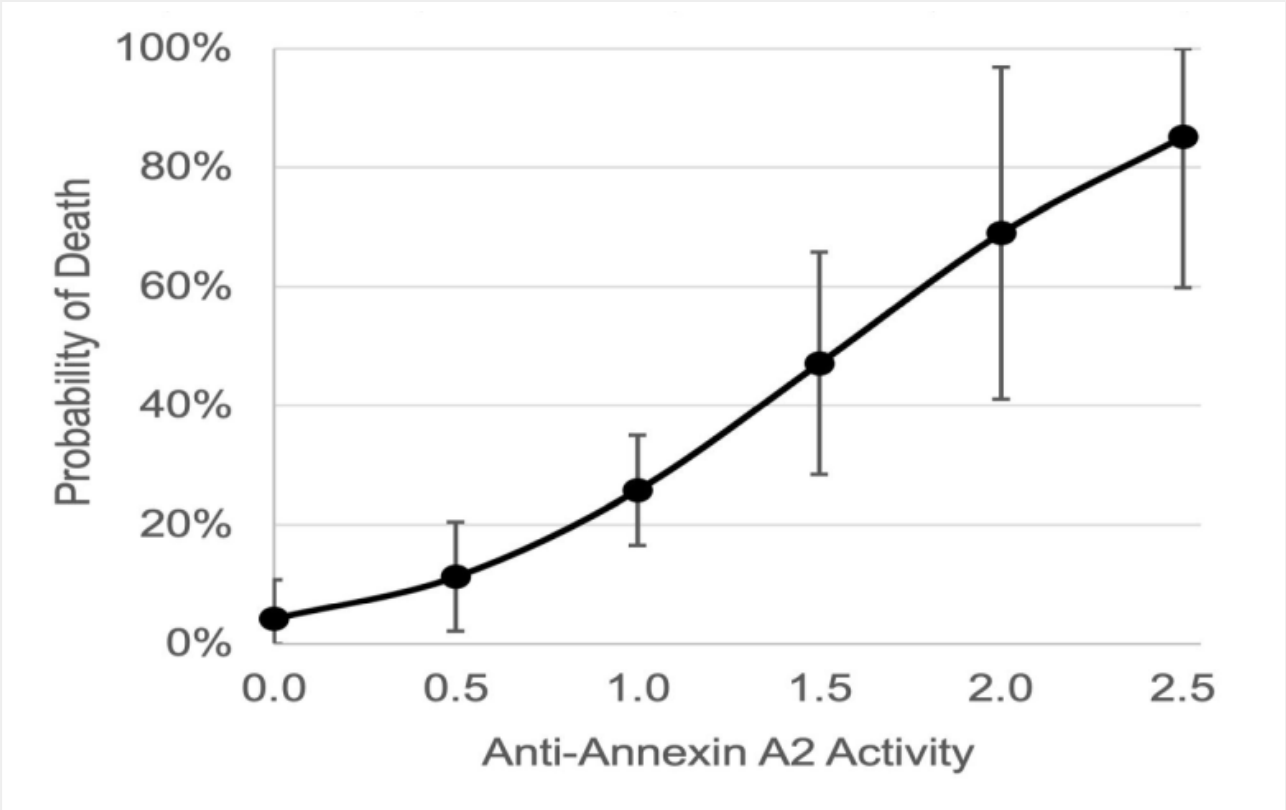
Swiss Policy Research

Severe covid: A postviral autoimmune attack



Autoimmunity to annexin A2 strongly predicts covid mortality (Zuniga et al)

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"Severe COVID-19 is not a viral pneumonia, but a post-viral autoimmune attack on the lung."

Note: Patients are asked to consult a doctor.

Already in 2020, multiple studies indicated that severe covid is not just a viral pneumonia, but might be a so-called anti-phospholipid antibody syndrome (APS), i.e. an autoimmune attack against phospholipids in endothelial cells causing hypercoagulation, thrombosis, and respiratory failure.

A new study, published in the European Respiratory Journal, now seems to confirm this hypothesis. Senior author Dr David Lee concludes: “Severe COVID-19 is not a viral pneumonia, but a post-viral autoimmune attack on the lung. The target of this autoimmune attack is Annexin A2, a phospholipid-binding protein () ensuring integrity of the pulmonary vasculature and promoting lung elasticity. Antagonism of Annexin A2 would cause lung blood clots, pulmonary edema, and ARDS.”

The chart above shows how anti-Annexin A2 auto-antibodies among hospitalized covid patients strongly predict mortality. Dr. Lee concludes: “It’s time for us to fully reassess how we define the pathophysiology of COVID-19 and consider alternative explanations.” Indeed, the antiphospholipid theory may well explain why most attempts at treating hospitalized covid patients have failed.

The anti-phospholipid syndrome (APS) is already well-known from chronic auto-immune diseases, notably rheumatoid arthritis and lupus (SLE). One of the standard medications to prevent APS is, of course, **hydroxychloroquine (HCQ)**. HCQ inhibits APS thrombotic events by inhibiting the anti-phospholipid autoimmune response (the immunomodulatory effect of HCQ) and by inhibiting platelet activation (similar to aspirin). In fact, in patients with chronic autoimmune disease, HCQ and aspirin are often used in combination to achieve optimal results.

In the highly politicized covid debate, both proponents and opponents of HCQ falsely assumed that HCQ would act as an anti-viral drug, which it does not (it also doesn’t act as a zinc ionophore, as a Spanish study showed). As a consequence of this, the design of most HCQ covid studies was misguided (or simply fake, as in the case of Surgisphere’s Lancet “study” that fooled the world).

If HCQ doesn’t act as an anti-viral drug, it cannot prevent covid infection, cannot prevent general covid symptoms (like fever or anosmia), cannot prevent covid pneumonia, and hence cannot even prevent hospitalization due to pneumonia. The only effect one can hope for is prevention of anti-phospholipid syndrome and death if HCQ is administered early enough (or as a prophylaxis). In low-risk patients, who won’t develop APS, HCQ will have no measurable effect at all.

It is far from certain that HCQ is indeed effective against covid APS, but some of the largest early treatment studies did indeed find a protective effect. For instance, a recent retrospective Iranian study with close to 30,000 participants, published in the Journal of

International Immunopharmacology, found a reduction in hospitalizations of 38% and a reduction in deaths of 73%. (1)

Furthermore, most covid trials studied only a single drug at a time, basically looking for a "wonder drug", whereas in many viral and autoimmune diseases, multiple drugs have to be applied to achieve optimal results. In the case of covid, it is obvious that one should try to inhibit viral replication as well as the hyperinflammatory, autoimmune and prothrombotic response. Indeed, this is what most early treatment protocols try to achieve. Once the damage is done, it is often too late to treat.

Finally, it should be added that thrombotic and thrombocytopenic events after covid vaccinations (e.g. blood clots, strokes or bleeding) may also be due to a vaccine-induced APS response.

Notes

1) The mistaken claim that HCQ caused heart failure in covid patients has long been refuted, unless patients received toxic overdoses, as in the horrendous Oxford Recovery and WHO Solidarity trials. Moreover, unlike the older chloroquine, HCQ doesn't cause anemia in people with favism, either.

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