Society for Endocrinology

Imperial College London

Imperial Pituitary Masterclass Meeting

Monday 13th September 2021

REMOTE IMPERIAL PITUITARY MASTERCLASS MEETING 2021

2 EXTERNAL CPD CREDITS
ROYAL COLLEGE OF PHYSICIANS LONDON

14:00-14:20: *Unusual presentation of diabetes insipidus during pregnancy.* N Kyaw Kyaw, A Chaudhry, A Rathore (Southend University Hospital).

14:20-14:40: *Treating Cushing's disease and its complications – a team effort.*
J Gan, M Modi, N Vanderpant, R Mohamed, N Mendoza, N El-Masry, N Hill, C Lim, E Hatfield, N Martin, K Meeran (Ashford and St Peter's Hospital NHS Foundation Trust and Imperial College Healthcare NHS Trust, London).

14:40 -15:00: *Giant functioning pituitary gonadotroph adenoma presenting with status epilepticus requiring three-stage surgery.* A Li, B Whitelaw, A Al Busaidi, E Maratos, J Gilbert, (King's College Hospital NHS Foundation Trust, London).

15:00-15:20: *Follicle-Stimulating Hormone-Secreting Pituitary Adenoma: a spectrum of clinical presentation.* N Papanikolaou, M Alameri, N Parker, C McNamara, L Dixon, J Yazbek, D Lyons, N Martin, K Meeran, M Al-Memar, C Jayasena (Imperial College Healthcare NHS Trust, London).

15:20-15:30: *BREAK*

15:30-15:50: *Dopamine agonist intolerance in an occult microprolactinoma – successful selective adenomectomy facilitated by 11C-Methionine-PET-MRI*. J MacFarlane, W Bashari, R Senanayake, D Gillett, O Koulouri, A Powlson, A Kolias, N Donnelly, R Mannion, M Gurnell (Cambridge University Hospitals NHS Foundation Trust).

15:50-16:10: *Apoplexy – a lesson in watchful waiting.* M Modi, R Mohamed, M Al-Ansari, N Vanderpant, D Papadopoulou, N Mendoza, L Dixon, E Hatfield, N Martin, K Meeran (Imperial College Healthcare NHS Trust, London).

16:10-16:30: *Reversing visual loss with pulse intravenous methylprednisolone in a case of granulomatous hypophysitis.* K Ayoub, S Barazi, N Sibtain, R Banatwalla, S Aylwin. (Kings College Hospital NHS Foundation Trust, London).

16:30-16:50: *Endocrinopathy behind the facemask*. S Nyunt, P Avari, C Mitchell, G Tarigopula, Y Ling (Hillingdon Hospitals NHS Foundation Trust).

Research to compare different steroids for replacement in patients with adrenal insufficiency.

At present, there is no evidence in support of any of the different steroids used in patients with primary or secondary adrenal failure.

Although the primary reason for the use of prednisolone once daily instead of hydrocortisone at Charing Cross was the huge difference in price in 2014 (Amin et al, 2014), it has become clear over the last five years that many patients prefer prednisolone once daily. The price of hydrocortisone has now fallen, but patients who have switched to once daily prednisolone are very reluctant to switch back and preliminary data is encouraging (Smith et al, 2017; Choudhury et al 2019). In addition, the DREAM study suggests that late doses of hydrocortisone may affect clock genes and might also have an effect on the immune system and hence thrice daily hydrocortisone might be harmful, especially an evening dose (Muller et al, 2018). It is thus possible that in addition to being cheaper and more convenient, when used at the correct dose, prednisolone has fewer undesirable effects than hydrocortisone thrice daily. The plasma profile of once daily prednisolone matches the natural circadian rhythm of plasma cortisol levels better than any other steroid (Williams et al, 2016).

At present, there is no evidence to support the use of either drug. We really need data to inform our future practice and to this end the NIHR have adopted three trials, one of which you can take part in through your NIHR Clinical Research Network.

Once your site is enrolled, the study is very simple although it needs someone to lead each site. Patients need to be stably replaced on either hydrocortisone or prednisolone for the preceding four months. When seen, the locally available markers of steroid exposure such as bone turnover markers, glucose, hba1c and lipids, weight, BMI, waist and hip measurements, blood pressure and the results of an SF36 well-being questionnaire need to be recorded. They are then switched to an equivalent dose of the alternate drug for a further four months and the data collected again at the end of that period. Checking for an appropriate dose can be supported by prednisolone levels if desired.

If you would like to take part in this study, please e-mail steroids@imperial.ac.uk

References:

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Smith DJF, Prabhudev H, Choudhury S, Meeran K. Prednisolone has the same cardiovascular risk profile as hydrocortisone in glucocorticoid replacement. Endocr Connect. 2017;6(8):766-772.

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Muller L, Quinkler M. Adrenal disease: Imitating the cortisol profile improves the immune system. *Nat Rev Endocrinol*. 2018;14(3):137-139.

Williams EL, Choudhury S, Tan T, Meeran K. Prednisolone replacement therapy mimics the circadian rhythm more closely than other glucocorticoids. *J Appl Lab Med*. 2016;1(2):152-161.

Unusual presentation of diabetes insipidus during pregnancy.

N Kyaw Kyaw, A Chaudhry, A Rathore (Southend University Hospital).

Abstract:
A 38 year old lady presented with polyuria and polydipsia for 7 months which started during the third trimester of her second pregnancy. Her symptoms improved immediately after she gave birth to a healthy child. Her symptoms returned within a few weeks and thereafter persisted. On presentation, her daily fluid intake fluctuated from 3 to 7 litres and was associated with significant nocturia. There was no reported weight loss or other systemic symptoms. She had previous medical history of pseudomyxoma peritonei of appendix which was under routing monitoring. Her only regular medication was oral contraceptive pill. She had two healthy children, ages 2 years and 6 months. Her first pregnancy was unremarkable.

She appeared euvolemic on clinical examination and her BP was 144/98. Her initial blood results were serum sodium 142mmol/L, Serum osmolality 258mmol/kg, serum creatinine 80mmol/L, urine osmolality 104 mmol/kg and urine sodium 21.6mmol/L, serum cortisol 288nmol/L, IGF1 17.2nmol/L, prolactin 258mU/L, TSH 2.74mU/L. Provisional diagnosis of Diabetes Insipidus was made and water deprivation test was then carried out for confirmation. The results were suggestive of partial Cranial Diabetes Insipidus. Her Serum osmolality rose to 301mmol/kg after water deprivation with corresponding urine osmolality of 323mmol/kg. After Desmopressin administration, urine osmolality rose to 614mmol/kg. This was more than 50% rise and supported the diagnosis of Cranial Diabetes Insipidus. After Desmopressin administration, her symptoms of thirst and polyuria disappeared completely for more than 24 hours which was another favourable factor suggesting Cranial Diabetes Insipidus rather than primary polydipsia.

She was then commenced on regular Desmopressin spray, 10mcg once at night which is nicely controlling her symptoms. MRI pituitary showed a 7.3 X 4.9 cm expansile lesion in posterior pituitary, isointense on T1 imaging with no demonstrable contrast enhancement and no involvement of pituitary stalk or optic chiasm. The case was discussed in Neurosurgical MDT which confirmed the finding of a non-enhancing cystic lesion in the posterior pituitary area, separate from pituitary stalk. No definite diagnosis could be made as to the nature of the lesion, but management plan was agreed as active monitoring.

Conclusion:
This is an unusual case of diabetes insipidus manifesting during pregnancy due to a posterior pituitary cystic lesion. The nature of the lesion is not entirely clear, and we are not sure whether removal/aspiration of lesion will improve patient's symptoms. Following questions are presented for discussion:

1) What is the nature of this posterior pituitary lesion?
2) Why did patient's symptoms start during late pregnancy?
3) Whether surgical intervention will improve or worsen her diabetes insipidus?

Treating Cushing's disease and its complications – a team effort.

J Gan, M Modi, N Vanderpant, R Mohamed, N Mendoza, N El-Masry, N Hill, E Hatfield, N Martin, K Meeran (Ashford and St Peter's Hospital NHS Foundation Trust and Imperial College Healthcare NHS Trust, London).

Abstract:
A 72 year old gentleman under investigation for suspected Cushing's syndrome was admitted to his local hospital with hypokalaemia. Over the preceding 8 weeks, he had developed progressive fatigue, lower back pain secondary to vertebral compression fractures, declining mobility due to leg weakness, easy bruising and poor wound healing. He also reported significant weight gain over the past eighteen months. On examination, he had an interscapular fat pad, central obesity, extensive bruising and a shallow wound on his left shin, with proximal myopathy and thin skin. Biochemical testing revealed ACTH-dependant cortisol excess, secondary hypothyroidism and secondary hypogonadism. He commenced metyrapone, spironolactone and potassium supplementation. An MRI confirmed a large pituitary macroadenoma. His case was discussed at the Pituitary MDT and in view of his clinical picture, the decision was made to proceed with urgent pituitary surgery. He was subsequently transferred to our centre.

Whilst awaiting pituitary surgery, his inflammatory markers increased, despite antibiotic therapy for a presumed lower respiratory tract infection. Subsequently, he developed acute abdominal pain, with peritonism. A CT scan demonstrated localised subacute bowel perforation, likely related to hypercortisolism (cortisol of 1227 nmol/L). An interval CT scan showed an increased volume of free intra-abdominal fluid, which was drained under radiological guidance, revealing pus. Given his deteriorating clinical condition, the decision was made for the Neurosurgeons to perform the trans-sphenoidal surgery as planned with the Colorectal Surgeons then performing a laparoscopic abdominal washout to treat the intra-abdominal sepsis immediately afterwards. The patient successfully underwent both surgeries in a single theatre attendance. Over the next few days, his cortisol levels fell dramatically. Histology confirmed a densely granulated corticotroph adenoma (Ki67 index less than 3%). One month after surgery, his early morning cortisol level was 35nmol/L, indicating remission of his Cushing's disease. He continued his recovery and was transferred back to his local hospital for ongoing rehabilitation.

Questions for discussion:

1) Could the pituitary surgery have waited until the intra-abdominal sepsis had resolved, or was it unavoidable due to the burden of complications related to cortisol excess?

Giant functioning pituitary gonadotroph adenoma presenting with status epilepticus requiring three-stage surgery.

A Li, B Whitelaw, A Al Busaidi, E Maratos, J Gilbert, (King's College Hospital NHS Foundation Trust, London).

Abstract:
A 34-year-old presented with a generalised tonic clonic seizure progressing to status epilepticus. A CT-Head scan showed a sellar mass with suprasellar extension into the third and lateral ventricles with expansion of the sella and tumour extension into the sphenoid sinus. There was a possible focus of haemorrhage within the tumour.

A collateral history revealed the patient had reported a headache on the day of presentation requiring paracetamol, which was unusual for him. However there had been no prior complaint of visual or endocrine disturbance. He had marked gynaecomastia and significantly increased testicular volume. Muscle bulk was normal.

His pituitary profile is shown in the table below:

Hormone	Value	Reference range
Prolactin	2496 mIU/L	86-324 mIU/L
TSH	1.89 mIU/L	0.27-4.2 mIU/L
T4	10.5 pmol/L	11-21.2 pmol/L
Cortisol	925 nmol/L	133-537 nmol/L
ACTH	< 5ng/L	0-46 ng/L
IGF-1	17.1 nmol/L	10.7-37.1 nmol/L

However, his gonadotropins were markedly elevated: FSH 108 IU/L (1.5-12.4 IU/L) and LH 28.4 (1.7-8.6 IU/L) with a testosterone level >52nmol/L (8.6-29 nmol/L). SHBG was normal. He was not polycythaemic and his liver function was not significantly abnormal.

Thus the diagnosis of a giant functioning gonadotroph adenoma was concluded. The hyperprolactinaemia was likely secondary to stalk effect and hypercortisolaemia due to administration of intravenous hydrocortisone at presentation.

His admission was complicated by rhabdomyolysis and acute kidney injury with a peak Creatinine Kinase 45353 IU/L (<150 IU/L), failed sedation wean and myocardial infarction evidenced by a troponin of 255 ng/L (<14 ng/L). Medical stabilisation was required prior to surgery. He underwent endoscopic transphenoidal debulking as a stage one procedure, followed by transventricular resection of the suprasellar lesion two weeks later. A third stage surgical pterional craniotomy to resect residual tissue was carried out after a further fortnight. Histology showed focal LH and FSH positivity with Ki67 index 1-2%.

Ophthalmic assessment was not attainable until three months' after his initial presentation due to neurological deficits. This showed significant visual loss in the left eye and super-temporal field loss in the right.

He remains on hydrocortisone and levothyroxine for replacement and his neurological status is improving significantly. He can now mobilise outside the house and he remains seizure-free on antiepileptic medication. He has maintained some gonadotroph function within normal limits: FSH 2.1 IU/L (1.5-12.4IU/L), LH 2.1 (1.7-8.6 IU/L) and testosterone 9.9 nmol/L (8.6-29nmol/L) although post-operative imaging demonstrates no evidence of residual tumour.

Functioning gonadotroph tumours are rare and the exact aetiology of the presentation with seizures remains unclear. Could supraphysiological testosterone levels have contributed to this patient myocardial infarction?

Follicle-Stimulating Hormone-Secreting Pituitary Adenoma: a spectrum of clinical presentation.

N Papanikolaou, M Alameri, N Parker, C McNamara, L Dixon, J Yazbek, D Lyons, N Martin, K Meeran, M Al-Memar, C Jayasena (Imperial College Healthcare NHS Trust, London).

Abstract:
Functional gonadotroph adenomas are rare tumours of the pituitary gland that secrete one or more of the gonadotrophins (FSH and/or LH). FSH-secreting pituitary adenomas can affect sexual and reproductive function and remains a challenge to diagnose due to variable clinical presentation. We would like to present 2 cases highlighting the different clinical presentations and outcomes of Follicle-Stimulating Hormone-Secreting Pituitary Adenoma.

First case is a 37-year-old lady presented with intermittent lower abdominal pain and bloating which were associated with nausea and vomiting. She also complained of irregular menstrual cycles and occasional headaches whilst she reported no galactorrhoea. Ultrasound pelvis revealed enlarged multicystic ovaries (Rt ovary 116 x 90 x 80 mm, Lt ovary: 90 x 60 x45 mm). She was referred to a tertiary center and a diagnosis of ovarian hyperstimulation syndrome (OHSS) was made. Hormonal investigation showed serum oestradiol 6992 pmol/L, prolactin 1808 milliunit/L, LH 2.4u/L and FSH 10.8 u/L. FT4 and IGF1 were borderline low (8.8 pmol/L and 9.9 nmol/L, respectively), TSH was within normal limits (1.68 milliunit/L). Based on the OHSS with high oestrogen levels, unsuppressed FSH and evidence of secondary hypopituitarism, she underwent an MRI of pituitary gland. The scan demonstrated an enlarged adenoma (41 x 41 x 24 mm) invading both cavernous sinuses and compressing the optic chiasm, consistent with a functioning gonadotroph adenoma. She had 2 doses of Lanreotide that resulted in no change in the hormonal profile. She underwent transsphenoidal surgery to debulk the tumour. Biopsy confirmed the presence of a functioning gonadotroph adenoma with LH and FSH expression approximately 20% and 30% respectively.
Progress**:** The patient is now 6 weeks post-surgery. Latest blood tests show undetectable oestradiol (<100), FSH: 6.4 and LH 2.

Second case is 68-year-old male who was diagnosed with incidental pituitary macroadenoma while investigating balance issues. He underwent a dedicated pituitary MRI revealing a macroadenoma with suprasellar extension. Blood tests showed elevated FSH of 94.9 IU/L, testosterone level of 30.5 nmol/L. The patient had normal-volume testes and there were no clinical manifestations of the high testosterone. Subtle visual filed defect was noted in the left eye. He declined surgical intervention. Progress: The patient remained well with conservative management during a 5-year follow up. No testicular enlargement was noted throughout this period. At 5 years, both pituitary MRI and visual fields did not show any major progression.

Conclusion:
These 2 cases highlight how differently a gonadotroph adenoma can manifest. Whilst in the first case OHSS had developed as a result of it, the second case represents a presumed “Silent” FSH-secreting pituitary adenoma with high FSH serum levels and without signs of testicular hyperstimulation. Spontaneous OHSS (without fertility treatment) is rare and usually caused by a gonadotroph adenoma. Surgical resection is the only known effective treatment. The extent of successful tumour resection will determine our patient’s future prognosis for motherhood.

Dopamine agonist intolerance in an occult microprolactinoma – successful selective adenomectomy facilitated by 11C-Methionine-PET-MRI.

J MacFarlane, W Bashari, R Senanayake, D Gillett, O Koulouri, A Powlson, A Kolias, N Donnelly, R Mannion, M Gurnell (Cambridge University Hospitals NHS Foundation Trust).

Background:
Medical management with dopamine agonists (DA) is currently first-line treatment for microprolactinomas. Surgery is generally reserved for patients with resistance or intolerance to medication. A key concern when considering surgery is the risk of postoperative hypopituitarism and, in women of childbearing age, its potential impact on fecundity. However, intolerance of DA therapy is being increasingly recognised with one study reporting that 68%-78% of women on DA experience adverse symptoms and these are sufficiently severe in 3%-12% to discontinue treatment. As such, the role of surgery in the management of microprolactinomas is being re-examined, particularly in light of recent advances in imaging and surgical techniques which may reduce the risk of consequent hypopituitarism.

Case summary:
A 37-year-old woman presented with secondary amenorrhoea after stopping the oral contraceptive; she also reported intermittent galactorrhoea. Serum prolactin was elevated on serial samples (*1498 – 1653 mU/L* [RR 59-619]). Once other causes were excluded (stress, exercise, pregnancy, drugs, renal insufficiency, liver disease and hypothyroidism) a pituitary MRI was performed. This was suggestive of a 3mm right-sided adenoma on standard spin-echo (SE) sequences with gadolinium enhancement.

The patient wished to achieve pregnancy and so was started on DA therapy to reverse hyperprolactinaemia and associated hypogonadism. Bromocriptine, cabergoline and quinagolide were each trialled but she experienced significant nausea and nasal congestion with all of the preparations. Despite this, she elected to restart cabergoline, and although this allowed restoration of menses, she then developed significant mood disturbance and all DA were withdrawn, with subsequent return of amenorrhoea.

Given the patient's ongoing desire for pregnancy, surgery was considered. A volumetric FSPGR MRI suggested a subtle 5mm abnormality in the left side of the pituitary. Given the discordance with earlier imaging and the desire to minimise the risk of new post-operative hypopituitarism, an ^{11}C-Methionine PET/CT was performed and co-registered with the MRI images (Met-PET-MRI$^{CR)}$). This showed a focus of intense tracer uptake on the left of the fossa.

The patient underwent endoscopic transsphenoidal selective adenomectomy. Prolactin normalised post-operatively with confirmatory histological findings.

Discussion and conclusions:
Normoprolactinemia may be achieved in 71–100% of patients with microprolactinomas following transsphenoidal surgery and thus may provide a better option than DA for some patients.

Met-PET-MRI^{CR} may offer a useful alternative for confirming/refuting the location of a microprolactinoma when pituitary MRI findings are equivocal, and thus guide selective adenomectomy so as to preserve normal pituitary function as far as possible.

Apoplexy – a lesson in watchful waiting.

M Modi, R Mohamed, M Al-Ansari, N Vanderpant, D Papadopoulou, N Mendoza, L Dixon, E Hatfield, N Martin, K Meeran (Imperial College Healthcare NHS Trust, London).

A 33-year-old woman, with a background of obesity, primary hypothyroidism and PCOS, presented with sudden onset severe headache and subsequent inability to open or move her left eye. On clinical examination, she had a complete left ptosis, a dilated left pupil, with 3rd, 4th and 6th nerve palsies, but no deterioration in visual acuity. An initial CT intracranial angiogram showed appearances suspicious of a short segment dissection within the left internal carotid artery. However, a subsequent MRI showed a large sellar mass with small volume haemorrhage, extending into the left cavernous sinus, which was concerning for pituitary apoplexy. As emergency management, she received IV hydrocortisone. On further questioning, she reported weight gain of 45kg over three years, with associated acne, hirsutism, easy bruising and secondary amenorrhoea for the past year. On examination, she had a plethoric moon face, central obesity, an interscapular fat pad, multiple bruises on both arms and proximal myopathy. Her weight was 146kg. A pituitary profile on admission showed: free T4 8.4 pmol/L, TSH 7.02 mU/L, LH <0.1 U/L, FSH 1.2 U/L, prolactin 65 mIU/L, growth hormone 0.14 µg/L. Her cortisol was noted to be >3300 nmol/L but may have been measured after IV hydrocortisone was administered.

Her case was discussed at the Pituitary MDT and in view of subtle neurological improvement and no compromise of visual acuity, conservative management was recommended with close surveillance. She was discharged from hospital without glucocorticoid replacement as her cortisol level was 513 nmol/L, with a paired ACTH of 117 ng/L. She was reviewed regularly and demonstrated complete resolution of her left sided ptosis within six weeks, with normalisation of eye movements. A repeat MRI showed resolution of the pituitary haemorrhage with collapse of the gland. A small residual focal area of hypo-enhancement in the left side of the pituitary (5mm) was noted, consistent with a microadenoma. She has shown recovery of her normal diurnal rhythm of cortisol secretion on serial cortisol day curves. Her periods have not yet resumed but her gonadotropin levels are now normalising (LH 3.8 U/L, FSH 4.3 U/L). She has lost 6kg in weight since her presentation three months ago.

Questions for discussion:

1) How best can we monitor for relapse of suspected Cushing's disease in this patient?
2) Can we be sure that Cushing's disease was the initial diagnosis given lack of histological or biochemical confirmation?

Reversing visual loss with pulse intravenous methylprednisolone in a case of granulomatous hypophysitis.

K Ayoub, S Barazi, N Sibtain, R Banatwalla, S Aylwin. (Kings College Hospital NHS Foundation Trust, London).

Abstract:
Hypophysitis is one of the causes of pituitary pathology causing hypopituitarism and visual abnormalities. Surgical decompression of visual apparatus is indicated when vision is threatened. We present a 43 years old Eritrean lady who presented on Christmas Eve with worsening of visual acuity over 4 weeks to perception of light only in her right eye (RE) and seeing hand movements from 10 cm distance in her left eye (LE). Head CT and subsequent pituitary MRI showed diffuse enlargement of pituitary gland with extensive contiguous suprasellar enhancement in keeping with hypophysitis. There is no headache or signs or symptoms of endocrinopathy apart from 6 months of amenorrhea. Blood tests showed panhypopitutiarism (TSH 0.22 mIU/L, FT4 2.9 pmol/l, FSH 1.0 IU/L, LH <1.0 IU/L, oestradiol <92 pmol/l, testosterone <0.4 nmol/l, prolactin <20 mIU/L, ACTH 22 ng/L, random cortisol 134 nmol/l, IGF-1 5.4 nmol/l, GH <0.1 ug/L).

She was given 1g of IV methylprednisolone stat, and her vision improved dramatically next morning to RE 6/60 LE 6/24. She continued on 1g of methylprednisolone for another 2 days and pituitary MRI on day3 showed significant reduction in enhancing tissue volume. A trans-sphenoidal biopsy was done on day 3 and showed a granulomatous hypophysitis and negative Ziehl–Neelsen stain and culture. She was discharged on prednisolone 40mg and levothyroxine as well as anti-tuberculosis treatment as she is coming from an endemic area. Chest x-ray was clear. Subsequently QuantiFERON-TB test was positive. IgG subclasses were normal. She continued to improve clinically on follow up.

Questions for discussion:

1) What is the differential diagnosis for hypophysitis in this lady?
2) What are the circumstances that glucocorticoids could be given before pituitary biopsy?

Endocrinopathy behind the facemask.

S Nyunt, P Avari, C Mitchell, G Tarigopula, Y Ling (Hillingdon Hospitals NHS Foundation Trust).

A 44-year-old gentleman presented to the Emergency Department with a 2-week history of fevers and rigors. Past medical history was unremarkable other than an earlier diagnosis of hypertension. He was noted to have new onset atrial fibrillation with rapid ventricular response, and a new diagnosis of hypertrophic obstructive cardiomyopathy was made on echocardiography. In addition, a vegetation was identified on the mitral valve. Treatment for infective endocarditis (Streptococcus oralis) was initiated and he was subsequently transferred to a specialist centre for mitral valve replacement surgery. During the admission, a history of chronic headaches was investigated. MRI pituitary revealed a 3.8 x1.9 cm pituitary macroadenoma with suprasellar extension. There was no cavernous sinus invasion.

He was further evaluated in the outpatient endocrine clinic. On removal of his facemask, examination revealed typical acromegalic features with supraorbital ridge prominence, significant underbite and macroglossia. Visual fields were normal to confrontation testing and no organomegaly was present on bedside examination.

Urgent endocrine investigations including baseline pituitary function testing were performed. IGF-1 was significantly elevated at 140.3nmol/l (range 8.5-31.0), 9am cortisol was 352nmol/L (range 200-750), prolactin 1119mU/L (range 60-300), TSH 1.98mU/L (range 0.34-5.60), FSH <0.1U/L (range 1.7-8.0) and testosterone 8.7 (range 10.0-30.0). Acromegaly was confirmed with an oral glucose tolerance test showing a paradoxical rise in growth hormone. Glucose levels remained normal throughout the OGTT. The patient was commenced on monthly Lanreotide injections. He has subsequently been reviewed in the neuro-endocrine clinic at Charing Cross Hospital to determine the best course of further management.

Cardiovascular complications including myocardia hypertrophy, arrhythmias, arterial hypertension and valvulopathy, as well as colonic benign neoplasms such as polyps, are common complications of acromegaly. Early diagnosis and appropriate treatment of acromegaly are critical for prevention of further complications. The Covid-19 pandemic with requirements for facemask and increasing virtual telephone consultations may compound potential delays in diagnosis.

Questions:

1) Would earlier diagnosis of his acromegaly have changed his cardiovascular outcome?
2) What are the challenges in his ongoing management resulting from his significant cardiovascular comorbidity?
3) What is the most appropriate management (monthly injection, pituitary surgery or gamma knife therapy) in his case?