BILATERAL THIRD NERVE PALSY SECONDARY TO APoplexy
IN A PITUITARY MACROADENOMA CAUSING CUSHING’S DISEASE
– A VERY RARE COMPLICATION OF A RARE ENTITY

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Background:
• Bilateral 3rd nerve palsy is seen in many conditions (diabetes mellitus, neurosarcoidosis, Guillain-Barre syndrome, multiple sclerosis, anterior or posterior communicating artery aneurysm or mesencephalic bleed)
• Only single cases have been reported in association with pituitary adenomas or carcinomas, usually in the context of apoplexy.
• We describe a patient with Cushing’s disease and bilateral 3rd nerve palsy secondary to apoplexy in pituitary macroadenoma causing massive transient release of ACTH.

Case presentation:
• 54 year old man with recent-onset hypertension, hypokalaemia and type 2 diabetes presented with weight loss, proximal myopathy and peripheral oedema.
• Random cortisol was 1800nmol/L, midnight sleeping cortisol 3200nmol/L (normal <50nmol/L).
• ACTH 188ng/L (normal 0-45ng/L).
• CT of the abdomen revealed bilateral adrenal enlargement.
• MRI scan of the pituitary showed a pituitary macroadenoma with central necrosis and left cavernous sinus invasion (Fig. 1A & B).
• The patient rapidly developed a left-sided 3rd nerve palsy, which was followed by right 3rd nerve palsy 24 hours later.
• His serum cortisol increased to 7500nmol/L.
• A CT confirmed haemorrhage into the pituitary macroadenoma (Fig. 2C).
• The patient developed septicaemia and associated thrombocytopenia and was not fit for transsphenoidal surgery.
• Four days following the apoplexy his 9.00h serum cortisol fell to 270nmol/L; in view of his sepsis hydrocortisone replacement was added.
• The 3rd nerve palsy resolved gradually in the right eye over following month.
• Subsequently, patient developed CSF leak (Fig. 2D) and underwent transsphenoidal adenomectomy and repair of the sella turcica.
• The left 3rd nerve palsy persists but the patient remains biochemically cured.

Conclusions
Bilateral 3rd nerve palsy, though very rare, can occur in Cushing’s disease, and if of acute onset is suggestive of pituitary tumour apoplexy. In this patient the massive surge in plasma ACTH and serum cortisol was considered to be secondary to the pituitary apoplexy.

References: