*XI Advanced Course of Endocrinology • XI Curso Avançado de Endocrinologia [2023;18 (Supl. 4)]*

# REVISTA PORTUGUESA DE ENDOCRINOLOGIA, DIABETES E METABOLISMO

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# REVISTA PORTUGUESA DE ENDOCRINOLOGIA, DIABETES E METABOLISMO

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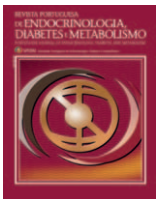

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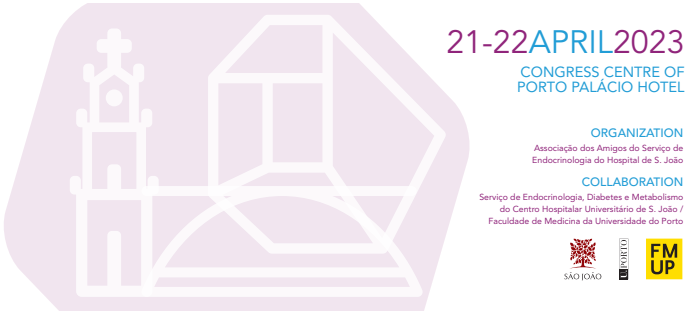


# SUMÁRIO

XI ADVANCED COURSE OF ENDOCRINOLOGY

# Welcome Words

Paula Freitas [a, b,*]

[a] *Editora-chefe da Revista da Sociedade Portuguesa de Endocrinologia, Diabetes e Metabolismo*
[b] *Serviço de Endocrinologia, Centro Hospitalar Universitário São João, Faculdade de Medicina da Universidade do Porto, I3S, Porto, Portugal*

Dear Friends,

Welcome to the XI Advanced Course of Endocrinology, Diabetes and Metabolism.

This year the Endocrinology Department of Centro Hospital Universitário São João and Faculty of Medicine of Porto University intended to do a total presential Course, but as the pandemic taught us, when it is not possible, virtual is a very good solution. Of course, we want to meet face-to-face, but the virtual taught us that everything is possible. And for various reasons, we will have a mixed course - face-to-face and virtual.

This course was planned to update our Knowledge in Endocrinology, Diabetes and Metabolism but essentially it is for the fellows and young doctors. For this reason, we have 3 Prizes to stimulate their research. But this course is a time of discussion and sharing of knowledge between young and expert doctors and local and foreigners scientists.

Undoubtedly, the more knowledge we have, the better we will treat our patients. This is our priority.

This year we have a program that seeks to touch on various areas of Endocrinology and show the most recent scientific evidence, namely in terms of treatment. In this way, we will address topics such as a new insulin glargine generation to optimize healthcare in people with diabetes; difficulties in the management of PitNets; new tools in diabetic nephropathy management; new perspectives in the treatment of diabetes, obesity and NASH; metabolic bone disease and X-linked hypophosphatemia – the role of burosumab and a panel of discussion and sharing of experiences – the vison of the Endocrinologist, the Nephrologist and Cardiologist, Ophthalmologist about the multidisciplinary management of patients with T2D in a Central Hospital.

This year is the first year without Prof. Davide Carvalho at the helm of this boat. As Prof. Davide Carvalho wrote last year in this Course. "I'm going to be walking around", we will always count on him and his knowledge and friendship.

This year we will try to grow and we want to be a milestone in national Endocrinology meetings, we want to held a short-term meeting (1 and a half days) but of great scientific intensity.

We want to acknowledge the work of all participants, speakers, moderators, panelists and awards juries.

A very special thanks to pharmaceutical industry in these difficult times.

Another thank you to the Norahevents team and leadership.

A very, very special thanks to all the elements of the Service of Endocrinology.

Paula Freitas

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Portuguese Society of Endocrinology, Diabetes and Metabolism
Rua Fernando Vicente Mendes, Nº 1B, 1º Dto., 1600-892 Lisboa, Portugal


21-22APRIL2023
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PORTO PALÁCIO HOTEL

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Associação dos Amigos do Serviço de Endocrinologia do Hospital de S. João

COLLABORATION
Serviço de Endocrinologia, Diabetes e Metabolismo do Centro Hospitalar Universitário de S. João / Faculdade de Medicina da Universidade do Porto

# 21st April Friday

**15h00** **EPoster Session** – Screen 1

**EP01** **USEFULNESS OF CELLBLOCK PREPARATION IN FINE NEEDLE ASPIRATION FOR THE DIAGNOSIS OF THYROID NODULES**
Francisca Marques Puga[1], Miguel Rodrigues[2], Catarina Eloy[3]
*1-Serviço de Endocrinologia, Diabetes e Metabolismo, Centro Hospitalar Universitário de Santo António; 2-Serviço de Anatomia Patológica, Hospital Distrital de Santarém; 3-Laboratório de Anatomia Patológica, Instituto de Patologia e Imunologia Molecular da Universidade do Porto | Instituto de Investigação e Inovação em Saúde (i3S) | Faculdade de Medicina da Universidade do Porto*

**EP02** **SECONDARY THYROID LYMPHOMA: A CASE REPORT**
Catarina Gama[1], Margarida Oliveira[1], Carolina Antunes[1], Paula Calvo[1], Leonor Lopes[1], Miguel Quintela[2], Bernardo Marques[1], João Sequeira Duarte[1]
*1-Serviço de Endocrinologia, Diabetes e Metabolismo, Hospital de Egas Moniz, Centro Hospitalar Lisboa Ocidental; 2-Serviço de Hematologia, Hospital de São Francisco Xavier, Centro Hospitalar de Lisboa Ocidental*

**EP03** **NEUROSARCOIDOSIS PRESENTING WITH PAN-HYPOPITUITARISM**
Carolina Monteiro Antunes[1], Leonor Guia Lopes[1], Catarina Gama[1], Olga Capontes[2], Paula Calvo[1], Margarida Oliveira[1], Manuel Salavisa[3], Francisco Sousa Santos[1], João Sequeira Duarte[1]
*1-Endocrinologia, Hospital de Egas Moniz; 2-Medicina Interna, Hospital de Egas Moniz; 3 - Neurologia, Hospital de Egas Moniz*

**EP04** **CONGENITAL DISORDER OF GLYCOSYLATION AND THE IMPORTANCE OF AN ENDOCRINOLOGY APPROACH: A CASE REPORT**
Margarida Oliveira[1], Catarina Gama[1], Carolina Antunes[1], Paula Calvo[1], Maria Leonor Lopes[1], Rute Ferreira[1], João Sequeira Duarte[1]
*1-Centro Hospitalar Lisboa Ocidental*

**EP05** **CARDIOVASCULAR AND METABOLIC RISK FACTORS IN PATIENTS WITH AUTONOMOUS CORTISOL SECRETION**
Sara Franco[1], Francisca Leitão[1], Ana Quítalo[1], Ana Gonçalves Ferreira[1], David Barbosa[1], Ricardo Capitão[1], Maria Manuel Costa[1], Henrique Vara Luiz[1], Isabel Manita[1], Maria Carlos Cordeiro[1], Luísa Raimundo[1]
*1-Hospital Garcia de Orta*

**EP06** **CORTISOL CO-SECRETION FROM ALDOSTERONE-PRODUCING ADENOMA: CLINICAL CASE**
Henrique Carmona Alexandrino[1], Marta Almeida Ferreira[1], Cristina Carneiro[2], Jorge Dias[3], Maria João Oliveira[1]
*1-Centro Hospitalar Vila Nova de Gaia / Espinho, Serviço Endocrinologia; 2-Centro Hospitalar Vila Nova de Gaia / Espinho, Serviço Medicina Interna; 3-Centro Hospitalar Vila Nova de Gaia / Espinho, Serviço Urologia*

**EP07** **DIABETIC KETOACIDOSIS IN THE ELDERLY: A NEAR-FATAL OUTCOME**
Bárbara Jesus[1], Gustavo Rodrigues[1], Joana Coelho[1]
*1-Centro Hospitalar e Universitário de Coimbra*

**15h00** **EPoster Session** – Screen 2

**EP08** **PREDICTORS OF READMISSION AND MORTALITY IN PATIENTS WITH DECOMPENSATED HEART FAILURE ACCORDING TO GLYCAEMIC STATUS**

Ana Brandão[2], Marta Borges Canha[1], Ana Rita Leite[1], João Sérgio Neves[1], Davide Carvalho[2]
*1-Serviço de Endocrinologia, Diabetes e Metabolismo do Centro Hospitalar Universitário de São João, Porto, Portugal.; 2-Faculdade de Medicina, Universidade do Porto, Porto, Portugal*

**EP09** **RELATIONSHIP BETWEEN PROBLEMATIC HYPOGLYCAEMIA AND HEALTH STATUS, GLOBAL COGNITION AND EXECUTIVE FUNCTIONS IN ADULTS WITH TYPE 1 DIABETES ATTENDING A TERTIARY DIABETES SERVICE IN PORTUGAL**

Eduardo Sepúlveda[1,2], Rui Poínhos[3], Davide Carvalho[4,5], Gil Nata[6], Selene G. Vicente[1], Stephanie A. Amiel[7]
*1-Centre for Psychology at Universidade do Porto, Faculty of Psychology and Educational Sciences, Universidade do Porto, Porto, Portugal; 2-Diabetes Research Group, King's College London, London, UK; 3-Faculty of Nutrition and Food Sciences, Universidade do Porto, Porto, Portugal; 4-Department of Endocrinology, Diabetes and Metabolism, Centro Hospitalar São João, Porto, Portugal; 5-Faculty of Medicine, Instituto de Investigação e Inovação em Saúde, Universidade do Porto, Porto, Portugal; 6-Centre for Research and Intervention in Education and Centre for Psychology at Universidade do Porto, Faculty of Psychology and Educational Sciences, Universidade do Porto, Porto, Portugal; 7-Department of Diabetes, School of Cardiovascular and Metabolic Medicine and Sciences, King's College London, London, UK*

**EP10** **ASSOCIATION OF GLYCEMIC VARIABILITY AND TIME IN RANGE WITH LIPID PROFILE IN TYPE 1 DIABETES**

Mariana Salsa Castelo[1], Celestino Neves[2], João Sérgio Neves[2], Davide Carvalho[2]
*1-Faculdade de Medicina da Universidade do Porto; 2-Centro Hospitalar Universitário de São João*

**EP11** **METABOLIC SYNDROME IS A MAIN DETERMINANT OF QUALITY OF LIFE AFTER METABOLIC SURGERY**

Catarina Abreu Loureiro[2], Andre Costa Pinho[1], Hugo Santos Sousa[1], Helena Urbano Ferreira[1], Juliana Gonçalves[1], Paula Freitas[1], Crio Group[1]
*1-Centro Hospitalar Universitário São João; 2-Faculty of Medicine of the University of Porto*

**EP12** **GLYCEMIC CONTROL AND METABOLIC PARAMETERS IN CHILDREN AND ADOLESCENTS WITH TYPE 1 DIABETES**

Marta Borges-Canha[1,2], Sofia Ferreira[3], Rita Santos Silva[3], Aida Correia De Azevedo[4], Ana Sofia Rodrigues[4], Cíntia Castro-Correia[2]
*1-Department of Endocrinology, Centro Hospitalar Universitário de São João, Porto, Portugal; 2-Faculty of Medicine, University of Porto, Porto, Portugal, Portugal; 3-Paediatric Endocrinology Unit, Paediatrics Department, Centro Hospitalar Universitário de São João, Porto, Portugal; 4-Paediatrics Department, Centro Hospitalar do Médio Ave, Portugal.*

**EP13** **QUALITY OF LIFE IN THYROID CANCER PATIENTS AND THE INFLUENCE OF CLINICO-PATHOLOGICAL CHARATERISTICS**

Ana Sarmento[1], Ana Carreira[2], Miguel Melo[2], Isabel Paiva[2]
*1-Faculdade de Medicina da Universidade de Coimbra; 2-Centro Hospitalar e Universitário de Coimbra*

**EP14** **ICPI-RELATED THYROID FUNCTION ALTERATION, COURSE AND MONITORING: A RETROSPECTIVE STUDY**

Raquel Calheiros[1], Sara Gil-Santos[1], Pedro Souteiro[1], Joana Oliveira[1], Isabel Inácio[1], Ana Paula Gil-Santos[1]
*1-Department of Endocrinology, Portuguese Oncology Institute of Porto (IPO Porto), Porto Comprehensive Cancer Centre (P.CCC)*

**EP15** **THYROID DYSFUNCTION AND HEART FAILURE WITH PRESERVED EJECTION FRACTION: A SYSTEMATIC REVIEW**

Luís Pedro Rosmaninho Miranda[1,2], Marta Borges Canha[1,2], Davide Carvalho[1,2], Ana Leite[1,2], João Ségio Neves[1,2]
*1-Faculdade de Medicina da Universidade do Porto; 2-Centro Hospitalar Universitário São João*

**15h50-16h00** **WELCOME WORDS** – Paula Freitas

**16h00-18h00** **WORKSHOP: CLINICAL CASES**
*Chairman:* Helena Cardoso
*Moderators / Speakers:* Helena Cardoso, João Sérgio Neves

**A NEW INSULIN GLARGINE GENERATION TO OPTIMIZE HEALTHCARE IN PEOPLE WITH DIABETES**
**General Discussion**
*Discussion Panel:* Celestino Neves, João Sequeira Duarte, Luísa Barros

*Sponsored by Sanofi*

**18h00-20h00** **CHALLENGES OF THE MULTIDISCIPLINARY MANAGEMENT OF PATIENTS WITH T2D IN A CENTRAL HOSPITAL. SHARE WITH US YOUR EXPERIENCE – THE VISON OF THE ENDOCRINOLOGIST, THE NEPHROLOGIST AND CARDIOLOGIST, OPHTHALMOLOGIST**

*Chairman:* Davide Carvalho
**General Discussion**
*Discussion of Experts:* Peter Rossing, Ricardo Neto, Angela Carneiro, Paula Dias, Paula Freitas

*Sponsored by Bayer*

# 22nd April Saturday

**08h30-10h30** **DIFFICULTIES IN THE MANAGEMENT OF PITNETS**
*Chairmen:* Davide Carvalho, Maria João Bugalho
*Moderators:* Leonor Gomes, Ana Paula Marques

08h30-08h50 **Genetics of Pituitary Adenomas: Do They Matter for Management?**
Pedro Marques

08h50-09h10 **When and How to Withdraw Dopamine Agonists in Prolactinomas?**
Pedro Souteiro

09h10-09h30 **Cushing Disease: Pitfalls in the Diagnosis and Management.**
Adriana Lages

09h30-10h00 **How do They Grow so Tall.**
Albert Beckers

10h00-10h30 **General Discussion**
*Discussion Panel:* Jorge Pedro, Josué Pereira, Jorge Pinheiro

*Sponsored by Recordati*

**10h30-11h00** **Coffee Break and EPoster Discussion** – Screen 1

**EP16** **SPONTANEOUS PREGNANCY IN A WOMAN WITH HYPOGONADOTROPIC HYPOGONADISM: A CASE REPORT**
Marta Vaz Lopes[1], José Vicente Rocha[1], Carolina Peixe[1], Mariana De Griné Severino[1], Ana Coelho Gomes[1], Maria João Bugalho[1,2]
*1-Serviço de Endocrinologia do Centro Hospitalar Universitário Lisboa Norte; 2-Faculdade de Medicina da Universidade de Lisboa*

**EP17** **TIME IN RANGE AND COMPLICATIONS OF DIABETES: A CROSS-SECTIONAL ANALYSIS OF PATIENTS WITH TYPE 1 DIABETES**
Marta Bezerra[1], Celestino Neves[2,3], João Sérgio Neves[1,2,4], Davide Carvalho[1,2,3]
*1-Faculty of Medicine of the University of Porto, Porto, Portugal; 2-Department of Endocrinology, Diabetes and Metabolism, Centro Hospitalar Universitário de São João, Porto, Portugal.; 3-Instituto de Investigação e Inovação em Saúde, Universidade do Porto, Porto, Portugal.; 4-Cardiovascular R&D Centre - UnIC@RISE, Department of Surgery and Physiology, Faculty of Medicine of the University of Porto, Porto, Portugal.*

**EP18** **GALACTOSEMIA: A RARE CAUSE OF HYPERGONADOTROPIC HYPOGONADISM**
Carolina Peixe[1], Marta Vaz Lopes[1], José Vicente Rocha[1], Mariana Griné Severino[1], Ana Paula Barbosa[1], Anabela Oliveira[1], Ana Coelho Gomes[1], Maria João Bugalho[1]
*1-Centro Hospitalar Universitário de Lisboa Norte, Lisboa, Portugal*

**EP19** **A UNIQUE CASE OF SUBCENTIMETRIC PAPILLARY THYROID CARCINOMA WITH DISTANT METASTASIS: WHAT CAN WE LEARN FROM THAT?**
Diogo Ramalho[1], Andreia Amado[1], Elisabete Teixeira[2], Sule Canberk[2], Henrique Carmona[1], Helena Alves[1], Barbara Castro[1], Hugo Pereira[1], João Varandas[1], Susana Graça[1], Carlos Soares[1], Paula Soares[2], Manuel Sobrinho Simões[2], Andreia Póvoa[1]
*1-Centro Hospitalar de Vila Nova de Gaia / Espinho; 2-i3S - Instituto de Investigação e Inovação em Saúde, Universidade do Porto, Portugal*

**10h30-11h00** **Coffee Break and EPoster Discussion** – Screen 2

**EP20** **QUALITY OF LIFE IN PATIENTS WITH METABOLIC SYNDROME – THE WORSE, THE WORST?**
Marta Borges-Canha[1,2], Joana Chaves[1], Ana Rita Leite[1,2], Rodrigo Liberal[3], Inês Lourenço[1], Madalena Von Hafe[1], João Sérgio Neves[1,2], Mariana Fragão-Marques[1,4], Pedro Pimentel-Nunes[1], Adelino Leite-Moreira[1], Davide Carvalho[2,5], Paula Freitas[2,5]
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**EP21** **EFFECTIVENESS OF GLICAZIDE IN TREATING MODY 13: A CASE REPORT**
Juliana Gonçalves[1,2], Helena Ferreira[1,2], Selma B. Souto[1,2], Jorge Pedro[1,2], Paula Freitas[1,2]
*1-Endocrinology, Diabetes and Metabolism, Centro Hospitalar Universitário de São João, Porto; 2-Medicine Faculty and Instituto Investigação e Inovação em Saúde (i3s), Universidade do Porto, Porto, Portugal*

**EP22** **HYPOCALCAEMIA IN PRE-EXISTING HYPOPARATHYROIDISM AFTER BARIATRIC SURGERY - A CHALLENGE FOR CLINICAL MANAGEMENT.**
Inês Meira & João Menino[1], Sara Ribeiro[1], Ana Varela[1], Paula Freitas[1]
*1-Centro Hospitalar Universitário de São João*

**EP23** **ECTOPIC ACTH SYNDROME – A CASE SERIES**
Sara Ribeiro[1], Telma Moreno[1], Ana Varela[1], Paula Freitas[1]
*1-Serviço Endocrinologia, Diabetes e Metabolismo,CHUSJ*

**11h00-11h45** *Chairmen:* Daniel Braga, Ricardo Neto
*Moderators:* Maria João Oliveira, Pedro Melo

11h00-11h30 **New Tools in Diabetic Nephropathy Management.**
Peter Rossing
11h30-11h45 **General Discussion**
*Discussion Panel:* Elisabete Rodrigues, Duarte Pignatelli, Pedro Rodrigues

*Sponsored by Bayer*

**11h45-13h15** **NEW PERSPECTIVES IN THE TREATMENT OF DIABETES, OBESITY AND NASH**
*Chairmen:* João Jácome de Castro, Jorge Dores
*Moderators:* Luisa Raimundo, Ana Maria Maia

11h45-12h15 **The Role of GIP in Diabetes and Obesity**
Alex Miras
12h15-12h45 **From Screening of Individuals at Risk to Type 1 Diabetes Prevention.**
Chantal Mathieu
12h45-13h00 **MAFLD: Do You Agree?**
Marta Canha
13h00-13h15 **General Discussion**
*Discussion Panel:* Sandra Belo, Selma Souto, Diana Festas-Silva

*Sponsored by Lilly*

13h15-14h30 **Lunch**

**14h30-16h30** **METABOLIC BONE DISEASE**
*Chairmen:* Valeriano Leite, Isabel Torres
*Moderators:* Mafalda Marcelino, Mário Mascarenhas

14h30-14h45 **Clinical Case: a Child with X-linked Hypophosphatemia**
Sofia Ferreira

14h45-15h00 **Clinical Case: an Adult with X-linked Hypophosphatemia**
Sara Ribeiro

15h00-15h30 **X-linked Hypophosphatemia – the Role of Burosumab**
Adalbert Raimann

15h30-16h00 **All the Patients with Hyperparathyroidism Should be Operated? Results of our Center**
João Capela

16h00-16h15 **General Discussion**
*Discussion Panel:* Margarida Almeida, Leonilde Coelho

*Sponsored by Kyowa Kirin*

**16h15-16h45** **Coffee Break and EPoster Discussion** – Screen 1

**EP24** **HYPONATREMIA – SHOULD WE CALL THE ENDOCRINOLOGIST?**
Sara Gil-Santos[1], Raquel Calheiros[1], Pedro Souteiro[1], Joana Oliveira[1], Isabel Inácio[1], Ana Paula Santos[1], Isabel Torres[1]
*1-Instituto Português de Oncologia Francisco Gentil do Porto*

**EP25** **TYPE 3 AUTOIMMUNE POLYGLANDULAR SYNDROME: A RARE COMBINATION OF MULTIPLE AUTOIMMUNE MANIFESTATIONS**
Sofia Monteiro Lopes[1], Mafalda Ferreira[1], Tânia Carvalho[1], Luísa Ruas[1], Leonor Gomes[1], Isabel Paiva[1]
*1-Centro Hospitalar e Universitário de Coimbra*

**EP26** **POSACONAZOLE-INDUCED PSEUDOHYPERALDOSTERONISM – A CLINICAL CASE**
Telma Moreno[1], Sara Ribeiro[2], Ana Varela[2], Pedro Rodrigues[2]
*1-Seviço de Endocrinologia, Diabetes e Metabolismo do Centro Hospitalar Universitário de São João; 2-Serviço de Endocrinologia, Diabetes e Metabolismo do Centro Hospitalar Universitário de São João*

**EP27** **PANHYPOPITUITARISM AND CRANIOPHARYNGIOMA REMOVAL SURGERY: IS CONSERVATIVE RESECTION BETTER?**
Maria João Dantas[1], Patrícia Polónia[1,2]
*1-Faculdade de Medicina da Universidade do Porto (FMUP), Porto, Portugal; 2-Department of Neurosurgery, Centro Hospitalar Universitário de São João (CHUSJ), Porto, Portugal*

**16h45-17h00** **Closing and Awards** – Paula Freitas

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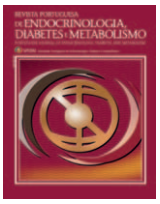
XI ADVANCED COURSE OF ENDOCRINOLOGY



# Conference Abstracts

## L1. GENETICS OF PITUITARY ADENOMAS: DO THEY MATTER FOR MANAGEMENT?

Pedro Marques

Most pituitary adenomas are sporadic, although up to 5% of these may arise in a familial context occurring either in isolation or as part of a syndrome. Remarkable advances in the field of genetics of pituitary adenomas were achieved in the past years, resulting in a deeper understanding of their pathophysiology, discovery of new conditions such as the X-linked acrogigantism (XLAG) syndrome, and recognition of pituitary adenomas as part of previously known tumor syndromes, such as *SDH*-related or *DICER1*-related disease, Lynch syndrome and neurofibromatosis. Isolated pituitary adenomas can be observed in *AIP* gene mutation-positive cases as well as in XLAG due to *GPR101* duplications, but in most patients with familial isolated pituitary adenomas, the disease-causing mutations have not been identified. On the other hand, pituitary adenomas presenting in a syndromic context may occur in MEN1, MEN4, Carney complex, McCune-Albright syndrome, and rarely, mutations in the *DICER1* and *SDH/MAX* genes can also predispose to pituitary adenomas. Familial pituitary adenomas may display a more aggressive biological and clinical behavior, as well as they are often refractory to conventional therapies, including medical therapy, and thus requiring multimodal and multiple treatment approaches in order to achieve disease control. For instance, somatotropinomas emerging in the context of *AIP* mutation-positive disease or in XLAG are particularly resistant to medical therapy with somatostatin analogues, and multiple surgeries and radiotherapy are often needed. Familial pituitary adenomas should be suspected in case of: i) family history of pituitary adenomas; ii) pituitary adenomas diagnosed age ≤18 years (micro ou macroadenomas); iii) pituitary macroadenomas diagnosed between ages 18-30 years; iv) coexistence of other syndromic manifestations such as kidney stones, primary hyperparathyroidism or neuroendocrine tumors (MEN1 or MEN4 syndromes); cardiac mixomas, nevi, thyroid or adrenal tumors, schwannomas (Carney complex syndrome); pheocromocytomas or paragangliomas (3PAs syndrome related to *SDHx* or *MAX* mutations); pituitary blastomas, lung blastomas, goiter, kidney cysts (DICER1 syndrome); colon, brain, uterine, pancreatic or ovarian cancers (Lynch syndrome). Genetic screening and clinical follow-up of individuals carrying germline mutations in genes predisposing to pituitary adenomas may lead to an early diagnosis of the disease and better long-term outcomes, whereas better understanding of the pituitary tumorigenesis may lead to further improvements in the management of familiar pituitary adenomas. Hence, the genetics of pituitary adenomas do really matter for management!

## L2. WHEN AND HOW TO WITHDRAW DOPAMINE AGONISTS IN PROLACTINOMAS?

Pedro Souteiro

Dopamine agonists (DAs) are the first-line agents in the treatment of prolactinomas. The two main goals are achieving tumoral shrinkage and normoprolactinemia, thereby avoiding central hypogonadism, visual field impairment and symptoms like galactorrhea and headaches.

Studies in the 1980s, mainly using bromocriptine, started to defy the paradigm that DAs should be used as a lifelong therapy. Since then, the introduction of the more effective agent cabergoline led to an additional increase in the remission percentages. Meta-analysis on this topic refer that remission rates after DAs withdrawal can range from 15% in macroprolactinoma patients treated with bromocriptine to 41% in those with microprolactinomas treated with cabergoline. When more stringent criteria were applied before attempting withdrawal, sustained remission was possible in more than 50% of the individuals.

The establishment of criteria to select which patients can be safely withdrawn from DAs and to ascertain when and how to do it are the main challenges when approaching this topic. Treatment duration for more than 24 months, the achievement of normoprolactinemia, marked reduction (≥ 50%) in tumoral size and DAs tapering till a low maintenance dose (e.g. cabergoline 0.5 mg/week) have been identified as the most consistent predictors of success. In addition, some evidence suggests that the post-pregnancy/breastfeeding period and menopause are also reasonable timings to re-access the need for continuing DAs.

Considering that the achievement of sustained normoprolactinemia is still far from being universal after DAs withdrawal, even in highly selected cohorts, future studies should continue to address this issue.

## L3. CUSHING DISEASE: PITFALLS IN THE DIAGNOSIS AND MANAGEMENT

Adriana de Sousa Lages[1]

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Endogenous hypercortisolism (EH) represents a major challenge for endocrinologists since its first description in 1932 by Harvey Cushing.

Diagnosis is often delayed for several years, in part due to lack of knowledge of the insidious and progressive course of the disease and the high complexity from diagnosis to lifelong management.

Despite many recent advances, diagnosis involves a rigorous clinical evaluation and a logical systematic approach with special

attention to the less typical features of glucocorticoid excess in certain populations (such as in advanced age) and the definition of the probability of pre-selecting patients with an appropriate likelihood of EH.

No screening or diagnostic test is 100% sensitive or specific and there is no single preferred diagnostic test, and for this reason, medical teams need to individualize decisions about timing and selection for diagnostic testing based on the clinical scenario, patient comorbidities, and laboratory availability.

With respect to imaging, which should only be done when the biochemical diagnosis has been established, assuming several constraints (such as small or ephemeral magnetic resonance imaging-MRI abnormalities), recent advances in MRI technical refinements or the use of functional imaging (such as PET $^{11}$C-methionine) may improve detection and allow better localization of microadenomas.

Although transsphenoidal surgery is recommended as first-line therapy for patients with Cushing's disease (CD), recurrence rates after initial remission range from 5% to 35%, with half appearing after up to 10 years or more, justifying the need for lifelong monitoring, with late-night salivary cortisol being the test with the highest sensitivity in this context.

Medical therapy may be used to treat hypercortisolism in patients with persistent or recurrent CD and those who are not candidates or refuse surgery, and to control cortisol levels in patients undergoing radiation therapy (RT). Although there is longest clinical experience for adrenal steroidogenesis inhibitors (as with ketoconazole and metyrapone), therapeutic options such as the use of osilodrostat and somatostatin analog pasireotide have broadened the range of available options with significant efficacy and an advantageous safety profile.

Concerns regarding quality of life and complications are a continuum that are not always resolved by establishing biochemical normality, highlighting the importance of focusing on some data such as the risk of thrombotic events and the impact on bone mineral density in a particular manner in individuals with CD.

Within this line, an optimal approach for patients with CD require a comprehensive and close proficiency of all professionals of the management team embracing the particular features of the disease journey.

## L4. HOW DO THEY GROW SO TALL?

Albert Beckers[1,2]

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Pituitary gigantism is a rare disorder caused by excess of GH/IGF-1 due to GH-secreting lesions, that occurs before epiphyseal closure leading to increased linear growth. This disease is accompanied by a significant reduction in life expectancy. Until very recently, little scientific research had been specifically dedicated to patients with gigantism, probably because of the rarity of the condition and the consequent difficulty of assembling large series. Following works on FIPA and *AIP* gene mutations, it became evident that patients with these mutations have more aggressive pituitary adenomas preferentially secreting growth hormone, and develop the disease much earlier in life (frequently before the end of puberty), thereby permitting the development of gigantism. These works have stimulated scientific, and particularly genetic, studies on patients with gigantism. These cases have more aggressive features of pituitary disease than sporadic acromegaly, including a younger age at disease onset and larger tumor size, and they can be challenging to treat. Over the past decades several molecular defects that cause GH-secreting pituitary adenomas have been identified, including multiple endocrine neoplasia syndromes type 1 and 4, Carney complex, McCune-Albright syndrome, familial isolated pituitary adenoma (FIPA) and *AIP* mutations, pituitary adenoma with paraganglioma/pheochromocytoma, and the recently identified X-linked acrogigantism syndrome (X-LAG). About half of pituitary gigantism cases have genetic predisposition, and AIP mutations represent the most frequent genetic cause of pituitary gigantism (30%). In 10% the cause is a duplication involving the GPR101 gene, which is responsible for X-linked AcroGigantism (X-LAG). This syndrome, discovered very recently, includes the most extreme forms of human gigantism (with heights greater than 2.50 m), such as Julius Koch, alias the giant Constantin (2.59 m), who died at the age of 30 in 1902. A careful study of his DNA allowed the diagnosis to be considered. X-LAG can be caused by variable degrees of somatic mosaicism for *GPR101* duplication in sporadic males. Hypothalamic GHRH hypersecretion can accompany the pituitary abnormalities seen in X-LAG, and *in vitro* studies showed that GHRH receptor antagonist can significantly reduce GH release. Besides sporadic cases, X-LAG represents a new genetic cause of non-*AIP* FIPA, transmission from affected mother to affected son was reported in 3 FIPA families. X-LAG is more frequent in females, and associated with early-onset pituitary disease (in most cases during the 1 year of life, and always before age of 5) and extremely accelerated linear growth. X-LAG is usually associated with markedly elevated GH and prolactin secretion by mixed pituitary adenomas/hyperplasia. Response to somatostatin analogs is poor and multimodal treatment is frequently required, including neurosurgery, GH receptor antagonist and radiotherapy, which however increase the risk of hypopituitarism. These studies on X-LAG have advanced our understanding of the physiology of growth and the pathological hypothalamic-pituitary mechanisms that govern the formation of somatotropic adenoma and development of the tallest forms of pituitary gigantism.

## L5. NEW TOOLS IN DIABETIC NEPHROPATHY MANAGEMENT

Peter Rossing

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Unfortunately, diabetes is a growing problem with now more than 537 million people worldwide having diabetes according to International Diabetes Federation. Excess morbidity and mortality in diabetes is related to the development of diabetic kidney disease seen in 30%-40%, which not only causes end stage kidney disease, but also increases risk for cardiovascular events and mortality, which is the leading cause of death. For treatment to be initiated the diagnosis has to be made, so screening with annual control of urinary albumin excretion and eGFR is mandatory but often missing.

Treatment of diabetic kidney disease has been control of glucose, lipids, and blood pressure including blockade of the renin angiotensin system, but the residual risk on optimal treatment was large. Multiple interventions have been tested and failed in this condition, but in the past few years, we have suddenly been able to find new treatment options. First cardiovascular outcome trials in

diabetes found the SGLT2 inhibitors reduced progression of kidney disease or heart failure, and GLP1 receptor agonists reduced cardiovascular events in people with CKD, whereas potential kidney benefits remain to be demonstrated in dedicated kidney studies.

Most recently the nonsteroidal mineralocorticoid receptor antagonist finerenone demonstrated reduction in progression of kidney and cardiovascular disease. Finerenone was tested in the complementary studies FIDELIO-DKD and FIGARO-DKD including over 13 000 patients with type 2 diabetes and chronic kidney disease (CKD) and the studies examined cardiovascular and kidney outcomes in different, overlapping stages of CKD. The studies were combined in the FIDELITY analysis were an individual patient-level prespecified pooled efficacy and safety analysis across a broad spectrum of CKD was performed to provide more robust estimates of safety and efficacy of finerenone compared with placebo. For this prespecified analysis, the two phase III, multicentre, double-blind trials involving patients with CKD and type 2 diabetes, randomized 1:1 to finerenone or placebo, were combined. Main time-to-event efficacy outcomes were a composite of cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, or hospitalization for heart failure, and a composite of kidney failure, a sustained ≥57% decrease in estimated glomerular filtration rate from baseline over ≥4 weeks, or renal death. Among 13 026 patients with a median follow-up of 3.0 years (interquartile range 2.3-3.8 years), the composite cardiovascular outcome occurred in 825 (12.7%) patients receiving finerenone and 939 (14.4%) receiving placebo [hazard ratio (HR), 0.86; 95% confidence interval (CI), 0.78-0.95; $p = 0.0018$]. The composite kidney outcome occurred in 360 (5.5%) patients receiving finerenone and 465 (7.1%) receiving placebo (HR, 0.77; 95% CI, 0.67-0.88; $p = 0.0002$). Overall safety outcomes were generally similar between treatment arms. Hyperkalaemia leading to permanent treatment discontinuation occurred more frequently in patients receiving finerenone (1.7%) than placebo (0.6%). Subsequent analyses demonstrated that the benefits of finerenone were independent of concomitant treatment with SGLT2i or GLP1 receptor agonists and independent of baseline HbA1c or CVD. Thus, the Fidelity analysis demonstrated that Finerenone reduced the risk of clinically important cardiovascular and kidney outcomes vs. placebo across the spectrum of CKD in patients with type 2 diabetes.

New guidelines for management of type 2 diabetes with CKD from American Diabetes Association and Kidney Disease Improving Global Outcomes (KDIGO) recommend the use of these new treatment options. Thus, we suddenly have the opportunity to improve on the most deadly complications to diabetes with several interventions, and as they work on different disease pathways (hemodynamic, metabolic, inflammatory) data suggest they can be combined with extra benefit for the person with diabetes. Now implementation is key, and quality monitoring systems should be in place to ensure this.

## L6. THE ROLE OF GIP IN DIABETES AND OBESITY

Alex Miras[1]

[1] *Imperial College Healthcare NHS Trust*

In this presentation we will go through the physiology of GIP secretion and its effects on glucose and body weight regulation. This will then be linked with the pharmacological action of GIP agonism and antagonism for diabetes and obesity.

## L7. FROM SCREENING OF INDIVIDUALS AT RISK TO TYPE 1 DIABETES PREVENTION

C. Mathieu

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With the arrival of the first disease-modifying therapy (teplizumab) into the therapeutic arsenal for treatment of type 1 diabetes, we are entering a new era. However, it is not only new therapeutics that are entering the clinical world. Based on evolving insights into the pathophysiology of type 1 diabetes, we are discovering that type 1 diabetes is an autoimmune disease that does not start when clinicians typically diagnose it on the basis of clinical signs and symptoms (polyuria, polydipsia, weight loss and the feared complication of diabetic ketoacidosis). We have learned that the disease starts many months even years before this clinical picture; thus, a new classification of type 1 diabetes has been proposed, with the typical clinical disease clinicians treat being the final stage (stage 4). New insights show that an interplay between genetic predisposition and environmental triggers (like viruses) lead to the onset of an autoimmune attack (stage 0). This first stage is characterized by the presence of a single type of autoantibody, directed against one of 4 antigens (insulin/proinsulin, glutamic acid decarboxylase (GAD), islet tyrosine phosphatase 2 (IA2) or zinc transporter 8 (ZnT8). Stage 1 is characterized by presence of two or more autoantibodies and is highly predictive of evolution to type 1 diabetes (>80% of evolution to new onset clinical diabetes). Stage 2 is the stage that occurs in many individuals: presence of more than two autoantibodies, with loss of functional beta-cell mass that leads to dysglycemia, particularly upon challenge with a mixed meal or with an oral glucose load. This stage is what used to be called 'pre-diabetes' and is the stage where at present the FDA has approved teplizumab (Tzield®) for disease modification. A challenge however is to find the individuals at risk. Risk can be determined by genetic screening (neonatal, genetic risk score based on HLA genotypes; initiatives like GPPAD), antibody screening in higher risk populations (family members of people with type 1 diabetes; initiatives like INNODIA) or general population screening for presence of autoantibodies (children at moment of vaccination or other clinical visit; initiatives like the German Fr1da study). At the moment, intervention studies have only rarely moved to stages 1 or 2 because of the logistic demand of screening general population with most still targeting newly diagnosed people (stage 3). In order to be able to rescue as many functional beta-cells as possible, fast diagnosis and rapid intervention is needed. Still, the new concepts of type 1 diabetes diagnosis, moving towards a preclinical diagnosis open a new path of therapeutics and should be embraced rapidly by clinicians to allow prevention of clinical type 1 diabetes in the near future.

## L8. MAFLD: DO YOU AGREE?

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Fatty liver is a metabolic liver disease, associated with several other metabolic disorders, and it is considered as the hepatic coun-

terpart of metabolic syndrome. Its prevalence is increasing worldwide. In 2020 a new definition of fatty liver, MAFLD (metabolic dysfunction□associated fatty liver disease), has been proposed by an international expert panel, replacing the designation NAFLD, non-alcoholic fatty liver disease. The authors proposed a clear set of positive criteria (the presence of hepatic steatosis of ≥5% and at least one of the following criteria: overweight/ obesity; type 2 diabetes; or evidence of metabolic dysregulation, characterized by the presence of at least 2 factors amongst increased waist circumference, hypertriglyceridemia, low serum HDL-cholesterol levels, hypertension, impaired fasting plasma glucose, insulin resistance and chronic subclinical inflammation), leaving the formerly adopted exclusion diagnosis (NAFLD diagnosis requires hepatic steatosis of ≥5% without a secondary cause of liver injury or excessive alcohol consumption). However, the new definition of MAFLD is not yet globally accepted.

On one hand, MAFLD term more properly reflects the key nature of this disease. The diagnosis of MAFLD may coexist with other types of chronic liver disease, and the reported prevalence of its coexistence is high. So, rather than excluding such diagnosis, it is important to assess fatty liver regardless from other etiologies of chronic liver disease. In the research setting, the positive diagnostic criteria of MAFLD are less confounding for patient selection. Additionally, the negative prefix "non-" in NAFLD carries a perception that the disease is not as important as other causes of liver disease, and evidence suggests that trivialization arises through an inappropriate name of the condition. Also, the use of alcohol-associated terms in its denomination may associate to social stigma, which can induce fear and lead to adverse health behaviors.

On the other hand, MAFLD concept is based on empirical clinical practice and, therefore, scientific evidence is still required for the widespread use of this new definition. Also, evidence shows that the new definition has a low specificity for the detection of patients with a higher hepatocellular carcinoma risk, making the surveillance in this population challenging. Likewise, lean patients with hepatic steatosis may not be evaluable using current MAFLD criteria in the real world. Further, past clinical trials would need to be adapted when using the new definition.

Given the above information, future global consensus is needed, involving not only the international organizations for the study of liver diseases, but also other medical experts such as endocrinologists and primary care physicians. MAFLD criteria might need further development and adaptation.

## L9. CLINICAL CASE: A CHILD WITH X-LINKED HYPOPHOSPHATEMIA

Sofia Ferreira[1]

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***Introduction:*** X-linked hypophosphatemia (XLH) is caused by an inactivating mutation in the gene encoding phosphate-regulating endopeptidase homolog X-linked (*PHEX*), which leads to increased secretion of fibroblast growth factor 23 (FGF-23). Excess FGF-23 impairs renal phosphate reabsorption, which leads to hypophosphatemia and, consequently, rickets, osteomalacia and skeletal deformities.

In 2018, burosumab (a recombinant human IgG1 monoclonal antibody that targets FGF-23 was approved for pediatric patients with XLH.

***Case Report:*** A 2-year-old female child was referred to the pediatric endocrinology outpatient clinic due to broad-based gait with varus knees deformity. Her father had XLH and was treated with calcium and calcitriol with clinical and analytical improvement. On examination, she presented short stature (<3th percentile of length). Her blood analysis showed hypophosphatemia, elevated alkaline phosphatase and normocalcemia. She presented phosphaturia and radiographic evidence of rickets. A *PHEX* gene study was performed and revealed a c.1174-1G>A variant in heterozygosity, which was compatible with XLH diagnosis.

She was treated with Joulie's solution and active vitamin D until 2020. Since 2020, because of incomplete healing of rickets and residual skeletal deformity, persistent short stature and gastrointestinal side effects, she is treated with subcutaneous burosumab every 2 weeks.

Currently, the patient is 7 years old and her growth velocity increased (15th percentile of height). Serum phosphorus levels increased and serum alkaline phosphatase decreased, radiological changes improved and no side effects were reported.

## L10. X-LINKED HYPOPHOSPHATEMIC RICKETS: CLINICAL MANIFESTATIONS AND A CASE OF LATE DIAGNOSIS

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X-linked hypophosphatemic (XLH) rickets is the commonest inherited form of rickets. It results from the impaired regulation of FGF23 due to mutations in the *PHEX* gene. Elevated levels of FGF23 lead to decreased renal phosphate reabsorption and reduced 1α-hydroxylation of 25-hydroxyvitamin D [25(OH)D] to the active hormone calcitriol, resulting in impaired skeletal and dental mineralization.

Hypophosphatemia with increased renal phosphate wasting, normal levels of serum calcium, intact parathormone (PTHi) and 25-hydroxyvitamin D represent the major biochemical findings in affected patients. These findings, together with the evidence of clinical and radiological signs of rickets is suggestive for the diagnosis of XLH.

The characteristics and severity of XLH vary greatly between patients, with most usually developing clinical symptoms during the first or second year of life.

In children, the main clinical features included rickets, which occur only with open metaphyses, and osteomalacia, typically persisting throughout life, resulting in severe and progressive deformities of the lower limbs, disproportionate short stature, gait abnormalities and bone and muscular pain and weakness. Patients might present with dolichocephaly, dental abscesses and excessive dental caries. Extraskeletal complications include hearing loss and symptomatic Chiari malformations. The heavy burden of disease associated with XLH frequently result in psychosocial distress and reduced quality of life.

In undiagnosed adults, typical findings include short stature, osteomalacia, bone pain, osteoarthritis, pseudofractures, stiffness and enthesopathies.

XLH is a lifelong progressive disease; even treated adults suffer from both the consequences of childhood disease as well as ongoing disease manifestations, as conventional treatment with inorganic phosphate salts and active vitamin D ameliorates some, but not all the clinical manifestations.

Because of its rarity, the diagnosis and specific treatment of XLH are frequently delayed, which has a detrimental effect on patient outcomes.

Here we will describe a case of a 26-year-old men with a late diagnosis of XLH, presenting with multiple bone deformities from childhood, with debilitating bone pain, joint stiffness and severely impaired mobility. With this case we aim to raise awareness for this rare condition and highlight the importance of early diagnosis.

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## L11. X-LINKED HYPOPHOSPHATEMIA: THE ROLE OF BUROSUMAB

Adalbert Raimann[1]

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X-linked hypophosphatemic rickets (XLH, OMIN 307800) is a rare genetic disorder that results in skeletal hypomineralization, deformities, and short stature. The condition is caused by inactivating mutations in PHEX, leading to an increase in serum levels of the major regulator of phosphate homeostasis, fibroblast growth factor 23 (FGF23). This dysregulation results in renal phosphate depletion and profound chronic hypophosphatemia, the key disease mechanism in the development of rickets. While the clinical presentation of XLH can vary, skeletal symptoms typically appear in early childhood with the development of lower limb deformities after onset of walking. Short stature, chronic pain, enthesopathies and arthritis are among the scope of symptoms not only affecting children but causing severe impacts on quality of life for the patients' entire life. Managing patients with XLH can be challenging, as conventional therapies such as phosphate supplements and active vitamin D derivatives can improve skeletal symptoms but may also cause complications such as nephrocalcinosis and hyperparathyroidism.

Burosumab, a monoclonal antibody that inhibits FGF23, is the first targeted therapy for XLH patients. Clinical trials in both paediatric and adult populations have demonstrated a normalization of phosphate excretion and significant improvements in functional and pain scores.

With the approval of burosumab for children and adults with XLH a novel horizon in the treatment of this vulnerable patient cohort has become available.

## L12. ALL THE PATIENTS WITH HYPERPARATHYROIDISM SHOULD BE OPERATED RESULTS OF OUR CENTER

João Capela

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Primary hyperparathyroidism (pHPT) has a prevalence of 0.2%, but more than 90% of the cases are undiagnosed. It is traditionally characterized by hypercalcemia and elevated or inappropriately "normal" levels of parathyroid hormone (PTH). More recently, a new entity with isolated elevation of PTH, normocalcemic HPT, has emerged. The only definitive treatment for pHPT is parathyroidectomy (PTX), which is safe and cost-effective compared to conservative treatment and has a >95% cure rate if the surgeon is experienced. Elderly patients are less likely to be admitted for surgery, despite the excellent results reported in this age group. Of course, the potential benefits of curative treatment should not be outweighed by the risks of surgery or anesthesia.

Parathyroid adenomas are the most frequent cause of pHPT and, therefore, surgical treatment could evolve from the classic bilateral cervical approach to mini-invasive excision of the anomalous parathyroid, with consequent reduction in pain, scar size, morbidity, operative time, hospital costs and fibrosis that may condition future surgical interventions. The preoperative glandular location must be performed by ultrasound and scintigraphy and, eventually, by 4D computed tomography. We must remember, however, that this imaging is a procedure that only facilitates the surgical intervention and is not a diagnostic method of pHPT.

In 2022, at the The Fifth International Workshop on the Evaluation and Management of Primary Hyperparathyroidism, the following indications were defined:

- All patients with symptomatic pHPT.

Among those patients who are classified as asymptomatic:

- Serum calcium >1 mg/dL above the upper limit of normal
- Osteoporosis (T-score of less than -2.5 at any site: lumbar spine, hip, distal one third radius) or a vertebral fracture.
- Renal insufficiency (creatinine clearance less than 60 mL/min), kidney stones or nephrocalcinosis.
- Hypercalciuria (eg, >250 mg/day in women; >300 mg/day in men).
- Under 50 years of age.
- Patient's choice.

The indications for surgery of normocalcaemic pHPT are similar, but the results of the localization studies and surgery are worse, because a higher proportion of small adenoma and multiglandular disease have been reported.

Most series with experienced surgeons show very low surgical complication rates, including a 1%-2% incidence of permanent recurrent laryngeal nerve injury and a <1% risk of neck hematoma. Post-PTX hypoparathyroidism occurs in <10% of patients undergoing multiple gland resections.

After PTX, bone density increases and the incidence of neph-

rolithiasis decreases. The results of several studies have demonstrated a reduction in blood pressure and in the decline of renal function. Improvements in nonclassical aspects of pHPT are not so clear, so surgery cannot be recommended to improve neurocognitive function, quality of life and/or cardiovascular indices.

We studied the last 50 patients operated on in our service with a minimum follow-up of 12 months. There were 40 women and 10 men, with a mean age of 65 years (34-84). In a total of 51 operations, 48 were first operations and 3 were reinterventions. We perfomed 6 bilateral cervical explorations in cases with negative or discordant localization studies: 1 subtotal PTX in MEN1 patient, 3 biglandular in normocalcemic HPT and 2 uniglandular PTX. In the remaining 45 uniglandular resections, the approach was unilateral or selective with minicervicotomy. We only noticed a temporary vocal cord paralysis and 2 transitory hypoparathyroidisms. Three patients had persistent HPT, one of them already cured in a second PTX.

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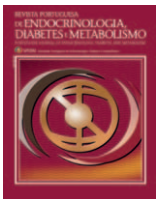

# E-Posters

**JURY OF AWARDS:**

**MANUEL PINHEIRO HARGREAVES AWARD**
Davide Carvalho, Cláudia Freitas, Maria João Matos
(EPosters: EP01 – EP07; EP16 – EP19; EP24 – EP27)

**LUIS MARQUES AWARD**
Luís Raposo, Elisabete Rodrigues, Joana Oliveira
(EPosters: EP20 – EP23)

**JOSÉ LUIS MEDINA AWARD**
Duarte Pignatelli, João Sérgio Neves, Luís Cardoso
(EPosters: EP08 – EP15)

## EP01. USEFULNESS OF CELLBLOCK PREPARATION IN FINE NEEDLE ASPIRATION FOR THE DIAGNOSIS OF THYROID NODULES

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[2] *Serviço de Anatomia Patológica, Hospital Distrital de Santarém*
[3] *Laboratório de Anatomia Patológica, Instituto de Patologia e Imunologia Molecular da Universidade do Porto | Instituto de Investigação e Inovação em Saúde (i3S) | Faculdade de Medicina da Universidade do Porto*

***Introduction:*** Thyroid nodules are common in the general population. The current diagnostic method is the ultrasound guided fine needle aspiration (US-FNA). The aim of the study was to evaluate the usefulness of cellblock preparation in addition to routine US-FNA in the diagnosis of thyroid nodules.
***Methods:*** A retrospective study of patients with thyroid nodules submitted to US-FNA, with collection of material using both smears and cellblock preparation. A pathologist reviewed the smears and cellblock slides of each case.
***Results:*** A total of 12 360 thyroid nodules were submitted to US-FNA. Cellblock was prepared in 153 (1.2%) in addition to smears. Among the satisfactory cellblocks (80.5%, 120), 31.7% (38) provided additional morphological information in comparison with smears. No significant differences were found between the smear and the combined smear and cellblock evaluation concerning the number of unsatisfactory (12.1% *vs* 11.4%, $p$=0.85) and indeterminate (27.5% *vs* 24.2%, $p$=0.52) results. Ten samples (6.7%) had their diagnosis changed after cellblock evaluation, nine of them due to immunohistochemistry that confirmed parathyroid origin in six cases.
***Conclusion:*** Cellblocks did not contribute to increase cellularity or to reduce indetermined results. Immunohistochemistry was essential to characterize rare cases without follicular histogenesis. Cellblock must only be prepared when considering immunohistochemistry.

## EP02. SECONDARY THYROID LYMPHOMA: A CASE REPORT

Catarina Gama[1], Margarida Oliveira[1], Carolina Antunes[1], Paula Calvo[1], Leonor Lopes[1], Miguel Quintela[2], Bernardo Marques[1], João Sequeira Duarte[1]

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***Introduction:*** Thyroid lymphoma is rare, accounting for 2% of all thyroid malignancies. Thyroid involvement secondary to a lymphoma of another location is even more uncommon (incidence of 0.2%). It usually presents as a rapidly growing thyroid mass, which might cause compressive symptoms. Classic B symptoms are less common.
***Case Report:*** We report a case of a 75-year female patient with history of stage IIIA follicular non-Hodgkin lymphoma (NHL). She did not undergo any treatment and was under active surveillance for 10 years. One year after discharge, she presented with a right cervical mass. Neck ultrasound showed an 18 mm solid, hypoechoic nodule, with microcalcifications on the right thyroid lobe and a level III right cervical adenopathy with 21 mm. Laboratory tests showed normal thyroid function. Fine needle aspiration cytology of the thyroid nodule and adenopathy revealed small lymphocytes and blasts, which were in favour of NHL recurrence. She performed a fluorodeoxyglucose-PET (FDG-PET), which revealed disseminated disease. Despite progression of disease, the patient remains asymptomatic and under active surveillance.
***Discussion:*** Although rare, thyroid involvement should be considered in patients with thyroid nodules and/or cervical adenopathies and personal history of lymphoma. Treatment of follicular NHL should be initiated only upon the development of symptoms.

## EP03. NEUROSARCOIDOSIS PRESENTING WITH PAN-HYPOPITUITARISM

Carolina Monteiro Antunes[1], Leonor Guia Lopes[1], Catarina Gama[1], Olga Capontes[2], Paula Calvo[1], Margarida Oliveira[1], Manuel Salavisa[3], Francisco Sousa Santos[1], João Sequeira Duarte[1]

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***Introduction:*** Hypothalamic-pituitary sarcoidosis is an uncommon form of neurosarcoidosis and is not usually its presenting manifestation.

***Case Report:*** We report the case of a 60-year-old-man with previous history of diabetes mellitus, sarcoidosis (cutaneous and musculoskeletal involvement), hypertension and cerebral vascular disease. He was taking metformin, dapagliflozin, detemir insulin, deflazacorte, telmisartan and hydrochlorothiazide. The patient presented to the emergency department (ED) with altered mental status associated with hypoglycemia. While in the ED he maintained recurrent episodes of hypoglycemia, despite IV glucose fluids, and developed a distributive shock being subsequently admitted to the intensive-care unit. He was started on IV hydrocortisone 50 mg qid with clinical improvement, however later developed hypernatremia and polyuria with low urinary osmolarity. Pituitary MRI revealed findings suggestive of hypothalamic and pituitary involvement by neurosarcoidosis. Laboratory analysis, after hydrocortisone withdrawal, showed TSH 0,767 µUI/mL, fT4 9.00 pmol/L, ACTH 11.2 pg/mL, cortisol 3.00 µg/dL, IGF-1 49.50 ng/mL, PRL 17 ng/mL, FSH <1.00 U/L, LH 1.08 U/L and total testosterone 7.1 ng/dL. After a multidisciplinary evaluation, the patient started methylprednisolone, levothyroxine, and desmopressin. Methotrexate was then initiated and the patient was discharged to a rehabilitating facility.
***Conclusion:*** Hypopituitarism is a rare manifestation of neurosarcoidosis and endocrinological assessment and follow-up are essential.

## EP04. CONGENITAL DISORDER OF GLYCOSYLATION AND THE IMPORTANCE OF AN ENDOCRINOLOGY APPROACH: A CASE REPORT

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***Introduction:*** Congenital disorders of glycosylation (CDG) are a group of rare metabolic diseases characterized by a defect in the protein glycosylation process. It can affect various tissues and organs and hormonal abnormalities are frequent.
***Case Report:*** An 18-year-old girl was referred to the Endocrinology department due to delayed development of secondary sexual characteristics and primary amenorrhea. She had a history of CDG type 1, diagnosed at 1 year, in the context of delayed psychomotor development. As a consequence, she also presented a prothrombin VII deficit. At observation, her height was 169.6 cm, her weight 51 kg and her BMI 18 kg/m$^2$. The breast development was at Tanner stage 1, pubic hair development at Tanner stage 3 and had no axillary hair. The hormonal profile confirmed hypergonadotropic hypogonadism. Pelvic ultrasound showed no changes in the ovaries or uterus. DEXA scan was compatible with severe osteoporosis. She started pubertal induction with transdermal 7ß-oestradiol and after her first vaginal bleeding, it was introduced vaginal cyclic progesterone.
***Conclusion:*** This case reports a late referral to Endocrinology despite an early diagnosis of a rare syndrome. The initiation of estrogen replacement therapy earlier could have prevented the psychological consequences and the onset of severe osteoporosis.

## EP05. CARDIOVASCULAR AND METABOLIC RISK FACTORS IN PATIENTS WITH AUTONOMOUS CORTISOL SECRETION

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***Introduction:*** Adrenal incidentaloma is a mass discovered on imaging not performed for that purpose. Possible autonomous cortisol secretion (pACS) or ACS are common findings, that can be associated with metabolic disorders and increased incidence of cardiovascular events.
Our objective was to compare comorbidities related to cortisol excess, and cardiovascular events between patients with non-functional adenoma (NFA) and patients with pACS/ACS.
***Methods:*** Retrospectively, we analyzed patients followed at our endocrinology clinic, diagnosed with adrenal incidentaloma that performed 1mg dexamethasone test. pACS and ACS were defined by cortisol 1.8-5 and >5 µg/dL, respectively.
***Results:*** We enrolled 70 patients, 44 NFA, 25 pACS and 1 ACS. Mean BMI was 30±4 in NFA and 30±8 kg/m$^2$ in pACS/ACS. Prevalence of hypertension was 73% and 85%; $p$=0.25 (mean age diagnosis 56±14 *vs* 54±10; $p$=0.65, controlled with a mean of antihypertensive drugs 1.7±1.2 *vs* 2±1.4; $p$=0.32), type 2 diabetes 39% and 50%; $p$=0.56 (mean age diagnosis 61±10 *vs* 60±10; $p$=0.79), dyslipidemia 80% and 88%; $p$=0.34, in NFA and pACS/ACS respectively. Prevalence of myocardial infarction was 9% and 8 %; $p$=0.6; ischemic stroke 5% and 11%; $p$=0.26 in NFA and pACS/ACS respectively.
***Conclusion:*** Although not statistically significant, patients with pACS/ACS tend to have more comorbidities, that can be more difficult to control, as well as a higher prevalence of ischemic stroke.

## EP06. CORTISOL CO-SECRETION FROM ALDOSTERONE-PRODUCING ADENOMA: CLINICAL CASE

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A 61-year-old man, with metabolic syndrome (poorly-controlled hypertension, diabetes mellitus and obesity) and previous diuretic-induced hypokalemia, was referred because of bilateral adrenal nodules: 43 mm in the left adrenal gland (AG); 27 mm and 20 mm in the right AG. The patient was on four anti-hypertensive medications (including a diuretic).
He was diagnosed with primary hyperaldosteronism based on hypokalemia and raised aldosterone-renin ratio: renin < 0.78 pg/mL (1.0-8.2); aldosterone 321.5 pg/mL (42-201.5). Urinary metanephrines were normal. Mild autonomous cortisol secretion was

observed: cortisol level after 1mg dexamethasone (DXA) suppression test > 4 ug/dL, suppressed ACTH (<5 pg/mL), normal urinary cortisol (11.1 ug/24h), and salivary cortisol with rhythm. Further testing with DXA-CRH test confirmed endogenous hypercortisolism.
Adrenal vein sampling was considered, but was deemed unnecessary due to the possible co-secretion of aldosterone and cortisol. Iodine-131 MIBG scintigraphy was performed, which confirmed uptake on the left adrenal gland. The patient underwent left adrenalectomy, and histopathology revealed cortical adenoma with cells mostly composed of zona glomerulosa and fasciculata.
Prior to discharge, the patient was prescribed glucocorticoids (currently tapering) and his hypertension and diabetes seems to be improving (currently two anti-hypertensive drugs). Follow-up with aldosterone-renin ratio and DXA suppression test (pending) will confirm the efficacy of the surgery.

## EP07. DIABETIC KETOACIDOSIS IN THE ELDERLY: A NEAR-FATAL OUTCOME

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***Introduction:*** Diabetic ketoacidosis (DKA) is a serious complication of diabetes, often triggered by infections or inappropriate use of insulin. It is more common in type 1 diabetes (T1D) and is characterized by hyperglycemia, ketonemia and metabolic acidosis.
***Case Report:*** A 83-year-old man brought to the emergency department for hyperglycemia, hypothermia and vomiting. Partially dependent patient, living alone, with T2D, treated with basal-bolus insulin therapy, with difficulties with compliance. He had HIGH capillary blood glucose, 7.0 mmol/L ketonemia and severe metabolic acidosis (pH 6.96; 5.5 mmol/L bicarbonate). He performed 10 units of glulisine and started fluid therapy, insulin perfusion and bicarbonate administration. The analytical study revealed glycemia of 1167g/dL and elevation of inflammatory markers, with right pneumonia on chest x-ray. Five hours after admission, he initiated respiratory distress and desaturation, which was followed by two episodes of cardiac arrest at a non-shockable pace, both with recovery after life advanced support.
***Conclusion:*** This case draws attention to the true emergency that DKA constitutes, as it can have a fatal outcome. It also highlights that, although more common in T1D, it can occur in elderly people with T2D, which are more predisposed to infections and therapeutic noncompliance, the main precipitating factors, that can coexist.

## EP08. PREDICTORS OF READMISSION AND MORTALITY IN PATIENTS WITH DECOMPENSATED HEART FAILURE ACCORDING TO GLYCAEMIC STATUS

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***Introduction:*** This study aimed to identify predictors of rehospitalization and all-cause mortality in patients with normoglycaemia, prediabetes, and diabetes admitted with decompensated heart failure (HF).
***Methods:*** A retrospective cohort study was conducted on patients admitted with HF during the first 6 months of 2015 (n=311). Clinical, demographic, and laboratory variables were evaluated, with the primary outcome being the composite of time to readmission for HF or all-cause mortality.
***Results:*** The average age was 77.8±10.8 years, with 44.1% being male. Sixteen percent had normoglycaemia, 19% prediabetes, and 65% diabetes. Older age, higher New York Heart Association (NYHA) class, atrial fibrillation (AF), lower total cholesterol levels, and use of loop diuretics and mineralocorticoid antagonists were predictors of the primary outcome. Glycemic status did not significantly predict the composite outcome. The risk of readmission or mortality was higher among women with normoglycaemia and among patients with AF with normoglycaemia or diabetes (but not prediabetes).
***Conclusion:*** Irrespective of glycemic status, older patients with higher NYHA class, AF diagnosis, and lower total cholesterol levels are at increased risk of readmission for HF or mortality. Female sex predicted higher risk only in normoglycaemia.

## EP09. RELATIONSHIP BETWEEN PROBLEMATIC HYPOGLYCAEMIA AND HEALTH STATUS, GLOBAL COGNITION AND EXECUTIVE FUNCTIONS IN ADULTS WITH TYPE 1 DIABETES ATTENDING A TERTIARY DIABETES SERVICE IN PORTUGAL

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***Introduction:*** To explore relationships between impaired awareness of hypoglycaemia (IAH) and severe hypoglycaemia (SH) and health status and cognition in type 1 diabetes (T1D).
***Methods:*** Adults with T1D completed Portuguese-language Gold score (GS) and Minimally Modified Clarke Hypoglycaemia Survey (MMCHS), ≥4 in either indicating IAH; Diabetes Health Profile (DHP) assessing health status; Montreal Cognitive Assessment (MoCA) and INECO Frontal Screening (IFS) measuring global cognition and executive functions (EF). EF data were also compared with data from a non-diabetic population database.
***Results:*** In 190 participants (age and diabetes duration 38±13 and

20±11 years respectively), prevalence of IAH by GS, by MMCHS awareness factor 1 (≥2 = IAH) and SH by MMCHS factor 2 were 24%, 29% and 37%. Participants with SH and/or IAH by MMCHS factor 1 had worse DHP total and psychological distress scores. Participants with SH had lower barriers to activity. Neither SH nor IAH were related to cognitive impairment. T1D was related to worse performance on IFS.
***Conclusion:*** These data suggest that while T1D may be associated with impairments of higher cognitive processes, this is not driven by problematic hypoglycaemia. However, SH is associated with behavioural dysfunction and both SH and/or IAH are related to emotional distress.

## EP10. ASSOCIATION OF GLYCEMIC VARIABILITY AND TIME IN RANGE WITH LIPID PROFILE IN TYPE 1 DIABETES

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***Introduction:*** Hyperglycemia is associated with lipoproteins' abnormalities. The study aimed to analyze the association of glycemic control with lipid profile, focusing on glycemic variability and time in range obtained from continuous glucose monitoring (CGM), in patients with type 1 diabetes (T1D).
***Methods:*** A retrospective cohort was performed in patients with T1D, analyzing clinical parameters, HbA1c, CGM and lipid profile in cross-sectional (n=242) and longitudinal (n=90) analyses.
***Results:*** The mean age of the study population was 36.6 ± 12.6 years, 51.7% were male, and the mean diabetes duration was 16.8 ± 10.3 years. In the cross-sectional analysis, higher HbA1c, higher glucose management indicator (GMI), higher time above range and lower time in range were associated with higher triglycerides. In the longitudinal analysis, an increase in time below range was associated with a decrease of HDL cholesterol. In both analyses, an increase in the coefficient of variability (CV) was associated with a significant decrease of HDL cholesterol.
***Conclusion:*** We observed a negative association between CV and HDL cholesterol levels and a positive association between hyperglycemia and triglyceride levels, suggesting that CGM parameters may be helpful to guide the improvement of glycemic control and lipid profile in T1D.

## EP11. METABOLIC SYNDROME IS A MAIN DETERMINANT OF QUALITY OF LIFE AFTER METABOLIC SURGERY

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***Introduction:*** Obesity is a major health problem, affecting over 800 million adults worldwide, and represents a major risk factor for developing metabolic syndrome. Bariatric surgery, besides significant weight loss, has provenly been responsible for the remission of obesity-related diseases. Our aim is to evaluate postoperative quality of life (QoL) and its association with metabolic syndrome.
***Methods:*** In a specialized bariatric unit, 454 patients were recruited during postoperative follow-up appointments and answered the "Moorehead-Ardelt II questionnaire" for evaluation of QoL. Patient's data were retrieved retrospectively, using patient's electronic records.
***Results:*** Postoperative QoL was significantly better in the metabolically healthy patients at the time the assessment occurred ($p$=0.016). Within pre-operative metabolically unhealthy patients, QoL is better in the subgroup that suffered changes in their metabolic status and has ≥3 years of follow-up ($p$=0.029). The long-term follow-up subgroup (≥3 years) has consistently better results in the QoL score in every factor evaluated.
***Conclusion:*** Having metabolic syndrome after undergoing metabolic surgery appears to be related to worse quality of life. Change in metabolic status has a delayed effect on quality of life, with benefits appearing after 3 years of follow-up. Metabolic syndrome is a main determinant of QoL, especially when analyzing longer follow-up times.

## EP12. GLYCEMIC CONTROL AND METABOLIC PARAMETERS IN CHILDREN AND ADOLESCENTS WITH TYPE 1 DIABETES

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***Introduction:*** The association between glycaemic control and metabolic status is poorly defined in children and adolescents with T1D, besides being biologically plausible. We aimed to evaluate the association between glycaemic control and several metabolic parameters in children and adolescents with T1D.
***Methods:*** Observational cross-sectional study including children and adolescents (5-18 years-old) followed in our outpatient clinic with the diagnosis of T1D for over a year. We used linear regression models to evaluate the association between glycated haemoglobin (A1c) and time in range (TIR), and prespecified metabolic parameters, demographic and clinical characteristics.
***Results:*** A total of 144 patients were included, 51% being female. The population had a mean age of 12.7±3.4 years old. We report a positive association between A1c and BMI, systolic and diastolic blood pressure, total- and LDL-cholesterol and triglycerides. Females and patients diagnosed at a younger age presented with higher A1c values. There is a tendency for a negative association between TIR and the former parameters. Higher A1c levels and lower TIR associated with higher glycemic variability and were treated with higher basal insulin/kg dose.
***Conclusion:*** Our results support an association between a worse glycemic control and an unhealthier metabolic profile in children and adolescents with T1D.

## EP13. QUALITY OF LIFE IN THYROID CANCER PATIENTS AND THE INFLUENCE OF CLINICO-PATHOLOGICAL CHARATERISTICS

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***Introduction:*** Differentiated thyroid carcinoma (DTC) usually has a good prognosis, but the quality of life (QoL) of these patients is impaired.
Our objective was to assess the QoL of patients with DTC and understand its relationship with clinicopathological characteristics.
***Methods:*** Cross-sectional and observational cohort study, using questionnaires to assess QoL of patients with DTC (EORTC-QLQ-C30 and EORTC-QLQ-THY34).
***Results:*** We included 69 patients, mainly female (78.3%) with papillary thyroid carcinoma (87%) and submitted to total thyroidectomy (92.8%): 17% developed permanent hypoparathyroidism, 11.6% transient hypoparathyroidism, and 4.4% dysphonia. Patients who underwent hemithyroidectomy did not developed these complications. Lymphadenectomy was performed in 10 patients; among them, frequency of permanent ($p$=0.001) and transient ($p$=0.012) hypoparathyroidism was higher. The median global score for the EORTC-QLQ-C30 was 74.5% and for the EORTC-QLQ-THY34 was 78.4%. Dysphonia was associated with lower QoL ($p$=0.023). Permanent hypoparathyroidism was not associated with QoL, but the median global score of EORTC QLQ-THY34 is lower in these patients (77.0 *vs* 79.4). Distant metastases were associated with lower QoL ($p$=0.014).
***Conclusion:*** Treatment of DTC, namely the type of surgery, may have a significant impact on the patient's QoL. The presence of distant metastasis is the clinicopathological characteristic more associated with QoL.

## EP14. ICPI-RELATED THYROID FUNCTION ALTERATION, COURSE AND MONITORING: A RETROSPECTIVE STUDY

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***Introduction:*** Immune checkpoint inhibitors (ICPI) enhance immunological response and they're associated with multiple autoimmune side effects. Among these, autoimmune thyroiditis (AIT) stands out due to its high prevalence.
We aimed to evaluate the clinical course in patients with ICPI-related AIT and the need for posterior therapy.
***Methods:*** We conducted a retrospective data analysis of patients with ICPI-related AIT, without previous thyroid dysfunction, followed at IPO-Porto between March/2021 and December/2022.
***Results:*** We included 100 patients; 57 patients presented an initial thyrotoxicosis phase and 27 (47%) of those had clinical thyrotoxicosis, with elevated fT4 or fT3. Hypothyroidism developed in 75 patients and 56 (76%) of them needed long-term supplementation with levothyroxine. Thyrotoxicosis group showed a median time to develop thyroid function alterations of 5 (3-9) weeks; the group who developed hypothyroidism, a median time of 7 (4-18) weeks; the thyrotoxicosis group had thyroid function alterations significantly earlier than the hypothyroidism group, $p$>0.01. Also, 95% of patients who developed thyrotoxicosis, did so before 25 weeks. We could not find a correlation between thyrotoxicosis magnitude (lowest TSH and the maximum fT3 and fT4 values) and posterior need of levothyroxine adjusted to weight (X2of 0.04, 0.01, and 0.01, respectively).
***Conclusion:*** ICPIs are a frequent cause of iatrogenic AIT, with a significant proportion of patients needing long-term levothyroxine therapy. The patients need frequent monitoring of thyroid function and comprehensive approach of this endocrine adverse effect.

## EP15. THYROID DYSFUNCTION AND HEART FAILURE WITH PRESERVED EJECTION FRACTION: A SYSTEMATIC REVIEW

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Thyroid hormones (TH) play a major role in cardiovascular homeostasis. Thyroid dysfunction (TD) has been linked with diastolic dysfunction. The link between TD and HFpEF is poorly determined. Our aim is to review the evidence regarding the relation between TD and HFpEF.
A literature search was performed on the PubMed, Web of Science and ClinicalTrials.gov databases. A total of 107 articles was retrieved. Nine studies were included. Data were extracted using a predesigned form.
Several studies found evidence of TD being linked with HFpEF. TD was more prevalent in patients with HFpEF than in patients with heart failure (HF) without preserved ejection fraction. TD was also linked with a worse prognosis in patients with HFpEF. Lower TH levels were correlated with echocardiographic changes, higher B-type natriuretic peptide (BNP) and worse clinical outcomes in patients with HFpEF. Subclinical hypothyroidism (SCH), low T3 syndrome and thyrotoxicosis were specifically found to be related with the development and progression of HFpEF.
Conclusive evidence shows that TD seems to be an important etiologic and prognostic factor in HFpEF. Nevertheless, more studies are required to firmly establish this relation. It is predictable that new therapeutic and prophylactic strategies can arise from this comprehensive view of HFpEF.

## EP16. SPONTANEOUS PREGNANCY IN A WOMAN WITH HYPOGONADOTROPIC HYPOGONADISM: A CASE REPORT

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***Introduction:*** Female patients with a history of craniopharyngioma often experience amenorrhea and infertility due to hypogonad-

otropic hypogonadism (HH). Consequently, successful pregnancy in these patients is rarely achieved.
In the largest published study about fertility in craniopharyngioma patients, only six out of 133 women who had undergone craniopharyngioma ressection became successfully pregnant; all pregnancies in patients with HH were achieved through assisted reproductive techniques.
***Case Report:*** A 40-year-old woman was diagnosed with craniopharyngioma at nine years old. She underwent neurosurgery with complete tumoral ressection and, as a result, developed hypopituitarism, including secondary hypothyroidism, central diabetes insipidus, growth hormone deficiency and HH.
Due to HH, she had always been in amenorrhea but had not been treated with estroprogestative drugs. However, she became naturally pregnant at 38 years old. During pregnancy, levothyroxine and desmopressin doses were adjusted, there were no complications and she delivered a healthy baby.
After the delivery, she was not able to breastfeed and the amenorrhea persisted, therefore she started an estroprogestive pill.
***Conclusion:*** This is, to our knowledge, the only reported case of a natural successful pregnancy in a craniopharyngioma patient with HH. We presume that an occasional rise in gonadotropin levels triggered ovulation, making this pregnancy possible.

## EP17. TIME IN RANGE AND COMPLICATIONS OF DIABETES: A CROSS-SECTIONAL ANALYSIS OF PATIENTS WITH TYPE 1 DIABETES

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***Introduction:*** Our objective was to evaluate the association of CGM-parameters and HbA1c with diabetes complications in patients with type 1 diabetes (T1D).
***Methods:*** Patients with T1D using CGM-system Freestyle-Libre were included in this analysis. The association of CGM-metrics and HbA1c with diabetes complications was assessed using logistic regression unadjusted and adjusted for age, sex, and diabetes duration (model 1), and further adjusted for hypertension and dyslipidemia (model 2).
***Results:*** 161 patients with T1D were included. The median T1D duration was 17.7±10.6 years. Time in range (TIR) was associated with any complication and microvascular complications in the unadjusted and in the adjusted models. TIR was associated with retinopathy in the unadjusted model as well as in model 1, and was associated with macrovascular complications only in the unadjusted model. HbA1c was associated with any complications, microvascular complications, and retinopathy in the unadjusted model but not in the adjusted models. HbA1c was associated with macrovascular complications in unadjusted model and in model 1.
***Conclusion:*** In this cross-sectional analysis of patients with T1D using CGM, TIR, and HbA1c were associated with diabetes complications. TIR may be a better predictor than HbA1c of any complication and microvascular complications, while HbA1c may be a better predictor of macrovascular complications.

## EP18. GALACTOSEMIA: A RARE CAUSE OF HYPERGONADOTROPIC HYPOGONADISM

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***Introduction:*** Hypergonadotropic hypogonadism (HH) is characterized by failure of the gonads to respond to pituitary gonadotropins, leading to impaired gonadal function. Female HH can result from various causes, including genetic syndromes, chromosomal abnormalities, autoimmune disease, surgery, or chemotherapy/radiation exposure. Galactosemia is a rare disorder (1/40 000 to 1/60 000 annual incidence in Western countries) caused by an enzyme deficiency (Gal-1-P uridyl transferase) in the major galactose metabolism pathway. HH is common in female patients with galactosemia.
***Case Report:*** A female patient, born to consanguineous parents, was diagnosed with galactosemia during the neonatal period. At the age of 13, she was referred to a pediatric endocrinology appointment in our hospital due to delayed pubertal development. Upon physical examination, external female genitalia were observed, and there was no breast development (Tanner stage 1). Pelvic ultrasound revealed normal ovaries and uterus. Laboratory tests showed hypergonadotropic hypogonadism, attributed to galactosemia diagnosis. Treatment with transdermal estrogen was initiated to induce puberty, followed by oral contraceptives.
***Conclusion:*** Galactosemia is a rare cause of HH with an unknown etiology. It has been admitted that galactose or its metabolites may be toxic to the ovarian parenchyma. Clinical surveillance includes screening for ovarian function abnormalities at an early age and treatment consists of estrogen/progesterone supplementation.

## EP19. A UNIQUE CASE OF SUBCENTIMETRIC PAPILLARY THYROID CARCINOMA WITH DISTANT METASTASIS: WHAT CAN WE LEARN FROM THAT?

Diogo Ramalho[1], Andreia Amado[1], Elisabete Teixeira[2], Sule Canberk[2], Henrique Carmona[1], Helena Alves[1], Barbara Castro[1], Hugo Pereira[1], João Varandas[1], Susana Graça[1], Carlos Soares[1], Paula Soares[2], Manuel Sobrinho Simões[2], Andreia Póvoa[1]

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***Introduction:*** Subcentimetric papillary thyroid carcinomas (SPTC) are typically indolent tumors, but when lymph node metastases(LNM) are the initial presentation in older patients, vital prognosis worsens.
***Case Report:*** In 2011, a 61-year-old man evidenced a cervical mass whose biopsy identified a SPTC LNM. He underwent total thyroidectomy with central and ipsilateral lymphadenectomy. Histopathology identified a 2 mm follicular variant of SPTC and

LNM in 25/25 lymph nodes. He underwent 150 mCi of radioactive iodine (RAI), followed by levothyroxine suppressive therapy. In 2016, a retro-tracheal mass was diagnosed, suggesting local recurrence. The patient was submitted to surgical excision and RAI therapy (120 mCi). In 2019, due to seizures, he performed a brain CT that detected brain metastases. The main lesion was submitted to debulking surgery and histopathology analysis confirmed a classical SPTC and features displaying follicular, hobnail and tall cell. Molecular analysis revealed only BRAFV600E in the LNM at presentation and BRAFV600E and TERT promoter mutations in the recurrent LNM and brain metastasis.
***Conclusion:*** Although of exceptional occurrence, brain metastases point towards aggressive phenotypic features. Patient-risk stratification of SPTC based on histopathological and genetic analysis may have a major impact on patient prognosis through the assessment of therapeutic biomarkers, and prediction of disease progression and survival.

## EP20. QUALITY OF LIFE IN PATIENTS WITH METABOLIC SYNDROME – THE WORSE, THE WORST?

Marta Borges-Canha[1,2], Joana Chaves[1], Ana Rita Leite[1,2], Rodrigo Liberal[3], Inês Lourenço[1], Madalena Von Hafe[1], João Sérgio Neves[1,2], Mariana Fragão-Marques[1,4], Pedro Pimentel-Nunes[1], Adelino Leite-Moreira[1], Davide Carvalho[2,5], Paula Freitas[2,5]

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***Introduction:*** Metabolic syndrome (MetS) is associated with sociopsychological diseases. It remains to be established whether MetS components are associated with QoL in these individuals. Aim: To evaluate the association between QoL of patients with MetS and prespecified metabolic parameters.
***Methods:*** Cross-sectional study including patients from microDHNA cohort. This cohort includes patients with MetS, 18-75 years-old. The recruitment includes filling out a QoL questionnaire (Short-Form Health Survey, SF-36), physical examination, blood sampling and hepatic elastography. We used linear regression models to evaluate the associations between QoL domains and prespecified parameters.
***Results:*** We included a total of 65 participants (53.8% female, average age 61.2 ± 9.6 years-old). Patients with higher BMI and hip circumference had worst scores regarding physical functioning. The ones with lower HDL-cholesterol had worst results regarding general health perception. Higher Fatty Liver Indexes (predictor of hepatic steatosis) associated with worst scores on energy, role limitations due to physical health and general health perception. Higher values of Controlled Attenuation Parameter (assesses hepatic steatosis) associated with worst QoL concerning role limitations due to emotional problems, emotional well-being, social functioning, and general health perception.
***Conclusion:*** Our data supports an association between worst QoL and a poorer metabolic profile in patients with MetS.

## EP21. EFFECTIVENESS OF GLICAZIDE IN TREATING MODY 13: A CASE REPORT

Juliana Gonçalves[1,2], Helena Ferreira[1,2], Selma B. Souto[1,2], Jorge Pedro[1,2], Paula Freitas[1,2]

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***Introduction:*** Maturity-onset diabetes of the young (MODY) is a rare type of diabetes accounting for 1% to 2% of cases and is often underdiagnosed. The importance of its diagnosis lies in the implications it can have on disease management and offspring.
***Case Report:*** A 24-year-old Caucasian woman with no relevant past medical history and normal weight (BMI 22.8 kg/m$^2$) was referred to an Endocrinology appointment due to newly diagnosed diabetes mellitus. Her family history was relevant for diabetes in three grandparents with diagnosis after the age of 50. Blood tests due to fatigue and polydipsia revealed hemoglobin A1c of 10.7%, fasting glucose of 278 mg/dL, and negative anti-GAD and anti-insulin antibodies. The medical team initiated insulin therapy, and requested C-peptide analysis, which showed normal levels (2.75 ng/mL). Medical records from the primary care revealed, eleven years before, pre-diabetes (hemoglobin A1c 6.2%). Genetic test identified a mutation c.679G>A, p.(Glu227Lys) in the *KCNJ11* gene, compatible with MODY 13 diagnosis. Gliclazide 60 mg was started, and basal-bolus insulin was discontinued. Excellent glycemic control was achieved (TIR 100%, %CV 13.9% and GMI 5.9%), and the patient reported a significant improvement in quality of life.
***Conclusion:*** MODY 13 is an extremely uncommon subtype of MODY, with only a few reported cases worldwide. Studies showed that sulfonylurea is safe and effective for long-term use in children with neonatal diabetes caused by a mutation in the *KCNJ11* gene. With the correct diagnosis and treatment, patients can achieve excellent glycemic control and improve their quality of life.

## EP22. HYPOCALCAEMIA IN PRE-EXISTING HYPOPARATHYROIDISM AFTER BARIATRIC SURGERY - A CHALLENGE FOR CLINICAL MANAGEMENT.

Inês Meira & João Menino[1], Sara Ribeiro[1], Ana Varela[1], Paula Freitas[1]

[1] *Centro Hospitalar Universitário de São João*

***Introduction:*** Treatment of hypoparathyroidism relies on gastrointestinal absorption of calcium and calcitriol. Information on postoperative management of bariatric surgery in patients with established hypoparathyroidism remains scarce.
***Case Report:*** A 67-year-old woman underwent a sleeve gastrectomy for treatment of obesity. She had a past history of postsurgical hypoparathyroidism, previously managed with oral elemental calcium (elemCa) 2.4 g/day and calcitriol 1 µg/day. She was admitted with a total serum calcium (TCa) of 8.7 mg/dL. She presented with postsurgical hypocalcemia and elemCa 3.2 g/day and calcitriol 1 µg/day were started. On day 2 post surgery, TCa reached a nadir of 6.6 mg/dL and add-on infusion of calcium and increasing doses of elemCa were needed. Several dose adjustments were re-

quired, with fluctuating levels of TCa. After 12 days, calcium infusion was interrupted and oral treatement was increased up to 7.2 g/day of elemCa, 2 µg/day of calcitriol and 3 mg of indapamide. She was discharged after 19 days, with a TCa of 8.9 mg/dL, on an increased dose of calcitriol (2 µg/day) and elemCa 5 g/day. She remained normocalcaemic 2 weeks after discharge.
***Conclusion:*** Recalcitrant postoperative hypocalcemia should be anticipated in patients with pre-existing hypoparathyroidism undergoing bariatric surgery, regardless of surgical approach.

## EP23. ECTOPIC ACTH SYNDROME – A CASE SERIES

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***Introduction:*** Ectopic ACTH secretion accounts for 10%-20% of endogenous Cushing's syndrome (CS). More often its source is located to the lung, associated with highly malignant tumors or with less aggressive variants of neuroendocrine tumors.
***Methods:*** We retrospectively reviewed the inpatient and outpatient records of patients seen at the endocrinology department of a tertiary hospital over the last 20 years.
***Results:*** A total of 12 patients were included (5 men and 7 women). The median age at diagnosis was 45 (32-75) years. Seven patients had lung carcinoids (LC), one had a medullary thyroid carcinoma (MTC), one a parotid carcinoma, one a small cell carcinoma (SCC), two patients had an occult source of ACTH (one is still under investigation). Four patients presented with metastatic disease at diagnosis; in one patient metastasis were observed months after diagnosis. The most common clinical features at presentation were de novo or aggravated hyperglicemia, hypokalemia and lower leg edema. Two patients had substantial weight loss (SCC and one occult).
The median 8 am ACTH levels was 108.85 ng/L (range 32.2-698) favoring ACTH-dependent CS. The median 8 am cortisol was 45.3 ug/dL (range 28-140.3) and the median 24 hour urinary free cortisol was 3209 ug/day (range 436.4-6210).
Seven patients undergone surgical intervention to address the primary tumor with five achieving a sustained cure on follow up (all LC). Three patients died of the primary disease (SCC, parotid carcinoma, MTC), one of unrelated causes (LC).
Two of the patients required bilateral adrenalectomy for uncontrolled CS.
***Conclusion:*** In this work we will review the diversity of clinical features at presentation, strains in tumour location and response to therapy.

## EP24. HYPONATREMIA – SHOULD WE CALL THE ENDOCRINOLOGIST?

Sara Gil-Santos[1], Raquel Calheiros[1], Pedro Souteiro[1], Joana Oliveira[1], Isabel Inácio[1], Ana Paula Santos[1], Isabel Torres[1]

[1] *Instituto Português de Oncologia Francisco Gentil do Porto*

***Introduction:*** Among the many causes of hyponatremia, SIADH is one of the most challenging to diagnose and treat.
***Case Report:*** A 54-year-old male, with a history of laryngeal squamous cell carcinoma, submitted to surgery and radiotherapy in 2016, in remission since then. He was admitted to our hospital due to hemoptysis and hyponatremia (115 mmol/L), which prompted an Endocrinology referral. He was clinically euvolemic, hyperglycemia, thyroid disease and adrenal insufficiency were excluded, and lab tests revealed plasma and urinary osmolality of 244 and 328 mOsmol/Kg, respectively, and urinary sodium of 80 mmol/L. The diagnosis of SIADH was made and the patient was discharged on water restriction, increased salt intake, furosemide and urea (30 mg 3id). At the first Endocrinology Clinic visit, the sodium levels had improved (128 mmol/L). In this context, an ectopic ADH source was suspected and a neck CT was requested which showed no signs of laryngeal cancer recurrence, but a 34x21 mm mediastinal mass was detected. An $^{18}$F-FDG PET/CT was then performed revealing pulmonary malignant lesions with extensive lymph nodes and bone metastasis.
***Conclusion:*** Endocrinologists have a major role in approaching hyponatremia and understanding its etiology. In this case, the SIADH work-up led to the diagnosis of a hidden stage IV tumor.

## EP25. TYPE 3 AUTOIMMUNE POLYGLANDULAR SYNDROME: A RARE COMBINATION OF MULTIPLE AUTOIMMUNE MANIFESTATIONS

Sofia Monteiro Lopes[1], Mafalda Ferreira[1], Tânia Carvalho[1], Luísa Ruas[1], Leonor Gomes[1], Isabel Paiva[1]

[1] *Centro Hospitalar e Universitário de Coimbra*

***Introduction:*** Autoimmune polyglandular syndromes (APS) comprise endocrine autoimmune disorders that occur in subjects with immune dysregulation. Type 3 APS (APS-3) consists of thyroid autoimmune disease (TAD) with another autoimmune illness, excluding Addison's disease, hypoparathyroidism or mucocutaneous candidiasis. Few patients have been described with more than two autoimmune diseases complicating TAD.
***Case Report:*** We report a case of an 18 year old female, in which skin depigmentation at 3 years of age advanced vitiligo diagnosis. When she was 8y, TAD with hypothyroidism was diagnosed: TSH 22. 5 mU/L, FT4 8.3 pmol/L, positive anti-thyroid antibodies and thyroid heterogeneous echostructure on ultrasound. Later, she presented with polyuria and polydipsia. Water deprivation test confirmed central diabetes *insipidus*. MRI detected stalk thickening and neurohypophyseal signal loss. Anti-pituitary autoantibodies are pending. Recent evaluation identified sideropenic anemia. Autoimmune gastritis (AG) testing revealed positive anti-parietal cell antibodies. She is waiting gastric biopsy. Celiac disease screening was negative. Regular A1C and glycaemia have been normal.
***Conclusion:*** This patient is suspected of having four autoimmune conditions, which compound a rare presentation of APS-3. The anticipation of multiple autoimmune glandular involvement enabled autoantibody and hormonal evaluation leading to early replacement therapy. Long-term follow-up is mandatory, as the onset age of associated conditions is unpredictable.

## EP26. POSACONAZOLE-INDUCED PSEUDOHYPERALDOSTERONISM – A CLINICAL CASE

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***Introduction:*** Posaconazole is commonly used for prophylaxis and treatment of fungal infections. Similar to ketoconazole, used in Cushing's syndrome for hypercortisolism, it can interfere with the steroid synthesis pathway leading to a syndrome of apparent mineralocorticoid excess.
***Case Report:*** A 69 year-old man diagnosed with lung adenocarcinoma is admitted for persistent purulent cough, weight loss and anorexia. Blood tests showed anemia and leukocytosis, normal renal function and electrolytes (potassium 3.7 mEq/L) and elevated C-reactive protein. Sputum and bronchoalveolar lavage were positive for *Aspergillus fumigatus* and treatment with posaconazole 300 mg OD was initiated. Hypokalemia was noted shortly after this. A suspicion was raised for posaconazole-induced pseudohyperaldosteronism confirmed by a low serum aldosterone (<1.0 ng/dL) and normal renin (12.7 µU/mL) and cortisol levels (27.3 µg/dL). Spironolactone 25 mg OD was initiated and a switch to isavuconazole was made. One week after discharge, the patient had stopped potassium replacement and presented with normokalemia.
***Conclusion:*** This case demonstrates posaconazole-induced pseudohyperaldosteronism with hypokalemia. Its mechanisms occur via inhibition of adrenal 11ß-hydroxylase and/or 11ß-hydroxysteroid dehydrogenase type 2, resulting in accumulation of 11-deoxycorticosterone and elevated cortisol-to-cortisone ratios with mineralocorticoid effects. Patients taking posaconazole should be regularly monitored for development of hypokalemia and/or hypertension.

## EP27. PANHYPOPITUITARISM AND CRANIOPHARYNGIOMA REMOVAL SURGERY: IS CONSERVATIVE RESECTION BETTER?

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***Introduction:*** This study aims to predict if a craniopharyngioma (CP) removal surgery that spares brain structure, has a lower incidence of panhypopituitarism (PH), considering patients pituitary deficits prior surgery.
***Methods:*** A retrospective cohort study of patients who underwent CP removal surgery over a period of 17.5 years at Centro Hospitalar Universitário de São João, considering pituitary deficits prior surgery, was performed. The type of removal surgery the patients underwent, was classified as total, subtotal or partial, according to medical reports. The occurrence of PH was assessed based on medical reports as well.
***Results:*** A total of 43 patients were identified and then divided in a no pituitary deficits prior surgery group, 23 cases, and in a pituitary deficits prior surgery group, 20 cases. In the first group, PH occurred in four cases (17.4%) of total removals, five cases (21.7%) of subtotal removals and in three cases (13.0%) of partial removals. However, the type of removal surgery performed did not demonstrate to be associated to the *novo* occurrence of PH (5%, $p$=0.742). Furthermore, regarding other subgroups analyses performed, postoperative complications were associated with the occurrence of PH after CP removal surgery.
***Conclusion:*** Regarding the fact this study was not able to demonstrate that the type of CP removal surgery performed is related to PH, nowadays, there is the evidence that pituitary conservative surgeries, followed by radiotherapy, may also treat CP, with less morbidity and higher quality of life (QoL). Furthermore, we were able to observe that postoperative complications are associated with PH.

## EP28. TRATAMENTO CIRÚRGICO DO HIPERPARATIROIDISMO PRIMÁRIO: CASUÍSTICA DE UM SERVIÇO

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***Introdução:*** O hiperparatiroidismo primário (HPTP) é uma condição que afecta o metabolismo do cálcio devido à hipersecreção da hormona paratiroideia (PTH) resultando em hipercalcemia. Classicamente as formas de apresentação clínica mais frequentes eram doença renal ou esquelética sintomática com hipercalcemia moderada ou grave; entretanto, atualmente, a maioria dos pacientes apresenta poucos sintomas e hipercalcemia leve, associada a PTH sérica elevada ou inapropriadamente normal. Uma nova forma de apresentação chamada HPPT normocalcémica também foi descrita. A localização glandular pré-operatória deve ser realizada por ultrassonografia, cintilografia e eventualmente por tomografia computorizada (TC) 4D. A paratireoidectomia é a forma de tratamento curativa quando efectuada por cirurgiões experientes e tem baixa morbilidade. Sempre que for possível deve ser realizada uma abordagem minimamente invasiva.
***Métodos:*** Análise retrospetiva dos casos de HPP operados de 07/2020 e 12/2021.
***Resultados:*** Operaram-se 50 pacientes, 80% mulheres (n=40) e 20% homens (n=10), com idade média de 65 anos. Num total de 51 operações, 48 foram primeiras operações e 3 foram reintervenções. Realizamos 6 explorações cervicais bilaterais em casos com estudos de localização negativos ou discordantes: 1 PTX subtotal em paciente MEN1, 3 biglandulares em HPT normocalcêmico e 2 PTX uniglandulares. Nas 45 ressecções uniglandulares restantes, a abordagem foi unilateral ou seletiva com minicervicotomia. Notamos apenas uma paralisia temporária das cordas vocais e 2 hipoparatireoidismos transitórios. Três pacientes apresentaram HPT persistente, um deles já curado em um segundo PTX.
***Conclusão:*** Podemos concluir que o tratamento cirúrgico foi bastante eficaz, resultando numa elevada taxa de cura (94%) associada a uma reduzida taxa de complicações.


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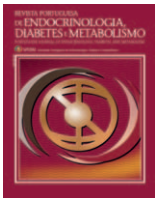

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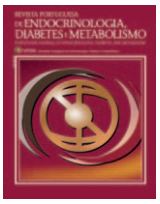

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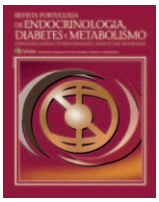

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Pode ser solicitado aos autores que disponibilizem os dados brutos em que basearam o seu artigo durante o processo de revisão e até 10 anos após a publicação.

**Submissão dos Trabalhos**

A submissão de um manuscrito implica que o trabalho descrito não tenha sido publicado previamente (excepto na forma de um resumo ou como parte de uma palestra publicada ou de uma tese académica), e que não está sendo considerado para publicação em outra revista, que o manuscrito foi aprovado por todos os autores e, tácita ou explicitamente, pelas autoridades competentes onde o trabalho foi realizado e que, se for aceite para publicação, não será publicada em outro lugar na mesma forma, em inglês ou em qualquer outra língua, incluindo electronicamente.

Todos os manuscritos devem ser acompanhados por uma carta de apresentação. Deve ser dada garantia na carta de apresentação de que o manuscrito não está sob consideração simultânea por qualquer outra revista. Na carta de apresentação, os autores devem declarar seus potenciais conflitos de interesse e fornecer uma declaração sobre a autoria.

Para verificar a originalidade, o artigo pode ser verificado pelo serviço de detecção de originalidade.

As submissões que não estejam em conformidade com estas instruções podem ser devolvidas para reformulação e reenvio.

**Submissão do Manuscrito**

Submeta o seu manuscrito em: http://spedmjournal.com/

**Contacto**

Em caso de dúvidas durante a submissão, contacte:
scientific.landscape@gmail.com

**Preparação do Manuscrito**

**Uso de programa de processamento de texto**

É importante que o arquivo seja guardado no formato nativo do processador de texto usado. O texto deve estar no formato de coluna única. Mantenha o *layout* do texto o mais simples possível.

Para evitar erros desnecessários, aconselhamos o uso das funções "verificação ortográfica" e "verificação gramatical" do seu processador de texto.

**Tipologia dos Artigos**

A Rev Port Endocrinol Diabetes Metab aceita a seguinte tipologia:

a) Artigos originais reportando investigação clínica ou básica;
b) Artigos de revisão (incluindo sistemáticas revisões e meta-análises);
c) Estudos de Caso/Casos Clínicos;
d) Imagens em Endocrinologia;
e) Editoriais, que são escritos a convite do Editor-Chefe e consistem em comentários sobre artigos publicados na revista ou sobre temas de relevância particular;
f) Cartas ao Editor, que consistem em pareceres concisos sobre artigos recentemente;
g) Perspectivas

h) *Guidelines*.

Os autores devem indicar na carta de apresentação qual o tipo de manuscrito que está a ser submetido para publicação.

Na primeira página/ página de título:

**I. Título**

Título em português e inglês, conciso e descritivo, sem abreviaturas e não excedendo os 120 caracteres. O título pode incluir um complemento de título com um máximo de 40 caracteres (incluindo espaços).

**II. Autores e afiliações**

Na linha da autoria, liste o Nome de todos os Autores (primeiro e último nome) e respectiva afiliação (departamento, instituição, cidade, país).

**III. Subsídio**

Todos os subsídio(s) ou bolsa(s) que contribuíram para a realização do trabalho.

**IV. Autor Correspondente**

Indicar claramente quem vai lidar com a correspondência em todas as fases de arbitragem e publicação, também pós-publicação. Endereço postal e *e-mail* do Autor responsável pela correspondência relativa ao manuscrito.

**V. Resumo e *Keywords***

Um resumo conciso e factual é requerido. Um resumo é frequentemente apresentado separadamente do artigo, por isso deve ser capaz de ficar sozinho.

Resumo escrito em português e inglês. Nenhuma informação que não conste no manuscrito pode ser mencionada no resumo. O resumo não pode remeter para o texto, não podendo conter citações nem referencias a figuras.

No fim do resumo devem ser incluídas um máximo de 5 *Keywords* em inglês utilizando a terminologia que consta no Medical Subject Headings (MeSH), http://www.nlm.nih.gov/mesh/MBrowser.html,

**VI. Resumo Estruturado**

Um resumo estruturado, com as etiquetas de secção apropriadas, deve fornecer o contexto e objectivo do estudo, procedimentos básicos (seleção dos sujeitos de estudo ou animais de laboratório, métodos observacionais e analíticos), principais resultados (significância estatística, se possível) e principais conclusões. Deve enfatizar aspectos novos e importantes do estudo ou das observações. Secções: Introdução, Métodos, Resultados e Conclusão.

**VII.** Os autores também incluirão nesta página de título, sob a designação "Considerações éticas" a declaração de "**Protecção de pessoas e animais"**, **Confidencialidade dos dados e consentimento informado e Conflitos de interesse.**

**Prémios e Apresentações prévias**

Devem ser referidos os prémios e apresentações do estudo, prévias à submissão do manuscrito

**Texto**

**Artigos Originais**

Os artigos originais devem incluir as seguintes secções: Introdução, Material e Métodos, Resultados, Discussão e Conclusão, Agradecimentos (se aplicável), Referências, Tabelas e Figuras.

Os artigos originais não devem exceder 4000 palavras, até 6 tabelas ou figuras e até 60 referências. Um resumo estruturado com o máximo de 350 palavras.

**Article structure**

***Introduction***

State the objectives of the work and provide an adequate background, avoiding a detailed literature survey or a summary of the results.

***Material and methods***

Provide sufficient detail to allow the work to be reproduced.

| Article type | Abstract | Keywords | Main text structure | Max. words | Tables/figures | References |
|---|---|---|---|---|---|---|
| Original Article | Max. 350 words; structured (Introduction and Objectives, Methods, Results and Conclusion(s)) Portuguese and English | Up to 6 Portuguese and English | Introduction; Methods; Results; Discussion; Conclusion(s); Acknowledgments, if any; References; and figure legends, if any | 4000 | Total up to 6 | Up to 60 |
| Review Article | Max. 350 words; unstructured Portuguese and English | Up to 6 Portuguese and English | Introduction; thematic sections at the discretion of the authors; Conclusion(s); Acknowledgments, if any; References; and figure legends, if any | 4000 | Total up to 6 | Up to 100 |
| Systematic Review | Max. 350 words; structured Portuguese and English | Up to 6 Portuguese and English | PRISMA | 4000 | Total up to 6 | Up to 100 |
| Case Report | Max. 150 words; unstructured Portuguese and English | Up to 6 Portuguese and English | Introduction; Case report; Discussion; Conclusion(s) (optional); References; and figure legends, if any | 2000 | Total up to 4 | Up to 25 |
| Images in Endocrinology | None | Up to 6 Portuguese and English | Unstructured | 500 | Total up to 4 | Up to 5 |
| Editorial | None | None | Unstructured | 1500 | Total up to 2 | Up to 20 |
| Letter to the Editor | None | Up to 6 Portuguese and English | Unstructured | 600 | Total up to 1 | Up to 10 |
| Current Perspectives | None | Up to 6 Portuguese and English | Unstructured | 1200 | Total up to 2 | Up to 10 |

Methods already published should be indicated by a reference: only relevant modifications should be described.

***Results***

Results should be clear and concise.

***Discussion***

This should explore the significance of the results of the work, not repeat them. A combined Results and Discussion section is often appropriate. Avoid extensive citations and discussion of published literature.

***Conclusion***

The main conclusion of the study may be presented in a short Conclusion section, which may stand alone or form a subsection of a Discussion or Results and Discussion section.

**Artigos de Revisão**

Os artigos de revisão são artigos abrangentes que sintetizam ideias antigas e sugerem novas. Abrangem áreas amplas. Podem ser de ciência clínica, investigação ou básica. Embora geralmente por convite do Editor-Chefe, ocasionalmente aceitamos artigos de revisão não solicitados sobre assuntos importantes ou sobre avanços recentes. Antes de submeter uma revisão, pedimos que envie ao Editor-Chefe um breve esboço (não mais de 500 palavras) indicando a importância e novidade do assunto, e por que está qualificado para escrevê-lo. Um convite para submissão não garante aceitação.

Os artigos de revisão não devem exceder 4000 palavras, até 6 tabelas ou figuras e até 100 referências. Um resumo não estruturado com o máximo de 350 palavras.

**Revisões Sistemáticas e Meta-Análises**

As revisões sistemáticas podem ou não utilizar métodos estatísticos (meta-análises) para analisar e resumir os resultados dos estudos incluídos.

As Revisões Sistemáticas podem ser apresentadas no formato Introdução, Métodos, Resultados, Discussão. O assunto deve ser claramente definido. O objectivo de uma revisão sistemática deve ser produzir uma conclusão baseada em evidências. Nos Métodos devem fornecer uma indicação clara da estratégia de pesquisa da literatura, extracção de dados, classificação das evidências e análise. Deve ser seguida a normativa PRISMA (http://www.prisma-statement.org/).

O texto não deverá exceder 4000 palavras, excluindo um resumo estruturado (máximo de 350 palavras). Não poderá incluir mais de 10 referências, e até 6 tabelas ou figuras**.**

**Caso Clínico**

O relato de Casos Clínicos deve incluir as seguintes seções: Introdução, Caso Clínico e Discussão.

O texto não poderá exceder 2000 palavras, e não poderá exceder as 25 referências bibliográficas. Deve incluir um resumo não estruturado, que não exceda 150 palavras.

Deve ser seguida a normativa CARE (http://www.care-statement.org/).

**Editoriais**

Os Editoriais são da responsabilidade do grupo editorial ou solicitados por convite do Editor-Chefe e constituirão comentários sobre tópicos actuais ou comentários sobre artigos publicados na revista. Não devem exceder as 1200 palavras, um máximo de 20 referências bibliográficas e podem conter uma tabela e uma figura. Não têm resumo.

**Cartas ao Editor**

As cartas ao Editor consistem em comentários críticos sobre um artigo publicado na revista ou uma nota curta sobre um determinado tópico ou caso clínico. Cartas ao Editor não devem exceder 600 palavras e 10 referências e pode conter uma figura ou tabela. Não têm resumo.

**Imagens em Endocrinologia**

Esta secção destina-se à publicação de imagens clínicas, radiológicas, histológicas e cirúrgicas relacionadas com casos de endocrinologia, diabetes ou metabolismo.

O título não deve ter mais de oito palavras. Os autores devem ser no máximo quatro. As imagens devem ser de alta qualidade e valor educativo. São permitidas até 4 figuras**.** As legendas devem ser breves e informativas. Setas ou outros símbolos devem ser incluídos conforme necessário para facilitar a compreensão das imagens. O texto não deve exceder 500 palavras, até cinco referências, e deve incluir uma breve história clínica e dados relevantes do exame físico, testes laboratoriais e progressão clínica, conforme apropriado. Não têm resumo.

**Perspectiva**

Este é o tipo de manuscrito é submetido a convite do Conselho Editorial. Pode abranger uma ampla diversidade de temas relacionados com endocrinologia, diabetes, metabolismo e saúde: problemas atuais ou emergentes, políticas de gestão e saúde, história da medicina, questões de sociedade e epidemiologia, entre outros. Um Autor que deseje propor um manuscrito nesta seção deverá enviar um resumo ao Editor-Chefe, incluindo o título e a lista de autores para avaliação. O texto não deve exceder 1200 palavras, até 10 referências, e até 2 tabelas ou 2 figuras. Não têm resumo.

***Guidelines***

Os guias de prática clínica não devem exceder 4000 palavras, até 6 tabelas ou figuras e até 100 referências. Resumo até 350 palavras.

**Referências**

**I.Citação no texto**

Certifique-se de que todas as referências citadas no texto também estão presentes na lista de referências (e vice-versa). As referências devem ser listadas usando algarismos árabes pela ordem em que são citados no texto.

As referências a comunicações pessoais e dados não publicados devem ser feitas diretamente no texto e não devem ser numeradas. Citação de uma referência como "in press" implica que o item tenha sido aceite para publicação. Os nomes das revistas devem ser abreviados de acordo com o estilo da Medline.

As referências a artigos publicados em revistas devem incluir o nome do primeiro autor seguido dos nomes dos restantes autores, o título do artigo, o nome da revista e o ano de publicação, volume e páginas.

Certifique-se de que os dados fornecidos nas referências estão corretos. Ao copiar referências, tenha cuidado porque já podem conter erros.

A lista de referências deve ser adicionada como parte do texto, nunca como uma nota de rodapé. Códigos específicos do programa de gestão de referências não são permitidos.

**II. Formato**

Uma descrição detalhada dos formatos de diferentes tipos de referência pode ser consultada em ICMJE *Recommendations* (http://www.icmje.org/recommendations/). Liste todos os autores se houver seis ou menos. *Et al* deve ser adicionado se houver mais de seis autores. Título do artigo, nome da revista, ano, volume e páginas.

**III. Estilo de referência**

*Texto:* Indicar as referências no texto por número (s) em expoente. Os autores podem ser referidos, mas o número de referência deve ser sempre dado.
*Lista:* Ordene as referências na lista pela ordem em que aparecem no texto
*Exemplos:*
Referência de artigo:

1. Isidori AM, Sbardella E, Zatelli MC, Boschetti M, Vitale G, Colao A, et al. Conventional and nuclear medicine imaging in ectopic Cushing's syndrome: a systematic review. J Clin Endocrinol Metab. 2015;100:3231-44.

Referência de livro:

2.Ware JE, Kosinski M, Dewey JE. How to score version 2 of the SF-36 Health Survey: standard & acute forms. Lincoln: Quality Metric Incorporated; 2000.

Referência de capítulo de livro:

3. Castellano Barca G, Hidalgo Vicario M, Ortega Molina M. Transtorno del comportamento alimentário. In: Castellano Barca G, Hidalgo Vicario M, Redondo Romero A, editores. Medicina de la adolescência – atención integral. 2ª ed. Madrid: Ergon; 2004. p.415-29.

Referências Web:

4. No mínimo, o URL completo deve ser dado e a data em que o documento foi consultado. Qualquer outra informação, se conhecida (nomes de autor, datas, referência a uma publicação de origem, etc.), também deve ser dada.

**Notas de Rodapé**

As notas de rodapé devem ser evitadas. Quando imprescindíveis, devem ser numerados consecutivamente e aparecer ao pé da página apropriada.

**Agradecimentos (facultativo)**

Devem vir após o texto, e antes das referências, tendo como objectivo agradecer a todos os que contribuíram para o estudo mas que não têm peso de autoria. Nesta secção é possível agradecer a todas as fontes de apoio, quer financeiro, quer tecnológico ou de consultadoria, assim como contribuições individuais.

**Abreviaturas**

Não use abreviaturas ou acrónimos no título e no resumo e limite o seu uso. Abreviaturas não consagradas devem ser definidas na primeira utilização, por extenso, logo seguido pela abreviatura entre parenteses. A menos que a sigla seja uma unidade padrão de medição. Uso excessivo e desnecessário de acrónimos e abreviaturas deve ser evitado.

**Unidades de Medida**

Devem ser utilizadas as unidades Sistema Internacional de Unidades. As medidas de comprimento, altura, peso e volume devem ser expressas em unidades do sistema métrico (metro, quilograma ou litro) ou seus múltiplos decimais. As temperaturas devem ser dadas em graus Celsius (ºC) e a pressão arterial em milímetros de mercúrio (mm Hg) ou a hemoglobina em g/dL. Todas as medições hematológicas ou bioquímicas serão referidas no sistema métrico de acordo com o Sistema Internacional de Unidades (SI).

**Nomes de Medicamentos**

Identifique com precisão todos os medicamentos e produtos pelo nome genérico. Não é recomendável a utilização de nomes comerciais de fármacos (marca registrada), mas quando a utilização for imperativa, o nome do produto deverá vir após o nome genérico, entre parênteses, em minúscula, seguido do símbolo que caracteriza marca registada, em sobrescrito (®).

**Tabelas e Figuras**

Tabelas/Figuras devem ser numerados na ordem em que são citadas no texto e assinaladas em numeração árabe e com identificação, Figura/Tabela.

Cada figura e tabela incluídas no trabalho têm de ser referidas no texto: Uma resposta imunitária anormal pode estar na origem dos sintomas da doença (Fig. 2). Esta associa-se a outras duas lesões (Tabela 1).

Figura: Quando referida no texto é abreviada para Fig., enquanto Tabela não é abreviada. Nas legendas ambas as palavras são escritas por extenso.

Cada tabela e figura deve ser acompanhada da respectiva legenda, sucinta e clara. As legendas devem ser auto-explicativas (sem necessidade de recorrer ao texto).

Em relação aos gráficos deve ser explícito se a informação inclui valores individuais, médias ou medianas, se há representação do desvio padrão e intervalos de confiança e o tamanho da amostra (n).

As fotografias deverão incluir identificadores (setas e asteriscos). Poderão ser publicadas fotografias a cores, desde que consideradas essenciais.

Cada tabela deve ser utilizada para mostrar resultados, apresentando listas de dados individuais ou sumariando os mesmos, não devendo no entanto constituir duplicação dos resultados descritos no texto. Devem ser acompanhadas de um título curto mas claro e elucidativo. As unidades de medida usadas devem ser indicadas (em parêntesis abaixo do nome que encabeça cada categoria de valores) e os números expressos devem ser reduzidos às casas decimais com significado clínico.

Para as notas explicativas nas tabelas devem ser utilizados os seguintes símbolos e sequência: *, †, ‡, §, ||, ¶, **, ††, ‡‡ .

Se fotografias de doentes forem usadas, estes não devem ser identificáveis ou as fotografias devem ser acompanhadas de autorização por escrito para usá-las.

As imagens a cores são reproduzidas gratuitamente.

Princípios gerais:

• Numere as ilustrações de acordo com a sua sequência no texto.

• Forneça as legendas das ilustrações separadamente.

• Dimensione as ilustrações próximas das dimensões desejadas da versão publicada.

• Envie cada ilustração em ficheiro separado.

A inclusão de figuras e/ou tabelas já publicadas, implica a autorização do detentor de *copyright* (autor ou editor).

A submissão deve ser feita separadamente do texto, conforme as instruções da plataforma.

Os ficheiros das figuras devem ser fornecidos em alta resolução, 800 dpi mínimo para gráficos e 300 dpi mínimo para fotografias.

A publicação de ilustrações a cores é gratuita.

Material gráfico deve ser entregue em um dos seguintes formatos:
JPEG (. Jpg)
Portable Document Format (. Pdf)
PowerPoint (.ppt)
TIFF (. Tif)
Excel

**Permissão para publicação:** No caso de publicação de tabelas de livros ou revistas os autores são responsáveis por obter permissão, junto dos autores dos trabalhos de onde forem reproduzidos, para a referida publicação, e terão de a apresentar na submissão.

**Ficheiros Multimedia**

Os ficheiros multimedia devem ser enviados em ficheiro separado com o manuscrito. O material multimedia deve seguir os padrões de qualidade de produção para publicação sem a necessidade de qualquer modificação ou edição. Os ficheiros aceitáveis são: formatos MPEG, AVI ou QuickTime.

**Anexos/ Apêndices**

Quando necessário, os anexos devem ser utilizados para apresentar inquéritos longos ou detalhados, descrições de extensos cálculos matemáticos e / ou listas de itens. Devem ser colocados depois da lista de referências, se necessário, com legendas. Anexos longos, tais como algoritmos, pesquisas e protocolos, serão publicados apenas *online*; o URL será fornecido no artigo impresso onde o anexo é citado.

Se houver mais de um apêndice, eles devem ser identificados como A, B, etc. As fórmulas e equações em apêndices devem ser numeradas separadamente: Eq. (A.1), Eq. (A.2), etc.; Em apêndice posterior, a Eq. (B.1) e assim por diante. Da mesma forma para tabelas e figuras: Tabela A.1; FIG. A.1, etc.

**Estilo**

Rev Port Endocrinol Diabetes Metab segue AMA Manual Style (10ª edição).

Última revisão **Janeiro 2022**