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Theoretical Strategies in SARS-CoV-2 Human Host Treatment

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1. Abstract

The communication reports on the clinically tested hypothesis that SARS-CoV-2 Spike 1 (S1) and Spike 2 (S2) protein strands might pose pathogens independently in human physiology, and that they might extend inwards into the viral envelope structure. The hypothesis raises concern for vaccination as anti-SARS-CoV-2 strategic methodology. With the post-vaccination interventional trial, the communication reports on the treatment method developed and further improvements to be tested.

2. Theoretical Background

Current SARS-CoV-2 vaccine strategies have been proven inefficient [1]. HIV-1 vaccine developments target envelope proteins, and SARS-CoV-2's hydrophobic Spike 2 (S2) protein is inverted in structure from HIV-1 gp41 [2,3,4,5]. It is not currently studied on the spike helix's strand extension with the envelop and positive-sense single-stranded RNA [5,6], but the structural similarities of S2 to HIV-1 gp41 in membrane fusion human pathogens correspond to the negative-sense paramyxovirus with the abnormally over-lengthed fusogenic single-strand virus [5,7]. Gp41envelop protein has only recently been strategized in HIV-1 vaccine development, and SARS-CoV-2 vaccination and testing methods utilized its Spike 1 (S1) protein [8,9,10], with 10 to 20 times to SARS-CoV's targeting on angiotensin-converting enzyme 2 (ACE2) [5,11].

Dissimilar to HIV-1's gp120 target-cell recognition [5], SARS-CoV-2 S1 induces human pathogen severity by endocytosis, while S2 induces human immunological pathogens with vascular-neuronal infection potentials evidenced by Delta variant [12]. Signature symptomatic SARS-CoV-2 cases' CT images show patient's primary lung infection starts with the innate immunity stem regions [7,13] and proliferates to whole lung infection with "white lung" imaging; post-vaccination pericarditis and myocarditis cases' antibody-unbound S proteins inspired the hypothesis that S1 and S2 protein strands might pose pathogens independently in human physiology, if not contributed by quantitative factors of antibody levels against infection levels, and that they extend inwards into the viral envelope structure [14,15].

3. Interventional Analysis

The hypothesis has been tested in the interventional trial, and the data generated seen in tab. 1 is analyzed from adenosine triphosphatase (ATP) synthesis [16,17]. The post-vaccination pericarditis adult case, differentiated from the adolescent cases of platelet apoptosis instead of platelet binding [14], shares the same physiological symptoms and risks in other adult cases developing acute myocarditis and blood clots [16]. The data from continued intervention suggests that the use of ACE inhibitor in the treatment did not function against S1, but only reduced the risks in blood clot formation by platelet bindings with S2 proteins' fusogenic activities intervened by proton-pump inhibitor (PPI) [16]. The pericarditis cause is hence diagnosed to be over-sized platelet concentration with increased immune reflex below the blood-brain barrier (BBB) contributed by the gateway reflex [19,20], with corroboration by the electrocardiogram in (Figure 1) collected the same day as "continued" blood test result. The evidence suggests SARS-CoV-2 lung sediment may be contributed by S1 protein with ACE2 carrier and proton-motive force (PMF) [21], but the key lethal pathogen lies with S2 protein. Albeit Omicron variant showed less physiological pathogenic severity contributed by pathogenic fusion replication, its increased six unique mutations in S2 are concerning for neuronal infections [11,12,22].

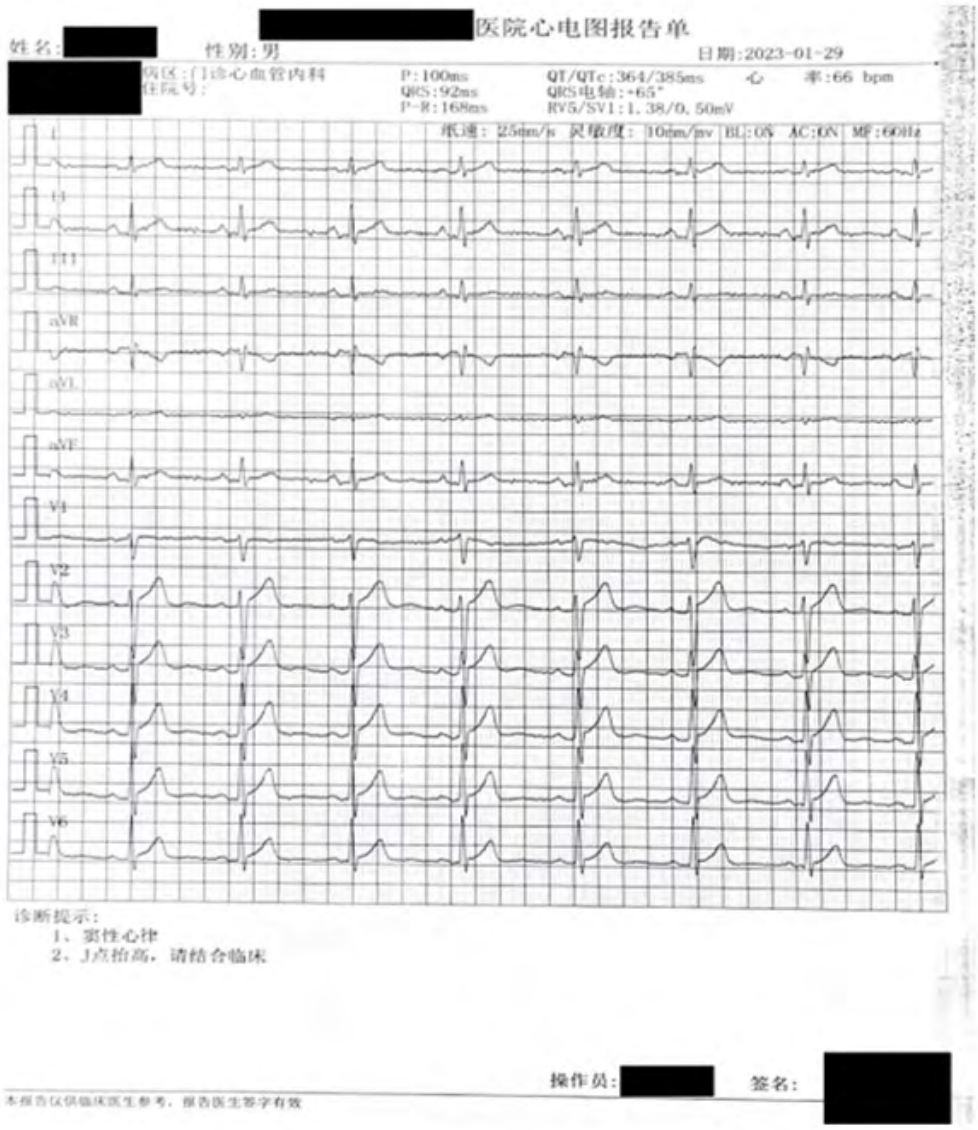

Figure 1: Electrocardiogram indicating to pericarditis cause during depolarization.

4. Methodological Concerns in Vaccination

Despite of HIV-1 gp41 targeted vaccine developments, the tested hypothesis raises methodological concerns in vaccination method for SARS-CoV-2 treatment before further confirmation on the virological RNA division survival capacities. Current vaccines have been functioning their designed purposes in the post-vaccination myocarditis studies, but their functions only serve for avoiding viral lung sediment [14]. Moreover, despite of the mass psychological placebo effect of vaccination, vaccination strategies have defused the viral concentration in vaccinated human pathogen and cut off the statistical advantages in pharmacokinetic treatment targeting [23]; severe lung infection is contributed mainly by keratinocyte-derived cytokine in reflex-based immune homeostasis, which also contributes to post-vaccination blood clots [11,14,24]. The human-induced viral pre-exposure only sent S2 deeper into the immune system from S1 designs, while further research into endocytic pathways with T cells may be beneficial for neurological pathogen assessments.

5. Initial Treatment Method

My current interventional clinical trial aimed at inhibiting fusogenic activities of S2 in the BBB with natural immunity preservation, regulating immune reflexes through symptomatic pathways in supplementing immune cell functions in innate immunity, and at viral excretion through renal hemodialysis [16,25]. With ACE inhibitor regulating cardiac input and beta blocker regulating cardiac output, for discretion on vein & artery scratches from platelet endocytosis, PPI was conceived to decrease fusogenic activities in the blood through the inhibition of extracellular protons [26]. The change in plasma membrane was effective with decreased platelet endocytosis and net growth in basophil absolute number seen in (Table 1), while the depolarization on synaptic acidification through the hydrogen channel was left unattended [27]. S2's hydrophobic characteristics make it unlikely to reach V0 in the vesicular ATP (V-ATPase) pathway with PMF [5,19], the normative goal in medicine-induced hemodialysis with ambient pH homeostasis is summarized from the clinical trial [16,27]: 1) monitor for

white blood cell regeneration activity / integrity; 2) turn around the over-acidified blood environment; 3) decrease the platelet binding activities; 4) excrete the excessive proteins through the artery circulation. The initial treatment method has been effective with room for improvement, especially considering unattended depolarization changes' impact to neurodiverse factors [16]. As long as the case's liquid excretion is functional and does not have any dehydration symptoms, the discrete treatment approach has been adhered to.

Table 1: Primary data collected in local hospital generated from the interventional clinical trial [16,18].

Indicator	Initial	Intervened	Continued	Reference Range	Unit	Relevant Indicator	Initial	Intervened	Continued	Reference Range	Unit
WBC	8.35	9.13	8.18	3.5-9.5	10^9/L						
NEU#	5.25	5.52	3.96	1.8-6.3	10^9/L	Neu%	62.9	60.5	48.4	40-75	%
LYM#	2.07	2.27	2.87	1.1-3.2	10^9/L	Lym%	24.8	24.9	35.1	20-50	%
MONO#	0.38	0.47	0.4	0.10-0.60	10^9/L	Mon%	4.5	5.1	4.9	10-Mar	%
EOS#	0.63	0.84	0.91	0.02-0.52	10^9/L	Eos%	7.5	9.2	11.1	0.4-8.0	%
BASO#	0.02	0.03	0.04	0.00-0.06	10^9/L	Bas%	0.3	0.3	0.5	0-1	%
RBC	5.22	5.12	5.32	4.3-5.8	10^12/						
tHb	166	166	171	130-175	g/L	Hct.	49.8	48.7	50.3	40-50	%
MCV	95.4	95.3	94.5	82-100	fL						
MCH	31.8	32.4	32.1	27-34	pg	MCHC	333	340	340	316-354	g/L
RDW-CV	14.1	13.9	13.8	11.0-16.0	%	RDW-SD	50.8	50.1	47.2	35-56	fL
PLT	162	193	192	125-350	10^9/L	PCT	0.2	0.23	0.22	0.108-0.282	%
PDW	17.1	16.7	16.7	15-17	fL	MPV	12.6	11.8	11.3	11-Jul	fL
HsCRP	0.98	<0.50	<0.50	0-3	mg/L						

Continued rapid acidification of SARS-CoV-2 infection and replication goes in parallel with improved basophil indicators and decreased platelet binding in the continued intervention.

6. Treatment Method Development

The methodological development on pathogen treatment has thence put physiological proton pathways into focus, with potentials in neurological applications in discretion of calcium carriers in pharmacokinetics [17,27]. Bafilomycin A1 has been located for the primary ingredient [23,28]. Bafilomycin A1's mechanism of action corresponds highly with the blood-borne pathogens in SARS-CoV-2 and apoptosis is not seen as negative factor for antiviral purposes [29]. Albeit the endocytosis and exocytosis process actions correspond [19,30], electrostasis changes from cardiac flow regulations through V-ATPase inhibitor may influence neurological activities, signified by the interventional trial's amplification on the patient's neurodiverse autism spectrum disorder symptoms from the vagus nerve arc [25,31]. However, the increased elaboration of neuronal dendrites, axons or decreased pruning or inflammatory responses are not seen as a disadvantage [22,31] for the applicability of the treatment or the treatment in development. Bafilomycin A1's neurological influences, along with oxidization changes, will need to be tested with animal experiments for possible adjustments before human clinical trials.

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