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COVID19 – the spike protein and blood clotting

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When COVID19 came along I was in the midst of writing my latest book on heart disease. What causes it – and what does not.

One section I was working on covers the wide range of conditions known as the vasculitis(es). I could immediately see a whole series of connections between COVID19, spike proteins, the immune system and blood clots. Some of which are deeply concerning, for reasons that should become apparent.

Before getting started, you can see an immediate problem here is there does not seem to be a plural form of vasculitis. A bit like octopus. You can have *one* octopus, but what happens then… two octupuses… or is it two octopi? Wars have been fought over less.

Anyway, a vasculitis is a condition whereby a factor, of some sort, causes damage to the vascular system. The vascular system being, essentially, the blood vessels and the heart. The suffix *itis* simplymeans inflammation. As in appendic*itis*, or tonsill*itis*. Or, in this case vascul*itis*.

There are many different vasculitis(es) or vasculiti? They range from Kawasaki's disease to antiphospholipid syndrome, rheumatoid arthritis, scleroderma, Sjogren's disease and suchlike. They are many, and varied, and quite fascinating. At least they are, to me.

In all of them you have two things in common… that are most relevant to this discussion. First, with any form of vasculitis, the body decides to attack the lining of the blood vessels – causing inflammation and damage. Second, the rate of death from cardiovascular disease goes up dramatically. In some cases, a fifty-fold increase. This was seen in young women with Systemic Lupus Erythematosus (SLE) with additional antiphospholipid syndrome[1].

Why does the body decide to attack itself? This is a good question that I cannot really answer. If I could, I would be claiming my Nobel prize, right now. However, I can say that, for various reasons, the immune system makes the decision that it doesn't like something about the lining of the blood vessels and believes it to have become 'alien' in some way. It then proceeds to attack. Which does not answer the question as to exactly *why* the attack happens? But it does tell you a bit about *what* happens.

Another major problem with vasculitis is that blood clots spring to life throughout the vascular system. This is because the blood is always ready to clot, at any time, and if you take away some of vital the anti-clotting mechanisms, the balance will be tilted firmly towards coagulation.

One of the most powerful anti-clotting mechanisms/systems is the protective layer that lines your entire vascular system, known as the glycocalyx. This is made up of glycoproteins (glucose and proteins stuck together). Under an electron microscope the glycocalyx looks like a tiny forest, or a badly mown lawn.

Many fish are covered with glycocalyx, which makes them very slippery, and difficult to get hold of. The glycocalyx also stops bacteria and viruses from gaining entry, in both fish and humans.

In your blood vessels, the glycocalyx protrudes out from endothelial cells, the cells that line all your blood vessels, and into the bloodstream. The layer of glycocalyx contains many, many, anticoagulant factors. Below is a short list of all the things the glycocalyx does:

The glycocalyx:

- Forms the interface between the vessel wall and moving blood.
- Acts as the exclusion zone between blood cells and the endothelium.
- Acts as a barrier against leakage of fluid, proteins **and lipids** across the vascular wall.
- Interacts dynamically with blood constituents.
- Acts as the "molecular sieve" for plasma proteins.
- Modulates adhesion of inflammatory cells and platelets to the endothelial surface.
- Functions as a sensor and mechano-transducer of the fluid shear forces to which the endothelium is exposed; thus, the glycocalyx mediates shear-stress-dependent nitric oxide production.
- Retains protective enzymes (e.g., superoxide dismutase).
- Retains anticoagulation factors, e.g.: Tissue factor inhibitor, Protein C, Nitric Oxide (NO), Antithrombin.

Complicated stuff – that hardly anyone has ever heard of.

Anyway, if you damage the glycocalyx, or damage the underlying endothelial cells that synthesizes the glycocalyx layer, you will tip the balance very strongly towards the creation of blood clots. These can then then stick to the artery, or vein, wall. Sometimes they will fully block a blood vessel, leading to such things as a stroke or heart attack.

The interaction between vasculitis and thrombosis has been a relatively unexplored area of medicine. But it remains critically important in many diseases:

'The relationship between inflammation and thrombosis is not a recent concept, but it has been largely investigated only in recent years. ***Nowadays inflammation-induced thrombosis is considered to be a feature of systemic autoimmune diseases such as Systemic Lupus Erythematosus (SLE), Rheumatoid Arthritis (RA), or Sjogren's Syndrome (SS)[2].***

In super-short version. If you damage the lining of blood vessel walls, blood clots are far more likely to form. Very often, the damage is caused by the immune system going on the attack, damaging blood vessel walls, and removing several of the anti-clotting mechanisms.

Sepsis

Moving sideways for a moment. There are other things that can damage the blood vessel wall, leading to widespread blood clot formation. One of them is the condition known as sepsis. Which used to be called blood poisoning.

In sepsis, bacteria gain entry to the bloodstream through such things as a cut, an insect bite, a severe urine infection, and suchlike. When bacteria get into the blood, and start multiplying, they release exotoxins. Which are, effectively, the waste products of the bacteria.

These exotoxins then attack blood vessel walls, damaging the glycocalyx and endothelial cells. This drives the formation of blood clots throughout the body. The medical term for this is disseminated intravascular coagulation (DIC) = widespread blood clots in the vascular system.

The attacks not only cause clots, they can also cause the smaller blood vessels to weaken and burst. Which is why one sign of an infection with the meningococcal bacteria (the one that causes meningitis), is a rash. The rash is made up of dark, almost black, bruises. Once these start to appear, things are very bad. Potentially fatal, it means blood vessels are under severe attack and are breaking apart. Creating both bleeding and clots.

In truth, the 'rash' in meningitis is not really a rash at all. It is a sign of underlying, severe, vasculitis. The individual small bruises can also be called petechiae. Just to be scientific.

Another sign of widespread blood vessel damage, with the formation of multiple blood clots, is that the level of platelets in the bloodstream falls dramatically. For those who have never heard of such things, platelets are small cells that float about in the bloodstream. Their primary role is to co-ordinate the blood clotting system. If a red blood cell was the size of the Earth, a platelet would be about this size of the Moon.

If there is damage to blood vessels, platelets fling themselves at the area, and stick together to form a solid plug. They also release chemicals and enzymes that cause fibrin to be formed. Fibrin

is the long sticky strand of protein that binds clots tightly together. Platelets also drag in red blood cells, and suchlike to make bigger and tougher clots. They have been called the conductors of the clotting orchestra.

In the process of doing all of these things, the number of platelets starts to fall. This is not surprising, as they are being used up to make blood clots/thrombi. Which means that one sign of widespread clot formation is a fall in the level of platelets (thrombocytopenia). This reliable sign of widespread coagulation, or disseminated intravascular coagulation (DIC).

Time for a quick re-cap.

What do we know?

What we now know, on the journey towards COVID19, are three important things.

- If you damage the endothelial cells/glycocalyx, blood clots will form and stick to the side of blood vessels.
- Damage is often caused by immune system attack.
- Falling platelet levels are a sign of widespread blood clotting.

COVID19

What do we know about COVID19? First, it can only enter cells that have a receptor known as the angiotensin II receptor (ACE2 receptor). Cells with these receptors are mainly found in the lining of the lungs, and endothelial cells that line all blood vessels. Also, the epithelial /endothelial cells than line the intestines. If a cell does not have an ACE2 receptor, COVID19 simply cannot gain entry.

This was known years ago, when SARS-CoV was identified, the precursor of SARS-Cov2. Here from a paper in 2004:

'The most remarkable finding was the surface expression of ACE2 protein on lung alveolar epithelial cells and enterocytes of the small intestine. Furthermore, ***ACE2 was present in arterial and venous endothelial cells and arterial smooth muscle cells in all organs studied****. In conclusion, ACE2 is abundantly present in humans in the epithelia of the lung and small intestine, which might provide possible routes of entry for the SARS-CoV.* ***This epithelial expression, together with the presence of ACE2 in vascular endothelium, also provides a first step in understanding the pathogenesis of the main SARS disease manifestations****[3].'*

So, SARS-CoV gets into the body through the lungs and bowels. These are the places where the virus can gain access because it is where ACE2 receptors can mainly be found. Of course, SARS-Cov2 gets into the body in exactly the same way.

What happens once SARS-Cov2 gets into cells? Well, it does what all viruses do. It takes over various cellular mechanisms and forces the cell to produce more SARS-CoV2 viruses. This then kills, or severely damages those cells. This mainly occurs when 'virions' start to escape from within the cell. This damages the cell membrane, and in some cases can cause the cell to burst apart.

Essentially, SARS-Cov2 starts by damaging endothelial cells in the lungs, because it usually arrives here first. Fluid is released, and there is the breakdown of small blood vessels in the lungs, and the small airways. In this situation, the lungs begin to fail, and oxygen levels in the blood can fall dramatically.

Infection can also cause diarrhoea, as the epithelial cells in the intestines are damaged. To quote from '*the COVID19 symptoms'* study:

'We think COVID-19 causes diarrhoea because the virus can invade cells in the gut and disrupt its normal function [4].'

As far as I know, no-one has died of COVID19 diarrhoea. However, COVID19 can create such severe lung damage that people have died from respiratory failure or lung damage… call this form of disruption what you will. However, many/most people survive this phase.

It is what happens next that that kills the majority of people who become severely infected.

What happens next is that SARS-Cov2 gets into the bloodstream. It then invades endothelial cells, also pericytes and myocytes in the heart. Both of which have a high level of ACE2 receptors. Both of which are kind of vital for heart function [5,6].

Then…

What we now have is a major widespread vasculitis on our hands, with severe endothelial cell damage and disruption and damage to the glycocalyx. Blood clots, blood clots, blood clots, everywhere.

'Coronavirus disease 2019 (COVID-19) causes a spectrum of disease; some patients develop a severe proinflammatory state which can be associated with a ***unique coagulopathy and procoagulant endothelial phenotype****. Initially, COVID-19 infection produces a prominent elevation of fibrinogen and D-dimer/fibrin(ogen) degradation products.* ***This is associated***

***with systemic hypercoagulability and frequent venous thromboembolic events**. The degree of D-dimer elevation positively correlates with mortality in COVID-19 patients. **COVID-19 also leads to arterial thrombotic events (including strokes and ischemic limbs) as well as microvascular thrombotic disorders** (as frequently documented at autopsy in the pulmonary vascular beds). **COVID-19 patients often have mild thrombocytopenia* and appear to have increased platelet consumption, together with a corresponding increase in platelet production**.[7]'*

*a low level of platelets

The spike protein

Then, of course, we have the spike protein to consider. If this is the thing that the immune system recognises and attacks – which it almost certainly is – then cells which are growing SARS-Cov2 inside them, which then express the spike protein on their surface as the virions escape, will be identified as 'the enemy'.

At which point, the immune system will start to attack the endothelium (and glycocalyx) in an attempt to wipe out the virus. This will tend to happen two or three weeks after the initial infection (sometimes sooner). This is after the immune system has had a real chance to identify the spike protein, then properly wind itself up to produce antibodies against it. This is the time of maximum attack on the endothelium.

This moment is often referred to as a cytokine storm. A point where every system in the immune system gets revved up and charges into action. At one point I wasn't sure if I really believed in the cytokine storm. But I do now think it is a real thing. It is almost certainly why steroids (which very powerfully reduce the immune response) have been found to reduce mortality in severely ill patients.

All of which means it may well be the body's own infectious disease defence system that creates much of the damage to the cardiovascular system. Not necessarily the virus itself.

Alternatively, it may be that the spike protein itself creates most of the blood clots. Here from the paper '*SARS-CoV-2 spike S1 subunit induces hypercoagulability*.'

'When whole blood was exposed to spike protein even at low concentrations, the erythrocytes (red blood cells) *showed agglutination, hyperactivated platelets were seen, with membrane spreading and the formation of platelet-derived microparticles*[8]*.'*

Translation. Introduce SARS-CoV2 spike proteins into bloodstream, and it makes it clot – fast. Which is a worry.

Vaccines

It is a worry because the entire purpose of vaccination against SARS-Cov2 is to force cells to manufacture the spike protein(s) and then send them out into the bloodstream.

So, quick recap again, what do we know?

We know that a very high percentage of the people who die following a COVID19 infection, die as result of blood clots. We also know that they can also suffer severe myocarditis (inflammation of the heart muscle), and suchlike.

We know that the spike protein can stimulate blood clots all by itself.

We know that the immune system attack on 'alien' proteins, such as the spike protein, can cause vasculitis.

We know that vaccines are designed to drive the rapid production of spike proteins that will enter the blood stream specifically to encounter immune cells, in order to create a powerful response that will lead to 'immunity' against future SARS-CoV2 infection.

We know that a number of people have died from blood clots following vaccination. To quote from the European Medicines Agency website report on the AZ COVID19 vaccine:

'The PRAC (pharmacovigilance risk assessment committee) noted that the blood clots occurred in veins in the brain (cerebral venous sinus thrombosis, CVST) and the abdomen (splanchnic vein thrombosis) and in arteries, together with low levels of blood platelets and sometimes bleeding [9].'

This was all pretty much predictable, if you understood what was going with SARS-CoV – nearly seventeen years ago.

My concern at this point is that, yes, we have identified very rare manifestations of blood clotting: cerebral venous sinus thrombosis (CVST) and splanchnic (relating to the internal organs or viscera) vein thrombosis (SVT). These are so rare that it is unlikely that anything else – other than a novel vaccine – could have caused them. I have never seen a case and I had never even heard of them before COVID19 came along. And I have spent years studying the blood coagulation system, and vasculitis, and suchlike.

So, if someone is vaccinated, then has a cerebral venous sinus thrombosis, or a splanchnic vein thrombosis, this is almost certainly going to be noted and recorded – and associated with the vaccination. Fine.

However, if there is an increase in vanishingly rare blood clots, could there also be an increase in other, far more common blood clots at the same time. If this was the case, then it would be far more difficult to spot this happening.

Millions and millions of people suffer strokes and heart attacks every year. Millions more suffer deep vein thrombosis and pulmonary emboli. In fact, around the world, tens of millions die each and every year as a result of a blood clots forming somewhere in the body.

That is a hell of a lot of background blood clotting noise. Which means that it could be extremely difficult to disentangle cause and effect, especially if you are not looking. If an elderly person is vaccinated, then dies of a stroke a couple of weeks later. What caused the blood clot that led to the stroke? It is unlikely that any doctor would record this as a post-vaccine adverse event.

To give you one example of the difficulty of disentangling cause and effect, when you are looking at very common events, a few years ago Merck launched a drug called Vioxx (an anti-inflammatory like ibuprofen, or naproxen but not exactly the same class of drug). It didn't go well. Here from the article '*Merck Manipulated the Science about the Drug Vioxx.'*

'To increase the likelihood of FDA (Food and Drug Administration) *approval for its anti-inflammatory and arthritis drug Vioxx, the pharmaceutical giant Merck used flawed methodologies biased toward predetermined results to exaggerate the drug's positive effects. Internal documents made public in litigation revealed that a Merck marketing team had developed a strategy called ADVANTAGE (Assessment of Differences between Vioxx And Naproxen To Ascertain Gastrointestinal tolerability and Effectiveness) to skew the results of clinical trials in the drug's favor.*

As part of the strategy, scientists manipulated the trial design by comparing the drug to naproxen, a pain reliever sold under brand names such as Aleve, rather than to a placebo.'

The scientists highlighted the results that naproxen decreased the risk of heart attack by 80 percent, and downplayed results showing that Vioxx increased the risk of heart attack by 400 percent. This misleading presentation of the evidence made it look like naproxen was protecting patients from heart attacks, and that Vioxx only looked risky by comparison. In fact, Vioxx has since been found to significantly increase cardiovascular risk, leading Merck to withdraw the product from the market in 2004.

Tragically, Merck's manipulation of its data—and the FDA's resulting approval of Vioxx in 1999—led to ***thousands of avoidable premature deaths and 100,000 heart attacks****.'*
https://www.ucsusa.org/resources/merck-manipulated-science-about-drug-vioxx

Yes, not exactly their finest hour. However, the point that I want to highlight from this sorry tale is that it is estimated that Vioxx caused 100,000 additional heart attacks, in the US alone, and *nobody noticed*. This figure was only worked out when researchers analysed the figures on increased risk, that had been seen in the clinical trials – at least the figures that were finally seen when Merck were forced to release the data.

You may think. How could one hundred thousand heart attacks simply be missed? Well, there are very nearly one million physicians in the US. If the heart attacks caused by Vioxx were evenly distributed, only one in five physicians would have seen anyone suffer because of taking Vioxx. In those physicians that did see one, or two, would they have made the connection? No, they would not. Not in a million years. There would not even be a record of any possible connection made.

Elderly person has a stroke, or heart attack. Elderly person took Vioxx. And…?

All of which means I am not gigantically concerned about CVST and SVT. Blood clots in these veins are rare, and remain rare, even after vaccination – and will never be missed, particularly when they happen in younger people. Because when younger people die, great efforts are made to establish the cause of death.

However, I can see no reason why these specific blood vessels would be targeted by blood clots. Perhaps there is some reason why clots only occur in the central venous sinus vein, or splanchnic vein following vaccination. If so, I have been unable to find out. I am more than willing to be educated on this.

Time to move on to the other worrying observation, that can be found within the report by the pharmacovigilance risk assessment committee (PRAC) – as mentioned above:

'The PRAC noted that the blood clots occurred in veins in the brain (cerebral venous sinus thrombosis, CVST) and the abdomen (splanchnic vein thrombosis) and in arteries, ***together with low levels of blood platelets and sometimes bleeding****.'*

One blood clot, in one relatively small vein, is not going to cause a low platelet level. Nor will it cause bleeding – a sign of very low platelet levels. Which means that those unfortunate people who developed CVST and SVT almost certainly had widespread problems with other clots as

well. Then, for reasons unknown, they triggered these forms of, vanishingly rare blood clot. The ones that killed them. The ones that were recognised – because they are so rare.

I shall finish here. You can join the dots yourself. Or not.

1: https://www.intechopen.com/books/pregnancy-thrombophilia-the-unsuspected-risk/thrombophilia-in-systemic-lupus-erythematosus-a-review-of-multiple-mechanisms-and-resultant-clinical

2: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4399148/

3: https://pubmed.ncbi.nlm.nih.gov/15141377/

4: https://covid.joinzoe.com/post/covid-symptoms-diarrhoea

5: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2614534/

6: https://academic.oup.com/cardiovascres/article/116/6/1097/5813131

7: https://www.karger.com/Article/FullText/512007

8: https://www.news-medical.net/news/20210310/SARS-CoV-2-spike-S1-subunit-induces-hypercoagulability.aspx

9: https://www.ema.europa.eu/en/news/astrazenecas-covid-19-vaccine-ema-finds-possible-link-very-rare-cases-unusual-blood-clots-low-blood

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