SPIKE CAUSES CLOTTING - MECHANISM & THERAPIES

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A. Spikes enter Circulation

For the first 7 days after the V, there is a secretion of S1 Spike proteins into the general circulation. This begins within hours after the V. This is caused by proteases cleaving spikes at the S1-S2 junction when they are presented ion the cell membrane surface. S1 segments then float freely through the blood circulation.

B. Spikes Bind to ACE2 Receptors

The Spike, as you well know, binds to and blocks ACE2 receptors. The spike has a great affinity for these receptors in the endothelial cells of -

lung

small intestine

vascular

kidney

heart

brain

liver

C. Angiotensin II Builds up

The job of ACE2 receptors is to trigger the production of Angiotensin II Converting Enzyme - which ,as the name suggests, reduces the level of Angiotensin II by converting it to something less harmful. When the Spike blocks these receptors, this causes Angiotensin II to build up.

D. Angiotensin II Releases Thrombin - Clotting Agent

Angiotensin II releases thrombin - a coagulation factor.

E. Angiotensin II Releases Endothelins - causing Vaso Constriction

Build-up of Angiotensin II causes release of endothelins which cause vaso-constriction. This is the main function of endothelins.

F. Endotheliitis Ensues

This pathological condition is called Endotheliitis (spelt with those 2 ii)

So you can see how this is going to cause clotting. We have -

- 1. narrowing of blood vessels
- 2. release of a clotting agent

In simple "plumbing" terms, if you narrow a pipe and increase the viscosity of the liquid, you cause a blockage.

Microvasculature is Main Area of Clotting

These events mainly occur in microvasculature - in the tiny capillaries. This is where the blood moves more slowly, and where blood vessels are already quite narrow. This results in edema and thrombosis. Hence the name thrombin (because it causes thrombi, or clots to form)

In a "plumbing" analogy, blockages are more likely to occur where the rate of flow is slower, and where the channel is narrower.

Potential Cure

- 1. **UPREGULATE ACE2**: It follows that drugs that can upregulate ACE2 receptor expression, will alleviate much of the clotting effects of the Spike proteins.
 - diminazine aceturate (DIZE) a small molecule that can be taken orally (200mg every 3 days)
- 2. **USE A VASO-DILATOR**: We could also use vaso-dilators to combat the vaso constriction. Vasodilators include
 - drugs such as sildenafil (viagra)
 - eating uncooked **beetroot** (high nitrate content converts to Nitric Oxide which dilates blood vessels quickly)
 - supplements such as arginine. Arginine increases the level of Nitric Oxide which dilates blood vessels
- 3. **USE A MILD ANTI-COAGULANT**: We could also use a mild anti clotting agent **baby aspirin** to combat the release of Thrombin.
- 4. **BLOCK THE SPIKE**: Drugs that bind to the spike and hence stop it from binding to ACE2 receptors in the first place will be of great benefit. These drugs include -
 - **Ivermectin** (sorry for the deliberate mis-spelling)
 - Suramin (found in pine needles can be taken as a pine-needle tea)
 - Epigallocatechin gallate found in green tea
 - Curcumin found in tumeric
 - **Apigenin** found in onions.

These four things would be really helpful for avoiding the formation of oedema and micro thrombi.

In Summary

Spike binds to ACE2 --> Build up of Angiotensin II --> release of Thrombin + release of endothelins ---> Clotting and narrowing of vessels --> Edema + Thrombosis

How You Can Help Others

If you know someone whom you suspect is suffering effects of clotting, or if they want to avoid clotting after the V, then it is important that **for 28 days** after the V, they reduce the probability of clotting by

- 1. **ASPIRIN**: taking an anti-coagulant such as **baby aspirin**
- 2. taking a vaso-dilator such as
 - a. **BEETROOT**: a small portion of raw uncooked **beetroot** each day. Note that cooking the beetroot breaks down the nitrate and nullifies its effect, so it needs to be uncooked.
 - b. **ARGININE**: an **arginine** tablet each day . Arginine is available at Holland and Barrets and at most health stores. It costs about £10 per bottle.
 - c. **SILDENAFIL**: A **sildenafil** tablet each day
- 3. Taking a Spike binder such as
 - a. **GREEN TEA**: Drinking plenty of green tea
 - b. **CURCUMIN**: Adding turmeric and curcumin to their meals.
 - c. **IVERMECTIN**: Obtaining Ivermectin.
 - d. SURAMIN: pine-needle tea.

How Long Are Spikes In The Circulation?

Most of the S1 (part of the Spike proteins) are produced in the first 7 days and peak in the blood stream on day 5.

By day 7 Spike antibodies start destroying the S1 particles.

However attack by T cells against human cells displaying the S1 fragments destroys the infected cells and causes release of the entire contents of the infected cells into the blood stream - which includes whole spikes. Consequently whole spikes appear in the blood stream after day 7 and persist up until day 28..

So the above recommendations need to be applied for 28 days after the V.

This information is provided to help those who have already taken the V. It should help reduce the damage caused by clotting.

Technical Ramble (Ignore this - just techy background stuff)

1. Notes on Mechanism of Endothelin Action

Endothelins activate the influx of Ca into cells by

the activation of Gq receptors leading to PIP2 conversion to IP3 and DAG. IP3 binds to ER ion channels to allow Ca entry.

the activation of Gq receptors that open cell membrane Ca ion channels, so Ca can flow in from outside Causes depolarisation of cells (increases their positive charge)

Influx of Ca is blocked by hyperpolarisation = decreasing the positive charge of cells.

Increasing a cells positive charge causes vaso-constriction.

Reducing a cells positive charge causes vaso-dilation

2. Notes on Mechanism of Sildenafil Action

Nitric Oxide causes vaso-dilation by activating Guanylate Cyclase to convert GTP into cGMP Nitric Oxide is produced by the body from Arginine and other nitrate compounds such as are found in beetroot.

Sildenafil is a PDE inhibitor. This means that it stops the breakdown of cGMP, therefore it increases the effect of Nitric Oxide

References

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