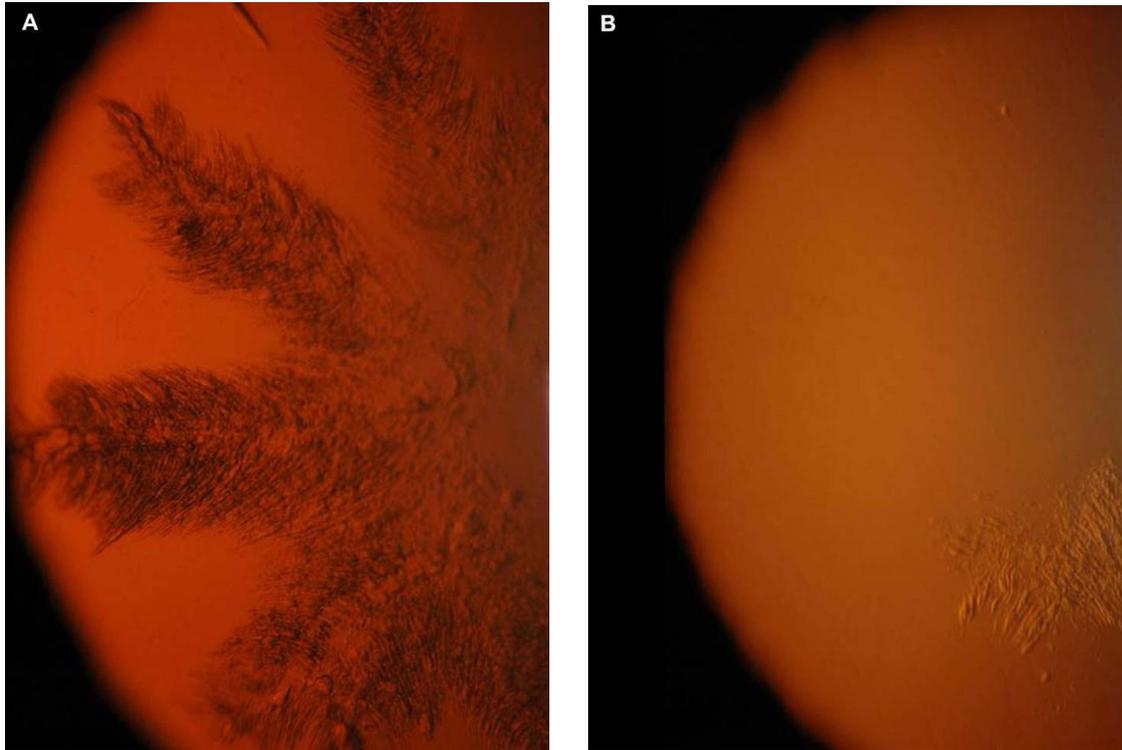


## Reversal of Bilateral Rosette Cataracts with Glycemic Control

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A 58-year-old woman diagnosed with type-2 diabetes mellitus 3 weeks prior to admission presents to the emergency room with a chief complaint of decreased vision for the past 2 weeks and polyuria. She reports blood sugars in the 400s for the preceding 3 weeks. Her blood glucose on admission was 500 and her HbA1c was 15.5%. Her ophthalmic exam was positive only for a visual acuity of 20/400 and feather-

shaped posterior subcapsular lens opacities (Fig. A) bilaterally. Glycemic control was quickly achieved with insulin and she was discharged. At the 2-week follow-up visit, her vision was 20/60 and the cataract had predominantly reversed (Fig. B). One month later, the lens was clear and her vision was 20/30. Two months later, her vision was 20/20. She reports maintaining blood sugars in the 100s since discharge.

The lens equatorial epithelium migrates first to the posterior subcapsular cortex and is affected most by hyperglycemia. Hypoinsulinemia causes sorbitol accumulation in the lens and fluid influx[1]. Cellular hydration delineates lens fibers, which are normally feather shaped, and impairs vision. Administration of insulin and fluids causes conversion of sorbitol to fructose, which diffuses out[2]. Reduced lens osmolarity allows vacuole resorption, cataract reversal, and vision improvement.

Acute hyperglycemia-induced cataracts are reversible with prompt glycemic control[3]. Uncorrected, hyperglycemia causes permanent cataracts that must be managed surgically. With acute worsening of vision due to cataract formation in the setting of hyperglycemia, supportive therapy with glycemic control and observation alone can provide a dramatic improvement in vision in a few weeks.

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