



Role of Imaging in the Management of Macular Edema Secondary to Retinal Vein Occlusion

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An understanding of the disease mechanisms underlying retinal vein occlusion (RVO) is fundamental to accurate interpretation of ocular imaging in patients with this disease.¹ Therefore, we begin this chapter with a simple overview of these mechanisms: first, the processes leading to occlusion of the vein; and second, the anatomic and visual consequences.

In most patients, RVO develops as a result of arteriosclerosis; hence, systemic cardiovascular risk factors (eg, hypertension) play a key role.^{2,3} Because retinal arterioles and venules share a common adventitial sheath at crossing points, thickening and hardening of the arterial walls impinges on the venules at these points, leading to venous narrowing. The resulting stasis—and then thrombosis—leads to venous occlusion. Less commonly, inflammation of the retinal veins (phlebitis) or hypercoagulable states can produce occlusion.^{4,5} Occlusion then causes elevation of first venous, and then intracapillary pressure, with subsequent slowing of arterial flow; the combination of these factors leads to extravasation of serous fluid and hemorrhage, as well as to capillary endothelial damage.⁶ Subsequent increases in interstitial pressure act as an impediment to capillary perfusion and leads to ischemia. Ischemia, in turn, leads to increased production of vascular endothelial growth factor (VEGF) with resulting increased hyperpermeability and creation of a vicious circle.⁷⁻⁹