Short Communication
Volume 6:4, 2022

Journal of Microbial Pathogenesis

ISSN: 2684-4931 Open Access

Growth Factors' Contribution to Dengue's Pathogenesis

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Introduction

The dengue viruses (DENV-1, DENV-2, DENV-3, and DENV-4) are a group of viruses that spread by mosquito bites and are the main cause of dengue. A wide range of clinical problems, both asymptomatic and symptomatic, are associated with dengue virus infection. Due to increased vascular permeability, plasma leakage, and shock, it can develop into a serious, life-threatening condition. Complex interactions between the virus, host genes, and host immune response play a role in the pathogenesis of dengue virus infection. Despite decades of research, it is true that comprehending the immunopathogenic processes is still difficult [1]. The development of severe dengue is linked to the overproduction of inflammatory mediators as cytokines, chemokines, and growth factors. Growth factors (GFs) are signalling proteins that encourage numerous biological processes. Growth, differentiation, proliferation, survival, inflammation, and tissue repair are cellular processes. The intracellular protein kinase domain of GFs is activated upon attachment to their particular receptors, which sets off a series of signalling processes [2].

However, some viruses have been found to require these receptors for entry, and GF-receptor signalling is involved in viral replication. There is no specific treatment for dengue, and the four dengue serotypes are currently only partially protected by vaccinations. In order to decrease severe disease and death caused by dengue, medicines that target viral targets or crucial host systems are needed. In order to create treatments that target these signalling proteins, a deeper comprehension of the role of GFs in dengue pathogenesis is necessary [3].

Description

Except for TGF-, which binds to serine/threonine receptors, GFs are proteins that either promote or inhibit mitosis and differentiation by binding to receptor tyrosine kinases (RTK). Growth factor (GF) receptors (GFR) can have several ligands with structural and functional similarity to activate various receptors to carry out their tasks, with the exception of HGF and its receptor Met, which are mutually exclusive [4]. Viruses can interact with RTKs, altering their activity to enhance cell entrance and encourage reproduction within the host, according to mounting evidence. The dengue growth factor that has been the subject of the most research, VEGF, has been proposed as a biomarker of severity. Regardless of the illness, this trend of rising levels of severe dengue was maintained. Classification, age range, or type of sample. VEGF-A, VEGF-B, VEGF-C, VEGF-D, VEGF-E, placental growth factor (PlGF), and endocrine-gland derived vascular endothelial growth factor are among the members of the VEGF family (EG-VEGF) [5].

VEGF plays a significant role in vasculogenesis and neoangiogenesis and is released by endothelial cells, macrophages, platelets, keratinocytes, leukocytes, dendritic cells, and bronchial and alveolar epithelial cells. Mastocytes are one of the sources of VEGF in dengue, according to in vitro research employing the human cell lines KU812 and HMC-1 [6]. When infected with DENV-2 in the presence of human dengue-immune sera and IL-9, KU812 and HMC-1 supernatant were found to contain high amounts of VEGF. These levels were even higher in the absence of IL-9. Three RTKs that are expressed mediate the biological effects of VEGF. The choroid plexus of the brain, which is a confined region, and resistance vessels, which are systemic regions of the body where fluid balance is crucially regulated. In essence, VEGFR-1 is mostly expressed in monocytes, macrophages, vascular smooth muscle cells, and neural cells, whereas VEGFR-2 is primarily expressed in endothelial cells and VEGFR-3 is primarily found in the lymphatic endothelium [7].

IL-1 and TNF- are two inflammatory cytokines that are frequently increased in dengue patients. Using the NF-B pathway, these cytokines can enhance VEGF production. By modifying the expression of P and E selectins, integrin-binding adhesion molecules (ICAM-1 and VCAM-1), which are implicated in leukocyte recruitment, and altering vascular permeability, VEGF may contribute to inflammation [8]. Additionally, during a DENV infection, coagulation and fibrinolysis processes are triggered. Through the elevation of VEGF receptor expression, thrombin production in this process can increase VEGF expression in endothelial cells and enhance its effects. Additionally, VEGF can boost the expression of tissue factors, and activation of the tissue factor pathway is another crucial aspect of coagulation problems caused by DENV [9].

Nevertheless, despite the fact that one study suggested a link between higher levels of VEGF and D-dimer however, another study found no correlation between plasma levels of VEGF and its soluble receptors and the occurrence of bleeding in patients without plasma leakage, indicating that bleeding is a complication independent of changes in endothelial barrier function, as determined by the WHO dengue case classification 2009. Additionally, it has been demonstrated that VEGF may play a significant role in the pathogenesis of viral illnesses, and numerous viruses use a variety of strategies to positively regulate VEGF. Some viruses, like DENV, trigger inflammatory mediators that increase the expression of VEGF, whereas others, like homologous viruses, have a similar structure [10].

Conclusion

The fundamental role of the GF pathway in maintaining tissue homeostasis during DENV infection, healing vascular damage and other damaged tissues, and controlling the expression and differentiation of monocytes, macrophages, lymphocytes, and neutrophils that could favour an intense inflammatory response and increase vascular permeability is summarised in this scoping review. In most investigations, severe dengue was associated with elevated levels of VEGF, GM-CSF, G-CSF, TGF and HGF as well as lower levels of PDGF and EGF. Biomarkers of severity have been found to include VEGF and HGF. Additionally, there is proof that the dengue virus can employ the GFs route to assist cell entrance and aid in viral reproduction, and the usage of GFs and their tyrosine kinase activity inhibitors can help to further support this theory. Are substitutes for dengue treatment? It will be possible to construct trials for new treatment protocols once there is a better knowledge of the function of the GF pathway.

Acknowledgement

None

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Date of Submission: 01 August, 2022, Manuscript No. jmp-22-77232; **Editor Assigned:** 03 August, 2022, PreQC No. P-77232; **Reviewed:** 17 August, 2022, QC No. Q-77232; **Revised:** 23 August, 2022, Manuscript No. R-77232; **Published:** 31 August, 2022, DOI: 10.37421/2684-4931.2022.6.127.

Conflict of Interest

None.

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How to cite this article: Solórzano, Elzinandes. "Growth Factors' Contribution to Dengue's Pathogenesis" J Microb Path 6 (2022): 127.