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Commentary

Levels of Serum Homocysteine in Retinal Vein Occlusion Patients

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DESCRIPTION

The central retinal vein can become blocked, most commonly due to thrombosis, which is known as Central Retinal Vein Occlusion (CRVO). The central retinal vein and the central retinal artery both have the potential to become blocked. Due to ischemia (restricted in blood flow) and edoema, which can cause serious retinal damage and blindness, the central retinal artery and vein are the only sources of blood supply and drainage for the retina (swelling).

Ocular ischemia syndrome may be brought on by CRVO. The less severe form of CRVO is no ischemic. It could develop into the more serious ischemia kind. Glaucoma may potentially resulted from CRVO.

A systematic review identified no higher prevalence of thrombophilia (an innate susceptibility to thrombosis) in patients with retinal vascular occlusion, despite the importance of thrombosis in the development of CRVO.

Pan-Retinal Laser Photocoagulation and anti-VEGF medications like Lucentis or intravitreal steroid implants (Ozurdex) are typically used as treatments. Treatment for underlying issues is also necessary. The visual prognosis of CRVO without ischemia is better than that of ischemic CRVO.

One of the most frequent reasons for vision loss in the elderly is Retinal Vein Occlusion (RVO), which has been linked to an increase in cardiovascular mortality. The primary etiopathogenic variables for RVO are classical Vascular Risk Factors (VRF). In fact, it is typically believed that the condition could be seen as a symptom of systemic atherosclerosis.

Nonetheless, a number of studies have hinted that some RVO patients may be affected by a hypercoagulable state, however this has not yet been proven. Thus, we recently demonstrated that genetic thrombophilia need to be ruled out in patients under the age of 50 whereas acquired thrombophilia (antiphospholipid syndrome and hyperhomocysteinemia) should be taken into account in the clinical assessment of patients with RVO. A sulfhydryl-containing amino acid called Homocysteine (Hcy) is produced as an intermediary byproduct of the metabolism of methionine and cysteine. McCully's 1969 report was the first to mention Hcy's proatherogenic effects. The risk of stroke, myocardial infarction, and venous thrombosis has since been linked to elevated Hcy levels.

The activation of protein C, the up-regulation of factor V, the enhanced oxidation of LDL, the inhibition of plasminogen activator binding, and other processes have all been put up to explain how serum Hcy may contribute to the development of RVO, and the activation of protein C. Studies have examined the connection between serum Hcy levels and RVO. However, the majority of them are retrospective in nature, show significant heterogeneity, lack well-characterized population-based controls, and lack adjustment for probable confounder. Because of this, case-series studies were included in meta-analyses on the relationship between serum Hcy and RVO, which only indirectly compared cases and controls and reported a significant level of study heterogeneity. The outcomes have so been called into question. Recent systematic reviews and meta-analyses of case-control studies provided some support for the hypothesis that plasma Hcy was connected to a little rise in RVO. However, it has been discovered that the same flaws highlighted for earlier meta-analyses exist significant heterogeneity among included papers, publication and selection bias, lack of adjustment for significant Confounding variables, a lack of population-based data, and an ambiguous definition or choice of controls.

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