29 (100%)	16 (100%)	49 (100%)	

* **Fisher's exact test**

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