Diagnosis and Management of Diabetic Retinopathy

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Abstract

Diabetic retinopathy is one of the most significant consequences of diabetes mellitus. The incidence of diabetes continues to rise globally. Diabetic retinopathy (DR) is typically asymptomatic, so early detection and yearly dilated eye examinations (DEE) with an ophthalmologist is imperative. When symptoms do occur, the patient may experience increased occurrence of floaters, blind spots in their vision and blurred vision. DR is classified into 2 types: Non-Proliferative Diabetic Retinopathy (NPDR) and Proliferative Diabetic Retinopathy (PDR). Diagnosis can be confirmed using fluorescein angiography (FANG) and DEE. Management of DR focuses on prevention: lifestyle modifications such as diet modification, checking Hemoglobin A1c levels, and increasing exercise levels. One of the mainstays of pharmaceutical management for moderate to severe DR and macula edema (DME) is anti-vascular endothelial growth factor (VEGF) intraocular injections. Anti-VEGF medications have a short half-life and patients need to see an ophthalmologist for repeat injections monthly or bi-monthly. Intravitreal corticosteroid injections have also proven to be efficacious, especially in cases where patients are not responsive to anti-VEGF treatment. Vitrectomy is performed when hemorrhage into the vitreous cavity is extensive.

Diabetic Retinopathy Diagnosis

DR signs and symptoms may include:

- increased occurrence of floaters, blind spots in their vision and blurred vision. Floaters are most often caused by vitreal hemorrhages from leaking blood vessels. Blind spots occur from DME and areas of protein exudation.
- premature cataract formation caused by the osmotic effect on the proteins in the lens. This occurs from diffusion of increased blood glucose from the aqueous and vitreous.
- Visual acuity (VA) is often affected. Diplopia can occur from diabetes-induced extraocular muscle palsies.

DR is classified into 2 types: Non-Proliferative Diabetic Retinopathy (NPDR) and Proliferative Diabetic Retinopathy (PDR). NPDR occurs when hemorrhages, microaneurysms, exudates, cotton wool spots (CWS), or venous beading are present (Figure 1). PDR is characterized by retinal ischemia, neovascularization, and pre-retinal and vitreal hemorrhages from vascular damage (Figure 2). When the central part of the retina, known as the macula is involved, the condition is referred to as DME. It is the leading cause of vision loss in type 2 diabetics. This can result alongside NPDR and PDE or as its own pathology. Diagnosis confirmed using fundus photography, FANG and optical coherence tomography (OCT) (Figure 3) during a DEE.

Diabetic Retinopathy Diagnosis

Dot-blot hemorrhages are formed in the arterioles located in the inner layers of the retina. They will have a small, pinpoint red macule appearance. Flame-shaped hemorrhages are named for their indistinct borders and run horizontally along the nerve fiber layer of the retina.

Diagnosis can be confirmed using FANG and a DEE. The fluorescein is injected in the arm and a series of photographs are taken with a retinal camera to evaluate the blood flow to the retina and choroid. Images from serial exams can also be used to track the progression or regression of any DR. OCT is another imaging modality in which an image of the retina is produced that can assess its thickness and areas of fluid accumulation (edema) (Figure 3). CWS are areas of ischemia that turn into a white or yellow appearing area on the retina. Persistent diabetic damage to retinal vessels causes pericyte loss which further weakens the tight junctions and results in protein leakage. As proteins leak out of the vessels, they form exudates which can be seen on the retina. Venous beading is the appearance of the retinal veins with outpouchings that look like beads on a string or a sausage casing (Figure 4). It is a sign of progression towards PDR as these form due to decreased perfusion (ischemia) of the retina. As the ischemic disease process progresses, it often leads to the development of neovascularization. Structurally, neovascular vessels are more tortuous in appearance and more fragile. They often grow on the surface of the retina and optic disc or even progress into the vitreous (Figure 2). Because they are fragile, these vessels leak and result in retinal edema and frequently, hemorrhage into the vitreous (Figure 5). The tension from the vessels tethering to the vitreous as well as the scarring that results can also cause parts of the retina to detach from the posterior surface of the eye. This retinal detachment can result in abrupt vision loss and if not addressed promptly may be irreversible. Neovascularization in the anterior chamber of the eye begins on the iris surface and eventually can obstruct fluid flow through the trabecular meshwork (Figure 6). This increases ocular pressure and can result in neovascular glaucoma.

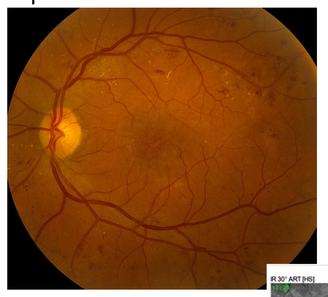

Figure 1

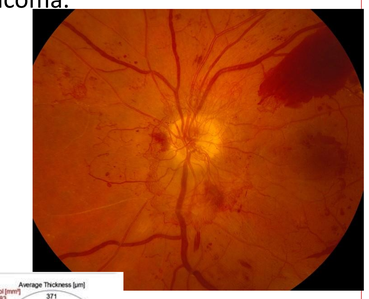

Figure 2

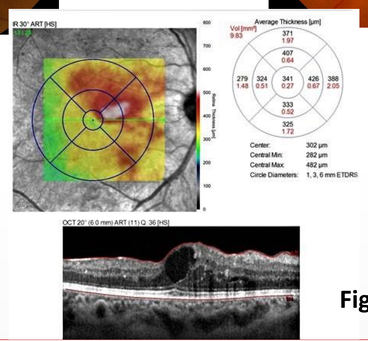

Figure 3

Management

Management of DR focuses much on prevention, which emphasizes lifestyle modifications such as diet modification, checking Hemoglobin A1c levels, and increasing exercise levels. Obtaining adequate control of blood glucose is the first line of management. Prevention can help preserve vision. The Hemoglobin A1c goal for diabetics should be under 7%.

Anti-VEGF intraocular injections:

- injected into the vitreous cavity and inhibit the growth of new blood vessels in the retina
- Mainstay of pharmaceutical management for moderate to severe DR and DME
- Ranibizumab (Lucentis 0.3 mg), bevacizumab (Avastin), and aflibercept (Eylea)
- Four out of ten patients with DME improved their VA by 3 lines on the Snellen eye chart after one year of anti-VEGF use
- Have a short half-life and patients need to see an ophthalmologist for repeat injections monthly or bi-monthly. Monitored with OCT and FANG.

Intravitreal corticosteroid injections have also proven to be efficacious, especially in cases where patients are not responsive to anti-VEGF treatment. These include triamcinolone (Triescence) which can last from 3 to 4 months or one of the intravitreal implants such as dexamethasone (Ozurdex) or fluocinolone (Iluvien). The advantage of the steroid implants is that they release low-dose corticosteroid for as long as 36 months with a single injection. All of these steroid products target cytokines involved in the inflammatory cascade and help treat DME. They also have undesirable side effects such as elevating IOP and cataract formation.

Vitrectomy is performed when hemorrhage into the vitreous cavity is extensive and impedes vision for an extended period of time.

Conclusion

Management of the patient's systemic diabetes is crucial in mitigating the effects of DR. This can be achieved by detecting early ocular signs and symptoms of the disease. Management of the patient's cholesterol, blood glucose, Hemoglobin A1c, blood pressure and smoking cessation are helpful initial steps. Annual DEE and potential treatment by an ophthalmologist is necessary to preserve vision.

Figure 4

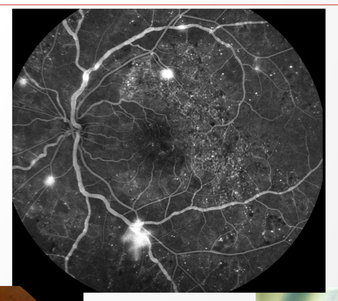

Figure 5

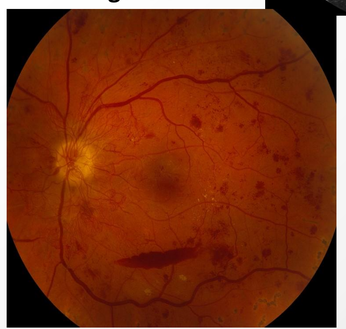

Figure 6