Society for Endocrinology

Imperial College London

Imperial Pituitary Masterclass Meeting

Monday 24th September 2018

IMPERIAL PITUITARY MASTERCLASS MEETING 2018

APPROVED FOR 6 EXTERNAL CPD POINTS (RCP, LONDON)

SUPPORTED AND ENDORSED BY THE SOCIETY FOR ENDOCRINOLOGY

Venue: Charing Cross Hospital, Imperial College Healthcare NHS Trust, London

08.30 - 09.00 Registration.

09.00 - 09.15 **Welcome.**
Dr Niamh Martin.

SESSION 1.

Chairs: Dr Rajee Baburaj and Dr Roselle Herring.

09.15 – 09.45 *Pituitary problems in pregnancy.*
Prof Catherine Williamson (King's College London).

09.45 - 10.00 Case presentation. *Management of macroprolactinoma during pregnancy and breast-feeding.*
M Parsad, M Silveira, M Al-Mrayat. (University Hospital Southampton NHS Foundation Trust).

10.00 - 10.15 Case presentation. *Cabergoline in management of large non-functioning pituitary macroadenoma during pregnancy.*
R Ramli, F Wernig, K Meeran, NM Martin, E Hatfield. (Imperial College Healthcare NHS Trust, London).

10:15 – 10.45 *Pituitary problems and male infertility – diagnosis and management*.
Dr Channa Jayasena (Imperial College London).

10.45 – 11.15 BREAK.

SESSION 2.

Chairs: Mr Ramesh Nair and Dr Ben Whitelaw.

11.15 - 11.45 *Hypophysitis - diagnostic challenges and new treatment options.*
Dr Florian Wernig, (Imperial College Healthcare NHS Trust, London).

11.45 - 12.00 Case presentation. *A case of IgG4-related hypophysitis.*
H Joshi, M Hikmat, SO Oyibo, SV Sagi. (Peterborough City Hospital).

12.00 - 12.15 Case presentation. *Rituximab induced long term remission from refractory lymphocytic hypophysitis. S* Raj S, BC Whitelaw, T Hampton, A King, SJB Aylwin. (King's College Hospital, London).

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12.15 – 12.30 Case presentation. *Metastasis to pituitary gland from renal cell carcinoma.* H Khan, S Vennam, T Dugal. (Royal Cornwall Hospital).

12.30 - 13.30 LUNCH.

13.30 - 13.40 LAPPS Update. Joy Ginn.

SESSION 3.

Chairs: Prof John Wass and Mr Nigel Mendoza.

13.40 - 14.30 Debate. *Patients with incidentally discovered pituitary macroadenomas should not have pituitary surgery unless they have abnormal visual fields.*
For the motion: Dr Paul Carroll (Guys and St Thomas's NHS Foundation Trust, London).
Against the motion: Miss Joan Grieve (The National Hospital for Neurology and Neurosurgery, London).

14.30 - 14.45 Case presentation. *Non-functioning pituitary adenomas in the elderly.* K Laycock, J Anderson. (Homerton University Hospital, London).

14.45 - 15.00 Case presentation. *An uncommon cause of infertility in a patient with a pituitary macroadenoma.* R Wilmington, R Russel, A Sinha, NJ Parr, U Srinivas-Shankar. (Wirral University Teaching Hospitals NHS Trust, Liverpool Women's Hospital, Walton Centre NHS Foundation Trust, Liverpool).

15:00 - 15:30 BREAK.

SESSION 4.

Chairs: Dr Marcus Martineau and Dr Emma Hatfield.

15.30 – 16:00 *Dopamine agonist withdrawal in prolactinomas – who should we offer this to and when?* Dr Antonia Brooke (Royal Devon and Exeter NHS Foundation Trust).

16.00 - 16:15 Case presentation. *Cabergoline therapy in macroprolactinoma after repeated surgery and radiotherapy.* M Silveira, M Parsad, M Al-Mrayat. (University Hospital Southampton NHS Foundation Trust).

16.15 - 16:30 Case presentation. *Acromegaly and associated pituitary macroadenoma resolution with cabergoline therapy.* N Thomas, A Brooke. (Royal Devon and Exeter NHS Foundation Trust).

16.30 - 16:45 Case presentation. *Late presentation of acromegaly in a patient with macro-prolactinoma.* AJ Beck, DE Flanagan. (University Hospitals Plymouth NHS Trust).

16.45 **CLOSING REMARKS AND FEEDBACK.**

Wireless Instructions

Find “Imperial” (open) on your list of connections.

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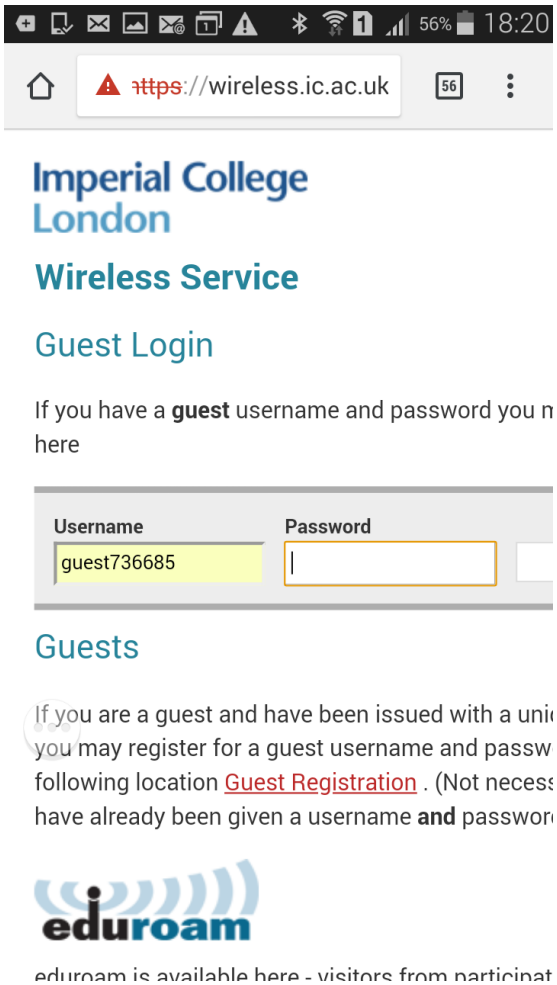
A guest login page will appear.

Put in guest736685 as the username, and Pituitary1 as the password.



Management of macroprolactinoma during pregnancy and breast-feeding.

M Parsad, M Silveira, M Al-Mrayat.
University Hospital Southampton NHS Foundation Trust.

We present a case of a 30 year old female who initially presented to her GP in April 2014 with galactorrhoea and intermittent headache. Initial prolactin level was 11714 mu/L (57.5-561 mu/L), oestradiol <73 pmol/L, FSH<0.2 iu/L (1.3-20), TSH 2.29mu/L (0.34-5.6) and cortisol 820 nmol/L. She was referred to the Endocrinology department and was commenced on cabergoline and titrated to 500 micrograms twice a week. We arranged for pituitary MRI and formal visual field testing. MRI revealed a macroadenoma measuring 12x11x15 mm with possibly a small haemorrhage. Optic chiasm was not touched or compressed. At 3 months follow-up, prolactin was 1882 mu/L and at 12 months, it was 584 mu/L.Cabergoline was titrated down to 500 micrograms once a week.

At 18 months follow-up, she expressed wishes of having a family but was finding difficulty to conceive. Repeat MRI pituitary in October 2015 showed shrinkage of the adenoma by 2mm at maximum diameter. At 2-year follow-up, hormone profile was LH 4 iu/L, FSH 3.9 iu/L, Prolactin 522 mu/L, TSH 1.4 mu/L, FT4 9.9 pmol/L, ostradiol 435 pmol/L. At 3 year follow-up, prolactin was 537 and MRI showed the prolactinoma had reduced further with a maximum diameter of 7mm, still with no success with conception.

In July 2017, the patient found out she was pregnant, at 4 weeks gestation, and cabergoline was discontinued straightway. She was clinically stable in endocrine clinic review at 8 weeks gestation with a prolactin level of 700 (blood tests were arranged prior to pregnancy being known). At 25 weeks gestation, she was reviewed in clinic having had normal visual fields in each trimester. A follow-up MRI scan 2 months post-partum showed the pituitary adenoma was at maximum diameter of 7mm and the patient continued to successfully stay off cabergoline whilst breastfeeding. Her visual fields remained intact.

Issues to consider:
1. Should we have switched from cabergoline to bromocriptine when planning for pregnancy?
2. Should we have continued with dopamine-agonist during pregnancy?
3. Should we have continued with dopamine-agonist whilst breastfeeding?

Cabergoline in management of large non-functioning pituitary macroadenoma during pregnancy.

R Ramli, F Wernig, K Meeran, NM Martin, E Hatfield
Imperial College Healthcare NHS Trust, London.

A 26-year-old woman presented with galactorrhoea and headache whilst taking the combined oral contraceptive pill (OCP) regularly. Initial biochemistry (off the OCP) showed hyperprolactinaemia (prolactin 2160 mIU/L; NR 50-500). MRI Pituitary showed a 2.5 cm predominantly cystic heterogeneous mass with suprasellar extension, elevating but not compressing the optic chiasm. Visual fields (VF) were normal. She was given the diagnosis of non-functioning pituitary macroadenoma with disconnection hyperprolactinaemia. On discontinuing the OCP, her oligomenerrhoea persisted, and as she failed to conceive, she was commenced on cabergoline 0.25mg twice weekly. A 6-month interval MRI Pituitary showed a similar sized pituitary macroadenoma, with slightly less suprasellar extension. Her case was discussed in the ICHNT Pituitary MDT, and as she was keen to become pregnant, two main treatment options were discussed. These were conservative management with clinical and radiological surveillance or surgical management (trans-sphenoidal hypophysectomy). She opted for conservative management, and fell pregnant with Clomid and cabergoline in January 2017. Due to the size of the adenoma, she continued on cabergoline 0.25mg twice weekly during pregnancy with a plan to discontinue it in the third trimester in preparation for breastfeeding. She was closely monitored, and visual fields throughout pregnancy were normal. An interval MRI Pituitary at 32 week gestation showed an increase in the cystic pituitary adenoma, with the optic chiasm stretched across it. At 35+5 weeks gestation, with normal visual fields, cabergoline was cautiously discontinued in preparation for breast feeding. She delivered a baby girl at 38+1 week gestation by normal vaginal delivery and successfully breastfed for a few weeks post-delivery. She is planning another pregnancy in the future.

Questions for discussion:
1. Would stopping cabergoline earlier in pregnancy, with close monitoring of visual fields, have been preferable to aid success of breastfeeding?
2. Could cabergoline have been stopped after conception?
3. Would similar conservative management be recommended for her next pregnancy?

A case of IgG4-related hypophysitis.

H Joshi, M Hikmat, SO Oyibo, SV Sagi.
Peterborough City Hospital.

Introduction Hypophysitis is a rare condition in which the pituitary gland becomes infiltrated with lymphocytes resulting in enlargement and impaired function. It could be autoimmune (AH) or secondary to systemic diseases or infections. Histopathological findings are classified into lymphocytic, granulomatous, xanthomatous, mixed forms, necrotizing and IgG4 plasmacytic types. Affected patients can present with headache, visual field impairment and more rarely, double vision. It is important to differentiate this condition from other non-secreting space-occupying lesions of sella turcica, since endocrine dysfunction and compressive effect may be transient. We report a case of IgG4-related hypophysitis.

Case A 39-yr old man with diabetes, presented with headache and double vision. Clinically he had left sided internuclear opthalmoplegia and right sided 6th nerve palsy. Pituitary profile was normal apart from a low testosterone level. Initial CT scan showed bulky pituitary gland but MRI was reported as normal-sized pituitary gland. A cerebral spinal fluid examination ruled out any demyelination process. He was commenced on antiplatelet therapy in case this was a brain stem ischaemic event. Six months later he was readmitted to hospital with severe headache and vomiting. His random cortisol and free thyroxine levels were very low. MRI showed a pituitary mass extending into super sellar space just touching optic chiasm with a thickened stalk. CT scan of chest, abdomen and pelvis was normal. Beta-HCG, serum ACE levels, α-fetoprotein, HIV and tests for TB were all negative. Serum light-free chains and immunoglobulin levels including IgG 4 were normal. Hypophysitis was suspected. Patient was started on hydrocortisone, testosterone and thyroxine replacement. Pituitary biopsy revealed inflammatory infiltrate with plasma cells likely IgG4 related. One month later repeat MRI showed stable disease but patient remained on hormone replacement.

Discussion We have presented an interesting case of autoimmune hypophysitis secondary to plasma cell infiltration. Glucocorticoids, anti-inflammatory and immunosuppressive drugs have been suggested as medical treatment however their long-term efficacy still needs to be confirmed. Hormone replacement is needed for pituitary dysfunction. Transphenoidal surgery to confirm diagnosis and save viable pituitary tissue may be required in individuals with symptoms or signs of severe compression. In some cases, pituitary biopsy is both diagnostic and therapeutic, as the procedure can induce a progressive recovery of pituitary function. Careful follow up is recommended.

Rituximab induced long term remission from refractory lymphocytic hypophysitis.

S Raj S, BC Whitelaw, T Hampton, A King, SJB Aylwin.
King's College Hospital, London.

In 1997 a female patient in her 40s developed symptoms of diabetes insipidus which was confirmed on biochemical testing. MRI scan showed a suprasellar mass surrounding the hypophyseal stalk. Transphenoidal biopsy demonstrated that this was an inflammatory lesion of uncertain aetiology. The patient was treated for panhypopituitarism with diabetes insipidus.

Three years later, she developed frontal headaches and MRI showed a recurrent suprasellar mass with optic chiasm distortion. Transphenoidal surgery was performed and the histology demonstrated lymphocytic hypophysitis. Over the next few months there was deterioration in visual fields due to recurrence of hypophysitis and this improved with dexamethasone. In 2002 fractioned radiotherapy was given which controlled headaches well and visual function was at that time normal.
From 2006 the patient developed recurrent episodes of bilateral worsening vision, each needing high doses of steroids to treat. She was sequentially trialled on steroid sparing agents Azathioprine, Mycophenolate Mofetil and Methotrexate but developed side effects to each of these.

In 2010 she had a further admission with worsening bilateral colour vision with only marginal response to methylprednisolone. She was taking a maintenance dose of 20mg Prednisolone and frequently higher doses. A decision was made to commence Rituximab therapy in view of steroid resistance. Weekly Rituximab infusions were given (375mg/m^2) for a total of 4 weeks in October 2010. Vision stabilised and gradually improved. There was a gradual radiological reduction in the suprasellar inflammatory mass. Over the following 8 years the hypophysitis has remained in remission. Prednisone has been weaned to a maintenance dose of 5mg.

Metastasis to pituitary gland from renal cell carcinoma.

H Khan, S Vennam, T Dugal.
Royal Cornwall Hospital.

Background: Symptomatic pituitary metastasis is rare. Less than 1% of pituitary surgical resections are found to be metastasis. It is difficult to differentiate from pituitary adenoma both clinically and radiologically. Renal tumours metastasis to pituitary is rarer than breast and lung primary malignancies. We present a case with no known malignancy and absence of hypopituitarism led to delayed diagnosis of pituitary metastasis.

Case
52 years old male was admitted to hospital with 1 week history of headache, dizziness and no neurological signs. MRI head showed sellar mass of 13x15mm in size with no contact to optic chiasm and features in keeping with pituitary adenoma. He was discharged with urgent outpatient MRI Pituitary, Visual field assessment and endocrine clinic follow up. Three weeks later he presented with worsening headache, and complete right ptosis.MRI scan showed non-haemorrhagic pituitary mass of increased size (21x16mm) abutting optic chiasm and invading cavernous and sphenoid sinuses. Blood test showed mild hyponatremia of 129mEq/L, 0900 Hrs Serum cortisol of 294nmol/L, TSH1.57Mu/L, Free T4 8.5pmol/L, Testosterone 0.1nmol/L, FSH 2.2mIU/ml, LH 1.9Miu/ml, prolactin 542IU/ml and normal IGF1. CT Thorax abdomen showed renal cell tumour with metastasis to liver and lung. Left kidney biopsy confirmed poorly differentiated carcinoma. Given significant metastatic disease process surgery on pituitary mass was not done. Stereotactic radiotherapy was deemed not suitable due to proximity with the chiasm. He was started on Dexamethasone, and later on levothyroxine and testosterone. He was treated with palliative radiotherapy to head and immunotherapy (Pazopanib) for renal cell carcinoma. His neurological symptoms improved with complete resolution of ptosis and stable disease on repeat imaging.

Conclusion: Our patient did not have the common presentations of pituitary metastasis: diabetes insipidus, raised prolactin or discriminative radiographic findings. Metastasis to pituitary gland, being rare and clinicoradiologically difficult to differentiate from adenoma can be missed. Yet sudden worsening of symptoms, ophthalmoplegia and increasing pituitary mass in a patient over 50 yr old should raise the suspicion for pituitary metastasis.

Non-functioning pituitary adenomas in the elderly.

K Laycock, J Anderson.
Homerton University Hospital, London.

Incidental radiological findings are an increasing source of endocrine referrals. In the case of substantially sized pituitary lesions, these are uncertainties associated with a lack of any marker to predict future behaviour. Decision making in elderly patients can be further complicated by the issue of fitness for anaesthesia with the passage of time if a clinical decision is made for a period of observation. We present the case of a common scenario to prompt discussion.

An 82 year old retired postman was found to have an abnormal pituitary appearance whilst being investigated for a nasal lesion by ENT. A focused MRI pituitary showed a pituitary macroadenoma measuring 16mm in height within 1mm of the optic chiasm.

He felt generally well with a stable weight. Visual fields were full to confrontation. He had a past medical history of benign prostatic hypertrophy and hypertension.

His pituitary function showed TSH 1.54mu/L (0.3-5), free T4 13.3pmol/L (9-19), FSH 8.2u/L (0.6-12), LH 4.6u/L (1.8-8.2), prolactin 177mu/L (54-380), testosterone 30.1nmol/L (7-26), IGF 16.3. His 9am cortisol was 316mol/L.

Given his age, lack of symptoms and the very small gap between the tumour and his optic chiasm, an expectant approach was taken. He was scanned sequentially at 6 months for one year and annually thereafter. He did not undergo dynamic testing to assess ACTH function as he was clinically well and the risk of investigation was felt to outweigh the benefit in his case. Thus far he has been followed up for 5 years with serial static MRI appearances and pituitary function.

Large case series have revealed that the mortality rate and duration of hospitalisation among patients undergoing transsphenoidal surgery increases with advancing age. This is particularly important in octogenarians. Grossman *et al*. showed that the odds of inpatient death increased in those over 80 (odds ratio, 1.85) compared to patients aged 65–69. Work published this year by Memel shows significantly more complications in those over 80. With this in mind, a high level of operative selectivity should be used in this cohort of patients. We hope that this case will stimulate a discussion of whether or when to intervene in such instances.

An uncommon cause of infertility in a patient with a pituitary macroadenoma.

R Wilmington, R Russel, A Sinha, NJ Parr, U Srinivas-Shankar.
Wirral University Teaching Hospitals NHS Trust, Liverpool Women's Hospital,
Walton Centre. NHS Foundation Trust, Liverpool.

A 32 year old man presented to endocrinology clinic after being unable to conceive with his partner. He had reduced libido and erectile dysfunction. He went through puberty normally and had a normal sense of smell. Clinical examination revealed that he was 203 cm tall, normally virilised, with normal visual fields to confrontation. He appeared euthyroid and did not have stigmata of hypercortisolism or growth hormone excess. He had normal body, pubic and axillary hair. There was no gynecomastia. The phallus was normal but the testicular volume was reduced at 15mls.
Pituitary hormone profile revealed elevated FSH (39 mIU/ml; nr 1-12 mIU/ml) and LH (23.6 mIU/ml; nr 2-9 mIU/ml), and normal testosterone levels (12.4 – 15.9 nmol/L; nr 10-28 nmol/L). Prolactin levels ranged between 243 & 375 mU/L (nr <350). Thyroid function tests, IGF1 and short synacthen test (baseline cortisol 298nmol/L and 30 minute cortisol 577nmol/L) were normal. Semen analyses revealed azoospermia. He was commenced on cabergoline 250 mcg per week and sildenafil.
MRI pituitary revealed a macroadenoma (12 x 10 x 9 mm) extending into the suprasellar cistern with optic chiasmal compression. Perimetry revealed normal left visual field and 2 spots missing in the periphery of the right visual field. Karyotyping revealed 47 XYY. Ultrasound testes showed a 1.5cm focal rounded area of ill-defined abnormality; he underwent left testicular orchidectomy and right testicular biopsy. Histology revealed Sertoli cell-only syndrome. There was no evidence of spermatogenesis in either testicle and no evidence of germ cell neoplasia.
Pituitary surgeon wanted to just observe the pituitary macroadenoma with annual MRI pituitary and continued monitoring of visual fields. Serial MRI pituitary scans did not reveal change in size of the pituitary macroadenoma.

Questions for discussion:
1. Would the panel have done an MRI pituitary in the first place given the clinical findings, high pituitary gonadotrophins and only mildly elevated prolactin levels.
2. Would you operate on this patient's pituitary macroadenoma given the proximity to the optic chiasm?
3. What if any, is the association between a pituitary macroadenoma in an XYY male with Sertoli cell-only syndrome?

Cabergoline therapy in macroprolactinoma after repeated surgery and radiotherapy.

M Silveira, M Parsad, M Al-Mrayat.
University Hospital Southampton NHS Foundation Trust.

We present a 50 year old male who was diagnosed in another institution with Macroprolactinoma in 2011, which was initially treated medically with normalisation of the prolactin and some reduction in tumour volume. 8 months later he developed pituitary apoplexy. Due to ongoing symptoms of headaches, photophobia and depression, he sought surgical opinion and underwent transphenoidal surgery in 2012 in Oxford. In 2013 he underwent a second operation there due to ongoing headaches aiming at further debulking tumour on the understanding this may not necessarily help his headaches. Unfortunately his symptoms persisted.

In 2016 he relocated to our catchment area and was reviewed in the joint pituitary clinic. He was on cabergoline 500micrograms twice weekly. His MRI from 2015 showed a significant amount of residual tumour in the sphenoid sinus and into the right clivus. His prolactin was controlled at 181mU/l (NR 55-276). However, he remained symptomatic with depression, headaches and low mood. The option of third surgery was discussed and collectively discounted due to previous poor responses. Radiotherapy was considered and subsequently he received VMAT radiotherapy 50.4 Gy in 28 fractions in 2016. .Repeat MRI in 2017 & 2018 showed stable appearance of the residual adenoma. His hormonal replacement optimised and is currently on Nebido, thyroxine, Vit D/ calcium supplements and recently switched from hydrocortisone to prednisolone, in addition to cabergoline 1mg twice weekly (higher dose now as his prolactin went up to 3071mU/L in Dec 2016). He is also on MST and Valproate for headaches, and sertraline for depression. His latest prolactin in is 465mU/l (NR: 55-276)

Issues to consider:
1. Could we stop his cabergoline now 2 years post radiotherapy, esp. given his depression? If not now, at what stage would it be reasonable to withdraw the treatment?
2. In view of his ongoing fatigue despite optimisation of other hormone replacement, is it safe to consider GH replacement?

Acromegaly and associated pituitary macroadenoma resolution with cabergoline therapy.

N Thomas, A Brooke.
Royal Devon and Exeter NHS Foundation Trust.

A 47 year old lady presented to the ophthalmology department in 2001 with decreased vision in her left eye and a superior bitemporal quadrantinopia. MRI revealed a 1.8 x 1.3 x 1.4cm pituitary adenoma with suprasellar extension causing chiasmal compression. Initial blood test revealed a prolactin elevated at 1099 mu/l (reference range 108-684 mu/l). IGF-1 and GH were awaited but the remainder of her mid cycle pituitary function was normal. She was felt to have no signs or symptoms consistent with a functioning pituitary adenoma and was referred and subsequently listed for surgical resection. Prior to surgery her IGF-1 returned elevated at 53.2 nmol/l (age related reference range 9-40 nmol/l) dynamic testing with a glucose tolerance showed non suppression of her growth hormone with a baseline of 19.2 miu/l and nadir of 13.6 miu/l (4.5 ng/ml). She had mild hypertension (blood pressure of 154/92 mm/hg) and impaired glucose tolerance Hba1c 6.3% (45mmol/mol). Surgery was postponed and in April 2002 she was started on Cabergoline 0.5mg weekly increased to 0.5 mg twice weekly, (January 2003 IgF1 22.5, growth hormone nadir 3.8 mu/). Her MRI showed no change with continued moderate compression of her optic chiasm and continued extension into the cavernous sinus although her visual fields had normalised. Surgery was re-discussed however she was keen to continue medical treatment to see if things might improve. In 2007 MRI showed shrinkage of her tumour with slight fullness of the sellar contents, marginal stalk deviation but no extension into the cavernous sinus. In May 2016 cabergoline was reduced then stopped with current 2018 biochemistry still off treatment showing a stable IGF-1 of 19nmol/l, improved BP off antihypertensives (142/83), improved HbA1c (39) and a stable pituitary appearance on MRI with no measurable residual tumor. She never reported symptoms suggestive of apoplexy and her other pituitary hormones remained normal. To our knowledge this is the first report of shrinkage of a growth hormone secreting pituitary adenoma with maintained remission of acromegaly off treatment.

Late presentation of acromegaly in a patient with macro-prolactinoma.

AJ Beck, DE Flanagan.
University Hospitals Plymouth NHS Trust.

A 70 year old man was found to have a pituitary macro adenoma having been found to have visual field defect by his optician. He complained of fatigue and had a previous history of obstructive sleep apnoea. He was otherwise asymptomatic.
Initial pituitary function tests showed a prolactin of 360316 [36-324miu/L], depressed testosterone at 4 [6.7-25.7], LH 3.3 [1.7-8.4iu/L], FSH 3.3 [1.5-12.4iu/L], TSH 0.92 [0.35-4.5 miu/L], borderline FT4 at 10 [10.5-26pmol/L] and mildly elevated IGF-1 at 43.4 [5-30nmol/L]. A short Synacthen test was normal with a peak of 834nmol/L. There were no clinical features of acromegaly or hypogonadism. He was commenced on Cabergoline 500mcg twice weekly and, in view of the marginally elevated IGF1, a glucose tolerance test (GTT) was performed (3 weeks after commencing Cabergoline). The growth hormone supressed to 0.24mcg/L [<1mcg/L]. The repeat IGF1 was normal (29.5nmol/l). His initial MRI showed a large mass compressing the optic chiasm, extending bilaterally into the cavernous sinus and inferiorly into the sphenoid sinus. Serial scans over the next 2 years showed a steady reduction in size of the mass (away from the optic apparatus) but with some remaining tumour laterally and inferiorly. There was a steady decline of the prolactin, to 757 miu/L. His IGF1 was followed serially. Despite a sustained fall in prolactin and a steady decrease in tumour volume his IGF1 steadily rose above the normal range to a peak of 8.2 nmol/L. He did not develop any new symptoms. He underwent endoscopic endo nasal resection of his pituitary 3 years after his initial presentation. It was felt unlikely that this would be curative because of tumour extension into both cavernous sinuses. A repeat IGF-1 measurement 3months post operatively (on Cabergoline) showed significant improvement to 31.5, and a GTT demonstrated successful suppression of GH to 0.68mcg/L. His prolactin dropped to 757. Repeat SST demonstrated adequate cortisol response on hydrocortisone replacement therapy.

Conclusion: It is well recognised that pituitary macro adenoma may co secrete growth hormone and prolactin. This person did not present with features of acromegaly and his initial GTT showed normal suppression of GH. This case illustrates the importance of continuing surveillance of the GH axis in this group of patients.