A 30 year old female presented with complaints of ocular discomfort for 8 months and increase in body weight, polyuria and fatigue for two years. There was associated pedal edema, truncal obesity, facial melanosis and hirsutism. Her best corrected visual acuity (BCVA) was 20/20 both eyes. Her baseline Intra ocular pressure (IOP) was 50 mmHg OD and 56 mmHg OS, and pachymetry was normal. There was no history of use of exogenous steroids or family history of glaucoma. On examination, she had normal anterior chamber depth, open angles on gonioscopy and a cup disc ratio of 0.4:1 OU. (Figure 1). Humphrey Field analyzer (HFA) 30-2 was within normal limits. Raised blood pressure (210/120 mmHg consistently) and persistently elevated blood sugars (between 301 and 480 mg/dl) were found. Endocrine investigations showed elevated plasma cortisol (35.2 ug/ ml) (range 4.3 – 22.4 ug/ ml). Overnight dexamethasone suppression test (2 mg) confirmed raised serum cortisol level. MRI depicted a small well-defined focal lesion of size 7 x 3 mm in the right adenohypophysis.

She underwent transsphenoidal hypophysectomy. Histopathology from excised specimen revealed basophilic monomorphic pituitary cells immunopositive for Adrenocortico tropic hormone (ACTH). (Figure 2 a and b) A diagnosis of ACTH-dependent Cushing Syndrome with endogenous steroid induced ocular hypertension was made. Two years postoperatively, she is off glaucoma medications, with a normal diurnal variation of intraocular pressure and no evidence of optic neuropathy.

**Question:** How does endogenous steroid induced glaucoma differ from intraocular pressure rise from exogenous steroid administration?

**Answer:** Patients with endogenous steroid induced glaucoma differ from patients with glaucoma caused secondary to exogenously administered corticosteroids, in running a lower risk of developing glaucomatous damage and having an overall better ocular prognosis. Endogenous steroid induced glaucoma should be suspected in patients with systemic features of hormonal imbalance,
in conditions when IOP is not controlled despite maximum medical therapy, or persistent headache. Systemic features like purple striae, facial plethora, proximal myopathy, hypertension, obesity, and uncontrolled diabetes along with ocular hypertension led to the suspicion of Cushing Syndrome.

**Question:** How does steroid exposure cause glaucoma?

**Answer:** Exposure to glucocorticoids induces metabolic changes in extracellular matrix, cytoskeletal re-organization, and changes in gene expression and cell function thus producing permanent changes in the human trabecular meshwork.

**Question:** How do you differentiate between visual field defects caused by neurological compression by pituitary tumor or due to glaucoma from hormonal secretion?

**Answer:** A compressive tumor etiology should be sought when one finds features suggestive of optic nerve pallor in excess of cupping, absent disc haemorrhages, poor disc/field correlation, vertically aligned field loss, visual acuity <20/40, poor colour vision, and age younger than 50 years.

**Question:** What is the treatment modality for endogenous steroid induced glaucoma?

**Answer:** Therapy in cases with hormone secreting pituitary tumours requires the removal of the source of these hormones, as well as IOP control. 26.5% patients with steroid induced glaucoma eventually require trabeculectomy for optimum IOP control. Our patient responded well to pituitary surgery.

**Question:** What are the causes of persistent IOP rise/glaucoma relapse after tumour resection?

**Answers:** Causes include an incomplete resection, tumour recurrence or enhanced steroidal sensitivity in conditions like high myopia, diabetes, Chronic open angle glaucoma (COAG), connective tissue diseases or familial predisposition. Rise in intraocular pressure along with progressive disc changes may serve as early markers of tumour recurrence.

**References**