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Rethrombosis in Covid-19 Patients

Guliko Kiliptari [1],Grigol Nemsadze [2]

[1] Head Of Critical Care Departament, Dr,Phd, Professor

[2]*Head Of Department* Computed Tomography And Magnetic Resonance Imaging,Dr ,Phd

Abstract – SARS-CoV-2 infection is associated with arterial and venous thrombotic complications. Autopsy findings revealed microthrombi in multiple organ systems, that may contribute to multisystem organ dysfunction in severe COVID-19. Thrombosis in patients with COVID-19 is due to a cytokine storm, hypoxic injury, endothelial dysfunction and increased platelet activity. Many difficult mechanisms contribute formation clots in vasculature of multi organs,

We presented two different cases of pulmonary artery thrombosis complicated by severe ARDS and rethrombosis in the branches of the pulmonary artery. Both patient was treated according PE(pulmonary embolism) management protocol (ESC)

Conclusion: The multisystem mechanisms: The complement system, part of the innate immune response, capable of activating the coagulation cascade, endothelial inflammation, loss of ACE2 and alteration of PAI/tPA balance, cytokine storm, high levels of the blood clotting protein factor, decreased concentrations of endogenous anticoagulant proteins increasis probability of new thrombosis, despite suitable treatment and prevention.

Keywords – Covid-19, PE, Rethrombosis.

I. INTRODUCTION

SARS-CoV-2 infection is associated with arterial and venous thrombotic complications. Autopsy findings revealedmicrothrombi in multiple organ systems, that may contribute to multisystem organ dysfunction in severe COVID-19.Thrombosis in patients with COVID-19 is due to a cytokine storm, hypoxic injury, endothelial dysfunction and increased platelet activity. Many difficult mechanisms contributes formation clots in vasculature of multi organs ,

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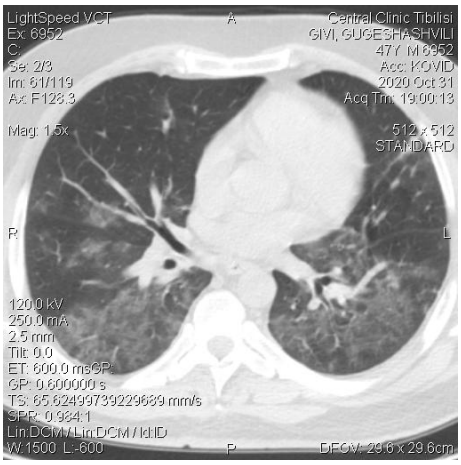
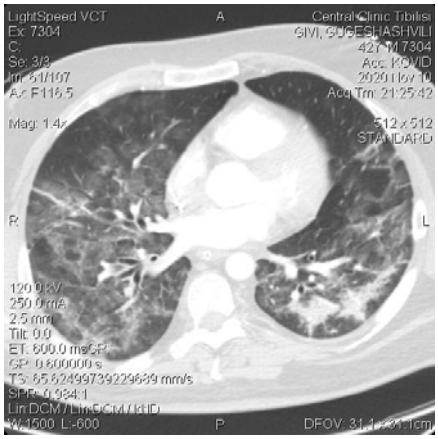
Patient N 1 .



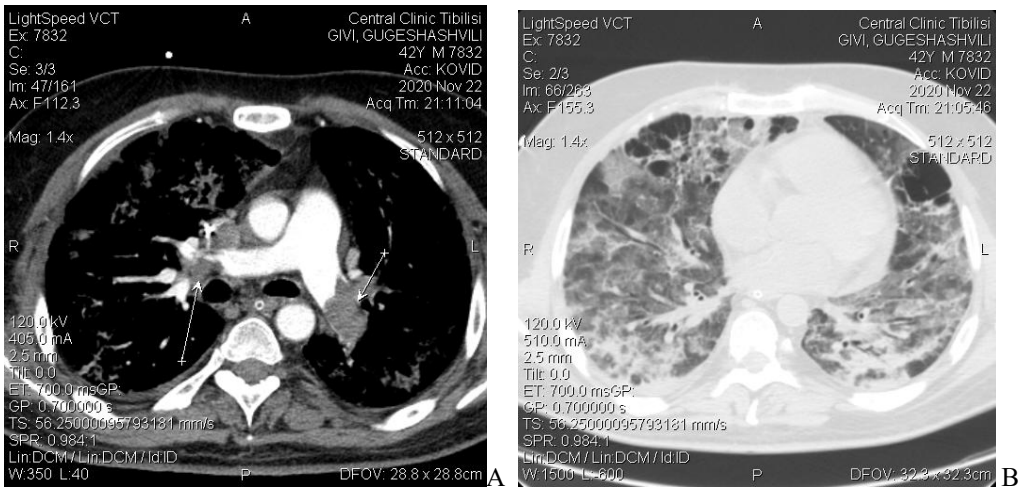
Pict 1. Computed tomography of the chest, axial section, pulmonary window.

On all sides, all fields are covered with foci of ground glass type infiltration and interstitial consolidation . Free fluid and air are not reflected in the bilateral pleural space. On semi-quantitative analysis of computed tomography data, the lung injury index was 18 points (0-24).[pict.1]



Pict. 2 Computed tomography of the chest, axial section of the pulmonary window.

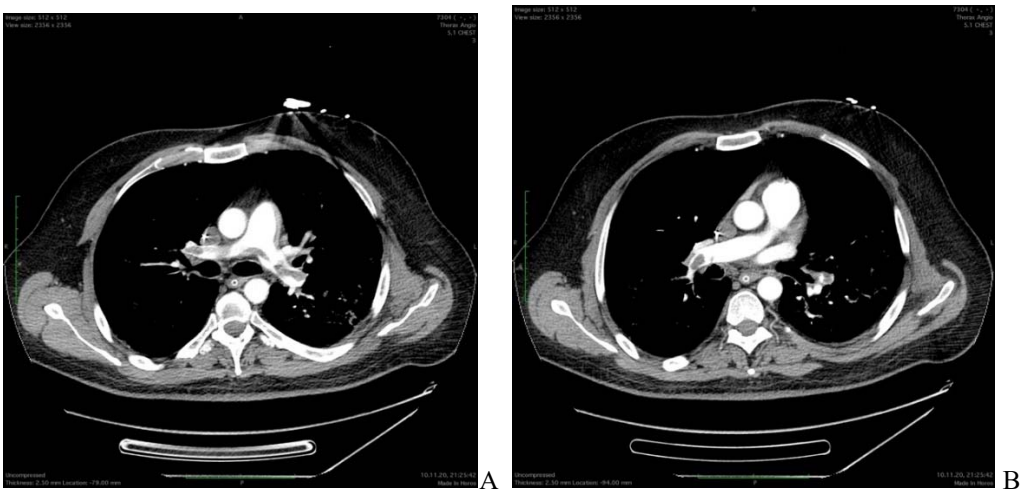
The volume and intensity of infiltrative changes increased in all bilateral lung fields. Areas of consolidation have been identified.In a semi-quantitative analysis of computed tomography data, the lung injury index was 21 points (0-24).[pict2]



Pict 3 Computed tomography of the chest, axial section of the mediastinal window

A. On both sides, thrombotic formations are observed in the right and left main arteries of the lung, in the lumen of the parietal and segmental branches.

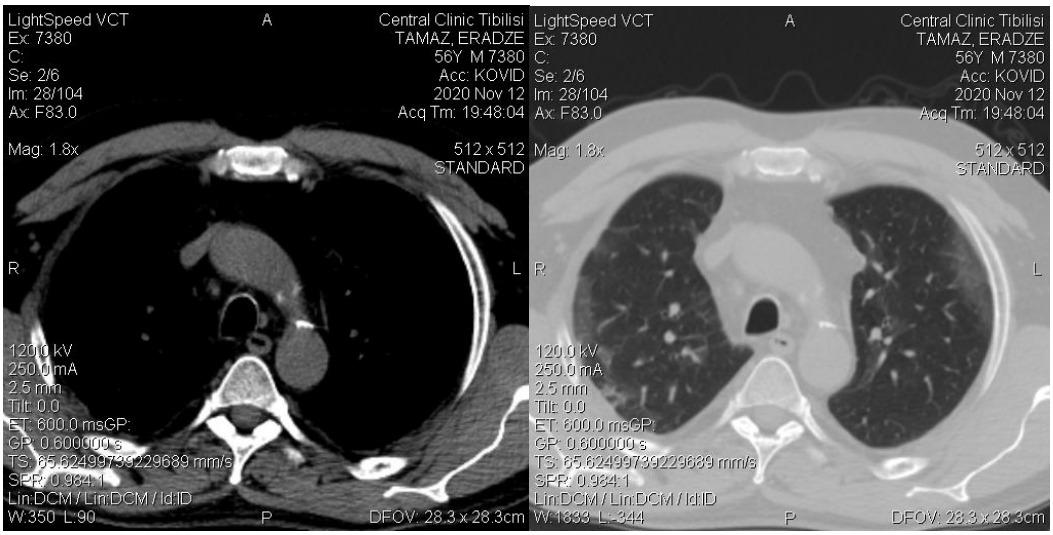
B. The volume and intensity of the infiltration increase. In the ventral segments, areas of lung tissue rupture were revealed. Semi-quantitative methodological analysis of computed tomography data, lung lesion index - 24 points (0-24).[pict.3]



Picture 4

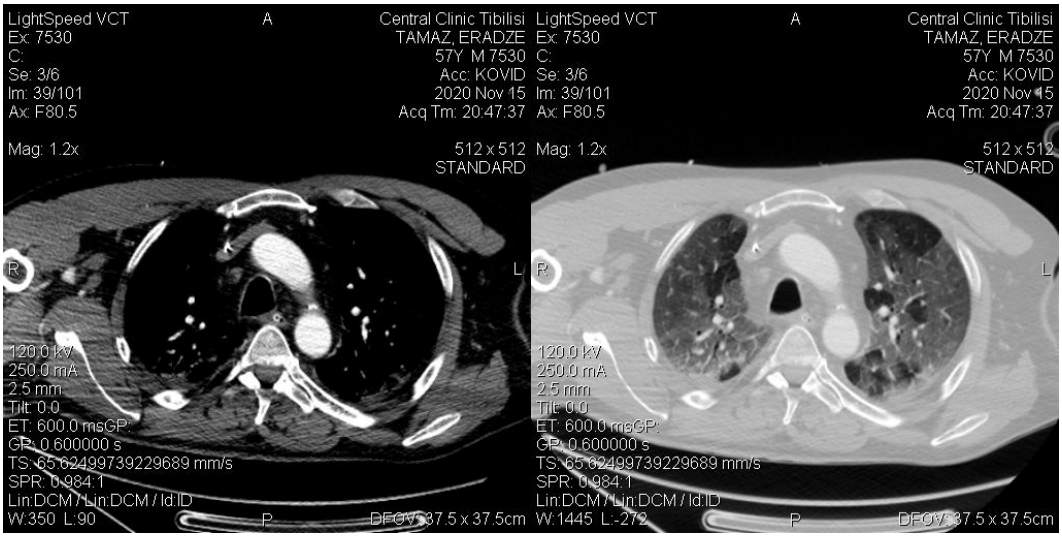
C. Angiographic examination does not reveal reliable signs of thrombosis of the lung trunk. Spreading from the level of the lung trunk bifurcation, thrombotic masses are reflected in the lumen of both large bronchi. Saddle thrombus. Thrombotic formations are reflected in the bilateral lobal and segmental arteries.

Patient N 2



Pict 1.

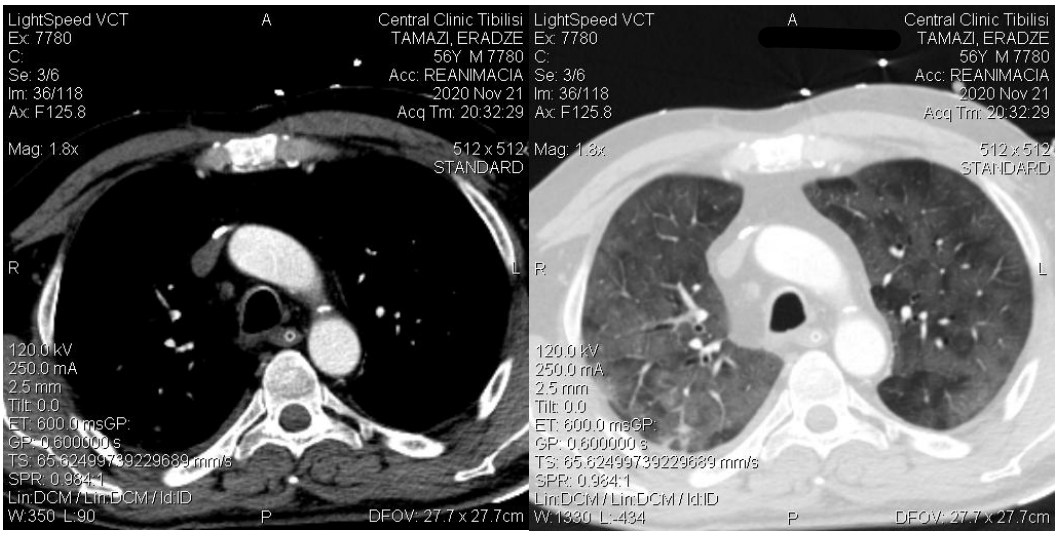
On both sides, there is an infiltrative change in the lungs in the form of a ground glass phenomenon without fluid in the pleural cavity. Semi-quantitative methodological analysis of computed tomography data, lung lesion index - 17 points (0-24).[pict1]



Pict .2

Angiographic examination does not reveal pulmonary thrombosis. The lumen of small arteries in the lower left corner is not completely contrasted, which suggests the presence of thrombotic masses. On both sides of the lung, there is a typical phenomenon of total ground glass, consolidation dorsally in the lower fields. Free fluid and air on both sides of the pleura are not detected

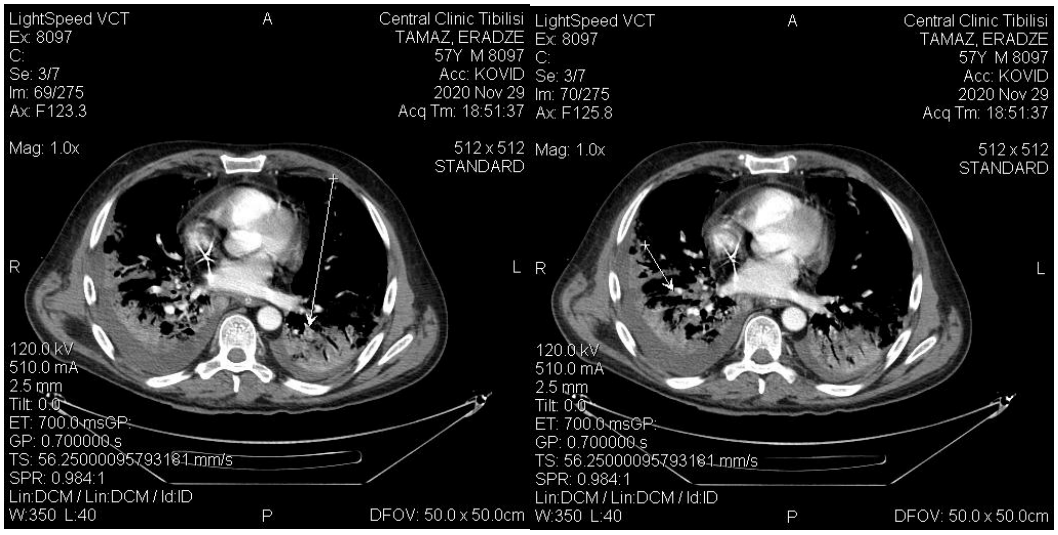
In a semi-quantitative analysis of computed tomography data, the lung injury index was 23 points (0-24).[pict.2]



Pict 3.

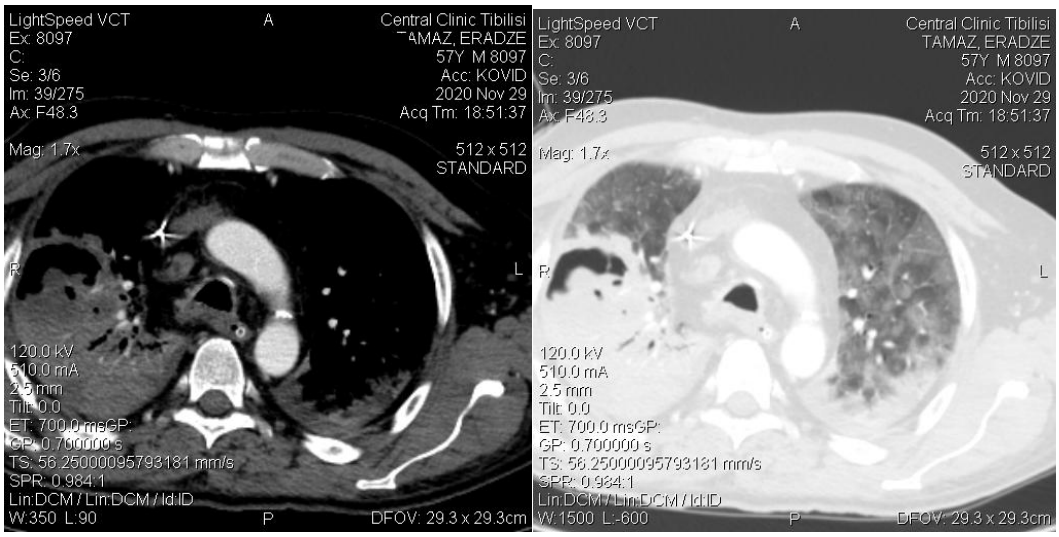
There is still lesion on both sides of the lungs, an almost entirely common graund glass phenomenon, although there is a relative decrease in intensity.

In the bilateral pleural cavity, a small liquid discharge, free air is not reflected. In a semi-quantitative analysis of computed tomography data, the lung injury index was 23 points (0-24).[pict.3]



Pict. 4

Angiographic examination showed no filling defect in the lung trunk and bilateral main artery. Thrombosis in the right lower artery is increased, in the left basal thrombus masses are reduced and reflected in the lumen of the segmental arteries. Shown by arrows.[pict.4]



Pict . 5

The intensity of the lesion was increased according to the type of ground glass phenomenon, which is totally widespread in both lungs; in the lower fields, extensive areas of consolidation appeared, against the background of which zones of avascular infarction pneumonia are revealed. Foci of destruction - 5.2 cm laterally in the right middle field.

The fluid is detected in the bilateral pleural cavity, the separation on the right is 2.2 cm, on the left is 1.0 cm, free air is not reflected.

In a semi-quantitative analysis of computed tomography data, the lung injury index was 24 points (0-24).[pict.5]

II. Discution

The pathophysiology of thromboembolism in COVID-19 related to endothelial inflammation, hypercoagulability associated with increased concentrations of coagulation factors, acquired antiphospholipid antibodies, and decreased concentrations of endogenous anticoagulant proteins. loss of ACE2 alters PAI/tPA balance High levels of the blood clotting protein factor V is at elevated risk from blood clots such as deep vein thrombosis to pulmonary embolism. Immune complex vasculitis is pathological mechanism of covid-19 pumonary artery thromboembolism , venous sinus thrombosis , cytokine storm. SARS-CoV-2 invasion of ACE-2 receptor expressing cells (airway epithelial cells, vascular endothelial cells, circulating monocytes), causes cellular damage, release of pro-inflammatory chemokines and chemoattractants (including C3a and C5a). The complement system is part of the innate immune response [1.2.], the complement system is an important host mediator of SARS-CoV-induced disease and that complement activation regulates a systemic proinflammatory response to SARS-CoV infection,complement system is overactivated and contributes to the dysregulated host immune response [3.4.5.]. the complement system activats the coagulation cascade, increases tissue factor activity [6.7.8.9.], increases platelet activity and aggregation [10.11.12.13.], as well as stimulate endothelial cells to release von Willebrand factor and express P-selectin [14.15.16]. Complement also regulates fibrinolysis, with complement cascade inhibitors [17], and activates the fibrinolysis inhibitors PAI-1(plasminogen activator inhibitor-1) and TAFI [Thrombin Activatable Fibrinolysis Inhibitor].

Such difficult and multisystem mechanisms contribute formation clots in vasculature of multi organs, Increases probability of new thrombosis, despite suitable treatment and prevention.

III. Conclusion

The multisystem mechanisms: The complement system, part of the innate immune response and capable of activating the coagulation cascade, endothelial inflammation, loss of ACE2 and alteration of PAI/tPA balance ,cytokine storm, high levels of the blood clotting protein factor, decreased concentrations of endogenous anticoagulant proteins increasis probability of new thrombosis , despite suitable treatment and prevention.

Abbreviation :

ARDS -Adult respiratory distress syndrome

ESC ----Europian society of cardiology

PAI-1----Plasminogen activator inhibitor-1

TAFI ---Thrombin Activatable Fibrinolysis Inhibitor.
ACE2 ---- Angiotensin-converting enzyme
t-PA-------Tissue plasminogen activator

PE——-pulmonary embolism

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