

A Review of the Cytokine Storm

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Abstract

Cytokine storm syndromes (CSS) are a broad category of diseases marked by extreme immune system overactivity. A combination of host variables, including genetic risk and predisposing diseases, as well as acute triggers such infections, cause CSS in the majority of patients. Compared to children, who are more prone to present with monogenic variants of these illnesses, CSS appear differently in adults. Individual CSS are uncommon, but together they are a significant contributor of serious sickness in both children and adults. We provide three uncommon, representative cases of CSS in young patients that illustrate the range of CSS. Chronic respiratory disorders (CRDs), particularly asthma and chronic obstructive pulmonary disease (COPD), are becoming more common and are carrying a heavier burden globally. Notably, the research on the global burden of disease notes that, on average, 545 million people worldwide experience chronic respiratory conditions. This is responsible for over 3.9 million fatalities per year. Additionally, with an 18% growth in the past few years, CRDs now account for a significant share of disability-adjusted life years globally.

Keywords: Cytokine storm syndromes; Macrophages; Immunomodulation; Hemophagocytic lymphohistiocytosis

Introduction

The phrase “cytokine storm” conjures up vivid pictures of an immune system gone haywire and an uncontrolled inflammatory reaction. Both the general public and the scientific community are interested in the word, which is increasingly utilised in both popular media and scholarly journals. Furthermore, there is a lack of knowledge regarding the molecular events that lead to a cytokine storm, the role that such a “storm” plays in pathogenesis, and possible treatment approaches to stop or stop the storm after it has begun [1].

Macrophages play a key role in inflammatory reactions and, depending on the environment, can polarise into either traditionally activated M1 with proinflammatory qualities or alternatively activated M2 with antiinflammatory properties. Numerous studies have shown that the phenotypic imbalance in macrophages causes an unchecked inflammatory response, which subsequently starts a cytokine storm that worsens the condition [2]. Natural plant polysaccharides have received a lot of interest lately in the areas of intestinal health, immunomodulation, antiviral, anti-inflammatory, and anticancer. This is because of their distinctive biological properties. The information at hand demonstrates that polysaccharides can reduce inflammation by stimulating macrophages. Acute colitis and colon cancer have been successfully treated in mice models with acetylmannan, which is abundantly contained in aloe vera [3].

Hemophagocytic lymphohistiocytosis (HLH) is a rare, severely fatal condition with a systemic inflammatory disorder and multiple organ dysfunction that can be caused by genetic defects (inherited form) or acquired risk factors (non-inherited form), such as infection (with a specific virus), autoimmune disease, and malignancy (with a specific lymphoma). Immune response that is highly stimulated but ineffectual, followed by a cytokine storm, are traits of HLH. In all forms of HLH, T cells and macrophage/monocytes are continuously activated and build up in various organs like the liver, spleen, and bone marrow to release overwhelming cytokines, which causes a variety of clinical symptoms like persistent fever, pancytopenia, hepatosplenomegaly, and hepatorenal dysfunction [4].

Associated cytokines in the cytokine storm

Interferons: A family of cytokines called interferons (IFNs) is

essential for innate immunity against viruses and other microbial pathogens (45, 75). On the basis of the receptor specificity, they are divided into three main categories (types I, II, and III). While type II IFNs (IFN- γ) signal through IFN-R1/IFN-R2, type I IFNs (IFN- α and IFN- β) signal through a heterodimeric receptor complex, IFNAR1/IFNAR2. A new type of IFNs known as lambda IFNs has been shown to have antiviral characteristics (127), protecting mice from the influenza A virus (106). IFN-1, -2, and -3 (also known as interleukin-29 [IL-29], IL-28a, and IL-28b) bind the IL-28R/IL-10R receptor complex. nonetheless, share a similar functional property with type I IFNs in that they both transmit signals via the Jak-STAT signalling pathway. Receptor binding causes the start of downstream signalling cascades, with the end result being [4,5].

Interleukins: In contrast to IFNs, a diverse family of immune system regulators called interleukins plays a major role in the development and activation of immune cells. Like all cytokines, they can either be pro- or anti-inflammatory and cause a range of reactions. Although many different cell types now understood to create interleukins, the term was originally coined to refer to cytokines produced by leukocytes that serve in intercellular communication. Despite the adoption of a standard nomenclature scheme, interleukins' categorization and name still cause confusion. For instance, the cytokine IL-1 is really transcribed by 11 different genes. As novel roles for several IL-1 family members are revealed, new interleukin designations have been suggested [5,6].

Infectious illness cytokine storms

The cytokine storm's pathology: In response to a concentration gradient, a variety of cytokines produced during proinflammatory immune responses can activate different populations of leukocytes

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Received: 04-May-2023, Manuscript No: jcb-23-100222; **Editor assigned:** 08-May-2023, PreQC No: jcb-23-100222 (PQ); **Reviewed:** 22-May-2023, QC No: jcb-23-100222; **Revised:** 24-May-2023, Manuscript No: jcb-23-100222 (R); **Published:** 31-May-2023, DOI: 10.4172/2576-3881.1000447

Citation: Chen GL (2023) A Review of the Cytokine Storm. J Cytokine Biol 8: 447.

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and draw them in via chemotaxis. Cytokines, including as interferons (IFNs), chemokines, and ILs, are involved in many proinflammatory reactions. The TNF, IL6, IL8, and IL1 families have received the most attention. By encouraging the detection of infections, the recruitment and eradication of threats, and the homeostasis of immune cells, cytokines play a critical role in inflammatory processes. Vasodilation and vascular permeation can be aided by TNF and IL1 β to boost leukocyte infiltration. IL6 can also encourage the production of proteins that are complimentary and are essential for innate immune responses. It can be challenging to research specific cytokines and their receptors, despite the fact that doing so can affect therapeutic interventions [6,7].

The effects of cytokine storm in severe infections: Cytokine storms cause morbidity and death in infections such as pneumonia, septic illnesses, and other infections. The endothelial and epithelial barriers in the lungs are damaged by ARDS, an acute inflammatory reaction. During ARDS, cytokines cause and amplify inflammation. Immunosuppressive medications like methylprednisolone, hydroxychloroquine, chloroquine, and leflunomide, proinflammatory cytokine inhibitors like IFN-5-007, IL1 β , IL6, IL17A, M-CSF, and TNF-5-007, and factor modulators that control innate and adaptive immune responses like C55 β can all be used to combat cytokine storms in extreme circumstances. Cytokine storms and the diseases they cause cannot be stopped by immune-modulating cytokines. Treatment strategies that target immune cells that control cytokine storms may be more effective in people with severe COVID-19 [7,8].

Result

Cytokine storm is strongly associated with the severity and dismal prognosis of acute pneumonia. Aloe Vera polysaccharides have received a lot of attention in recent years in research on anti-inflammatory and antiviral activities, which may be a useful technique for the treatment of cytokine storm. In this work, a polymeric acemannan (ABPA1) isolated from AVBEC was used, and it was discovered that both in vitro and in vivo, ABPA1 successfully reduced the cytokine storm [8-10].

Conclusion

We are currently in what could be a golden age of scientific discovery, thanks in large part to recent developments in next-generation sequencing (NGS) technology. By revealing a more complete perspective of the transcriptome, for instance, NGS has made it possible to identify large and small RNAs, splice isoforms, and new transcripts from unannotated genes. However, the overwhelming amount of data we can currently gather poses its own problems. One of the challenges associated with employing this technology is the requirement for

better methods for NGS data interpretation, including RNA-Seq bias correction and enhanced isoform quantification. By addressing the constraints brought on by a lack of training data, we have created a unique meta-analysis informed ML-based approach that might allow machine learning-based prediction algorithms to be employed in practise. We accomplish this by adding domain knowledge from a statistical meta-analysis to the seed training data. By enabling users to trace back predictions to scientific evidence from the knowledge base that supports the outcomes, the meta-analysis integration also grants the final technique the property of being explicable.

Acknowledgement

None

Conflict of Interest

None

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