The patient is an 82-year-old male with a longstanding history of hereditary chorioretinopathy and pseudophakia for 6 years, who initially presented with a visual acuity with correction of 20/150 OD and 20/60 −2 OS. There was no cell in the anterior or posterior segment. Optical coherence tomography (OCT) showed cystoid macular edema (CME) . Fluorescein angiography demonstrated retinal vascular leakage in the macula with fluorescein accumulation in a petaloid pattern surrounding the fovea There was some consideration that the CME was associated with ellipsoid-zone or retinal pigment epithelial disruption associated with his hereditary chorioretinopathy, but there was no evidence of ellipsoid-zone–retinal pigment epithelial disruption . Medical history included type 1 diabetes, congestive heart failure, hypertension, and HIV infection. Medications included darunavir, ritonavir, furosemide, insulin, and apixaban. There was no change in CME after 3 months of topical ketorolac, 0.5%, dministered 6 times daily and topical prednisolone acetate, 1%, administered 4 times daily. He then received STK, 20 mg, in each eye, which resulted in improvement in visual acuity to 20/40 OD and 20/40 OS and reduced OCT CME thickness. Within 2 months of starting STK, the patient started having progressively worsening exercise tolerance, dyspnea on exertion, lower extremity weakness, increased fat in his face and abdomen, easy bruising, muscle wasting, and confusion. He continued to receive STK, 20 mg, in each eye every 3 months, for a total of 3 doses in each eye. One month after the third dose of STK, the patient was seen in the emergency department for progression of all his symptoms including skin atrophy and admitted for further evaluation.

Corticotropin level was low at 3.2 pg/mL (normal, 7-63 pg/mL; to convert to picomoles per liter, multiply by 0.22) and early morning cortisol level was low at 2.0 µg/dL (normal, 3.7-19.4 µg/dL; to convert to nanomoles per liter, multiply by 27.588). Magnetic resonance imaging of the pituitary gland, and computed tomography of the adrenal glands were unremarkable. Cosyntropin (Cortrosyn [Amphastar Pharma]) stimulation test was normal. The patient received dexamethasone, 1 mg, orally at 11 PM and serum dexamethasone at 8 AM the following day was 855 ng/dL (reference range, 140-295 ng/dL), indicating that glucocorticoid metabolism was substantially inhibited and supported the diagnosis of iatrogenic Cushing syndrome caused by high circulating glucocorticoid level, presumably from STK, despite the low serum corticotropin and cortisol levels.