Endocrine Abstracts

17th European Congress of Endocrinology
16–20 May 2015, Dublin, Ireland

EDITORS
The abstracts were marked by the Abstract Marking Panel selected by the Programme Organising Committee

ECE 2015 Programme Organising Committee

W Artl Chair
J Visser Co-chair
F Beuschlein Co-chair

Members

P Beck-Peccoz M Korbonits N Pitteloud M Sherlock
P Burman M Laan M Pfeifer A Spada
J Christiansen E Lalli JA Romijn M Tena-Sempere
M Hewison D Marks R Ross C Thompson
J Huhtaniemi J Mittag D Schulte R Semple
E Husebye M Niedziela

Abstract Marking Panel

A Agha Ireland
M Alevizaki Greece
B Allolio Germany
J Argente Spain
G Assié France
S Babajko Canada
R Badenhoop Germany
D Bassett UK
E Baudin France
A Beckers Belgium
A Berrutti Italy
J Bertherat France
B Bilinska France
J Bluhm Germany
K Boelaert UK
A Boelen The Netherlands
R Bouillon Belgium
J-P Bourguignon Belgium
M L Brandi Italy
K Bretz France
G Brunetti Italy
C Buchanan UK
J S Carroll UK
J Castano Spain
K Chatterjee UK
B Chini Italy
L Chioldini Italy
J Chowen Spain
T Coll UK
C Daoust UK
W de Herder Netherlands
E de Koning The Netherlands
R de Krüger The Netherlands
W Dhillo UK
G Di Dalmazi Germany
E Diamanti-Kandarakis Greece
Dotta Italy
J Drouin Canada
The ESE would like to thank its Corporate Members and the ECE 2015 sponsors

**ECE Corporate Members**
Eli Lilly
Ipsen
Laboratoire HRA Pharma
Merck Serono (MSD)
Novartis Pharmaceuticals
Novo Nordisk
Pfizer
Sandoz International Gmbh
ViroPharma SPRL

**Gold Sponsors**
Ipsen
Novartis Pharmaceuticals

**Bronze Sponsors**
MSD International Gmbh

---

**ESE Office**
Euro House
22 Apex Court
Woodlands
Bradley Stoke
Bristol BS32 4JT, UK

Contact: Andrea Davis
Tel: +44 (0)1454 642247
Fax: +44 (0)1454 642222
E-mail: info@euro-endo.org
Web site: www.ese-hormones.org

---

**ECE 2015 Secretariat**
Bioscientifica Ltd
Euro House, 22 Apex Court
Woodlands
Bradley Stoke
Bristol BS32 4JT, UK

Contact: Claire Arrigoni
Tel: +44 (0)1454 642240
Fax: +44 (0)1454 642222
E-mail: conferences@bioscientifica.com
Website: http://www.bioscientifica.com
CONTENTS

17th European Congress of Endocrinology 2015

PRIZE LECTURES AND BIOGRAPHICAL NOTES

The European Journal of Endocrinology Prize Lecture ................................................................. EJE1
The Geoffrey Harris Prize Lecture ................................................................................................. GH1

PLENARY LECTURES

PI 3-Kinase: connecting diabetes, obesity and cancer ................................................................. PL1
European Hormone Medal Lecture: Obesity and insulin resistance: Lessons from human genetics ................................................................. PL2
The genomics of adrenocortical tumors .................................................................................... PL3
Congenital Adrenal Hyperplasia (CAH): Mechanisms and management across the life span ................................................................. PL4
Insulin signalling and action ........................................................................................................ PL5
Initiative for Science in Europe (ISE) - how can we lobby so that Europe listens? ................. PL6
Obesity and the skeleton ............................................................................................................... PL7
From base change to better care in diabetes ............................................................................. PL8

SYMPOSIA

Glucocorticoid action in health and disease ............................................................................... S1.1–S1.3
Management of type 2 diabetes: State of the art ......................................................................... S2.1–S2.3
Non-classical causes of hypopituitarism (Endorsed by Endocrine Connections) ...................... S3.1–S3.3
Thyroid hormone and cardiovascular system ............................................................................. S4.1–S4.3
Endocrinology of ageing men ...................................................................................................... S5.1–S5.3
Beyond Phosphorus: multiple actions of FGF23 ....................................................................... S6.1–S6.3
Novel mechanisms of central weight regulation ......................................................................... S7.1–S7.3
The endocrine gut (Endorsed by Endocrine Connections) ......................................................... S8.1–S8.3
Steroid hormone action in target tissues ...................................................................................... S9.1–S9.3
Thyroid and autoimmunity .......................................................................................................... S10.1–S10.3
Hot topics and IESP symposia .................................................................................................... S11.1–S11.3
Advances in phaeochromocytoma diagnosis and management (Endorsed by the European Journal of Endocrinology) ................................................................. S12.1–S12.3
New concepts in Vitamin D research .......................................................................................... S13.1–S13.4
Adipose tissue as an endocrine organ (Endorsed by Endocrine Connections) ......................... S14.1–S14.3
Puberty: new mechanisms ........................................................................................................... S15.1–S15.3
Pathogenesis of adrenocortical tumours ...................................................................................... S16.1–S16.3
Diabetes and bone (Endorsed by the European Journal of Endocrinology) ................................. S17.1–S17.3
New genetics of pituitary tumours (Endorsed by the European Journal of Endocrinology) .................................................................................................................... S18.1–S18.3
Metabolic dysfunction in PCOS .................................................................................................. S19.1–S19.3
Endocrine consequences of childhood cancer treatment ............................................................ S20.1–S20.5
Thyroid hormone in pregnancy .................................................................................................. S21.1–S21.3
Beta cell biology ......................................................................................................................... S22.1–S22.3
Adrenal insufficiency: advances in diagnostics and therapy (Endorsed by the European Journal of Endocrinology) ................................................................. S23.1–S23.3
Androgens and disease progression in prostate cancer ............................................................... S24.1–S24.3
Challenges in pituitary tumours .................................................................................................. S25.1–S25.3
Towards the bionic pancreas: will the journey end? .................................................................. S26.1–S26.3
Thyroid cancer: new development in diagnosis and treatment (Endorsed by the European Journal of Endocrinology) ................................................................. S27.1–S27.3
Hormones and immunity in pregnancy ...................................................................................... S28.1–S28.3
Management of endocrine transition .......................................................................................... S29.1–S29.5
Thyroid - hypothyroidism .................................................. GP.26.01–GP.26.10
Thyroid - hyperthyroidism and treatment ................................ GP.27.01–GP.27.08
Endocrine tumours and neoplasia - NETS ................................ GP.28.01–GP.28.09
Endocrine tumours and neoplasia - Adrenal Tumour ................. GP.29.01–GP.29.07
Endocrine tumours and neoplasia - General .......................... GP.30.01–GP.30.07

EPOSTER PRESENTATIONS

Steroids, development and paediatric endocrinology .................. EP95–EP130
Calcium and Vitamin D metabolism ................................... EP223–EP318
Diabetes (complications & therapy) .................................... EP399–EP541
Pituitary: basic and neuroendocrinology .............................. EP650–EP711
Thyroid cancer ............................................................ EP833–EP922
Thyroid (non-cancer) ..................................................... EP923–EP1082
Endocrine tumours ....................................................... EP1083–EP1151
Clinical Cases–Thyroid/Other .......................................... EP1247–EP1347
Endocrine nursing ........................................................ EP1348–EP1353

INDEX OF AUTHORS
E-Posters
Conclusions
An oral dose of 600 000 IU of cholecalciferol in HIV-1 postmenopausal women rapidly increases 25(OH)D and 1,25(OH)2D levels reducing PTH levels, regardless of the presence of PIs in the cART scheme.

DOI: 10.1530/endoabs.37.EP293

EP294
May the polymorphism of low molecular weight protein tyrosine phosphatase modulate metabolic and bone remodelling parameters associated with osteoporosis?
Joana Freitas1, Cristina Monteiro1, Ana Paula Barbosa2, Fátima Batista3, Maria José Lairés1, Manuel Bicho1,2 & Mário Rui Mascarenhas1,2
1Institute for Scientific Research Bento Rocha Cabral, Lisbon, Portugal; 2ISAMB, FMUL, Lisbon, Portugal; 3CIPER, FMUL, Lisbon, Portugal;

Aims
To study the association of protein tyrosine phosphatase (LMW–PTP/ACP1) polymorphism with bone mineral density and metabolic parameters of bone remodelling.

Methods
BMD (g/cm²) was measured by DEXA in 760 subjects: 448 normal BMD (359F/89M; 49.7 ± 12.9 years; 30.2 ± 5.4 kg/m²) and 312 osteoporosis (265F/47M; 63.9 ± 10.4 years; 27.16 ± 4.4 kg/m²). Metabolic bone remodelling parameters were analyzed: LDL, HDL, total cholesterol, AP, osteocalcin, and alkaline phosphatase (AP), and osteocalcin. ACP1 activity was measured by spectrophotometry. ACP1 polymorphism was evaluated by PCR.

Results
Association was found between the genetic polymorphism of ACP1 and its enzymatic activity with higher values for genotypes AC + BC, intermediate values for BB and lower values for AA + AB. Osteoporosis: i) increased LDL, total cholesterol, AP, osteocalcin and ACP1, and decreased HOMA; ii) association between genotypes BB + BC + AC and increased total cholesterol, LDL, and ACP1; and iii) positive correlation between AP and LDL.

Conclusion
In osteoporosis, ACP1 polymorphism appears to modulate some metabolic parameters associated with a decrease in BMD, including total cholesterol, LDL, and ACP1 activity.

DOI: 10.1530/endoabs.37.EP294

EP295
Pancreatitis in familial hypocalcuric hypercalcaemia
Iulia Potorac1, Olivier Malaise2, Adrian Daly1 & Albert Beckers1
1Department of Endocrinology, CHU de Liège, University of Liège, Liège, Belgium; 2Department of Rheumatology, CHU de Liège, University of Liège, Liège, Belgium.

Familial hypocalcuric hypercalcaemia (FHH) is a characteristic asymptomatic condition that is caused principally by calcium receptors gene (CASR) mutations and less frequently by GNA11 or AFAP2/1 mutations. We report a case of recurrent symptomatic pancreatitis in an FHH patient. The 17-year-old patient was hospitalized with abdominal pain and raised pancreatic enzymes due to acute pancreatitis. The only predisposing factor on investigation was a very elevated serum calcium level (3.8 mmol/l; NR: 2.15–2.60). This was associated with consistently moderately elevated PTH (33 ng/l; NR: 4–26), normal 25-OH vitamin D (44 ng/ml; NR: 30–80), elevated 1,25(OH)2 vitamin D (133 pg/ml; NR: 23–109), and undetectable urinary calcium. Family history revealed that the patient’s grandmother was also known to suffer from hypocalcuric hypercalcaemia, and that hypercalcaemia had been found in the patient’s mother, uncle, brother and sister. CASR sequencing revealed the patient (and family members) to be heterozygotic for a R185Q mutation, previously suggested to be a dominant negative mutation and leads to higher calcium levels than other known CASR mutations. Cinacalcet treatment lowered serum calcium to 2.95 mmol/l and the patient has not presented new pancreatitis episodes.

DOI: 10.1530/endoabs.37.EP295

EP296
Giant parathyroid adenoma with severe hypercalcaemia: case report
Cristina Spriou1, Aurelian Emil Ranetti & Claudiu Nistor
Central University Military Hospital ‘Dr Carol Davila’, Bucharest, Romania.

Introduction
Parathyroid adenomas are the main cause of primary hyperparathyroidism. They are usually small – weighing <1 g – and not easy to find – requiring meticulous imaging studies for localisation. Giant adenomas are uncommon; large tumours and high levels of PTH raise the suspicion of parathyroid malignancy.

Case presentation
A 68-year-old female presented in our clinic with polydipsia, polyuria, nausea, weight loss, and extreme muscular weakness – she wasn’t able to walk – and depressive mood. Clinical exam revealed dehydration and right cervical mass. Calcium was 21 mg/dl and PTH was 2238 pg/ml. The patient was also vitamin D deficient – 25OH vitamin D 14 µg/l. Radiographic study showed fracture of the first lumbar vertebra, CT scan showed multiple osteolytic areas of the skull. Osteodensitometry demonstrated osteoporosis (lumbar spine T score —2.9 s.d. and distal radius T score —6.7 s.d.). Ultrasonography revealed a hypoechoic inhomogeneous mass, 38/30/45 mm, laterally and caudally to the right thyroid lobe. Parathyroid scintigraphy (99Tc-MIBI) demonstrated a large area of high uptake in that region. The patient received intravenous fluids, loop diuretic, i.e. bisphosphonate (zoledronate) and calcitonin to reduce the level of calcium, then she was successfully operated. Calcium dropped after surgery and it was managed with i.v. calcium and alpha calcidol. Mild hypocalcaemia persisted for more than 6 months thereafter and so did the high levels of PTH, that raised to 506.5 pg/ml, then returned to normal – the hungry bones syndrome. The pathologic diagnosis was benign parathyroid tumour – parathyroid adenoma.

Conclusions
This is a rare case of giant parathyroid adenoma. The peculiarities of the case are the size of the tumour, the very high level of calcium and PTH – suggesting a malignant tumour, and the persistence of high levels of PTH and hypercalcaemia months after surgery.

DOI: 10.1530/endoabs.37.EP296

EP297
Primary hypoparathyroidism is common in adult patients with β-thalassemia and protect patients from osteoporosis
Anna Ansaloni1, Francesca Ferrara1, Chiara Dazzi2,3, Antonello Pietrangelo4, Manuela Simon1,2 & Vincenzo Rochira1,2
1Unit of Endocrinology, Department of Biomedical, Metabolic and Neural Sciences, University of Modena and Reggio Emilia, Modena, Italy; 2Azienda USL of Modena, Modena, Italy; 3Unit of Internal Medicine, Research Center ‘Mario Coppo’, University Hospital Policlinico, Modena, Italy.

Introduction
β-thalassemia (βT) is associated to several endocrine abnormalities mainly due to iron overload. With the increase in βT-patients life expectancy, due to progresses in iron chelation therapy, more patients enter into adulthood and so the prevalence of endocrine diseases is being reconsidered. The aim of the study is to investigate the prevalence of primary hypoparathyroidism (pHPT) in adult βT-patients and to characterize the relative clinical phenotype with particular regard to bone health.

Methods
We enrolled 26 adult patients with major or intermedia βT (12M and 14F; mean age of 38.1 ± 7.5 years). Serum PTH, 25-hydroxyvitamin D (25OHD), calcium, phosphorous, albumin, bone turnover markers, and bone mineral density (BMD) by dual-energy X-ray absorptiometry (Hologic) at lumbar and femoral site were measured.
Introduction
Hypoparathyroidism is most commonly observed following neck surgery and is characterized biochemically by deficient parathyroid hormone (PTH) and hypocalcaemia alongside hyperphosphataemia and reduced 1,25-dihydroxyvitamin D. Standard treatment with oral calcium and vitamin D aims to maintain serum calcium within the low-normal range whilst avoiding hypercalcemia due to over replacement. However, concerns remain over the presence of hypercalcemia and the associated risk of renal calcification.

Aim
To assess whether serum and urine biochemical parameters are associated with the presence of renal calcification in hypoparathyroid patients on Alfalcacidol therapy.

Method
A 12-month audit of the laboratory database was undertaken of paired requests for 24-h urine calcium (24 h-Ca), spot calcium:creatinine ratio (Ca:Creat), serum calcium, phosphate, urea, and creatinine. A review of case notes was performed to confirm aetiology of hypoparathyroidism, Alfalcacidol dose and results of renal ultrasound scan (USS).

Results
A total of 34 patients were identified as having hypoparathyroidism and receiving Alfalcacidol therapy. 24 h-Ca and Ca:Creat were not-normally distributed, however significant associations were found between 24 h-Ca and Ca:Creat when log-transformed (linear regression $\beta$-coefficient $=0.64$, 95% CI 0.36-0.92; $P<0.001$, $\beta=0.63$). 17 patients had documented hypercalcemia evidenced by elevated 24 h-Ca (five patients), Ca:Creat (eight patients), or both (four patients). 13 patients had undergone renal USS; four had evidence of renal calcification. Interestingly, these four patients each had an elevated Ca:Creat, in contrast with only one patient having elevated 24 h-Ca. No patient had hypercalcemia. However, 20 patients had low, or low-normal serum adjusted calcium (Ca < 2.2 mmol/l); nine of these patients having documented hypercalcemia evidenced by an elevated 24 h-Ca (78% of patients) or Ca:Creat (89% of patients).

Conclusion
Ca:Creat appears a sensible and convenient marker for the follow-up of patients on long term Alfalcacidol therapy to determine associated risk of renal calcification.

Endocrine Abstracts (2015) Vol 37

EP298
Renal calcification in hypoparathyroid patients treated with calcium and vitamin D: can biochemistry help?
Sivatharsihya Pathmanathan1, Scott Tolhurst2, Emma Illingworth1, Claire Higham1, Peter Traini1 & Philip Monaghan2
1Department of Endocrinology, The Christie NHS Foundation Trust, Manchester, UK; 2The Christie Pathology Partnership, The Christie NHS Foundation Trust, Manchester, UK.

Introduction
Parathyroid tissue in ectopic thyroid tissue is relatively rare situations: supernumerary parathyroid gland, in thyroid tissue in an ectopic location.

Conclusions
In this patient despite scintigraphy suspicion of a functioning parathyroid adenoma, since it is a tertiary hyperparathyroidism, we chose surgical exploration with resection of all parathyroid glands. It was found a fifth focus of parathyroid tissue within an ectopic thyroid tissue. This case presents the association of three relatively rare situations: supernumerary parathyroid gland, in thyroid tissue in an ectopic location.

EP299
Parathyroid tissue in ectopic thyroid tissue
João Silva, Catarina Ivo, Mafalda Marcelino, Dolores Passos, Hélder Simões, Luís Lopes & João Jácome de Castro
Armed Forces University Hospital, Lisbon, Portugal.

Results
pHPT (PTH < 15 pg/ml) was found in seven of the 26 patients (27%). Of them, four patients (57%) had hypocalcemia and two were on chronic calcitriol therapy. Lumbar BMD was significantly higher in patients with pHPT (0.884 ± 0.189 g/cm²) than in patients without pHPT (0.731 ± 0.124 g/cm²) ($P=0.023$). No significant difference was found in femoral BMD, even though a trend for higher BMD was present in pHPT (0.704 ± 0.117 vs 0.670 ± 0.143 g/cm² in pHPT and no-pHPT respectively) ($P=0.578$). The prevalence of osteoporosis was higher in patients without pHPT (68%) than in patients with pHPT (29%). Two patients had a history of bone osteoporotic fractures and both of them did not present pHPT. Bone turnover markers were not different in the two groups.

Conclusions
The prevalence of pHPT in adult bT patients is higher if compared to that observed in Pediatric bT patients, the latter ranging from 8% to 11%. Moreover we found an higher prevalence of pHPT compared to that reported in literature on bone turnover markers.

EP300
Anti-diabetic treatment as an additional factor in a FRAX based evaluation of osteoporotic fracture risk
Maria P Yavropoulou, Athanasios Moutsios, Vasiliki Kolokouri, Pelagia Kolimpianaki, Athina Dimitriou, Petros Papalexis, Danieliliis & Kalliopi Kotisa
Department of Endocrinology and Diabetes, AHEPA University Hospital, Thessaloniki, Greece.

Introduction
Postmortem studies have shown that a fifth parathyroid gland may be present in about 5% of patients with hyperparathyroidism. 1% of parathyroid glands are located in thyroid tissue. There’s a prevalence of 7–10% of thyroid ectopic tissue.

Case report
A 53-year-old male, submitted to bilateral nephrectomy due to a Grawitz tumour at the age of 25. Under haemodialysis since then (with a rejected renal transplant in the past), he was recently referred to our department with a tertiary hyperparathyroidism diagnosis. Treated intra-hemodialysis with alfalcacidol 0.25 µg and cinacalcet. Analytically had a PTH 1604 pg/ml, calcium 9.5 mg/dl, phosphorus 5.6 mg/dl, and creatinine 11.4 mg/dl. Cervical ultrasound did not identify parathyroid gland and thyroid scintigraphy suggested parathyroid adenoma in the bottom right. PET-scan showed bone lesions suggestive of brown tumours. The patient was submitted to surgery and has removed four parathyroid glands (9–20 mm) with an histology of ‘nodular hyperplasia of the parathyroid gland’. On one of the four parathyroid glands was ‘focus of parathyroid in parenchyma thyroid (intra-thyroid parathyroid?)’. Three months after surgery he’s treated with 1 g of calcium carbonate (3 + 3 +3) and 0.25 µg calcitriol (1 + 0 + 1), with PTH 139 pg/ml, calcium 8.2 mg/dl, and phosphorus 2.6 mg/dl.

Conclusions
In this patient despite scintigraphy suspicion of a functioning parathyroid adenoma, since it is a tertiary hyperparathyroidism, we chose surgical exploration with resection of all parathyroid glands. It was found a fifth focus of parathyroid tissue within an ectopic thyroid tissue. This case presents the association of three relatively rare situations: supernumerary parathyroid gland, in thyroid tissue in an ectopic location.