Hypercoagulability, without Antiphospholipid Antibodies Disseminated Intravascular Coagulation in Covid-19

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COVID-19 is an infection with a status of hypercoagulability not well understood in which a critical vision is needed to understand it. The virus beta coronavirus contains an RNA of double lipidic chain with an S specular protein. The incubation period is for four days with a range of four to fourteen days. Fever is present in 77%, the cough in 46% to 52%, the dyspnea in 3 to 31% and the loss of smell and the taste by 5%.

Covid-19 may be presented in asymptomatically way, without symptoms until it reaches the moderate form in two weeks. The findings of lymphocytes of lymphocytosis and normal calcitonin, which indicates a normal saturation of oxygen and positive PCR that the case is mild to moderate. To understand the immunothrombosis in this pandemic detected by necropsies performed by the Italian and American groups. It is necessary to study the two immune systems.

To understand immune thrombosis in this pandemic detected by necropsies performed by the Italian and American groups it is necessary to study the two immune systems that are in humans. They are innate immunity and adaptive immunity.

This immunity is compromised in those patients with comorbidities as cancer patients, hematological diseases, immuno-suppressed patients with chronic pulmonary obstructive broncho disease, diabetes, high blood pressure, neurological diseases such as Parkinson’s, multiple sclerosis, amyotrophic lateral sclerosis, elderly, obese, patients receiving immunosuppressant’s such as transplant patients, dialysis patients, leukemia patients with myelocytic leukemia, lymphocytic, multiple myelomaemia, macroglobulinemia, myelodysplasia syndromes, no Hodgkin lymphomas Hodgkin, low-weight patients, elderly cough patients, patients with chronic ischemic heart disease, dilated cardiomyopathy, valvulopathies, cardiac post-surgical patients, coronary angioplasty, post replacement valvular percutaneous and patients with disabilities.

Innate immunity involves the first defense in which the crown is introduced to the epithelium or endothelium through the ACR receptors and the corona S protein in a process of endocytosis that process incorporates it into the cytoplasm and activation of macrophages interleukins occurs 16, 21 necrosis factor tumor MSP, cytotoxic-T cells and T-lymphocytes help them as well as the presence of interferon. These receptors like this are abundant in the lung so Covid-19 has pulmonary preference neurotropic, intestinal, blood vessels, kidneys, pancreas, heart, brain.

The introduction of Covid-19 into the endothelium, epithelium or alveolus works as the key is the protein from the crown and the lock is the receptor makes.

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This first attack system tries to self-limit the Covid-19 not always succeeding. If he escapes because the patient fails to self-delimit the disease, the adaptive response begins with the production of antibodies and destruction of the virus.

The crazy invasion of the virus causes a storm of destructive cytokines that causes it to reproduce to produce an exaggerated immune self-destructive response or storm with tissue and virus destruction.

The adaptive response may leave memory this would be important if you repeated the Covid-19 as an apoptosis occurred that is a programmed cell death response.

The great thing about our immunity is that we have a large army of Cytotoxic T, Lymphocyte Macrophages, Interleukins that cause 80% of patients to become infected and asymptomatic and 20% would be moderate mild symptomatic, or severe to the point of entering intensive therapy with multi-organic failure.

The Covid-19 has the ability to also block the interferon and minimize it is defense. Now as it is possible for a beta virus to crown spherical 100 to 160 gauges, polar positive 22 to 23 kilobase in length. Have a four-protein-encoded genome called a protein spike called the M protein called membrane and N protein that is in the nucleus.

This virus is able to disrupt the aiding T cells the CD4 the CD8 interleukin six the stimulant factor of granulocytes interleukin 10 causing a cytokine storm and producing micro vascular damage and activation of the coagulation system and inhibition of fibrillar by causing the antithrombin to be reduced in the coagulation cascade and achieving D-dimer levels and high fibrinogen.

The cause of the activation of interleukin six and the activation of coagulation the first mechanism is by stimulation in the liver of thrombopoietin that increases the fibrinogen increases the growth factor of vascular endothelium increases the expression of tissue monocytes and activation of the extrinsic system.

Thrombin can induce the vascular endothelium to produce more interleukin six and another place there is a feedback between cytokines and bleeding disorders so the theoretical use of the very map bacon to block interleukin six and decrease cytokines storm and coagulation. Other speculative damage would be platelet damage attacking directly from the bone marrow and activating the supplement.

How will we understand as a theoretical model the activation of the coagulation system?

Let’s see...How to activate the antiphospholipid antibodies fibrinogen degradation products?

ICO antiphospholipid syndrome is an autoimmune disorder that has a venous and arterial thrombosis clinic.

Laboratory findings include antibodies against anionic phospholipids such as anticardiolipin anti-antibody, antiphosphatidyl serine and other plasma proteins predominantly associated with beta-2 glycoprotein apolipoprotein H or evidence of circulating anti-coagulant antibodies.

Pathophysiology or thrombosis mechanism is not well defined. One hypothesis is a cellular defect in apoptosis, which exposes the phospholipid membrane that binds to plasma proteins such as beta-2 glycoprotein.

Once attached this phospholipid complex the protein works covertly and becomes the target of the auto antibodies. There is evidence to suggest that oxidized beta-2 glycoprotein is able to bind to dendritic cells in a similar way to 4PL receptor activation - four that would amplify the production of autoantibodies.

The hypercoagulability mechanisms with effect of these antibodies may not be dependent on beta one beta two glycoprotein.

We can say that there is production p or other mechanism of antibodies against coagulation factors including prothrombin protein is protein S and annexins also platelet activation increasing platelet adhesion activation of the vascular endothelium that facilitates the binding of platelets and monocytes and another mechanism is the reaction of antibodies to oxidized low-density lipoproteins that predispose to atherosclerosis and myocardial. Another mechanism would be activation of the supplement this would increase the positivity of anticardiolipin and antiphospholipid ICO antibodies this has been documented in repetitive loss in failed pregnancies.

There are 5% of patients with ICO antiphospholipid syndrome. That is 40 - 50 cases per 100,000 now with the Covid-19 pandemic two this has changed.

In ICO antiphospholipid syndrome fatal manifestations can be more than 50% with the presence of multi-organic heart attacks over a period of days and weeks. This syndrome is most common in African Americans and Hispanic populations that will have to be documented that the most serious and critical patients are the ones who have it. Differential diagnosis would be with hypercoagulability states such as malignancy contraceptive drugs, hyperhomocystinemia, antithrombin deficit three, protein deficiency S, Mutation five of Leiden, prothrombin.

Atherosclerotic disease even in cholesterol embolism, necrotizing vasculitis.

How is cid produced?

The intravascular coagulation disseminated by Covid-19 is characterized by the activation of coagulation and generation and deposit of fibrin with the presence of vascular micro thromboses in COVID-19 and it is appreciated is a coagulopathy of consumption without bleeding by activation of the vias procoagulants.

Laboratory tests in Covid-19 at baseline are leukocytosis thrombocytopenia, elevation of prothrombin, prothrombin time and elevated fibrinogen. Elevated anticardiolipin and GEA antibodies and anti-beta-2 glycoprotein one for IG and IGG. The international association of thrombosis and hemostasis classifies disseminated intravascular coagulation in Score from zero to four score zero.

They place the D dimer cut money in 2600 and with a thromboelastographic test, no thrombus is observed at 30 minutes. This is called fibrosis shutdown. These patients needed urgent hemodialysis 80%.

Prospective clinical studies are required to report that patients benefit from anticoagulation optimal anticoagulation regimens, which type which dose lasts.

The onset of disseminated intravascular coagulation ah includes decreased hemostatic components such as platelets, fibrinogen and coagulation factors. Disseminated intravascular coagulation can be acute asymptomatic and in Covid-19 what we have seen is that there is no bleeding but formation of intravascular thrombus that can give hyposular hypoxia multiorgan dysfunction and death.

By consumer coagulopathy or CID.

Excess thrombin production is the central element for intravascular coagulation as well as the conversion of fibrinogen into fibrin, thrombin has numerous relative effects on coagulation cascade. Thrombin contributes to the activation of factors five, seven, thirteen and direct effect on platelets.

The tissue factor thromboplastin and the intrinsic pathway are believed to be the one that initiates disseminated intravascular coagulation and this is the one that contributes to the pathophysiology of the latter unlike the initial part of Covid-19 that was a state of hypercoagulability without factor consumption.

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**Bibliography**


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