



I-PREVENTSM

VACCINE INJURY

**An approach to post-vaccine
cardiovascular and cancer care**

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ALLIANCE

Disclaimer

This document is primarily intended to assist healthcare professionals in providing appropriate medical care for patients who have received a COVID-19 vaccine. Patients should always consult a trusted healthcare provider before embarking on any new treatment.

There is very limited data on the clinical features, pathogenetic mechanisms, and pathological findings of patients who have had delayed complications related to the COVID-19 vaccine. In addition, there is no published guidance on how to avoid these complications. This guidance is, therefore, based on our assessment of the likely pathogenic mechanisms underlying these delayed complications (spike protein-related disease) and the limited available autopsy data.

Post-vaccine cardiovascular events and cancer

The vast majority of serious adverse events following vaccination occur in the two weeks immediately following a dose of the vaccine. Therefore, we previously suggested that patients who developed no adverse events in the 2 to 3 weeks following vaccination had ‘dodged a proverbial bullet’ and did not require specific interventions to prevent vaccine injury.

However, evolving data suggests this approach may not be optimal, for two reasons. First, some patients who otherwise had no adverse events from the vaccine appear to have delayed acute cardiac events (often leading to sudden death). This appears to peak between 4 to 6 months after the vaccine but may extend for at least one year. Second, there has been evidence of an emergence of “turbo” and relapsed cancers in the months following vaccination.

The approach to preventing these serious disorders is unclear and the developers and manufacturers of these ‘vaccines’ obviously did not develop an ‘antidote’. Nevertheless, we have developed this document to attempt to limit these complications and reassure those who have been vaccinated.

Essentially, both cardiac and cancer-related complications are related to the persistence of spike protein. Therefore, any intervention that reduces the persistence and the ‘load’ of spike protein will likely be beneficial.

The cause of the increased risk of cancers is less clear, however spike protein-induced alteration in the function of tumor suppressor genes, immune depression, altered mitochondrial function, and other metabolic pathways may be involved.

Delayed Sudden Cardiac Death

Sudden cardiac death has been recognized within weeks of the COVID-19 mRNA vaccination, likely due to an arrhythmia triggered by an acute myocarditis, [1-3] and/or a catecholamine surge and coagulative myocardial necrosis. [4]

More recently, delayed cardiac complications following COVID-19 vaccination have received attention in the “alternative media”. This devastating fatal condition has been ignored by the medical establishment. Typically, these are otherwise healthy individuals without features of the “vaccine injury syndrome” who “die suddenly” — usually 4 to 6 months after the last dose of the COVID-19 vaccine. However, the duration of the window for sudden death is unknown and likely extends up to 1 year or longer. As mentioned, many of these patients are otherwise healthy with no obvious risk factors or history of cardiac disease.

To date, the pathogenesis and pathology of this syndrome have not been reported in peer-reviewed publications. However, an unpublished autopsy of patients who died unexpectedly following vaccination demonstrated endothelial inflammation (most prominently in the heart, lungs, and brain), endothelial damage, rupture of atheromatous plaques, complex formation of amyloid-spike protein fibrin in vessels as well as platelet aggregates and thrombi leading to acute coronary events. In addition, lymphocytic vasculitis with medial necrosis and dissection of large vessels (aorta, coronary artery) was found. Expression of spike protein (and not nucleoprotein) was detected at high concentrations in the endothelium of capillaries, small arteries, and veins, as well as within the myocardium, aorta, and brain and aggregated in blood clots.

These findings suggest that spike protein-induced endothelialitis, with activation of clotting and medial necrosis of the blood vessel, are the major pathologic events leading to sudden death. It is important to emphasize that the pathology of this syndrome differs significantly from that of the typical finding of atherosclerotic coronary disease. However, it is unknown if these patients had underlying coronary artery disease (or risk factors for coronary artery disease) and the acute spike-related endotheliitis served to accelerate this process. It should be noted that subclinical atherosclerosis is common in Westernized populations. In the PESA study, subclinical atherosclerosis was highly prevalent in middle-aged individuals, with nearly half of the participants classified as having intermediate or generalized disease. [5]

As this catastrophic disorder is not recognized and has not been studied by the medical establishment, the true incidence and risk factors for sudden cardiac death remain unknown. Ideally, these asymptomatic patients would be risk stratified, with the initiation of prophylactic measures in the moderate to high-risk groups. Unfortunately, there is little data to allow for risk stratification. There are however two potential approaches to risk stratification, and these could be combined. The first is based on the lot number of the vaccine. It has been well established (from the VAERS data itself) that certain lots of the vaccine are associated with a much higher risk of adverse events than others; the adverse event rate of “hot” lots is increased

one thousand-fold. Presumably, these “hot” lots have a much higher concentration of mRNA, which translates into a much higher load of spike protein. The load of spike protein appears to be a major factor predicting the development of serious adverse events to the vaccine. Patients can check if they received a bad lot at the following website:

<https://www.howbadismybatch.com/>

The second approach to risk stratification involves using a composite of validated cardiac biomarkers. Dr. Gundry, a cardiac surgeon, performed a biomarker-based cardiac risk assessment score (the PULS Cardiac Test) in 566 patients 2 to 10 weeks following the second mRNA COVID shot and compared this score to the PULS score drawn 3 to 5 months prior to the jab. [6] The PULS score is a composite score based on the following tests: IL-16, MCP-3, Eotaxin, CTACK, Fas, Fas Ligand, HGF, HDL, HbA1C.

In this study, the 5-year Acute Coronary Risk Score (ACS) increased from a baseline of 11% to 25% after the jab. The PULS score is a cardiac risk assessment score reflecting the degree of atheromatous coronary disease and “chronic” endothelial inflammation rather than being a specific marker of acute endothelial inflammation and clotting. However, this is complex with some degree of overlap, as acute endothelial inflammation may precede alterations in the development of atherosclerotic change and progress to a chronic inflammatory process within the endothelial lining in atherosclerosis susceptible regions. [7] Furthermore, the components of the PULS score may be markers of insulin resistance, a major factor leading to coronary artery disease, rather than a direct index of endothelial injury. [8-10] In addition, the test is further limited by its expense and limited availability.

In the absence of data to guide risk stratification for delayed sudden cardiac death post “vaccination”, the following markers of endothelial activation and clotting may have utility (in combination) and require urgent investigation: [7,11]

- I. Transforming growth factor Beta (TGF- β)
- II. Matrix Metalloproteinase-9 (MMP-9)
- III. Thrombin-antithrombin complex
- IV. High-sensitivity C-reactive protein (HS-CRP)
- V. E-selectin
- VI. Interleukin-6 (IL-6)
- VII. Von Willebrand Factor (vWF)
- VIII. Hepatocyte growth factor (HGF)

Furthermore, those patients who have “classic” risk factors for coronary artery disease (i.e., metabolic syndrome (characterized by abdominal obesity and a BMI > 30 kg/m², prediabetes/type 2 diabetes), as well as a family history of type 2 diabetes, smoking, and hypertension) should be screened with the following tests:

- I. HbA1C
- II. TG/HDL ratio (triglyceride/high-density lipoprotein), which is the best predictor of coronary artery disease.
- III. Microalbumin/Creatinine ratio

Potential treatment approach

The primary approach to preventing delayed complications from vaccination is to enhance the body's ability to eliminate spike protein. This is best achieved by practicing intermittent fasting/time-restricted eating and with a supplement like resveratrol, which activates autophagy and encourages the removal of spike protein.

In addition, nattokinase, a naturally derived enzyme, breaks down extracellular spike protein and is a potent fibrinolytic agent, which breaks down blood clots.

Furthermore, treating hyperinsulinemia likely limits both endothelial inflammation and carcinogenesis.

We have added other interventions to this core treatment approach that likely have additional benefits. These include anti-platelet and fibrinolytic agents, which are central to the prevention of cardiovascular events following vaccination; the pharmacology, dosing, and precautions of these drugs are reviewed at the end of this document.

A suggested theoretical approach to limit the long-term complications of spike protein

- **Intermittent fasting/time-restricted eating** (activates autophagy and removal of spike protein). [12,13] For more detail, see [I-RECOVER: Post-Vaccine Syndrome](#) and an [FLCCC Guide to Intermittent Fasting](#). Fasting should be combined with a low-carbohydrate,

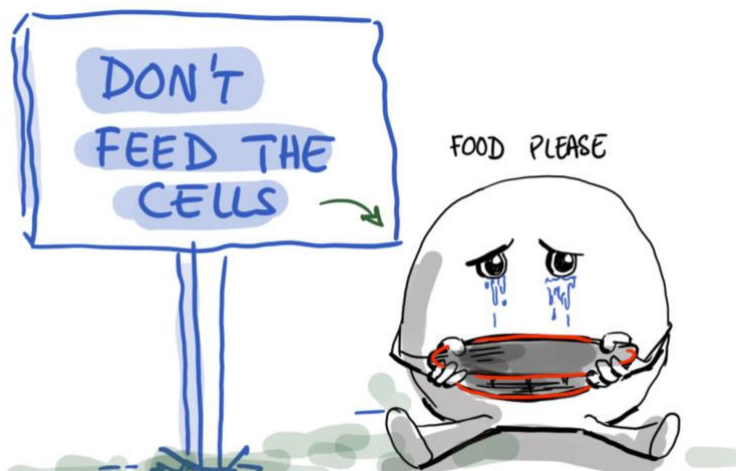


Figure 1 (Source: Dr. Mobeen Syed)

high-fat diet (ketogenic diet), low in Omega-6 vegetable oils (improves insulin resistance).

- **Nattokinase**; 100-200 mg twice daily.
- **Resveratrol**; 500 mg daily. Resveratrol has cardioprotective, anti-inflammatory, and anti-coagulant properties and augments autophagy. [14-19] Resveratrol also binds to spike protein, likely promoting spike removal. Generally, the oral bioavailability of resveratrol is poor. [20] However, a bio-enhanced formulation containing trans-resveratrol from Japanese Knotweed Root appears to have improved bioavailability.
- **Aspirin (ASA)**; 81 mg daily (in those with low risk of bleeding).
- **Magnesium**; 100-400 mg daily. A starting dose of 100 to 200 mg daily is suggested, increasing the dose as tolerated up to 300 mg (females) to 400 mg (males) daily. Generally, organic salts of Mg have a higher solubility than inorganic salts and have greater bioavailability. [21] Magnesium Malate, Taurate, Glycinate, and L-threonate have good bioavailability. Magnesium deficiency is associated with serious cardiac arrhythmia and all-cause cardiovascular mortality. [22,23]
- **Omega-3 fatty acids**; 2-4 g daily. Omega-3 fatty acids have anti-inflammatory properties and have been demonstrated to improve endothelial function and reduce cardiovascular events. [24-26]
- **Co-enzyme Q (CoQ)**; 200-400 mg/day. CoQ has antioxidant, anti-inflammatory, and cardioprotective effects. [27-32]
- **Melatonin**; 3-10 mg at night (slow release/extended release). Melatonin has anti-inflammatory and antioxidant properties and is a powerful regulator of mitochondrial function with proven cardioprotective effects. [33-38]
- **Bromelain 500 mg twice daily +/- N-acetyl cysteine (NAC) 600 mg twice daily.** *In Vitro* studies have demonstrated that bromelain cleaves the spike protein.[39,40] This effect appears to be enhanced by the addition of NAC.[41]
- **Berberine**; 500-600 mg twice daily. Berberine has anti-cancer, anti-diabetic, antioxidant, and cardioprotective properties. [42-44] Avoid in patients taking cyclosporine and during pregnancy and breastfeeding. For more information see [I-CARE: Insulin Resistance Treatment](#).

Figure 1. Time course of sudden death following vaccination

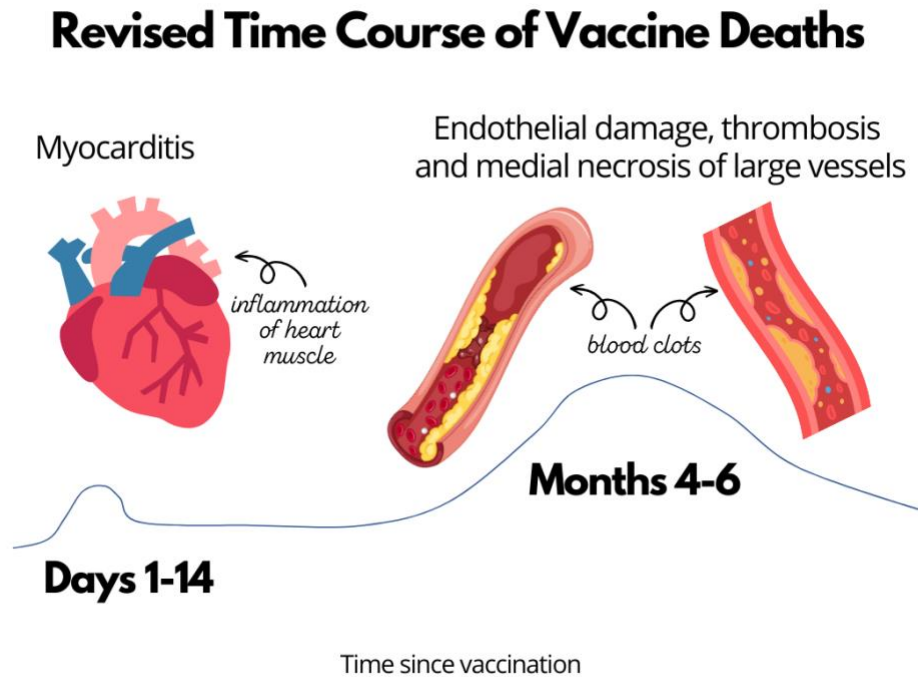


Figure 2 (Source: FLCCC)

Antiplatelet and Fibrinolytic Agents

Anticoagulants, anti-platelet drugs, and fibrinolytic agents are central to the prevention of cardiovascular events post-vaccine. The pharmacology of these agents is briefly reviewed below. The greatest risk with the use of anticoagulants, antiplatelet drugs, and fibrinolytics is clinically significant bleeding. A number of factors increase the risk of bleeding; [45-47] these include age (> 65 years; advanced age is a major risk factor for bleeding), hypertension, renal impairment, diabetes, previous stroke, a previous bleed, and male sex. Furthermore, the risk of bleeding increases exponentially as the number of anticoagulant/anti-platelet drugs is increased. [46,48] These risk factors need to be evaluated prior to embarking on any “anticoagulant” drug.

Antiplatelet drugs:

Aspirin (ASA): ASA produces a clinically relevant antiplatelet effect by irreversibly acetylating the active site of cyclooxygenase-1 (COX-1), which is required for the production of thromboxane A2, a powerful promoter of platelet aggregation. These effects are achieved by daily doses of 75 mg (and higher). The major adverse effect is bleeding. Bleeding most commonly occurs in the gastrointestinal tract and is rarely fatal. Bleeding also occurs at other sites, with intracranial bleeding being the rarest (approximately 4 per 10,000) but the most serious (with a 50% case fatality rate).

Clpidogrel (Plavix): Clopidogrel requires *in vivo* biotransformation to an active thiol metabolite. The active metabolite irreversibly blocks the ADP receptors on the platelet surface, which prevents activation of the GPIIb/IIIa receptor complex, thereby reducing platelet aggregation. Similar to ASA, platelets blocked by clopidogrel are affected for the remainder of their lifespan (~7 to 10 days). The usual dose is 75 mg daily.

Oral Fibrinolytic agents:

Nattokinase: Nattokinase (NK) is a serine protease purified and extracted from natto, a traditional Japanese (cheese-like) food produced from the fermentation of soybeans with the bacterium, *Bacillus subtilis*. [49-51] Recent studies demonstrated that a high natto intake was associated with decreased risk of total cardiovascular disease mortality and, in particular, a decreased risk of mortality from ischemic heart diseases. [52]

Nattokinase has potent fibrinolytic, antithrombotic, and antiplatelet activity. [49,50,53-56] NK degrades fibrin directly and also increases the release of tPA with a subsequent increase in the formation of plasmin. [57] Furthermore, NK enhances fibrinolysis through cleavage and inactivation of PAI-1. [51,56]

In a study comparing the antiplatelet effects of NK and aspirin, NK was shown to display excellent antiplatelet aggregation and antithrombotic activities *in vitro* and *in vivo*, inhibiting thromboxane B₂ formation from collagen-activated platelets. [58] In addition, in both animal and human studies, NK also has antihypertensive, anti-atherosclerotic, lipid-lowering, and neuroprotective actions. [50,56,59]

Of particular relevance to patients with spike-related clotting, nattokinase causes the proteolytic cleavage of both spike protein and amyloid proteins. [60,61] In a randomized study, NK proved to be more effective than statins (simvastatin) in reducing carotid artery atherosclerosis. [62]

Chen et al demonstrated that high dose NK (10 800 Fibrinolytic Units [FU]/day; ~ 500 mg/day) reduced the thickness of the carotid artery intima-media and the size of the carotid plaque. [63] The authors reported a synergistic effect between NK and aspirin/ASA. Studies indicate that an oral administration of NK can be absorbed by the intestinal tract. [59,64] NK, unlike most proteins, is more resistant to the highly acidic gastric fluids in the stomach and can be absorbed in the later sections of the digestive tract.

The optimal dose of nattokinase is unclear, however, a dose of 100-200 mg (2000-4000 FU/day) twice daily has been suggested.

Cautions and contraindications: While NK appears to have an excellent safety profile, [63,65] bleeding has rarely been reported in patients with risk factors for bleeding (advanced age, renal failure, hypertension, concomitant ASA, etc). [66,67] High concentrations of vitamin K₂ in natto can reduce the INR when co-administered with warfarin; this may also occur with nattokinase supplements if vitamin K₂ is not removed during the production process. Information regarding safety and efficacy in pregnancy and lactation is lacking.

Lumbrokinase: Lumbrokinase derives from a group of enzymes extracted from earthworms. The enzymes are sourced mostly from the earthworm *Lumbricus rubellus*. Lumbrokinase has very similar pharmacodynamic properties to Nattokinase, i.e., it directly breaks down fibrin clots, inhibits PAI-1 activity, enhances t-PA activity, has antiplatelet activity, and proteolytically cleaves amyloid. [68-70]

The recommended dose is 300,000 to 600,000 IU/day (20-40 mg).

Lumbrokinase has been widely used for patients with acute ischemic stroke in China; however, because rigorously designed studies are lacking, the safety and efficacy of lumbrokinase remains largely unknown. [71]

As the pharmacology, clinical effectiveness, and safety of nattokinase has been assessed in a number of experimental and clinical studies, this agent is preferred over lumbrokinase.

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