ISSN: 2573-9573

Case Report

Journal of Ophthalmology & Clinical Research

A Case of Bilateral Optic Edema in a 71 Year Old Diabetic

Tyler M Kaplan[1*], Bruce H Kaplan[1], Ira Garoon[1], Robert B Garoon[1] and Lisa Leishman[2]

[1]*Department of Ophthalmology, Chicago Medical School, Rosalind Franklin University of Medicine and Science, North Chicago, IL, USA*

[2]*Retina Consults LTD., Des Plaines, IL, USA*

[*]Corresponding author

Tyler M Kaplan, Department of Ophthalmology, Chicago Medical School, Rosalind Franklin University of Medicine and Science, North Chicago, IL, Address: 2 Burr Oak Trail, Riverwoods, IL, 60015, USA, Tel: 847-338-7600

Submitted: 09 Aug 2021; **Accepted:** 19 Aug 2021; **Published:** 13 Sept 2021

Citation: *Tyler M Kaplan, Bruce H Kaplan, Ira Garoon, Robert B Garoon and Lisa Leishman (2021) A Case of Bilateral Optic Edema in a 71 Year Old Diabetic. J Ophthalmol Clin Res 5(3): 103-105.*

Case Report

A 71 year old Hispanic male with a past medical history of hypertension, type II diabetes mellitus, and bilateral anatomically narrow angles status post bilateral peripheral iridotomies presented with the complaint of "looking through a film." The patient could still drive, work as a cashier, see his computer and television, and read with his glasses. It was worse in the morning and resolved when he washed his face. The patient denied headaches, jaw claudication, weight loss, and anorexia. Pt had no neurologic deficits including diplopia. The patients hemoglobin A1c 4 months prior to being seen was 7. The patient endorsed checking his blood sugars at home and most are less than 200. The patient routinely checks his blood pressure at home and it is usually 130s/70s. The patient's best corrected visual acuity was 20/20 in each eye at distance, intermediate, and near. His Ishihara color test was 11/11 in each eye as well. His intraocular pressure was 13 in each eye with corneal thickness of 642 OD and 626 OS. There was no relative afferent pupillary defect in each eye, extraocular muscle movements were full, and his confrontation fields were full as well. His anterior segment exam was unremarkable except for nuclear sclerosis of each lens. The fundus examination was remarkable for bilateral optic disc swelling with heme off both optic discs. Fluorescein angiography demonstrated bilateral optic disc leakage. OCT showed a flat sensory retina in each macula. The patient had an emergent MRI that demonstrated diffuse loss of normal high T2 signal, but no intracranial mass was present. CRP and ESR were both within normal limits when accounted for the patient's age. The patient's papilledema improved on one month follow-up exam without intervention. Due to the patient's history of diabetes, normal corrected visual acuity, reportedly well controlled blood pressure, and essentially unremarkable work-up, the patient was diagnosed with diabetic papillopathy.

Discussion

Diabetic papillopathy (DP) is self-limiting unilateral or bilateral optic disc swelling not associated with significant changes in a patient's vision. It can occur in either type I or type II diabetics and occurs independent of glycemic control or the severity of diabetic retinopathy [1]. The incidence of DP is approximately 0.5% [1]. A thorough work-up must be completed prior to diagnosing a patient with diabetic papillopathy and thus it is a diagnosis of exclusion. The differential for a patient who presents with new optic disc edema is broad including elevated intracranial pressure, hypertensive retinopathy, vasculities, trauma, toxicity, and various infectious and inflammatory etiologies [1,2]. As previously noted, the patient had an extensive work-up, reportedly well controlled hypertension, minimal visual changes, and most importantly optic disc edema that resolved without intervention thus making diabetic papillopathy the most likely etiology of the patient's self-limited optic disc swelling.

The etiology of diabetic papillopathy is not well defined. It can be associated with macular edema [3]. Risk factors for diabetic papillopathy include rapid improvement in the control of diabetes and a small cup to disc ratio [3]. Patients with a small optic disc are at greater risk because optic disc swelling within a smaller optic disc can induce a compartment syndrome like process [4]. DP is often considered a mild form of non-arteritic anterior ischemic optic neuropathy (NAION) [3]. However, this is complicated by the fact that it is considered a distinctive disorder as well. NAION is differentiated from DP by visual loss being worse and more persistent than in DP. In addition, fluorescein angiography generally demonstrates early disc hypofluorescence in NAION from hypoperfusion while DP generally demonstrates early disc leakage likely from telangiectasia of the optic disc [3].

DP is generally self-limited and resolves in 2-10 months. There is not a consensus on the treatment of DP. Some have noted expedited resolution of optic disk swelling following intravitreal injections of anti-VEGF or intraocular steroids [2,5]. One case report by Al-Dhibi and Khan argued for the efficacy of anti-VEGF in DP by providing treatment to only one eye. The eye that received treatment demonstrated complete resolution of optic disc edema in four weeks while the untreated eye had persistent optic disc edema after three months [5]. This supports VEGF playing a

role in the pathophysiology of diabetic papillopathy possibly from optic nerve ischemia inducing VEGF expression and increasing vascular permeability resulting in vasogenic disc edema [1,5]. In addition, intravitreal steroids have been utilized for the treatment of diabetic papillopathy [6]. Al-Haddad, et al. observed resolution of optic disc edema within 2 weeks of intravitreal injection. The major difference between our patient and the cases discussed is our patient had a visual acuity of 20/20, mild visual complaints, and no diabetic macular edema (DME) on presentation. While in the other discussed cases, the patient's had a decline in visual acuity due to DME. Therefore, there was a separate indication for the usage of anti-VEGF or intraocular steroids. Although, anti-VEGF or intraocular steroids may accelerate the improvement of optic disc edema in DP, if a patient does not have a significant decline in their vision it may not be worth the risks associated with therapy since disc edema in DP generally resolves without intervention.

It can be diagnostically challenging at times to discern disc neovascularization due to proliferative diabetic retinopathy (PDR) from diabetic papillopathy [2]. A case report by Choi et al demonstrated the value of OCT-A in the diagnosis of DP. In disc neovascularization, OCT-A demonstrates positive flow signals elevated about the vitreoretinal interface and signal flows of randomly oriented new vessels both of which are generally not present in DP [2]. Fine and radially orientated telangiectatic vessels often found in DP can be more easily visualized on OCT-A and that they are located below the vitreoretinal interface. It can be challenging on fluorescein angiography to identify neovascularization as it can be obscured by leakage of telangiectatic vessels found in both neovascularization and DP [2]. It is crucial to make the proper diagnosis as PDR requires intravitreal anti-VEGF therapy while DP generally only requires close follow-up. Due to the challenge of differentiating PDR from DP, it raises the possibility that improvement of disc edema following intravitreal anti-VEGF may be due to misdiagnosis of PDR as DP. DP is diagnostically challenging due to the myriad causes of optic disc edema and pathologies with overlapping presentations. However, if a patient does not have significant visual complaints, close follow-up is generally indicated to avoid complications from interventions and provide time for the underlying disease process to better present itself.

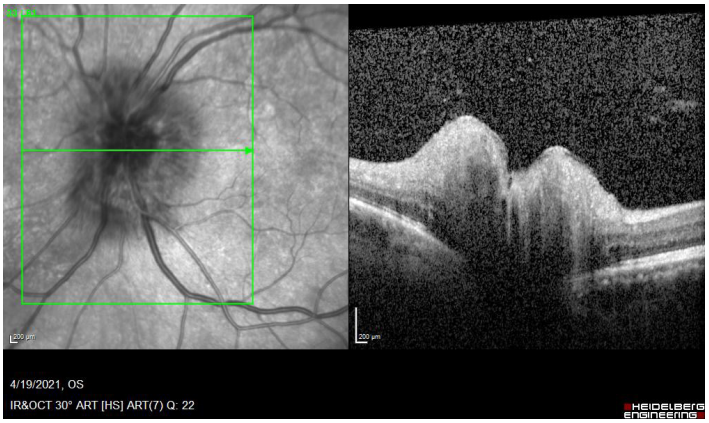
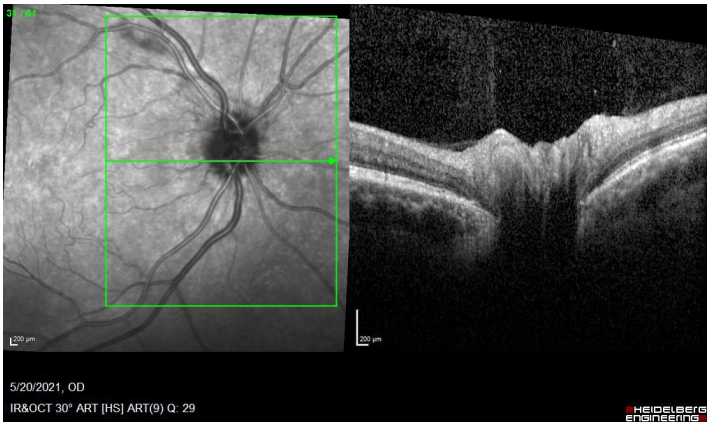


Figure 1: A) Right eye fluorescein angiogram and OCT demonstrating optic nerve edema B) Right eye fluorescein angiogram and OCT demonstrating optic nerve edema



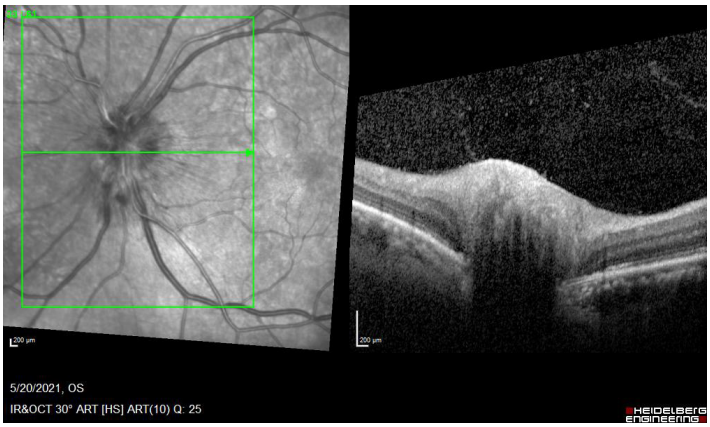


Figure 2: A) Right eye fluorescein angiogram and OCT demonstrating improvement of optic nerve edema B) Left eye fluorescein angiogram and OCT demonstrating improvement of optic nerve edema

References

1. Kim M, Lee JH, Lee SJ (2013) Diabetic papillopathy with macular edema treated with intravitreal ranibizumab. Clin Ophthalmol. 2013: 2257.
2. Choi JM, Lee HJ, Ma DJ (2020) Swept-source optical coherence tomography angiography of diabetic papillopathy: a case report. BMC Ophthalmol 20: 194.
3. Al-Hinai A, Al-Abri M, Al-Hajri R (2011) Diabetic papillopathy with macular edema treated with intravitreal bevacizumab. Oman J Ophthalmol 4: 135.
4. Atkins EJ, Bruce BB, Newman NJ, Biousse V (2010) Treatment of Nonarteritic Anterior Ischemic Optic Neuropathy. Surv Ophthalmol 55: 47-63.
5. Khan A, Al-Dhibi H (2011) Response of diabetic papillopathy to intravitreal bevacizumab. Middle East Afr J Ophthalmol 18: 243.
6. Ghasemi Falavarjani K, Nguyen QD (2013) Adverse events and complications associated with intravitreal injection of anti-VEGF agents: a review of literature. Eye 27: 787-794.