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Review Article

Post-Exposure Prophylaxis During Pandemic - Rationale of a Concept with Narrative Review of Literature

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Abstract

With the devastations of COVID-19 pandemic still fresh in our memories and speculations about possible pandemic X in the future ongoing, mankind is not yet fully prepared to combat a possible future pandemic. In this backdrop, the recent outbreaks of Hantavirus and Ebola virus in two different parts of the globe that made global headlines almost around the same time once again points to how vulnerable mankind still is against the possible surge of a virus. If and while a virus strikes, the prime concern becomes to strike an effective vaccine in order to strike an end to it's progression. However, till that time comes devising post-exposure prophylaxis also becomes important and immediate concern. Here we discuss two such options, namely NASVAC and Favipiravir, that may prove useful for post-exposure prophylaxis in case of a virus once going beyond control anytime in the future.

Keywords: Hantavirus; Ebola Virus; NASVAC; Favipiravir; Post-Exposure Prophylaxis

Introduction

Although now contained from the public health perspective, the severity and magnitude of Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-Cov-2) related Coronavirus Disease-2019 (COVID-19) pandemic, not only still haunt our memories, many across the world continue to suffer from post-COVID syndrome to date.

Hantavirus (HTNV) is a negative-sense, single-stranded, tri-segmented RNA virus that belongs to order *Bunyavirales*, family *Hantaviridae* and genus *Orthohantavirus* [1]. HTNV maintains persistent infection in rodents, their natural host and shed in their excreta, urine, saliva, aerosols and rarely bites [2,3]. HTNV is primarily transmitted to humans through inhalation of airborne particles contaminated by urine, excreta or saliva of infected wild rodents [4]. It produces 2 types of febrile diseases in humans namely, Hantavirus Pulmonary Syndrome (HPS) and Hemorrhagic Fever with Renal Syndrome (HFRS) [5]. Worldwide approximately 200,000 people are infected annually with HTNV and the numbers are steadily on the rise [6,7].

The first outbreak of HTNV occurred during the Korean war from 1950-1953, where more than 300 US troops were infected [8], while the second outbreak was reported in South-Western USA in 1993 [9]. HTNV infection is encountered worldwide, with case fatality being 1%-5% from HFRS, but up to 40%-60% in case of HPS [6,10]. HTNV recently made global headlines following an outbreak linked to a cruise ship named MV Hondius, which travelled to the sub-Antarctic region. The outbreak led to 8 cases and 3 deaths in The Netherlands, Switzerland, South Africa and USA [11]. Spread of the virus was primarily from rodent excreta.

Ebola virus, on the other hand, belongs to family *Filoviridae* along with genus *Marbugvirus*. The genus Ebola virus has 6 distinct strains namely, *Zaire ebolavirus (EBOV)*, *Sudan ebolavirus (SUDV)*, *Tai forest ebolavirus (TAFV)*, *Bundibugyo ebolavirus (BDBV)*, *Bombali ebolavirus (BOMV)* and *Reston ebolavirus (RESTV)* [12,13]. It spreads through direct contact through broken skin of mucous membrane of eyes, nose or mouth, with body fluid of a symptomatic infected person or animal [14,15]. Ebola Hemorrhagic Fever (EHF) caused by *EBOV* has the highest death rate of 57%-90% followed by *SUDV* 41%-65% and *BDBV* 40% [16-18].

The first outbreak of Ebola virus was reported in Yambuku in northern Democratic Republic of Congo (DRC) and South Sudan in 1976 [15]. However, the deadliest Ebola outbreak started from Guinea and effected western Africa in 2013-2015 claiming several thousand lives [19-21]. Around the same time as the recent HTNV outbreak, an Ebola outbreak, caused by *BDBV*, was reported in DRC and Uganda. There were more than 800 confirmed and suspected cases and over 170 deaths [22]. It is assumed that the outbreak originated in Mongbwalu, a high-traffic mining area in DRC and spread to Uganda through cross-border movements. Unlike HTNV, although Ebola virus cannot spread through aerosol, nasal route still remains an important for entry of Ebola virus in the human body too.

Role of immune-response in HTNV pathogenesis

HTNV infection begins with binding of Gn and Gc surface proteins of the virus with β-integrin receptors on epithelial cells. Although the spread of HTNV in the body is still illusive, it is assumed that immature dendritic cells (DCs) are important in the spread of the virus in the human body [23]. DCs introduce their dendritic projections into airway lumen and contract HTNV in the lungs [24-26]. Besides, monocytes that are exposed to HTNV transform into DC-like cells that assist spread of the virus throughout the human body [27,28]. During HTNV infection, stimulated endothelial cells (EC) produce chemokines like interleukin-8 (IL-8), intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1) [29,30]. There is also activation of natural killer (NK) cells [31]. Cytotoxic T-cells and NK cells remove HTNV infected as well as non-infected ECs leading to vascular leakage [32]. In acute HFRS, there is complement activation and IL-8 production through pentraxin-related protein-3 (PTX-3), which in turn facilitate vascular inflammation [33,34]. HTNV-related macrophages, DC, NK cells, neutrophils and CD8+ T-cells release tumor necrosis factor-α (TNF-α), which remove HTNV from infected cells by non-cytopathic mechanism [26,35]. This can have a two-way sword-like effect as on one hand, this can help prevent HTNV propagation, but on the other hand, vascular leakage and breathing problems may aggravate [36-38]. Although not yet fully established, disrupted immune response may be important in HTNV-pathogenesis [39].

Role of immune-response in Ebola pathogenesis

Ebola virus enters humans monocytes, macrophages and DCs using it's spike glycoproteins following entry into the human body,

which in turn plays important role in spread of the virus inside the body [40,41]. A cascade of events is thus initiated including failure of chemokine stimulation and upregulation of major histocompatibility complex (MHC), lymphocyte differentiation and interferon (IFN) production [42-44]. There is persistent systemic vascular inflammation leading to increased capillary permeability, capillary leakage, alteration in blood flow, microthrombi formation, microvascular stasis and oedema [41,45]. In case of Ebola virus infection failure of host adaptive immune response ultimately leads to multiple organ dysfunction syndrome (MODS).

How does the human body fight viruses?

The human body's first-line defense against any viral infection is innate immune response. Viral components or viral replication intermediates called pathogen-associated molecular patterns (PAMPs) are recognized by pattern recognition receptors (PRRs) located on plasma and endosomal membranes and cytoplasm, which in turn leads to activation of innate immunity. There is complex interplay between viruses, viral receptors, PRRs, PAMPs, NK cells, NK T-cells, DCs, neutrophils and macrophages that prevent viral attachment to host receptors thus inhibiting viral replication or facilitating viral elimination [46]. Therefore, progression of a viral infection occurs when the host innate immunity fails to contain the virus.

What may be the Initial Strategy in a Pandemic X?

In case of any unforeseen pandemic in the future, one of the obvious strategies will be post-exposure prophylaxis (PEP) till an effective vaccine and curative drugs are available. Getting a curative drug may remain a far cry and may never be obtained, as we have experienced with COVID-19. Vaccine development is also a challenge, especially when a pandemic is ongoing. It may take years. In case of COVID-19, we had vaccines very fast, but that too was after millions lost lives across the globe and after nearly a year of waiting. Besides as the causative organism is likely to undergo mutations, keeping the vaccine updated and effective remains challenging which was evident during the Delta COVID-19 pandemic. Therefore, the necessity of effective PEP can never be undermined at any stage of an ongoing pandemic.

The million dollar question is, how to design efficient PEP. The answer is possibly two folds. One is to potentiate the host innate immunity that will prevent spread of the causative organism in the human host and reduce severity of illness. For a RNA virus induced pandemic, the other option may be to bring out an anti-viral drug from the shelves having broad spectrum anti-RNA viral effect without inducing of long-term host genetic mutation.

NASVAC - Can it be a novel answer to known threat?

NASVAC can possibly help in PEP against HTNV and/or Ebola virus by potentiating innate immunity in the nasal cavity. It has been demonstrated that hepatitis B virus core antigen (HBcAg) in NASVAC induces production of inflammatory cytokines in humans infected with hepatitis B virus (HBV) and COVID-19 [46,47]. Moreover, HBcAg-induced proinflammatory cytokine production does not lead to organ damage, a challenge that has hindered immune-therapy against different pathogens for long. Besides, NASVAC stimulates both innate and adoptive immunity both *in vivo* and *in vitro* [48,49]. NASVAC probably can also help HTNV and Ebola virus infected patients by inducing IFN production and also by improving detection of viral RNA by innate immunity [46].

Although there is no published data on the effect of NASVAC against HTNV or Ebola virus, this novel therapeutic vaccine has shown promise against HBV and SARS-Cov-2. Increase in innate immune receptors namely, RNA-sensing TLR3, TLR7 and TLR8 in tonsils of chronic hepatitis B (CHB) patients have been demonstrated in a study after administration of NASVAC. Significant survival benefit was demonstrated in lethally SARS-Cov-2-infected mice model following administration of TLR3, TLR7/8 and TLR9 agonists [50,51]. HBcAg has been shown to be a potent adjuvant for glioblastoma, chlamydia infection [46]. Also, when flu vaccine is combined, cross protection and adjuvant property of HBcAg becomes evident [46]. In a Japanese study, *in vitro* neutralizing assay showed that plasma samples from NASVAC-treated chronic hepatitis B (CHB) patients had high titers of anti-HBs demonstrating strong viral neutralizing capability of NASVAC [52].

A non-randomized, single-arm, prospective clinical trial of NASVAC in SARS-Cov-2 infection during the COVID-19 pandemic demonstrated that following intra-nasal and sublingual NASVAC administration, none of the study participants, who were all at high risk of contracting the virus, got infected within 14 days. Three patients got mild COVID-19 during 6-months follow up [53,54]. This suggests possible role of NASVAC in post exposure prophylaxis against SARS-Cov-2. NASVAC was also evaluated for treatment of COVID-19 patients. This further revealed that mean duration of

symptoms were longer in control patients than those treated with NASVAC [55,56].

Favipiravir - Can it be an answer too?

Favipiravir (FPV) is a prodrug that undergoes metabolic activation through ribosylation and phosphorylation inside tissues into an activated metabolite called FPV-fibofuranosyl-5'-triphosphate (T-705RTP) [57,58]. FPV has a novel mechanism of action by binding to and inhibiting RNA-dependent RNA polymerase (RdRp) and ultimately preventing transcription of viral RNA and viral replication [57-59]. RdRp domain is absent in human cells and conserved only in RNA viruses. This mechanism of action makes FPV an attractive drug candidate for RNA viruses like HTNV and Ebola.

Although the molecular mechanism of the broad-spectrum antiviral effect of FPV is yet to be fully understood, several mechanisms have been proposed. One possibility is that T-705RTP inhibits viral RNA synthesis through nascent viral RNA strand chain termination preventing extension of the RNA strand [60,61]. It has also been suggested that FPV induces mutagenicity inducing mutations in viral genome leading to progressive decrease in viable viral progeny [62].

There is publication in the scientific literature evaluating FPV against Ebola virus infection in mouse model. It was demonstrated that early FPV treatment, before the onset of viral detection in blood and initiation of liver damage, cured 100% mice from lethal Ebola infection suggesting efficacy of FPV in PEP against Ebola virus infection [63]. A non-randomized, single-arm study from Guinea further demonstrated non-statistically significant reduced mortality in Ebola-infected humans with low viral load compared to historical controls [64]. Another non-randomized, single-arm, human study conducted during the 2014 Ebola epidemic in western Africa reported better survival among FPV-treated Ebola-infected patients compared to historical controls [65]. On the other hand, in case of HTNV, it has been demonstrated in hamster model that FPV protects against HTNV-related HPS, if treatment is initiated before the onset of viremia [66].

Conclusion

Although it is still premature to comment with limited evidence in hand, it can possibly be safely stated that both NASVAC and FPV may be effective PEP tools. With the COVID-19 pandemic still bright in our memories and ongoing speculations about possible pandemic X, the recent outbreaks of HTNV and Ebola infections are wake up calls for human civilization. In this review, we have tried to share our own experience, as well that of others to identify possible PEP candidates against eminent threats like HTNV and Ebola virus, that have the potential to shake up human civilization once again as SARS-Cov-2 did in the recent past.

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