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CASE REPORT

Massive embolism in Covid-19 patient: A Case Report

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ABSTRACT
Hospitalized patients with COVID-19 were characterized by a high rate of thromboembolic complications and in hospital mortality. The exact mechanisms of COVID-19 induced thrombosis have not beenelucidated.The early pathogenesis in COVID-19 (Huertas **et al***.) pneumonia defined by a widespread endotheliitis (5)affecting multiple organ systems ,viral inclusion are observed within endothelial cells accompanied by apoptosis, inflammatory cell infiltration and microvascular thrombosis(1.2).The primary infection initiates alveolar injury and the resulting inflammatory response, including production of inflammatory cytokines, including IL-6, as well as activation and recruitment of mononuclear cells and neutrophils causing more tissue damage, including damage to the capillary endothelium. In addition to the procoagulant effectors derived as the result of inflammation the usual thrombo-protective state of the vascular endothelial cells is disrupted; Both pathophysiologic changes lead to the development of microvascular thrombosis(1). Over time the pathology of ARDS progresses to a proliferative and then a fibrotic state, which is fatal. We presented one case when the patient developed severe respiratory failure after massive pulmonary embolism and coma after ischemic stroke . Patient had many comorbidities with COPD , heart failure (HFrEF) and diabetes mellitus . High values of d-dimer could be related to a higher activation of blood coagulation in COVID-19 patients secondary to a systemic inflammatory response syndrome – or as a direct consequence of the SARS-CoV-2 itself. Pulmonary thrombosis was the confluence of processes, endothelial inflammation with no evidence of DVT . Tissue factor, upregulated on platelets, leucocytes during inflammation, leading to activation coagulation pathways and promote the formation of fibrin. The profound hypoxaemia is a likely driver of vasoconstriction, inflammation and thrombosis. The origin of Covid-19-associated pulmonary emboli and lung microcirculatory thrombotic disease: Interaction of inflammation and coagulation*
Key words: Thrombosis, Pulmonary embolism, inflammation

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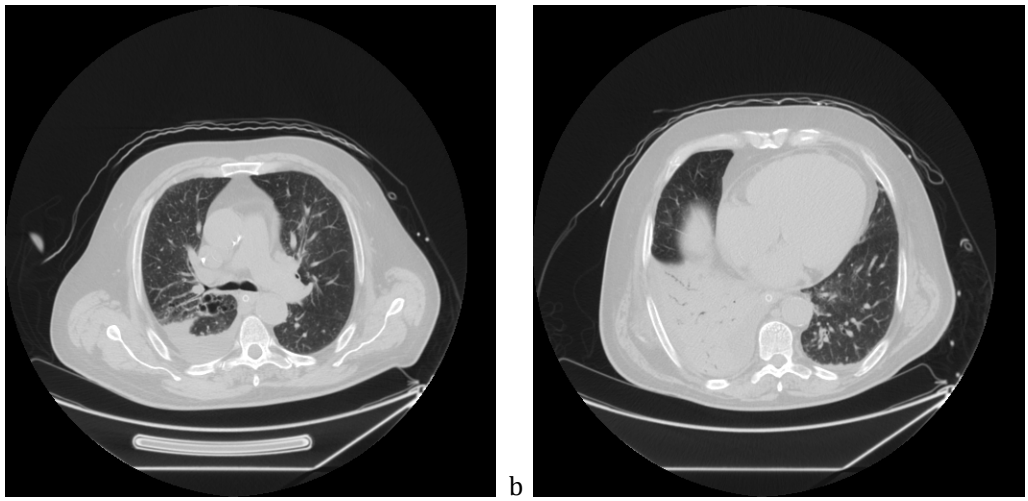
INTRODUCTION

Hospitalized patients with COVID-19 were characterized by a high rate of thromboembolic complications and in hospital mortality.The exact mechanisms of COVID-19 induced thrombosis have not been elucidated.The early pathogenesis in COVID-19 (Huertas ***et al***.) pneumonia defined by a widespread endotheliitis affecting multiple organ systems (5),viral inclusion are observed within endothelial cells accompanied by apoptosis, inflammatory cell infiltration and microvascular thrombosis(1.2).The primary infection initiates alveolar injury and the resulting inflammatory response, including production of inflammatory cytokines, including IL-6, as well as activation and recruitment of mononuclear cells and neutrophils causing more tissue damage, including damage to the capillary endothelium. In addition to the procoagulant effectors derived as the result of inflammation the usual thrombo-protective state of the vascular endothelial cells is disrupted. Both pathophysiologic changes lead to the development of microvascular thrombosis(1). Over time the pathology of ARDS progresses to a proliferative and then ultimately a fibrotic state, which is fatal. We presented one case when the patient developed severe respiratory failure after massive pulmonary embolism and coma after ischemic stroke . Patient

had many comorbidities with COPD , heart failure (HFrEF), chronic renal failure and diabetes mellitus.

Patient , male , 70 y. old, was admitted in our hospital with respiratory failure and coma . Patient was started mechanical ventilation. CTPA revealed massive pulmonary embolism and bilateral infiltrates. Thrombotic masses are reflected at different levels in the bilateral pulmonary arteries. Areas of infarction-pneumonia are detected against the background of the right basal infiltration.



a b

Figure 1. 05/10/2020. Computed tomography, axial section. Lung window. Incision at the level of the tracheal bifurcation

a. Incision at the level of the basal segments of the lung

b. In the right parenchyma of the lung, there are foci of bronchiectasis and extensive basal compaction-infiltrative changes

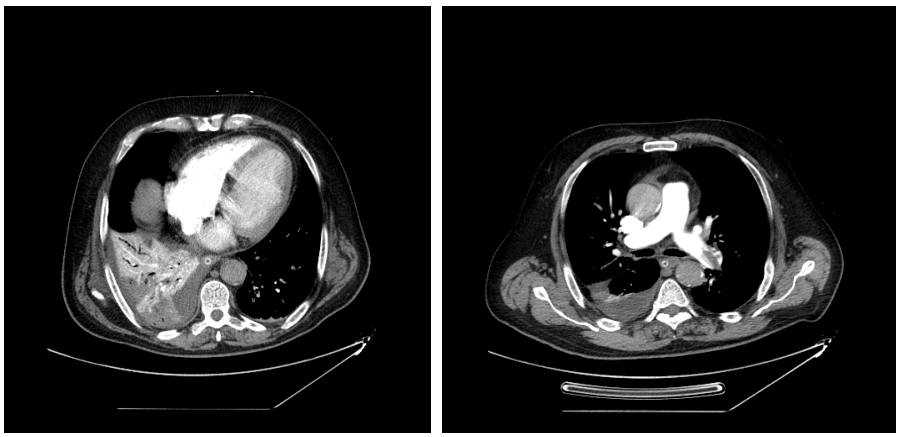


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Figure 2. 10.05.2020. Computed tomography, axial section. Vascular window. Incision at the level of basal segments

a. Incision at the level of the main arteries of the lung

b. Thrombotic masses are reflected at different levels in the bilateral pulmonary arteries. Areas of infarction-pneumonia are detected against the background of the right basal infiltration.

Echocardiographic findings of RV overload and/or dysfunction not detected , but was revealed left ventricle dysfunction, EF -20 %,. RV dilation was not found on transthoracic echocardiography (TTE). The combination of a pulmonary ejection acceleration time (measured in the RV outflow tract) with a peak systolic tricuspid valve gradient Was not present(9) . PASP -40 mm.Hg. “Buble” test was negative. Pulmonary Embolism Severity Index (PESI) to assess a patient’s overall mortality risk and early outcome, was >125 points(Class V), was identify of very high mortality risk(10-24.5%). Haemodynamic instability(pressure ,supporting by norepinephrine), combined with PE confirmation on CTPA was sufficient to classify a patient into the high-risk PE category,but calculation of the PESI and

measured of troponins (cardiac biomarker was high) identified the patient like in high mortality risk. Very elevated levels of D dimer have been observed, that was correlated with illness severity , like a marker of PE ,infectious and inflammatory diseases. Venous thromboembolism (VTE) , including deep vein thrombosis was not detected .

Treatment was followed the ESC guidelines focusing on the clinical management of pulmonary embolism (PE) published in 2019.

CT scan of brain was detected acute haemorrhagic infarction (Hemorrhagic transformation after cerebral infarction) in the right parietal lobe. There was a hypodenseous zone 5-6 cm, with blood-density inserts in the cortex and the phenomenon of periventricular luminescence, without displacement of the middle structures. Picture of cortical venous thrombosis and venous infarction in the right parietal lobe of the brain. Leukomalacia, leukoencephalopathy, cortical atrophy.(Fig.3).

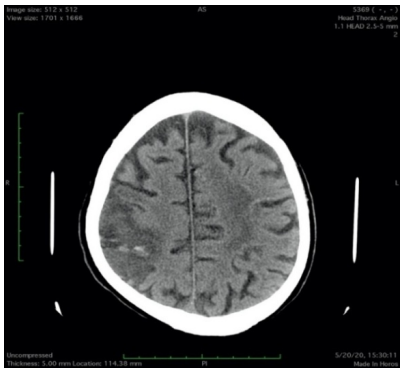
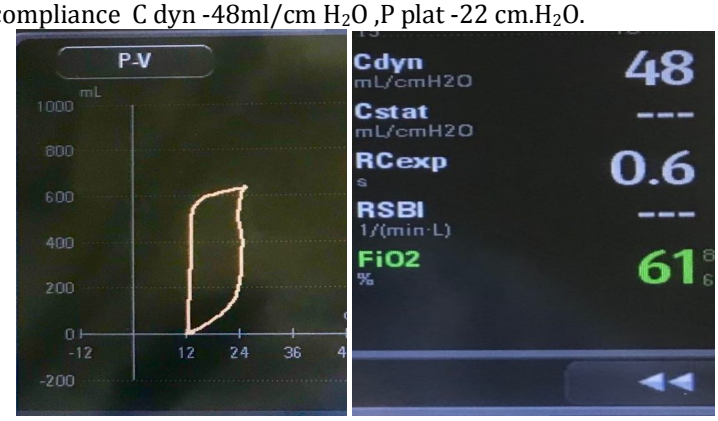


Fig .3: CT scan of brain was detected acute haemorrhagic infarction(Hemorrhagic transformation after vein cortical thrombosis and cerebral infarction) in the right parietal lobe

Initial level of D dimer was high -20 mkg/ml ,Hs Troponin --24 ng/ml, Ferritin—430 ng/ml, IL-6—28.24 mkg/l, CRP-70mg/L. PaO_2/FiO_2 <150 , patient was ventilated with DUOLEVEL mode and High PEEP-- 12 cm.H_2O , compliance C dyn -48ml/cm H_2O ,P plat -22 cm.H_2O.



LABORATORY FINDING

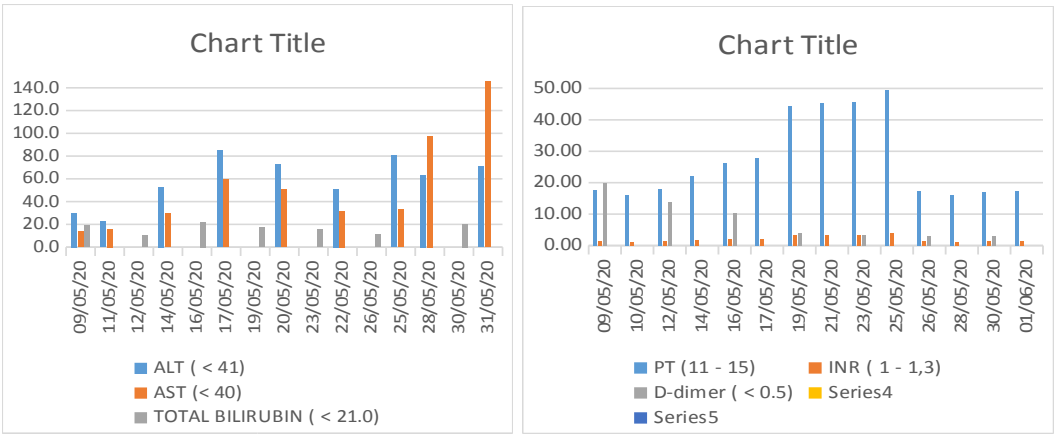


Table 1: Liver Profile and Various Blood Laboratory Finding

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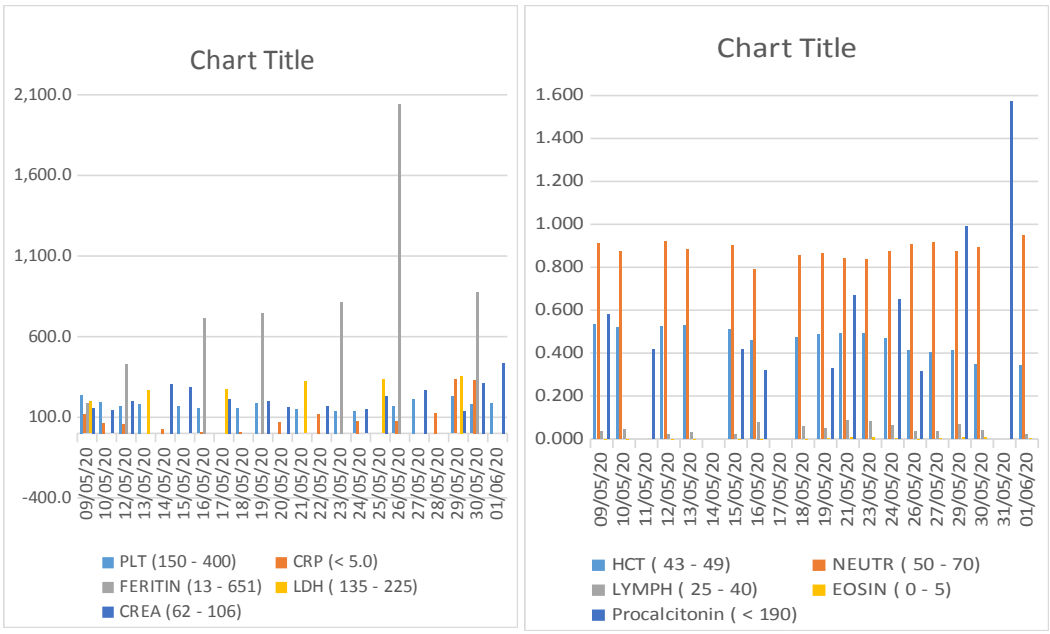


Table 2: Blood parameters

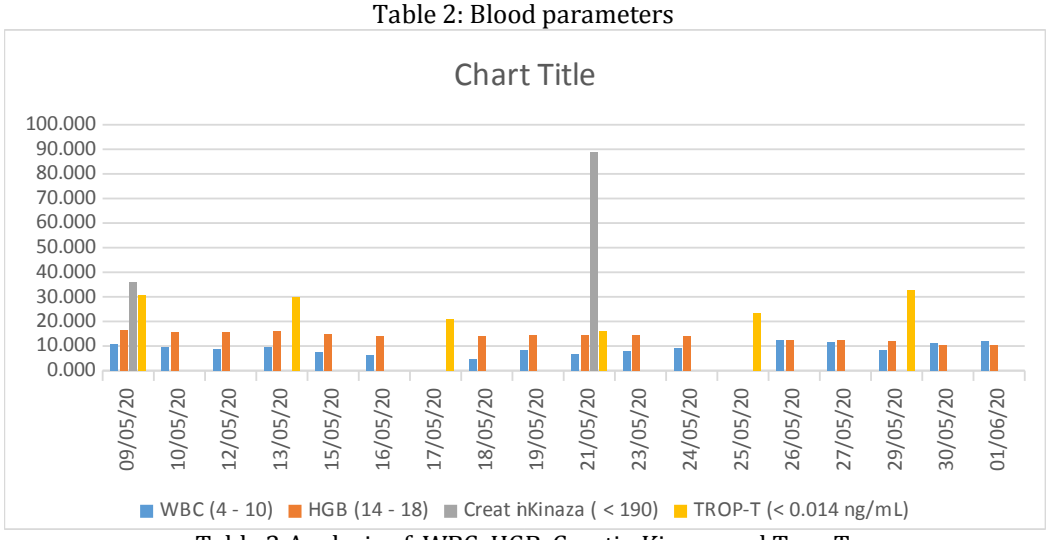


Table 3:Analysis of WBC, HGB, Creatin Kinase and Trop T

Patient state was improved. On the CT scan of brain was observed Blood density areas reduction in right parietal lobe .The density of haemorrhagic area is reduced(- positive X-ray dynamic). Fig.4

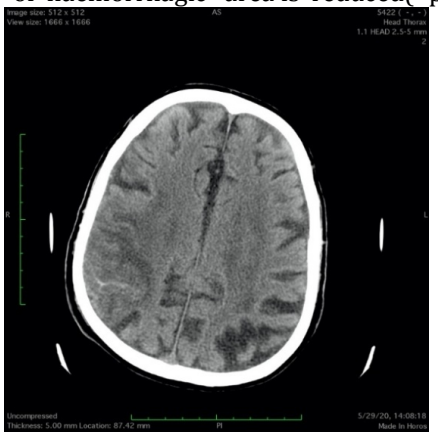


Fig.4

In the trunk of the pulmonary artery and in the main arteries a thrombus does not revealed (Fig 5), but The volume of extensive inflammatory changes was reduced with thickening of interlobal pleuras .

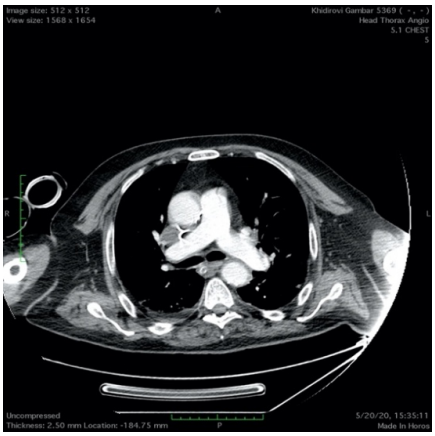
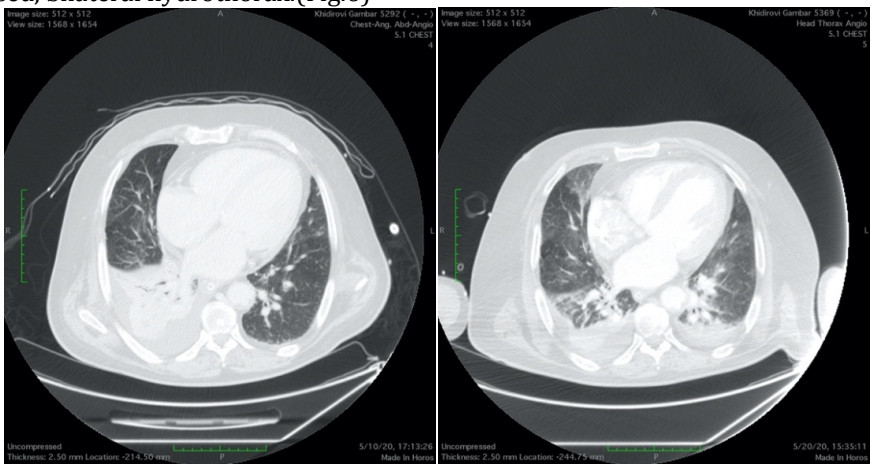


Fig .5. Thrombotic masses are no longer reflected in the lumen of the bilateral main artery in the pulmonary trunk. Against the background of the existing consolidation, a small triangular avascular zone is revealed

Extensive consolidating infiltrative changes in the lower right part are reduced, it is observed the interlobar pleura is thickened on the same side, bronchiectasis in the upper part and bullous changes in the apex, mixed infiltrative changes in the middle lobe. The infiltration volume of the upper lobe was slightly increased, bilateral hydrothorax.(Fig.6)



a b

Fig.6. a.In the lower part of right lung extensive inflammatory and consolidation lesions ,with airal bronchogramm. b.. The volume of extensive inflammatory changes is reduced with thickening of interlobal pleuras.

We presented case of vein embolism in the brain and in the bilateral pulmonary arteries in patient where comorbidities was different .Laboratory finding has shown changes of base parameters on different stage of illness and with correlation of disease severity(Table 1.2.3.) .

Bilateral pneumonia, systemic inflammation, endothelial dysfunction, coagulation activation, massive embolism, acute respiratory distress syndrome, coma and multiorgan failure we have described as key features of severe COVID-19 illness patient .

Hypothesis of the origin of Covid-19-associated pulmonary emboli and lung microcirculatory thrombotic disease: Interaction of inflammation and coagulation(1) . active replication and release of the virus may cause the host cell to undergo pyroptosis (pro-inflammatory apoptosis) and release damage-associated molecular patterns, activating oxidant stress, and generating pro-inflammatory cytokine and chemokine release from nearby epithelial cells, endothelial cells and alveolar macrophages. Tissue factor, from the subendothelium, is upregulated on platelets, leucocytes and EC during inflammation, leading to activation of both the extrinsic and intrinsic coagulation pathways [1, 2]. Occluded small pulmonary blood vessels are likely to contain fibrin, platelets and coagulation factors, as well as neutrophils that pass through the lung. The infection initiates alveolar injury and the resulting inflammatory response, production of inflammatory cytokines, IL-6, which has been demonstrated significantly elevated in our patients, as well as activation and neutrophils causing more tissue damage(7), including damage to the capillary endothelium, resulting in microvascular thrombosis and VTE [4.7.8.].

CONCLUSION

High values of d-dimer could be related to a higher activation of blood coagulation in COVID-19 patients secondary to a systemic inflammatory response syndrome – or as a direct consequence of the SARS-CoV-2 itself. Pulmonary thrombosis was the confluence of processes, endothelial inflammation with no evidence of DVT . Tissue factor, upregulated on platelets, leucocytes during inflammation, leading to activation coagulation pathways and promote the formation of fibrin. The profound hypoxaemia is a likely driver of vasoconstriction, inflammation and thrombosis. The origin of Covid-19-associated pulmonary emboli and lung microcirculatory thrombotic disease: Interaction of inflammation and coagulation.

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