WWJMRD 2020; 6(9): 23-27
www.wwjmrd.com
International Journal
Peer Reviewed Journal
Refereed Journal
Indexed Journal
Impact Factor MJIF: 4.25
E-ISSN: 2454-6615

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COVID-19 Case Complicated By Massive Embolism

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Abstract
Objective:
In COVID-19 patient high rate of thromboembolic complications has a high hospital mortality. The early pathogenesis in COVID-19 pneumonia " defined by a widespread endotheliitis affecting multiple organ systems, viral inclusion are observed within endothelial cells accompanied by apoptosis, inflammatory cell infiltration and microvascular thrombosis" (Huertas *et al*.). 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.
Conclusion: High values of d-dimer reveales activation of blood coagulation in COVID-19 patients, like a result of systemic inflammatory response syndrome. Hypoxaemia itself lead to vasoconstriction, inflammation and thrombosis. Presented case of Covid-19-associated pulmonary massive embolism, was a result of inflammation and coagulation, consequence of this processes without revealed evidence of DVT.

Keywords: Thrombosis, Pulmonary embolism, inflammation

Introduction

In COVID-19 patient high rate of thromboembolic complications has a high hospital mortality. The early pathogenesis in COVID-19 pneumonia " defined by a widespread endotheliitis affecting multiple organ systems, viral inclusion are observed within endothelial cells accompanied by apoptosis, inflammatory cell infiltration and microvascular thrombosis" **(**Huertas *et al*.). The infection initiates inflammatory response, production of inflammatory cytokines, IL-6, activation neutrophils causing tissue and capillary endothelium damage.

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 aur 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.

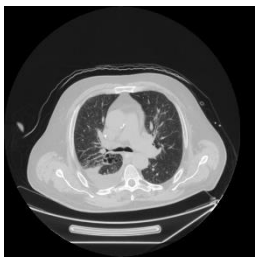
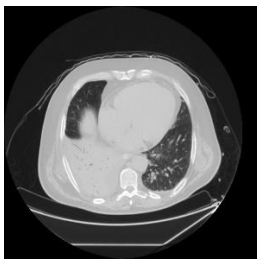
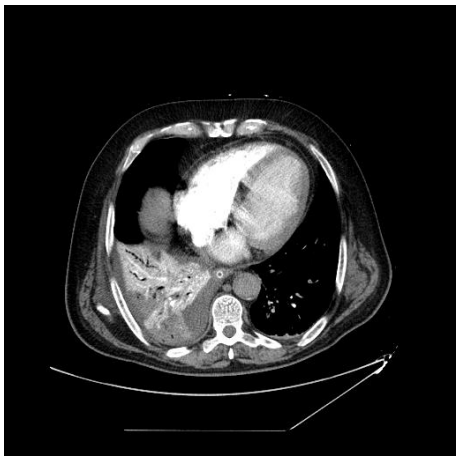
a.  b. 

Fig. 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

a.



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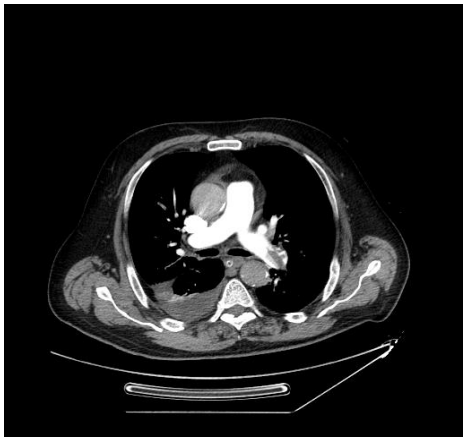


Fig. 2: 10.05.2020. Computed tomography, axial section. Vascular window. Incision at the level of basal segments.

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 disfunction, 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. PASP -40 mm.Hg. “Buble” test was negative.
Pulmonary Embolism Severity Index (PESI) 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) essesed 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

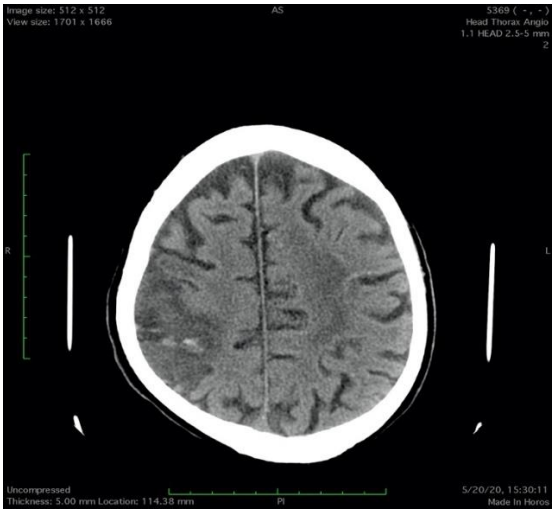


Fig. 3: CT scan of brain was detected acute haemorrhagic infarction (Hemorrhagic transformation after vein cortical thrombosis and cerebral infarction) in the right parietal lobe.

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

Laborattory finding:

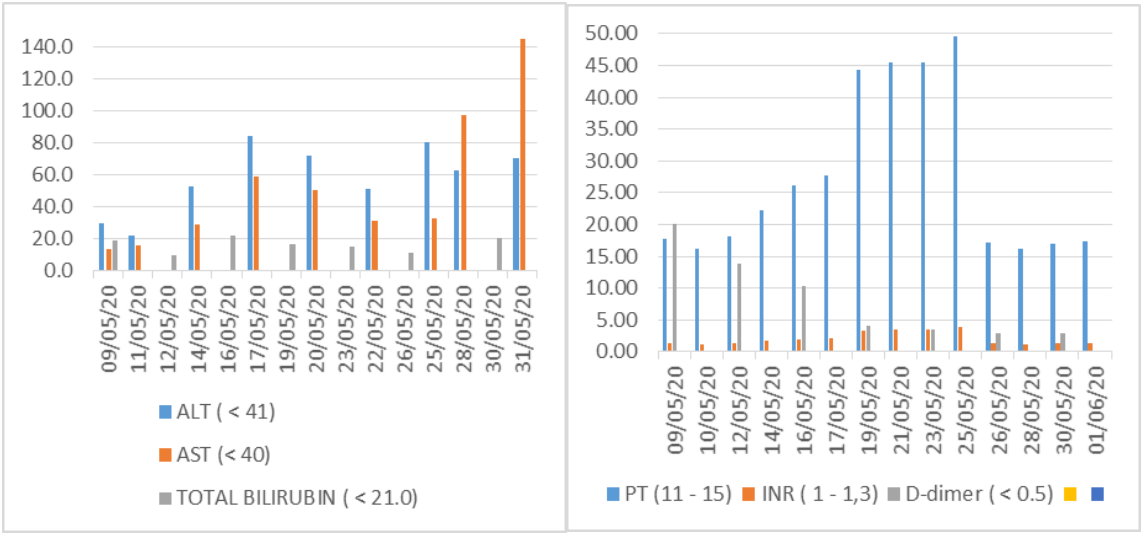


Table 1

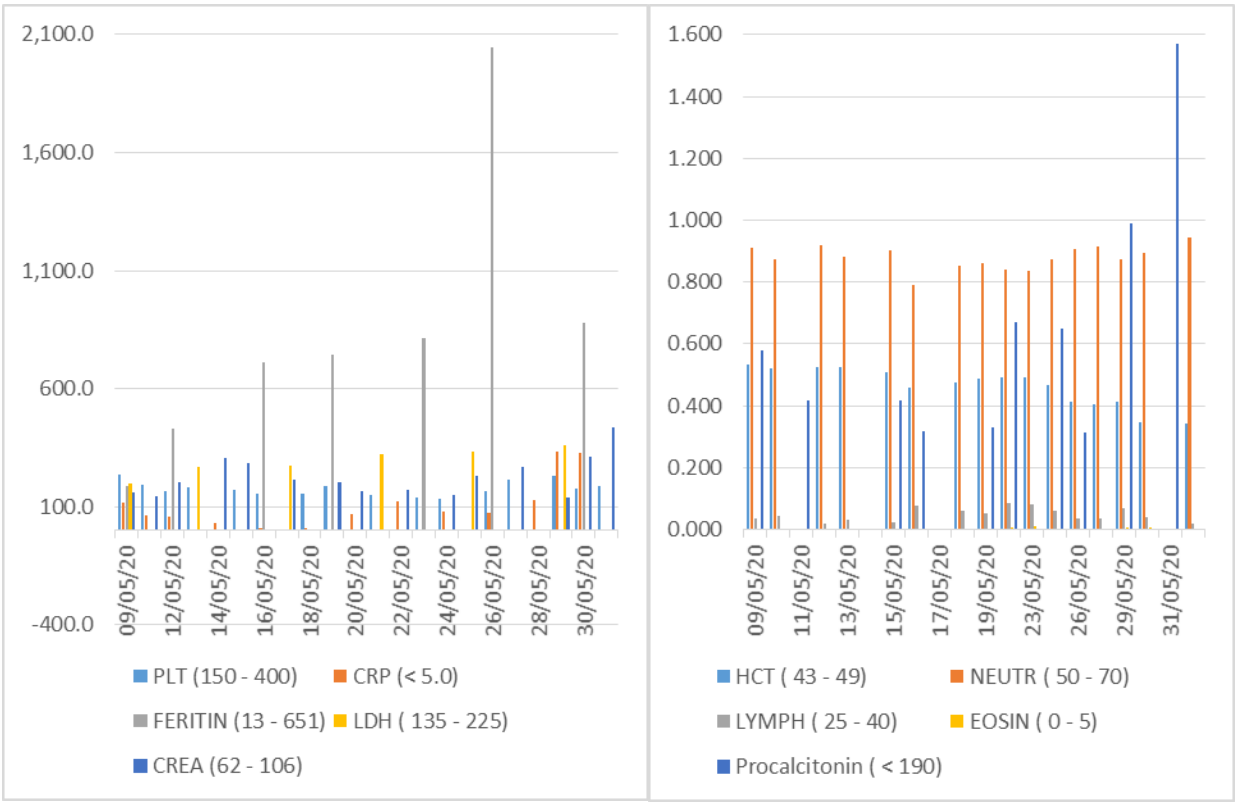


Table 2

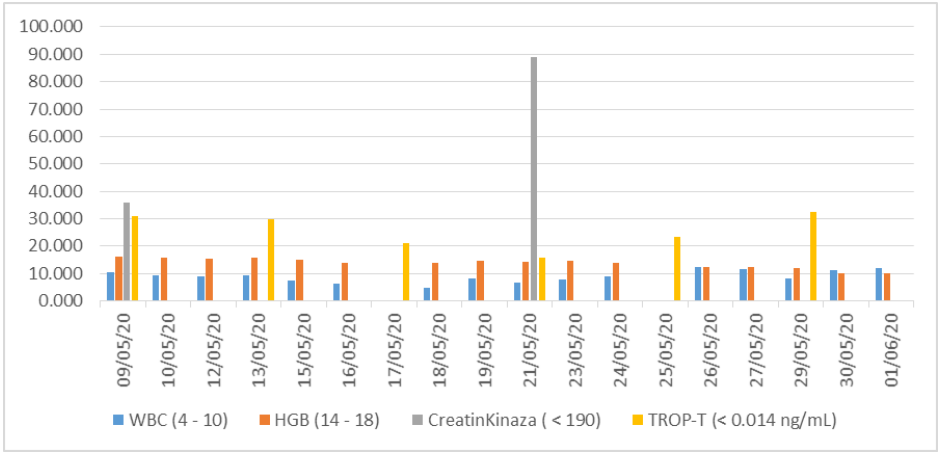


Table 3

Patient state was improuved. 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

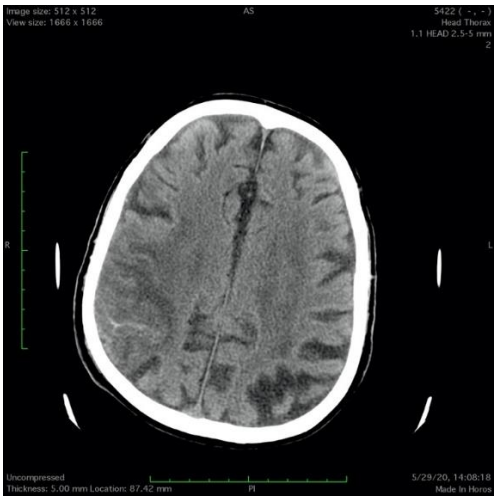


Fig.4: In the trunk of the pulmonary artery and in the main arteries a thrombus doas not revealed (Fig 5), but the volume of extensive inflammatory changes was reduced with thickening of interlobal pleuras.

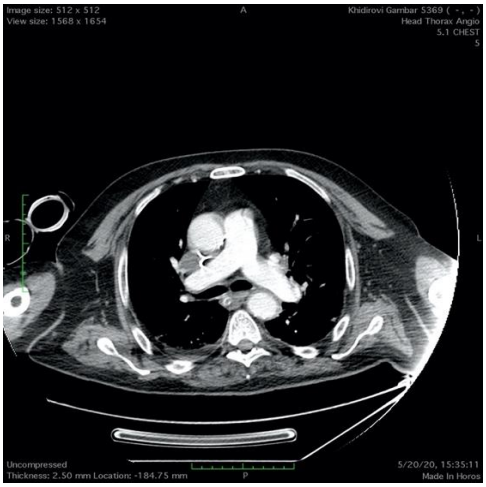
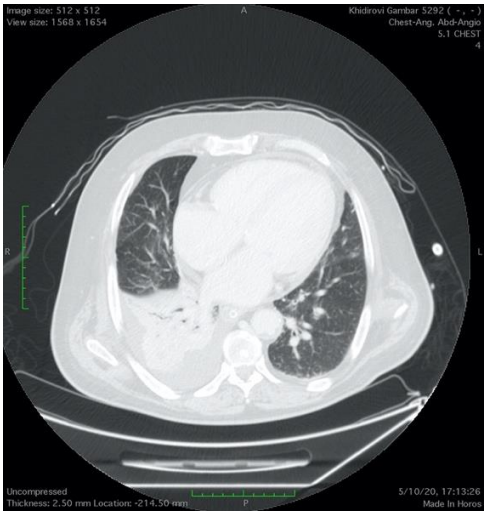


Fig. 5: Thrombotic masses are no longer reflected in the lumen of the bilateral main artery in the pulmonary trunk. Against the background of the existing consolidation, a small triangular avascular zone is revealed.

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.

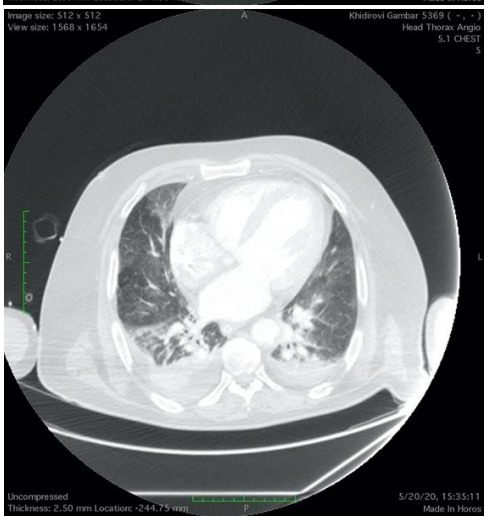


Fig.6: a. in the lower part of right lung extensive inflammatory and consolidation lesions, with airal bronchogramm. b. The volume of extensive inflammatory changes is reduced with thickening of interlobal pleuras.

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, coagulation activation, massive embolism, acute respiratory distress syndrome,coma and multiorgan failure we have described as main features of this severe COVID-19 ilnes patient.

The infection initiates cells injury and results production of inflammatory cytokines, IL-6, which has been detected elevated in our patient.

Conclusion: High values of d-dimer reveales activation of blood coagulation in COVID-19 patients, like a result of systemic inflammatory response syndrome. Hypoxaemia itself lead to vasoconstriction, inflammation and thrombosis. Presented case of Covid-19-associated pulmonary massive embolism, was a result of inflammation and coagulation, consequence of this processes without revealed evidence of DVT

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