Rapidly reversing bilateral macular edema associated with fluid overload in a young type 1 diabetic

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We report a case of sudden onset bilateral diminution of vision in a young lady with type 1 diabetes. She was administered intravenous fluids for correction of diabetic ketoacidosis (DKA) prior to onset of her ocular symptoms. Dramatic resolution of macular edema was noted within a very short period after correction of fluid input–output ratio. Visual acuity was restored to baseline after 3 days without any active ocular intervention.

Key words: Diabetic ketoacidosis, diabetic retinopathy, fluid overload, macular edema, type 1 diabetes

Macular edema in a diabetic patient is multifactorial. Adequate metabolic control and monitoring of systemic parameters are equally important as compared to other conventional treatment modalities, like anti-vascular endothelial growth factor (VEGF) injections and laser photocoagulation. Here, we report a unique case of bilateral macular edema aggravated by fluid overload in a type 1 diabetic and its resolution without any active ocular intervention.

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Case Report

A 27-year-old lady was referred to the ophthalmology department with mild blurring of vision in the left eye (LE) for last 6 weeks. She was admitted in medicine ward at a tertiary care hospital few days back for a urinary tract infection (UTI). She was also recently diagnosed with type 1 diabetes. Ocular examination at this point revealed best-corrected visual acuity (BCVA) of 6/6 in the right eye (RE) and 6/9 in the LE. Intraocular pressures were normal. Anterior segment examination was unremarkable. Fundus revealed few microaneurysms and hemorrhages in both eyes (BE) suggestive of mild non-proliferative diabetic retinopathy (NPDR). Optical coherence tomography (OCT) revealed normal foveal contour in the RE [Fig. 1a] and a few cystoid spaces in the LE [Fig. 1b]. The laboratory evaluation revealed fasting blood sugar (FBS) 186 mg/dL; hemoglobin 10 g/dL; white blood cell (WBC) count 9170/µL (polymorphs 66%); urea 57 mg/dL; creatinine 1.35 mg/dL. Lipid profile and serum electrolytes were normal. She was kept under observation from ocular point of view and managed conservatively at this moment.

Eleven days after her initial ocular evaluation, she was referred again with a sudden onset of painless marked diminution of vision in BE since last 2 days. On examination, her BCVA was 4/60 in the RE and 5/60 in the LE. Fundus revealed marked increase in macular edema, disproportionate to background diabetic retinopathy changes. OCT showed multiple large intraretinal cystoid spaces with small serous detachment in BE [Fig. 1c and d]. Central macular subfield thickness (CMT) increased from 220 µm at baseline visit to 448 µm in second visit in the RE [Fig. 1c] and from 255 µm to 483 µm in the LE [Fig. 1d]. She was also diagnosed as diabetic ketoacidosis (DKA) 4 days prior to second ocular visit (maximum recorded blood sugar was 553 mg/dL and HbA1c 19%) and managed with intravenous fluid, insulin,

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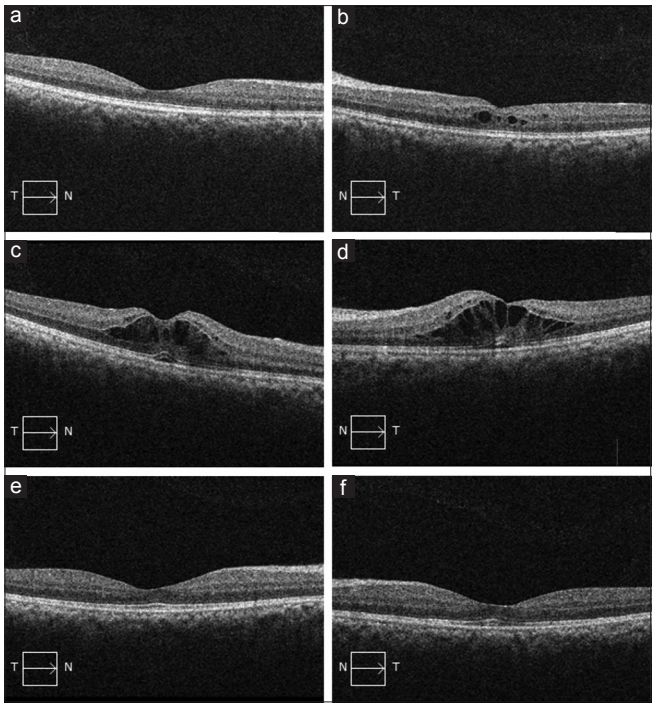

Figure 1: (a and b) Optical coherence tomography (OCT) of the right and the left eye, respectively, at first visit showing normal foveal contour in the right eye and few cystoid spaces in the left eye; (c and d) OCT of the right (c) and the left (d) eye at second visit (after fluid overload; 11 days after first visit) showing multiple large intraretinal cystoid spaces with small serous detachment in both eyes; (e and f) OCT of the right (e) and the left (f) eye at third visit (14 days after first visit) showing resolution of macular edema following correction of fluid overload

Table 1: Intake output chart of the patient during admission. Day 1 refers to first ocular visit and corresponds to Figure 1 (a and b). Day 12 refers to second ocular visit after sudden worsening of vision and corresponds to Figure 1 (c and d). Note significant positive fluid balance in Day 9, 10, and 11. Day 15 refers to third ocular visit and corresponds to Figure 1 (e and f)

	Input (ml)	Output (ml)	Balance (ml)
Day 1 (first visit)	1400	1200	200
Day 2	1700	1300	400
Day 3	Records not available		
Day 4	1400	1000	400
Day 5	1200	1150	50
Day 6	1200	1100	100
Day 7	1300	1000	300
Day 8	Records not available		
Day 9	5000	1700	3300
Day 10	3100	1300	1800
Day 11	3250	1100	2150
Day 12 (second visit)	1950	1250	700
Day 13	1750	1300	450
Day 14	1650	1250	400
Day 15 (third visit)	1400	1150	250

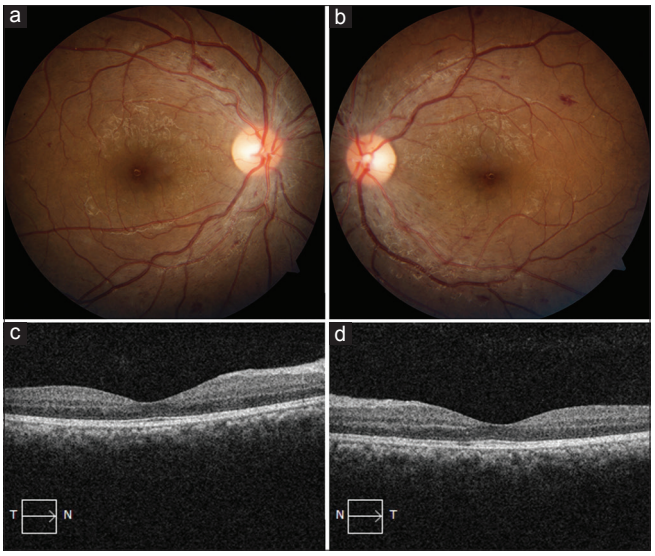

Figure 2: (a and b) Fundus photo of the right and left eye, respectively, at 3 months follow up, showing few microaneurysms, dot blot, and flame-shaped hemorrhages suggestive of mild non-proliferative diabetic retinopathy; (c and d) optical coherence tomography showing normal foveal contour in both eyes at 3 months of follow up

and electrolyte replacement. Systemic examination revealed ascites, bilateral mild pleural effusion, and pedal edema. Marked macular edema and systemic findings were attributed to fluid overload. Strict monitoring of intake and output was carried out for next few days without any active ocular intervention and it resulted in rapid resolution of macular edema. Repeat OCT at third visit (just 3 days after worsening) showed remarkable decrease in CMT in BE [Fig. 1e and f] with restoration of BCVA to 6/12 in BE. She was discharged after systemic stabilization on stable blood sugar levels. At follow up after 3 months, her visual acuity improved to 6/6 in the RE and 6/9 in the LE; fundus had bilateral mild NPDR changes [Fig. 2a and b] without any macular edema [Fig. 2c and d].

Discussion

The hallmark of pathogenesis of diabetic macular edema (DME) is alteration of blood retinal barrier (BRB) which is guided by complex interplay between various biochemical and hemodynamic factors.[1] Edema is governed by Starlings principle which states the hemodynamic relationship between vascular and tissue compartments.[2] If the hydrostatic pressure and oncotic pressure gradient between the intravascular and extravascular compartment is maintained optimally, there is no edema. But in case of fluid overload due to rapid intravenous administration, there can be imbalance between hydrostatic and oncotic pressure leading to edema. We believe macular edema in our case is due to the above reason, which is further substantiated by collection of fluid in other extravascular spaces (ascites, pleural effusion, and pedal edema).

The other probable explanation of marked macular edema is biochemical perturbation of the retinal microvasculature during acute insult of DKA. However, Martin *et al.* evaluated

the structural and functional aspects of the retina immediately following the correction of DKA in seven young patients and concluded that the BRB may act as a relatively protected site as compared to blood–brain–barrier (BBB).[3] The authors attributed greater stability of the retinal microvasculature to the increased number of pericytes in the BRB as compared to BBB.[3]

Moreover, careful analysis of intake output chart [Table 1] shows fluid imbalance in favor of positive tilt just prior to onset of ocular symptoms (Day 9, 10, and 11 in Table 1 shows positive balance of 3300 ml, 1800 ml, and 2150 ml for three consecutive days). Once this fluid overload got corrected as evidenced by decrease in positive fluid balance in next 3 days (700 ml, 450 ml, 400 ml in Day 12, 13, and 14 in Table 1), the equilibrium between hydrostatic and oncotic pressure was restored leading to resolution of macular edema. Few studies have reported that fluid overload might be associated with exacerbation of DME.[4-6] Ciardella reported marked improvement of DME after systemic treatment with furosemide.[4] Ryan *et al*. described DME in patients who developed fluid retention as a consequence of glitazone use in a series of 30 patients.[5] Kameda *et al*. have reported a case of a giant retinal pigment epithelial (RPE) tear associated with fluid overload in a patient with DME and kidney disease.[6] However, such rapid development and resolution of macular edema over few days associated with fluid overload in a type 1 diabetic patient with DKA have not been reported in the literature earlier to the best of our knowledge.

Conclusion

A period of observation and optimal control of deranged systemic parameters by a multidisciplinary team are of vital importance before more invasive treatment modalities such as intravitreal injection and laser photocoagulation are considered for management of macular edema.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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