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Opinion Article

The Impact of COVID-19 on Vascular Endothelial Growth Factor

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DESCRIPTION

Vascular biology is significantly influenced by Vascular Endothelial Growth Factors (VEGF). Cardiomyocytes, fibroblasts, stem cells, neurons, vascular smooth muscle cells and pericytes, endothelial cells, lymphatic endothelial cells, and the placenta are all affected by VEGFs. For example, certain VEGFs may cause arrhythmias. Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS) are thought to be significantly influenced by VEGF. VEGF levels are raised by severe inflammation and hypoxia. Nearly all COVID-19 patients had significantly higher VEGF levels than healthy controls. Compared to controls, VEGF concentrations were consistently greater in COVID-19 patients in the ICU as well as those who were not. During acute lung injury, Angiotensin-Converting Enzyme 2 (ACE2) inhibited VEGF-A, thereby lowering vascular permeability. VEGF-A expression was downregulated in breast cancer cells by ACE2. It is known that SARSCoV-2 downregulates ACE2. Consequently, it appears that SARS-CoV-2 neutralizes the VEGF-Antagonizing action of ACE2, which exacerbates endothelial damage and increases vascular permeability.

Tissues with ACE2 upregulation may already exist in certain patient groups. For instance, it has been noted that the hearts of patients with obstructive hypertrophic cardiomyopathy had a notable elevation of ACE2. Improved glycemic management, prevention of myocardial fibrosis, and improved heart function following myocardial infarction has all been linked to upregulated ACE2. It has been demonstrated that iodide inhibits the transcription of VEGF in cultured thyroid cells. Iodide inhibited the transcription of VEGF and NIS concurrently; the effect started to fade after 6 hours and persisted for 24 hours. Comparably, gene expression was restored in the same amount of time after iodide was removed from the culture medium. Iodide raised the expression of VEGF-C and VEGF-D, which are linked to the lymphatic system, while it lowered the expression of NIS, VEGF-A, and VEGF-B in another investigation involving human thyroid follicles. Moreover, it reduced the expression of genes involved in energy metabolism while increasing the expression of anti-angiogenic proteins. VEGF-D may have therapeutic potential because it can promote angiogenesis without also increasing cell proliferation. It has been demonstrated that iodide inhibits the transcription of VEGF in cultured thyroid cells. Iodide inhibited the transcription of VEGF and NIS concurrently; the effect started to fade after 6 hours and persisted for 24 hours. Comparably, gene expression was restored in the same amount of time after iodide was removed from the culture medium. Iodide raised the expression of VEGF-C and VEGF-D, which are linked to the lymphatic system, while it lowered the expression of NIS, VEGF-A, and VEGF-B in another investigation involving human thyroid follicles. Moreover, it reduced the expression of genes involved in energy metabolism while increasing the expression of anti-angiogenic proteins. VEGF-D may have therapeutic potential because it can promote angiogenesis without also increasing cell proliferation. Endogenous VEGF levels rise in ischemic brain tissue after an Acute Ischemic Stroke (AIS). VEGF promotes angiogenesis, neurogenesis, and neuroprotection, but it also causes vascular leakiness and permeability, which is harmful during the early stages of recovery.

Because systemic exogenous VEGF infusion promotes blood-brain barrier breakdown in conjunction with vascular development, it may worsen brain injury during the acute phase. An *in vitro* investigation found that there was a correlation between twofold levels of hippocampus VEGF and systemic inflammation. However, VEGF may help with blood vessel repair in the latter stages. Thus, there are significant time limits that need to be taken into account. Several low-cost methods have been suggested to improve host response besides iodide, such as intravenous sodium ascorbate infusion. It has been recommended as the standard treatment for sepsis, has been used regularly for a decade in Shanghai's critical care unit for a variety of indications, and is currently the main approach for treating COVID-19 patients there. It is well known that ascorbate inhibits VEGF and stops endothelial dysfunction. Some studies has suggested employing statins and Angiotensin Receptor Blockers (ARBs) to address endothelial dysfunction in sepsis and pneumonia brought on by new viruses, taking into account a broad approach to COVID-19 therapies.

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