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Covid-19 as a Cause of Pneumonia and Diffuse Peripheral Pulmonary Embolism. Early Anticoagulant Treatment to Prevent Thrombi Formation

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The literature describes that viral infections can influence homeostatic balance. This means that during or immediately after virus infection, the ability of blood to clot in humans can be drastically altered, leading to thrombotic or hemorrhagic complications.

The direct or indirect inflammation of the endothelium of the vascular wall by viruses can cause, in fact, alterations of the coagulation. It is not yet clear why some viruses cause bleeding, while others cause thrombosis.

The COVID-19 seems to determine mainly the formation of thrombi. At the current state of knowledge, it is possible, in these patients, to identify two clinical pictures. In 80% of cases, the symptomatology corresponds to that of flu [1-3].

In comparison, in the remaining 20%, there is mono or bilateral pneumonia that can evolve towards a respiratory distress syndrome with diffuse damage to the alveolar capillaries. Despite the use of ventilatory supports, the clinical picture can precipitate quickly, and in about 2.5% of cases, it can lead to death.

The chest X-ray and above all the CT examination, performed in ICU patients, shows pulmonary thickening pictures and the presence of diffuse streaks that cannot be modified with the pronation and recruitment technique.

These radiological pictures and the presence of a positive Di dimer suggest that pulmonary complications may be related to a diffuse pulmonary embolism.

Due to the close anatomical relationship between blood vessels and pulmonary alveoli, it is, in fact, conceivable that the COVID-19 from the pulmonary alveoli reaches the capillary network and triggers an inflammatory process affecting the vascular endothelium. Endothelial inflammation can activate coagulation processes and the formation of progressive thrombosis.

This hypothesis could explain both the radiological pictures, commonly found in intensive care and the sudden death. Recent autopsy studies performed on 50 patients, who died for covid19, at the Brescia Hospital (Italy), have detected remarkable distension (up to 20 times) of the pulmonary vessels and the presence of thrombi inside them. Vascular dilatation and thrombi could be an expression of severe obstruction of the downstream pulmonary circulation.

Currently, in Italy, in many hospitals, in patients hospitalized for COVID-19 and pneumonia, for the suspicion of a possible onset of pulmonary embolism, anticoagulant therapy with low molecular weight heparin is administered.

It is conceivable that, from the beginning of the disease, the virus passes from the pulmonary alveoli to the microcirculation. In these positions, it simultaneously causes pneumonia and the formation of diffuse thrombosis. Subsequently, the thrombotic process can extend to a vast territory of the lung and cause widespread embolism of the microcirculation.

It could be the cause of many symptoms accused by patients and of sudden death. Likely, the thrombotic process in the alveolar capillaries starts simultaneously with the inflammatory process in the pulmonary alveoli.

To prevent, from the beginning, the formation of thrombi could be useful, in patients with positive buffer, the administration of anticoagulant drugs for 20 days. A large-scale study would be necessary, but currently, in Italy, this is not technically possible.

Pending these studies, in patients with COVID-19 positive buffer, in the absence of contraindications, the intake of enoxaparin 4.000U I think it is useful. This drug avoids the formation of thrombi and can prevent the onset of many serious complications.

Covid19 as a Cause of Diffuse Peripheral Pulmonary Embolism and Pneumonia. Early Anticoagulant Treatment to Prevent Thrombus Formation.

It has been described in the literature that viral infections of any kind can influence hemostatic balance. This means that during or immediately after infection with some viruses, the ability of blood to clot in humans can be drastically changed, which can lead to thrombotic or hemorrhagic complications. Direct or indirect activation of the endothelium by viruses can cause changes in coagulation and fibrinolytic systems. It seems that normally a well-regulated activation of coagulation is part of the body's defense against infectious agents. However, inflammation caused by some viral infections can lead to these changes in clotting. The clinical picture of impaired coagulation, in various viral infections, is manifested by thrombosis or bleeding. It is not yet clear why some viruses cause bleeding, while others cause thrombosis. (1,2,3) The covid19 seems to determine mainly the formation of thrombi. From the clinical behaviour observed in patients with Covid19, two clinical pictures can be identified. The first is similar to that of classical flu located in the upper respiratory tract, while the second, often serious and fatal, is attributable in the initial phase to pneumonia. This affects 20% of patients. A decrease in oxygen saturation in the blood, detected with an oximeter, seems to characterize the clinical picture in the initial phase. Subsequently, increasingly severe dyspnea appears followed by heart failure, which often requires the administration of norepinephrine.

The CT scan often shows a thrombotic impairment of the microcirculation and many of these patients have positive D-Dimer present usually when pulmonary embolism occurs.

These data suggest that complications caused by covid19 are mainly related to diffuse pulmonary embolism. It is conceivable

that the covid19, from the upper respiratory tract, reaches the venous bloodstream and the right heart from where it spreads into the pulmonary microcirculation. Here it alters the mechanisms of coagulation and leads to the formation of thrombi which progressively obstruct the microcirculation and cause a picture of diffuse pulmonary embolism. Pulmonary embolism quickly leads to desaturation and subsequently to right heart failure which can be the cause of death. To prevent thrombi formation in pulmonary microcirculation, we propose a large-scale study consisting of the administration of enoxaparin 4,000 U or a new oral anticoagulant for 20 days. The clinical behaviour of this group will be compared with the same number of patients with similar but untreated pathological characteristics. If clinical improvement in the group undergoing anticoagulant therapy is confirmed, we can reasonably say that this therapy can prevent the onset of all the serious complications that characterize this pathology.

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