A delicate exchange of homeostatic mechanisms is in place with the vitreous, retina, retinal pigment epithelium (RPE), and choroid receiving their circulation through the retinal and choroidal vasculature. A variety of risk factors may disrupt the normal interactions affecting the retinal environment. There is an intrinsic balance amongst the osmotic force, hydrostatic force, capillary permeability, and tissue compliance that occur within the vasculature [3][4]. Specifically, the capillary filtration rate should equal the rate of fluid removal from extracellular retinal tissue, such as glial and RPE cells. Once these forces are disrupted an imbalance occurs and accumulation of fluid is seen in cystoid spaces within the inner layers of the retina, most commonly the outer plexiform layer (OPL). The OPL is more prone to fluid collection due to the watershed area that exists between the retinal and choroidal circulation, especially within the central retina due to its anatomical avascular zone [5]. Accumulation of the fluid commonly occurs in the Henle's fiber layer causing the classic petaloid pattern. Specifically, a common factor that can cause CME is vitreomacular traction (VMT). VMT can cause stress at the Muller cell end-feet, exerting tractional forces and contributing to the release of inflammatory factors such as basic fibroblastic grown factor (bFGF), vascular endothelial growth factor (VEGF), and platelet-derived growth factor (PDGF). This results in blood-retinal barrier breakdown from separation of the retina and RPE, lysis of muller cells, leakage and edema [6][7][8][9]. However, typically CME associated with VMT does .not demonstrate leak on FFA