X Advanced Course of Endocrinology • X Curso Avançado de Endocrinologia [2022;17 (Supl. 2)]

REVISTA PORTUGUESA DE ENDOCRINOLOGIA, DIABETES E METABOLISMO

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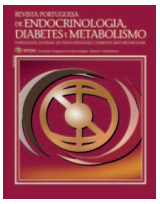

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COLLABORATION
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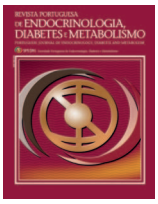

Welcome Words

Davide Carvalho[1]

[1] *Head of the Department of Endocrinology, Diabetes and Metabolism Centro Hospitalar Universitário de S. João; Associate Professor Faculty of Medicine University of Porto (UP), Portugal; Core Researcher i3s UP; Full Academic of the National Academy of Medicine; President of the Association of Friends of the Endocrinology, Diabetes and Metabolism Department, Centro Hospitalar Universitário S. João.*

"To forget a friend is sad. Not everyone has had a friend"

Antoine Saint-Exuperry

Dear friends,

Welcome to the X Advanced Course of Endocrinology.

Creating an Endocrinology training programme is an important challenge for all department directors. Supporting interns and young doctors in training and the less young in updating their knowledge is also a priority.

Back to the Advanced Course. The pandemic forced us to cancel the 2020 edition when it was virtually ready to launch. This will be its 10th edition. Due to the circumstances of life, it will also be my last Advanced Course. However, I have no doubt that the department will be able to carry on providing the same line of training and updating that I built upon and expanded when it was handed over to me by Manuel Pinheiro Hargreaves, Emílio Peres, Maria Luisa Vila-Cova, and José Luis Medina.

Everything is ephemeral and only friendship remains. We recently lost one of the leaders of our department, José Luis Medina, who was one of the creators of the department. He was probably one of its most influential members. Firstly, because of the friendly atmosphere that he created and cultivated before, during, and after his departure from the department, and secondly, due to the scientific quality of his contribution. He imparted to us a legacy of a demanding and always up-to-date clinic that has constituted the paradigm for the various generations that he trained who came to follow him. His sudden departure leaves us all with a feeling of loss that cannot be surpassed, as he was irreplaceable. To keep him alive in our memory, we have created an award in his name for the best communication in the area of research presented during the Advanced Course.

This course brings together residents, young specialists of Endocrinology, and well-known specialists, and is a melting pot of different perspective and diverse interests, albeit with one focal challenge: to improve the quality of care of patients.

We are going to discuss diabetes, including insulin pump therapy and continuous glucose monitoring, craniopharyngiomas, pheo/paragangliomas, and neuroendocrine tumors in a relaxed and friendly atmosphere.

I hope that you will all enjoy the X Course and I look forward to participating in the XI edition with all of you! As a Portuguese prime minister said a few years ago when he left the government: I'm going to be walking around!

Davide Carvalho

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8-9APRIL2022
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8th April Friday

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EP01. A RARE AND CHALLENGING DIAGNOSIS: HYPOPHYSITIS AND PARASELLAR LIPOMA – TWO RARE ASSOCIATIONS
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Telma Moreno, Sara Ribeiro, Pedro Rodrigues, Davide Carvalho

EP03. EFFICACY, SAFETY AND QUALITY OF LIFE OF PASIREOTIDE IN FIRST GENERATION SOMATOSTATIN ANALOGS RESISTANT ACROMEGALIC PATIENTS
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Sara Ribeiro, Telma Moreno, Ana Varela, Davide Carvalho

EP07. ACROMEGALY: GLYCEMIC METABOLISM MAY PREDICT A MORE AGGRESSIVE TUMOR
Maria João Ferreira, Marta Canha, Jorge Pinheiro, Josué Pereira, Davide Carvalho, Irene Bernardes

EP08. TSH-SECRETING PITUITARY ADENOMAS: A REPORT OF 5 CASES FROM A TERTIARY CENTRE
Fernando Mendonça, Selma Souto, Josué Pereira, Jorge Pinheiro, Irene Bernardes, Davide Carvalho

EP09. LARGEST DIMENSION OF THE PHEOCHROMOCYTOMA IS ASSOCIATED WITH MORTALITY: RESULTS OF THE ANALYSIS OF 62 CASES
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Moderators: Luís Sá Vinhas, Maria Teresa Faria
Discussion Panel: Elisabete Rodrigues, Sandra Belo

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17h45 – 18h15 **Paraganglioma – Pituitary adenomas – SDHB saga** – Ana Saavedra
18h15 – 18h30 **Discussion**

18h30 – 19h45 **CRANIOPHARYNGIOMAS**
Chairmen: Isabel Paiva, Josué Pereira
Moderators: João Sequeira Duarte, Leonor Gomes, Maria João Oliveira
Discussion Panel: Luís Cardoso, Mariana Martinho, Patrícia Polónia

18h30 – 19h00 **From the diagnosis to medical management** – Kristi Alexandraki
19h00 – 19h20 **Surgical Management** – António Cerejo
19h20 – 19h45 **Discussion**

20h00 ***Dinner***

9th April Saturday

08h30 – 10h15 **TYPE 2 DIABETES. WHAT IS NEW IN DIABETES MANAGEMENT?**
Chairmen: João Jácome de Castro, Paula Freitas
Moderators: João Raposo, Silvestre Abreu
Discussion Panel: Olinda Marques, Rui César

08h30 – 09h15 **SGLT2 inhibitors: mechanisms of cardiorenal benefit beyond glycaemic control** – Miles Fisher
Sponsored by Astrazeneca

09h15 – 10h00 **GIP/GLP-1 receptor co-agonist tirzepatide: Clinical results and mechanism of action** – Michael Nauck

10h00 – 10h15 **Discussion**

10h15 – 10h45 **Poster Discussion 2** – Duarte Pignatelli, Ana Varela, Selma Souto, Eva Lau

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Moderators: Elisabete Barbosa, Cristina Sarmento
Discussion Panel: John Preto, António Gouveia

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11h15 – 11h45 **Imaging Diagnosis – review of the experience of Center** – Ana Paula Moreira
11h45 – 12h05 **Functional Imaging and PRRT in Metastatic Medullary Thyroid Carcinoma** – Pedro Souteiro
12h05 – 12h40 **Neuroendocrine Tumours Management** – Wouter de Herder
12h40 – 13h00 **Discussion**

Sponsored by AAA

13h00 – 14h15 ***Lunch***

14h15 – 15h45 **FROM THE SCIENTIFIC EVIDENCE TO THE CLINICAL PRACTICE: CSII EFFICACY AND SAFETY**
Chairmen: José Luís Castedo, Helena Cardoso
Moderators: Celestino Neves, Cíntia Castro Correia

14h15 – 14h45 **New systems of Continuous Glucose Monitoring** – Julia Mader

Sponsored by Menarini

14h45 – 15h45 **Optimising bolus insulin therapy: calculators, downloads and grey matter** – Mark Evans

Sponsored by Novo-Nordisk

15h45 – 17h00 **WORKSHOP: CLINICAL CASES** – Mark Evans, Julia Mader
Moderators: Celestino Neves, Jorge Dores

17h00 **Closing and Awards** – Davide Carvalho

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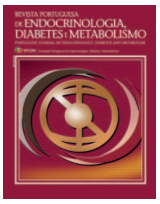

Conference Abstracts

L01. PHEOCHROMOCYTOMAS / PARAGANGLIOMA SYNDROME: WHAT'S NEW IN GENETICS

Jorge Lima[1]

[1] *i3S-Ipatimup, Porto, Portugal*

Pheochromocytomas (PCC) and paragangliomas (PGL) are neuroendocrine neoplasms derived from the chromaffin tissue of the adrenal medulla or from extra-adrenal sympathetic and parasympathetic paraganglia.

Recent studies have indicated that up to 40% of PCC/PGL could be attributable to an inherited germline variant in an increasing list of susceptibility genes. These genes can be grouped into three clusters: pseudohypoxia group (*VHL*, *SDHA*, *SDHB*, *SDHC*, *SDHD*, *SDHAF2*, *FH*, *EPAS1*), kinase signalling group (*RET*, *NF1*, *TMEM127*, *MAX* and *HRAS*) and Wnt signalling group (*CSDE1* and *MAML3*). On this basis, international guidelines recommend genetic testing to every patient with a PCC/PGL, since the inherited mutation has important clinical impact.

We will review the germline and somatic genetic features of PCC/PGL and address the clinical relevance of PCC/PGL genetic characterization.

L02. PARAGANGLIOMA – PITUITARY ADENOMAS: SDHB SAGA

Ana Saavedra[1]

[1] Department of Endocrinology - Centro Hospitalar Trás-os-Montes e Alto Douro

Multiple endocrine neoplasia (MEN) syndromes are characterized by the presence of tumours affecting two or more glands in a single patient. Type 1 and type 2 MEN were the main and earlier forms described, but other associations have been recognized. More recently, the presence of pituitary adenoma and paraganglioma/pheochromocytoma (PGL/Pheo) in the same patient have also been reported. This very rare clinical condition is known as "3 P association" (3PAs) and, despite it could be a result of coincidence, at least in some cases, a common pathogenic mechanism is involved and is mostly due to succinate dehydrogenase (SDH) defects. SDH is a multimeric four-subunit enzyme, which is bound to the inner mitochondrial membrane, where it participates in the Krebs cycle and electron transport chain. The genes encoding the four SDH subunits – *SDHA*, *SDHB*, *SDHC*, and *SDHD* – have been demonstrated to be tumour supressor genes. *SDHx* mutations are classically associated with familial PGL/Pheo syndromes.

PGL/Pheo have the highest reported degree of heritability among all tumours and more than 20 susceptibility genes have been identified. Therefore, it is recommended to consider genetic studies for any patient diagnosed with PPG. In the particular case of patients with *SDHB* mutations, they are highly predisposed to metastatic PGL/Pheo and are at risk of developing multiple tumours, which can be widely distributed from skull base to pelvic floor.

On the other side, most pituitary tumours are sporadic, but approximately 5% can be due to a hereditary disease (either isolated or as part of a syndromic condition, like 3PAs). Although only a very small percentage of all pituitary adenomas are associated with *SDHx* mutations, the identification of particular features that differentiate them from sporadic cases may be crucial. The literature suggests that SDHx-related pituitary adenomas can have different phenotypes (somatotropinomas, prolactinomas, and non-functioning adenomas), can have specific histological findings, be associated with more aggressive features and be more resistant to somatostatin analogues.

L03. CRANIOPHARYNGIOMAS – FROM THE DIAGNOSIS TO MEDICAL MANAGEMENT

Kristi Alexandraki[1]

[1] *Laiko University Hospital, Medical School, National and Kapodistrian University of Athens, Athens, Greece*

Craniopharyngiomas are rare benign intracranial neoplasms arising in sellar/parasellar region, presenting in two subtypes, adamantinomatous (ACP) or papillary (PCP), which are clinically, histologically and molecularly distinct. They present in a bimodal pattern; the ACP are manifested mostly during the childhood and the PCP during the adulthood with a wide range of clinical manifestations. Despite their benign nature they are characterized by non-specific symptoms due to pressure effects on vital structures, mostly visual disturbances and headaches, whilst their severity is due to the location, size, and growth potential of the neoplasms. In addition, hypothalamo-pituitary dysfunction may be seen at presentation characterized by hypothalamic syndromes or pituitary deficits. Hence, their management is usually challenging since the common therapies, the surgery and the radiotherapy, may be accompanied by debilitating complications because of the neoplasm proximity to vital brain structures. Additional therapeutic interventions include intracystic therapies but without excellent results. However, the progress on molecular pathogenesis understanding of craniopharyngiomas, unveiled new druggable molecular pathways. ACPs are mostly characterized by mutations of β-catenin, activating Wingless/Int (Wnt) pathway, by altering of the mitogen extracellular kinase (MEK)/extracellular signal-regulated kinase (ERK) pathway, and implication of the inflammatory, of the cellular senescence, of

the programmed cell death and of the sonic hedgehog (SHH) pathways. On the other hand, PCPs are mostly characterized by Ras/Raf/MEK/ERK pathway activation secondary to BRAF-V600E mutations. Firstly, BRAF inhibitors, either as monotherapy or in combination with MEK inhibitors have been administered to patients with PCP resulting in favorable responses in neoadjuvant, adjuvant or bridge therapy setting. Following the success of these molecular-targeted therapies, in ACPs, anti-inflammatory mediators, such as the traditional anti-inflammatory agent, interferon, but also novel anti-inflammatory agents such as the anti-IL-6 receptors or molecular-targeted therapy such as the MEK inhibitors, have been administered with variably favorable outcomes. A number of ongoing trials will unravel the utility of the drugs already tried or of other novel drugs in the therapeutic armamentarium of craniopharyngiomas. Concluding, the personalized medicine depicted by molecular-targeted therapies or mediators-targeted therapies, has gained ground on craniopharyngiomas management as in other rare neoplasms by using the facilities and benefits of molecular diagnosis.

L04. CRANIOPHARYNGIOMAS – SURGICAL MANAGEMENT

António Cerejo[1,2]

[1] *Department of Neurosurgery, Centro Hospitalar Universitário São João, Porto, Portugal*
[2] *Faculty of Medicine of the University of Porto, Porto, Portugal*

There are recent changes in the philosophy of the surgical treatment of craniopharyngiomas Although the goal for tumor resection still is gross total resection, the extent of tumor resection is planned according to patient factors, tumor extent and invasiveness of adjacent neural and vascular structures, to avoid tumor recurrence. Surgical subtotal resection is an alternative, and at times preferred, as 90% of progression-free survival at 5-years is achieved, with fewer comorbidities than with a complete resection. There is a continued need for alternative treatments with increased efficacy and fewer adverse postoperative complications.

Apparently, there seem no significant changes in craniopharyngioma patients' overall survival or progression-free survival with gross total resection when compared with surgical subtotal resection and radiation. They also showed that surgical subtotal resection alone was associated with lower survival than gross total resection alone or surgical subtotal resection plus radiation.

We presente our results in some of the most challenging craniopharyngiomas and show a video pointing the more important steps of this surgery.

L05. SGLT2 INHIBITORS: MECHANISMS OF CARDIORENAL BENEFIT BEYOND GLYCAEMIC CONTROL

Miles Fisher[1]

[1] *Consultant Physician, Department of Diabetes, Endocrinology and Clinical Pharmacology, Glasgow Royal Infirmary, and Honorary Professor, University of Glasgow, Glasgow, UK*

The first cardiovascular outcome trials (CVOTs) with SGLT2 inhibitors were performed in people with type 2 diabetes to satisfy the safety requirements of the FDA and EMA. Trials with empagliflozin, canagliflozin and dapagliflozin (EMPA-REG OUTCOME, CANVAS and DECLARE-TIMI 58) demonstrated clear reductions in major adverse cardiovascular events (MACE), a composite of cardiovascular death, nonfatal myocardial infarction, and nonfatal stroke. The VERTIS CV trial with ertugliflozin, however, did not reduce MACE. In all four CVOTs a reduction in hospitalisation for heart failure, which was a secondary outcome, was observed, and there were also reductions in composite renal endpoints. Further trials were performed in patients with baseline heart failure and chronic kidney disease (CKD), including in subjects with and without diabetes. In these trials heart failure outcomes and renal outcomes were significantly reduced, and the size of the reductions were similar in diabetic and non-diabetic subjects, suggesting that these were not mediated by changes in glucose as measured by HbA1c.

There are many mechanistic trials in animals and humans with diabetes, heart failure and renal disease to try and understand the mechanisms of benefit. Early natriuresis with a reduction in plasma volume, a rise in haematocrit, improved vascular function, a reduction in blood pressure and changes in tissue sodium handling are all likely to have a role. Additional mechanisms of SGLT2 inhibitors that might be beneficial include a reduction in adipose tissue-mediated inflammation and pro-inflammatory cytokine production, a shift towards ketone bodies as the metabolic substrate for the heart and kidneys, reduced oxidative stress, lowered serum uric acid level, reduced glomerular hyperfiltration and albuminuria, and suppression of advanced glycation end-product signalling. Indeed, almost all possible mechanisms that have been studied have improved with SGLT2 inhibitor therapy.

The licences of SGLT2 inhibitors have been changed to reflect the results of the outcome trials. Dapagliflozin is currently licenced for use in patients with diabetes, heart failure and CKD, empagliflozin for use in diabetes and heart failure, and canagliflozin for use in diabetes and diabetic kidney disease. As no further outcome trails are planned for ertugliflozin its licence is solely for treating people with type 2 diabetes.

Many guidelines now recommend a combination of metformin plus SGLT2 inhibitor for patients with baseline cardiovascular or renal disease, whereas for patients without these complications metformin alone is recommended. SGLT2 inhibitors may be added later if HbA1c targets fail to be reached. As SGLT2 inhibitors alone as a class of antidiabetic drugs have proven cardiovascular and renal benefits, there is sufficient evidence to recommend these drugs first line for all patients with type 2 diabetes regardless of HbA1c targets.

L06. NEW INSIGHTS IN DUAL AGONISTS GIP-GLP-1

Michael Nauck[1]

[1] *Bochum, Germany*

Bariatric surgery, in particular gastric Roux-en-Y bypass, reduces body weight and leads to diabetes remission in many patients previously diagnosed with type 2 diabetes. Originally, these were thought to be consequences of the "restrictive" nature of surgery, prohibiting propulsive peristalsis and, thus, nutrient absorption on the one hand, and food intake on the other. We have learned that after bariatric sur-

gery, a fulminant change occurs in the hormonal milieu created by nutrients coming into contact with enteroendocrine cells secreting, e.g., GLP-1, PYY, glicentin, but also GIP. In an attempt to mimic these mechanisms pharmacologically, unimolecular co-agonists have been developed, which stimulate not only GLP-1 receptors (as do selective GLP-1 receptor agonists), but in addition, one or two of the other hormones. The most advanced development has led to clinical trials with the GIP/GLP-1 receptor co-agonist tirzepatide, which uniformly have reported highly effective reductions in HbA_{1c} (often by > 2.0 %) and in body weight (often > 10 kg on average). Notably, the effects are greater than with selective GLP-1 receptor agonists (semaglutide and dulaglutide) in head-to-head comparison trials, and compare favorably to basal insulin treatment. Overall, tirzepatide seems to be the most effective single agent to treat type 2 diabetes across all stages from monotherapy to add-on to basal insulin. Reasons quoted to explain this outstanding effectiveness include additive effects of GLP-1 and GIP on insulin secretion and insulin sensitivity (perhaps going beyond what would be expected to follow tirzepatide-induced weight loss), and in suppressing glucagon, plus novel mechanisms involving hypothalamic GIP receptors for controlling appetite and energy intake, potentially interacting with known effect of GLP-1 agonism on these essential neural circuits.

The clinical results with tirzepatide are generally viewed as highly promising, and seem to confirm the development strategy of combining GLP-1 and GIP receptor stimulation as potentially superior approach to the treatment of type 2 diabetes, obesity, and their complications. However, based on present knowledge, a number of questions remain concerning important details of the mechanisms involved: (a) It has hitherto been almost impossible to stimulate insulin secretion with GIP in subjects with type 2 diabetes. (b) GIP has been associated with increased rather than decreased glucagon secretion. (c) GIP promotes triglyceride storage in adipose tissue and has been found essential for weight gain associated with hypercaloric feeding (such that even GIP receptor antagonists have been suggested to be an effective treatment of obesity). (d) Exogenous GIP reduced food intake and body weight in rodent models of type 2 diabetes, but was ineffective in several human studies. Thus, more research is needed to fully explain the impressive effectiveness of tirzepatide in the treatment of type 2 diabetes, and to guide the future development of glucose- and body weight-lowering medications.

L07. NEUROENDOCRINE TUMORS: A CLINICAL OVERVIEW

João Sérgio Neves[1, 2]

[1] *Department of Endocrinology, Diabetes and Metabolism, Centro Hospitalar Universitário de São João, Porto, Portugal*

Neuroendocrine tumors are defined as epithelial neoplasms with neuroendocrine differentiation and can arise in most organs. The main primary sites for neuroendocrine tumors are the gastrointestinal tract and the lung. Neuroendocrine tumors can be divided as well-differentiated neuroendocrine tumors and poorly differentiated neuroendocrine carcinomas. Proliferative rate (evaluated by mitotic count and Ki-67) is important to predict biologic behavior of well-differentiated neuroendocrine tumors. Well-differentiated neuroendocrine tumors can be divided into low (G1), intermediate (G2) and high (G3) grades based on the proliferative rate. Clinical manifestations of neuroendocrine tumors depend on the location of the primary tumor and its functionality. Insulinomas are the most common type of functional neuroendocrine tumor of the pancreas and are responsible for most cases of endogenous hyperinsulinemia. Patients usually present with autonomic and neuroglycopenic symptoms, and the presence of a hypoglycemic disorder is confirmed by the Whipple's triad (hypoglycemic symptoms, with low glucose during symptoms and relieve of symptoms by correction of the hypoglycemia). Nonfunctioning neuroendocrine tumors frequently present late, usually with symptoms of mass effect or liver metastasis. Carcinoid syndrome may develop in patients with metastatic disease, especially those with liver metastasis. Manifestations of carcinoid syndrome include flushing, diarrhea, abdominal cramping and valvular heart disease. A careful evaluation of clinical history and physical examination are essential for the diagnosis and management of neuroendocrine tumors.

L08. NEUROENDOCRINE TUMOURS: IMAGING DIAGNOSIS – REVIEW OF THE EXPERIENCE OF CENTER

Ana Paula Moreira[1, 2]

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Neuroendocrine neoplasms (NENs) can develop anywhere in the body and are heterogeneous in the classification based on the tumour grade and morphology. The increase in the knowledge of molecular drivers of tumorigenesis, and scientific/technological advancements support the accuracy of molecular classification and led the way to multiple drug approvals for the treatment of advanced NENs. Considered rare cancer groups without treatment option in the past, NENs are now discussed in specialized clinical programs or centres with focused research agendas.

Because of the large variability and heterogeneity of NENs, from proliferation index Ki67, SSR subtype profile, and clinical aggressiveness, there is no radiologic modality entirely effective, but nuclear medicine techniques with advanced functional imaging improve sensitivity and specificity on NEN detection. Unlike anatomical or structural imaging, functional imaging is designed to physiologically examine and measure qualitatively and/or quantitatively changes in metabolism, blood flow, chemical or cellular composition, and receptor density. Using tracers analogous to chemical compounds, like glucose (e.g. ^{18}F-FDG) or receptor binding ligands, bound to radionuclides, functional imaging is vital in managing cancer patients. Somatostatin receptor imaging, primarily positron emission tomography (PET) with radiolabelled somatostatin analogous is the most universal nuclear medicine technique for NENS.

^{111}In-Pentetreotide scintigraphy was the mostly used radiopharmaceutical for NENs at the Department of Nuclear Medicine of the Coimbra University Hospitals (SMN, HUC-CHUC) since the 1990s, followed by ^{99m}Tc-EDDA/HYNIC-TOC at the beginning of the century until early 2013, when the 68Ga-labeled somatostatin analogue 1,4,7, 10-tetraazacyclododecane-1,4,7,10-tetraacetic acid–1-NaI3-octreotide (^{68}Ga-DOTA-NOC) became available, due the close relationship between CHUC and the Institute for Nuclear Sciences Applied to Health - University of Coimbra (ICNAS-UC). Shortly thereafter, 6-Fluoro-(18F)-l-3,4-

dihydroxyphenylalanine (^{18}F-DOPA) was made available.

Summarised case examples of patients with NEN will be presented, illustrated with nuclear medicine techniques that highlight its unique role in diagnosing, staging, monitoring and assessing the suitability for peptide receptor radionuclide therapy (PRRT).

Radiopharmaceuticals potentially available in the near future for optimising the assessment of these tumours will also be mentioned.

L10. NEUROENDOCRINE TUMORS MANAGEMENT

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Significance of the Clinical Problem

- Reported incidence of gastroenteropancreatic neuroendocrine neoplasms (GEP-NENs) ranges from 1.4 to 7 / 100 000 persons and is rising.
- NEN of the GI tract: Formerly known as "Carcinoid".
- Pancreatic NEN types: Insulinoma.
  Gastrinoma.
  VIPoma (VIP = vasointestinal polypeptide).
  Glucagonoma.
  Somatostatinoma (?)
  Non-syndromic / non-functioning (NF) pancreatic NEN.

Barriers to Optimal Practice

- Orphan diseases, however incidence rapidly rising.
- Variable clinical manifestations.
- Variable clinical courses.
- Symptom control versus tumor control.
- Multidisciplinary diagnostic and therapeutic approaches.

The (Potential) Role of the Endocrinologist

- Rare functioning tumor syndromes.
- Duodenal NEN in the spectrum of MEN-I, pancreatic NEN are generally sporadic, but can be multiple and a component of familial syndromes: Multiple endocrine neoplasia type I (MEN-I), von Hippel-Lindau disease (VHL), or neurofibromatosis type 1 (NF1).
- Medical therapy and management of hormonal & peptide hypersecretion.
- Receptor-mediated therapies.

Learning Objectives

As a result of following lecture, learners have attained knowledge on:

- Treatment modalities for symptom control and tumor control in GEP-NEN patients.

L11. NEW SYSTEMS OF CONTINUOUS GLUCOSE MONITORING

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Continuous glucose monitoring (CGM) is becoming indispensable in everyday diabetes management. Especially real-time CGM systems with alerts reduce time in hypoglycemia more than CGM systems without alerts.

Within the study the accuracy of the new GlucoMen Day® CGM system (Waveform Cascade, launched by A. Menarini Diagnostics) was assessed by comparing it to a laboratory reference instrument and a self-monitoring blood glucose (SMBG) device.

Eight individuals with type 1 diabetes (three women, age 41.6 ± 13.3 years, BMI 28.0 ± 6.1 kg/m^2, HbA1c 55.6 ± 12.2 mmol/mol, diabetes duration 13.9 ± 6.5 years) spent 14 days at home simultaneously wearing two GlucoMen Day® CGM systems in the abdominal subcutaneous adipose tissue while also performing a minimum of five SMBG measurements (GlucoMen Day® Meter, A. Menarini Diagnostics, Italy) per day. On days four and ten of sensor wear, the participants underwent a 5-hour meal and insulin challenge at the research center. The plasma glucose concentration was determined in 20-minutes intervals with a laboratory reference instrument (YSI 2300 Stat Plus, Yellow Springs, OH).

During the investigational period spent at home, participants could follow their lifestyle but performed at least 4 finger-stick measurements per day.

The CGM accuracy was assessed by calculating the mean absolute relative difference (MARD) and by performing the consensus error grid analysis (CEG).

Overall, the 450 CGM/YSI matched pairs available for analysis were generated from glucose data collected within the range of 40–400 mg/dL. This resulted in a MARD of 9.7 (±9.4)% and a MAD of 20.5 (±18.7) mg/dL. It was observed that accuracy as assessed by MARD was better in the 201–400 mg/dL range compared to the 100–200mg/dL range (6.1 vs 10.7%), while MAD was lower in the 40–70-mg/dL range compared to the 71–99-mg/Ll range (19.5 vs 20.9 mg/dL). CEG analysis showed that 84.9% of CGM/YSI data pairs was in the clinically acceptable zone A (combined A and B: 97.8%).

The participants completed a questionnaire on patient reported outcomes. 50% stated that the insertion was painless, 25% claimed it to be less painful than finger-pricking. The remaining 25% found the insertion as painful as SMBG (12.5%) or more painful than SMBG (12.5%). There was also predominant satisfaction with sensor adhesive, wearability, calibration procedure, and user-friendliness of the dedicated mobile application.

Accuracy of GlucoMen Day® CGM meets the current clinical requirements for state-of-the-art continuous glucose monitors. High CGM accuracy is crucial for good diabetes management, especially when CGM readings are used for insulin dosing both alone or in combination with open/closed-loop systems. Accurate readings are also essential for precise calculations of time in range. The present analysis suggests that the GlucoMen® Day CGM is a user- and environmentally friendly system that meets the current clinical requirements for state-

of-the-art CGMs. The reduced ecological impact of this needle-free system has to be emphasized and may support further advances in sustainable diabetes technology.

L12. OPTIMISING BOLUS INSULIN THERAPY: CALCULATORS, DOWNLOADS AND GREY MATTER

Mark Evans[1]

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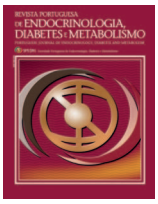
Approximately 50% of insulin dosing whether delivered by insulin pumps or by multiple daily injections (MDI) is "bolus" insulin. This component is usually more varied from dose to dose than background/ basal insulin. This flexibility carries benefits of course in allowing people with diabetes to adapt to daily changes but also challenges for clinical teams in supporting dosing decisions. The more widespread adaption of continuous glucose monitoring will mean that more data are available for interpretation. A common clinical problem is how to target "post prandial spikes". With near-future use of "smart pens" with connectivity, again this brings benefits and challenges in interpreting data and supporting dosing decisions.

I will describe common approaches to guide bolus insulin dosing with both insulin pumps and MDI. I will also discuss some of the more complex areas of bolusing such as how to approach non-carbohydrate fuels (fat and protein), the potential use of directional arrows in CGM devices to modify doses, use of complex bolus options with pumps (or split bolus with MDI), newer insulin analogues etc.

I will also discuss the advantages and drawbacks of bolus calculators supporting people with diabetes with dosing advice. These have moved from insulin pumps and "smart meters" into app-based support. Increasingly, connectivity allows clinicians to download and review insulin dosing decisions but again there are advantages and limitations. Professor Mader and I will show some exemplars in the following case based session. Finally and importantly, "grey matter" means that those living with diabetes still need to review critically any insulin dosing advice and modify as needed, for example for planned exercise/ activity, stress, alcohol, illness, travel/ time zones, environmental temperature, menstrual cycle, etc.

SPEDM

X ADVANCED COURSE OF ENDOCRINOLOGY



E-Posters

PRÉMIO PROFESSOR MANUEL PINHEIRO HARGREAVES / ENDOCRINOLOGIA

EP01. A RARE AND CHALLENGING DIAGNOSIS: HYPOPHYSITIS AND PARASELLAR LIPOMA – TWO RARE ASSOCIATIONS

Mafalda Martins Ferreira[1], Cátia Araújo[1], Mariana Lavrador[1], Patrícia Oliveira[1], Isabel Paiva[1], Bárbara Araújo[1], Sofia Lopes[1]

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Case Report: Thirty six year-old man presented with asthenia, decreased libido, erectile dysfunction, weight gain, decreased muscle mass, thinning of body hair and anedonia. He had a history of Hashimoto's thyroiditis.

He denied previous traumatic brain injury, drug consumption, opioid use, headaches, visual changes or anosmia. Testes were of normal volume and facial dysmorphism was not present.

Blood tests showed hypogonadotropic hypogonadism: FSH 2.7 mUI/mL (<15); LH 2.7 mUI/mL (<9.0); total-testosterone 1.7 ng/mL (2.7-11.0); normal prolactin; morning cortisol of 3.0 µg/dL; Synacthen test showed secondary corticoadrenal insufficiency.

Sellar magnetic resonance imaging exhibited intense homogeneous contrast uptake by the pituitary, without adenomas and a lipoma near the tuber cinerum.

Pituitary antibodies were positive.

Testosterone and hydrocortisone supplementation were started with clinical improvement.

Conclusion: Hypophysitis is a rare and poorly understood condition since pituitary antibodies are difficult to obtain in clinical practice. It typically involves the adenohypophysis. It can be idiopathic, associated with autoimmune diseases (Hashimoto's thyroiditis as one of the commonest) or parasellar lesions (such as lipomas) or secundary to immune-checkpoint inibitors. Histopathological confirmation is rarely performed and it is reserved for equivocal cases. This clinical case describes two rare associations (hypophysitis and parasellar lipoma) and aims to approach and review these two entities and the necessary approach to this diagnosis.

Keywords: Antibodies; Central Adrenal Insufficiency; Hypogonadism; Hypophysitis; Lipoma; Pituitary.

EP02. A RESISTANT PROLACTINOMA

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Introduction: The mainstay treatment of lactotroph adenomas are dopamine agonists. However, a minority of patients will not respond with normalization of prolactin levels and/or reduction in tumor size, even with maximal tolerable doses, and second-line therapeutic options must be considered.

Case Report: A currently 17-year-old woman was referred to Endocrinology due to complaints of galactorrhea and secondary amenorrhea. Initial prolactin levels were 181.4 ng/mL, a pituitary magnetic resonance imaging (MRI) showed a 10 mm adenoma and treatment with cabergoline 1 mg/week was started. During follow-up, prolactin levels kept rising to a maximum of 846.7 ng/mL and the tumor exhibited progressive growth becoming a 17 mm macroadenoma, despite increasing doses of dopamine agonist treatment, up to cabergoline 3.5 mg weekly and bromocriptine 10 mg daily. Headaches and bothersome galactorrhea persisted and the patient was submitted to transsphenoidal surgery in 2020 with an immunohistochemical study compatible with a lactotroph adenoma. A MRI reevaluation one year after surgery showed tumor growth along with prolactin levels on the rise while on cabergoline and bromocriptine therapy. Subsequent treatment options, including reoperation, are being evaluated.

Discussion: The management of drug-resistant prolactinomas remains a clinical challenge. Second-line treatment options include transsphenoidal surgery, radiotherapy and somatostatin analogs with varying reported remission rates.

Keywords: Liver Neoplasms/secondary; Thyroid Neoplasms

EP03. EFFICACY, SAFETY AND QUALITY OF LIFE OF PASIREOTIDE IN FIRST GENERATION SOMATOSTATIN ANALOGS RESISTANT ACROMEGALIC PATIENTS

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Acromegaly is caused by an excessive secretion of growth hormone (GH) by a pituitary adenoma. In most patients, transsphenoidal surgery is the primary approach. Medical therapy is recommended in patients with persistent or relapsing disease after surgery. First-generation long-action somatostatin analogues (LA-SSA), octreotide long-acting release (LAR) and lanreotide autogel (ATG), are suggested as first line medical therapy. Pasireotide LAR (PAS-LAR) is a second-generation LA-SSA that can be used in refractory cases to first-generation LA-SSA. We aimed to retrospectively investigate if T2 weighted qualitative signal identifies IGF-1 and tumor reduction after 1 year of initiation of pasireotide treatment. We evaluated quality of life using AcroQOL. In our center, 4 patients with acromegaly refractory to first-generation LA-SSA were initiated PAS-LAR. All underwent transsphenoidal surgery and were previously treated with ATG, octreotide LAR, pegvisomant, or a combination. No patient underwent radiotherapy. They were started on PAS-LAR three to ten years after diagnosis, and two of them had evidence of extrasellar extension on magnetic resonance imaging at the time of PAS-LAR initiation. Three patients experienced an average reduction in GH of 80.0% and in IGF-1 of 26.5% at 12-24 months after starting treatment with PAS-LAR. A fourth and last patient was recently initiated on PAS-LAR but has not been revaluated since then.

Keywords: Hydatidiform Mole; Hyperthyroidism.

EP04. PANHYPOPITUITARISM SECONDARY TO SELLAR AND SUPRASELLAR METASTASIS FROM BREAST CANCER: A CASE REPORT

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Pituitary metastasis are rare (only 1% of intracranial metastasis). Breast cancer in women and pulmonary cancer in man are the most common primary malignancy metastasizing to the pituitary. Symptoms such as headache, visual field, diabetes insipidus and hypopituitarism are rare.
We report a case of a 64-year-old Caucasian woman with a history of breast cancer, who underwent mastectomy, chemotherapy and hormonotherapy 20 years ago, without cancer recurrence. The patient presented to emergency department with vomiting, headache and stupor. Head magnetic resonance imaging (MRI) showed a massive sellar, suprasellar lesion (29.6x40.4x25.8 mm) that compress the third ventricle, body of the lateral ventricles and optic chiasm and causes hydrocephaly. Thoracic, abdomen and pelvic computed tomography (CT) demonstrated pulmonary, mediastinal and iliac bone lesions. Laboratory data showed low thyroid-stimulating hormone (0.21 µUI/mL), free thyroxine (0.68 ng/dL), luteinizing hormone (<0.10 mUI/mL), follicle-stimulating hormone (0.23 mUI/L), estradiol (<5.0 mUI/mL) and matinal cortisol (1.2 µg/dL), low-normal IGF-1 (53 ng/mL), and high prolactin (67.8 ng/mL). Histology of pulmonary lesions was compatible with metastasis from breast cancer. The patient underwent an external ventricular derivation and started dexamethasone 4 mg 8/8 hours and levothyroxine 75 µg. After multidisciplinary discussion, the therapeutic plan was established with radiotherapy and hormonotherapy with anastrozole. The patient is free of disease progression after 14 months.

EP06. A RARE CASE OF A SELLAR, SUPRASELLAR AND PARASELLAR IMMATURE TERATOMA

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Central nervous systems (CNS) teratomas are extremely rare tumors that can be classified into three types: mature, immature and teratoma with somatic-type malignancy. They occur more frequently in males, children and young adults. The most common symptoms at diagnosis are neurological, most prominently visual defects. When with a sellar, suprasellar or parasellar localization, diabetes insipidus (DI) and panhypotituitarism are frequently encountered. The optimal treatment includes neurosurgical excision. In cases of immature teratomas, a multistep treatment with excision and adjuvant radiotherapy and chemotherapy is usually performed. However, given the rarity of this entity, there are no specific guidelines to orient the treatment.
Here we present a case of a 10-year-old female presenting with headaches and vomits. Two weeks later she was referred to ER after an Ophthalmology routine evaluation that detected anisocoria and left eye palpebral ptosis. A magnetic resonance imaging (MRI) revealed a sellar lesion extending toward the optic chiasm and hypothalamus and bilaterally into both cavernous sinuses. A hormone panel showed corticotrophic and thyrotrophic deficiencies and replacement therapy was started. The patient underwent 2 neurosurgical interventions only achieving partial resection. She was subsequently subjected to chemotherapy and radiotherapy. Postoperative DI was diagnosed and desmopressin was started.
The patient is now 24 years old and doing clinically well under hormonal therapy. Annual MRIs show a stable lesion measuring approximately 4 x2 x3 cm.

EP10. BULLOUS PEMPHIGOID AND DPP4 INHIBITORS: EXPERIENCE FROM A TERTIARY CARE CENTER

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Background: Bullous pemphigoid (BP) is an autoimmune subepidermal blistering disease. The incidence of BP is increasing over the past two decades and there is evidence suggesting that the use of some drugs, particularly dipeptidyl peptidase-4 inhibitors (DPP4i) may be implicated. The exact pathogenesis of how DPP4i may induce BP remains unclear.
Objective: To evaluate the association between the diagnosis of BP and the use of DPP4i in a tertiary care center in Portugal.
Methods: Clinical data from patients with BP was retrieved and retrospectively analyzed using SPSS®.
Results and Conclusion: We collected information from 233 patients with BP, with mean age of 79.3 years, 55.2% of which were men. 113 (48.5%) individuals had type 2 diabetes mellitus (DM), 72 (63.7%) of which were taking DPP4i, mostly vildagliptin. The mean latency between the introduction of DPP4i and the manifestations of BP was 27.9 months. In 34.7% of the patients, the drug was discontinued, allowing reduction of the symptoms.
In the presented cohort of BP patients, there was a high prevalence of DM patients taking DPP4i. Endocrinologists should be aware of this association, since the morbidity of BP and its impact on quality of life are significant.
Keywords: Cutaneous Manifestations; Diabetes; DPP4 inhibitors,

EP14. SUBACUTE THYROIDITIS AND COVID-19

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Subacute thyroiditis is characterized by neck pain or discomfort, a tender diffuse goiter, and a predictable course of thyroid function evolution. It can be caused by a viral infection or a postviral inflammatory process. Many patients have a history of an upper respiratory infection prior to the onset of thyroiditis.
In a study carried out between March 2020 and July 2021, the results showed that 18.8% of diagnosed cases of subacute thyroiditis were associated with COVID-19 infection and 9.3% with the vaccine.
We describe the case of a 44-year-old healthy woman who, two weeks after infection with SARS-CoV-2, started fever, pain and swelling of the neck. She went to the emergency room (ER) and the blood tests showed an increase in inflammatory parameters with very high CRP, suppressed TSH (0.03 uUL/ml) and greatly increased T4L (30.0 pmol/L). A diagnosis was made of post COVID-19 subacute thyroiditis and she was discharged with symptomatic treatment and it was requested for a reassessment consultation.
Complete remission of subacute thyroiditis is common but we need more and longer studies to follow these patients and understand the real risk for long-term sequelae.
Keywords: COVID-19; Subacute Thyroiditis.

EP15. ANAPLASTIC THYROID CANCER: A DIAGNOSTIC AND THERAPEUTIC CHALLENGE

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Introduction: Anaplastic thyroid cancer (ATC) is a rare but highly aggressive tumor, accounting for approximately 50% of thyroid cancer-specific mortality, with a median survival time of about 5 months. Despite recent treatment advances, the prognosis remains poor.
Case Report: A 73-year-old male with a medical history of nodular thyroid disease presented with 1-month history of sore throat, dysphonia and dysphagia. Ultrasound of the neck showed a solid mass in the left thyroid lobe. Ultrasound-guided cytology and biopsy were non-diagnostic. Neck and chest computed tomography scan confirmed a thyroid mass extending into the mediastinum and revealed multiple nodules in the lungs. Laryngoscopy showed left vocal fold paralysis. Total thyroidectomy with central node dissection was performed, with R2 resection. The histological diagnosis was ATC. Molecular analysis (BRAF, ALK, NTRK, RET alterations and PD-L1 expression) was negative. The patient was submitted to neck and mediastinal radiotherapy and chemotherapy. He remained stable for 10 months since diagnosis. However, one month after the end of chemotherapy, he demonstrated disease progression with new metastatic lesions in the skin and scalp.
Conclusion: This case emphasizes the challenges in the management of ATC. A prompt diagnosis, adequate staging and multimodal treatment are critical to optimize patient outcome.
Keywords: Anaplastic Thyroid Cancer.

EP16. PITFALLS IN THE DIAGNOSIS OF PRIMARY HYPERPARATHYROIDISM: RACIAL DIFFERENCES IN VITAMIN D STATUS AND PARATHORMONE LEVELS

Bárbara Araújo[1], Cátia Araújo[1], Ana Carreira[1], Sandra Paiva[1], Isabel Paiva[1]

[1] *Serviço de Endocrinologia, Diabetes e Metabolismo, Centro Hospitalar e Universitário de Coimbra*

Introduction: Normocalcemic primary hyperparathyroidism (NPHPT) is a diagnosis of exclusion and can only be considered after a careful evaluation of causes of secondary hyperparathyroidism. Even physiological factors may influence normal PTH levels, such as BMI and ethnicity.
Case Reports: 55-year-old black female, referenced to Endocrinology for elevated PTH. She had hypertension, stable under valsartan plus hydrochlorothiazide, otherwise unremarkable medical history. At first evaluation: PTH 271 pg/mL(9-72), albumin-corrected-calcium 9.4 mg/dL(8.8-10.6), phosphate 2.9 mg/dL(2.5-4.5), 25OHD 14 ng/mL (insufficiency<30). Renal ultrasound and bone mineral density were normal. She stopped hydrochlorothi-

azide and initiated vitamin-D supplementation. Six months later: PTH 113 pg/mL, creatinine 0.86 mg/dL, calcium 9.4 mg/dL, magnesium 1.9 mg/dL (1.8-2.6), phosphate 3.0mg/dL, 25OHD 34ng/mL, urinary-calcium 162 mg/24h(<250). For one-year PTH levels were 90-115 pg/mL. Thereafter, she was told to increase calcium dietary intake and increase vitamin-D. At the last follow-up, six months after: PTH 71 pg/mL, calcium 9.6 mg/dL, phosphate 3.7 mg/dL, 25OHD 40 ng/mL.
Conclusion: PTH was highly elevated but became corrected after reaching 25OHD levels of 40 ng/mL. Previous studies showed that black patients have higher PTH levels and lower levels of vitamin-D compared to Caucasians, with the greatest difference among vitamin-D-deficient individuals. We emphasize the need for careful correction of vitamin-D in black patients with elevated PTH, before a NPHPT diagnosis
Keywords: Ethnicity; Hyperparathyroidism, Vitamin D.

EP17. PALLIATIVE TREATMENT OF REFRACTORY HYPERCALCEMIA IN PARATHYROID CARCINOMA: THE ROLE OF DENOSUMAB

Ana Carreira[1], Bárbara Araújo[1], Diana Silva[1], Diana Catarino[1], Carolina Moreno[1], Dírcea Rodrigues[1], Miguel Melo[1], Isabel Paiva[1]

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Introduction: Denosumab is an approved therapy for refractory hypercalcemia of malignancy, and its use in unresectable parathyroid carcinoma (PTC) has been described, with favourable results.
Case Report: A 72-year-old women with unresectable recurrence of PTC was admitted with severe hypercalcemia (15.5 mg/dL) and acute kidney injury. Additional investigation revealed PTH=195 1pg/dL (9-72), obstructive nephrolithiasis and cervical lymph node metastases. Two weeks later, she underwent urgent palliative surgery due to refractory hypercalcemia, with marked improvement (calcium=10.8 mg/dL, PTH=480 pg/dL). Five months later, she presented with symptomatic hypercalcemia (13.8 mg/dL), despite therapy with zoledronic acid 4 mg/month and cinacalcet 150 mg/day. Cervical ultrasound confirmed tumour recurrence. Intravenous saline and bisphosphonate were, again, ineffective, with calcium nadir on the second day (11.6 mg/dL) and subsequent increase (12.5 mg/dL). Then, denosumab was started and calcium levels reduced on the third day (11.5 mg/dL), with nadir on the seventh (10.8 mg/dL). She was discharged under cinacalcet 180 mg/day and denosumab 60 mg/month. Currently, at 4-month follow-up, calcium levels and symptoms are stable (10.0-11.3 mg/dL), without further hospitalizations, despite ascending PTH levels (11993 pg/mL). Additional palliative therapies (surgery/radiotherapy) are being discussed.
Conclusion: Denosumab led to rapid reduction and stabilization of calcium values, obviating the need for urgent surgery and reducing hospital stay. This case highlights denosumab's role in palliative treatment of PTC.
Keywords: Denosumab, Parathyroid Carcinoma; Palliative Treatment; Refractory Hypercalcemia.

PRÉMIO PROFESSOR JOSÉ LUIS MEDINA

EP05. IMPACT OF BASAL SERUM ANTI-MÜLLERIAN HORMONE LEVELS ON EMBRYO QUALITY: THE EXPERIENCE OF THE PORTUGUESE NATIONAL REFERENCE CENTRE FOR PREIMPLANTATION GENETIC TESTING

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Introduction: Anti-Müllerian hormone AMH) is an ovarian reserve predictor, although its association with embryo quality remains controversial.
Aim: To evaluate the predictive value of basal serum AMH (bsAMH) on embryo quality in women undergoing Intracytoplasmic Sperm injection(ICSI) with preimplantation genetic testing (PGT).
Methods: Retrospective study including women proposed for a first cycle, from September/2020(first time an embryo biopsy occurred at day 5[blastocyst]) until December/2021. Women under hormone therapy in the previous 3 months, ovarian surgery and cases of male infertility were excluded. The 93 women included were categorized according to bsAMH level: G1(<1 ng/mL)–n=21(22.6%); G2(1.1-2 ng/mL)–n=22(23.6%); G3(>2 ng/mL)–n=50(53.8%). Grade A(excellent)/B(good) blastocysts were considered of superior quality based on morphological criteria.
Results: Women in G3 were younger (p=0.024), presented higher numbers of antral follicles (p<0.001), total blastocysts (p<0.001) and A/B blastocysts (p=0.042), and lower levels of basal Follicle-stimulating hormone (FSH) (p=0.025) and total gonadotropin dose administered (p<0.001). There were no differences for PGT reason, cycle duration, body mass index, basal Luteinizing hormone (LH), estradiol and endometrial thickness. After multivariate analysis, g3 showed a non-significant trend of association with A/B blastocysts counts (p=0.085).
Conclusion: Greater bsAMH levels were correlated with greater numbers of total blastocysts, probably with higher quality. AMH may be a useful qualitative biomarker for these carrier/affected couples who desire to conceive a disease-free embryo.

EP11. INFLUENCE OF CYSTIC FIBROSIS-RELATED DIABETES ON THE SEVERITY OF CYSTIC FIBROSIS PHENOTYPE

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Aim: The aim of this study was to evaluate clinical and laboratorial factors that could be associated with CFRD. The potential impact of CFTR modulator therapy on optimization of glycaemic profile was also ascertained.
Methods: A retrospective observational study was conducted using data from electronic medical records of 48 adult CF patients. We compared patients with and without diabetes and examine the differences regarding pulmonary function, glycemic outcomes, genetic mutation, exocrine pancreatic insufficiency (PI), renal function and respiratory infections.
Results: Patients included in this study had a mean age of 31.7 ± 10.6 years. There was a significant higher prevalence of PI (66.7% *vs* 21.4%, *p*=0.007), pulmonary transplant (33.3% *vs* 7.1%, *p*=0.040) and homozygotic F508del mutation (66.7% vs 25.0%, p= 0.015) in CFRD patients. FEV1 tended to have a lower value in the group of patients with diabetes (CFRD:58.5±20.8%; non-CFRD: 75.4±26.3%, *p*=0.08). CFTR modulator Ivacaftor/tezacaftor/elexacaftor appears to have a positive influence on glucose metabolism.
Conclusion: CFRD is associated with a more severe CF phenotype. Early diagnosis and optimal glycemic profile may reduce the risk of future complications and improve quality of life. Further studies are needed to establish the role of CFTR modulation in glucose metabolism.
Keywords: CFTR Modulators; Cystic Fibrosis-Related Diabetes; Cystic Fibrosis; Glucose Metabolism.

EP12. INADEQUATE WEIGHT GAIN IN WOMEN WITH OBESITY AND GESTATIONAL DIABETES: WHICH ARE THE MATERNO-FETAL OUTCOMES?

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Introduction: Despite of the consensus around the negative impact of obesity in pregnant women, the ideal weight gain during pregnancy in this particular group of women is still under debate.
Objective: To investigate the impact of differential weight gain in the materno-fetal outcomes of pregnant women with obesity.
Methods: Retrospective study of the National Gestational Diabetes Registry from 2011 to 2018, being included all women with body mass index (BMI)≥30 kg/m2 and monochorionic pregnancy. They were divided in three groups according to their weight gain during pregnancy (considering the 2009 Institute of Medicine recommendations): Insufficient weight gain (IWG, <5 kg) versus adequate weight gain (AWG, 5-9 kg) versus excessive weight gain (EWG, >9 kg). Groups were compared considering multiple clinical and analytical variables that include gestational, peri and postpartum maternal-fetal outcomes.
Results: Of the included 4444 patients, 1750 (39.4%) presented IWG, 1143 AWG (25.7%) and 1551 EWG (34.9%). Women with EWG were younger, presented slightly higher third trimester HbA1c, lower BMI and lower rates of previous pregnancies while the opposite was observed with those with IWG (*p*<0.001). First endocrinology appointment was attended earlier in those with IWF and later in those with EWG (*p*<0.001). No difference between groups was found regarding insulin or metformin therapy usage. Patients with EWG delivered heavier babies (3377 *vs* 3262 *vs* 3164g, *p*<0.001), presenting higher rates of fetal macrossomy (10.0 *vs* 5.8 *vs* 4.2%, p<0.001) and pre-eclampsia (5.1 *vs* 4.7 *vs* 3.3%, *p*<0.001) with the opposite happening with those with IWG. There was a tendency towards higher cesarean rates among those with EWG (45.9 *vs* 42.5 *vs* 37.8%, *p*=0.28). There were no differences between groups regarding prematurity, trauma during delivery, polyhydramnios, neonatal hypoglycemia or respiratory distress syndrome, newborn intensive care admission and fetal or neonatal deaths.
Conclusion: Our data demonstrated that most of the major materno-fetal outcomes are not affected by differential weight gain among pregnant women with obesity, with the exception of higher newborn weight and pre-eclampsia rates among the EWG group.

EP19. EFFICACY, TOXICITY AND PREDICTORS OF RESPONSE OF RETREATMENT WITH PEPTIDE RECEPTOR RADIONUCLIDE THERAPY IN PATIENTS WITH PROGRESSING NEUROENDOCRINE TUMOURS: THE EXPERIENCE OF A SINGLE CENTER

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Introduction: Peptide receptor radionuclide therapy (PRRT) is an effective treatment of neuroendocrine tumours (NET). However, patients with metastatic NETs will invariably progress.
Objective: To evaluate the efficacy of a repeat 177Lu-DOTATATE-PRRT course in patients with progressive NET after initial 177Lu-DOTATATE (PRRT1).
Methods: This is a 9-year retrospective observational study of 20 patients who were retreated with PRRT (PRRTR) after PRRT1.
Results: The median progression free survival (PFS) following PRRT1 was 32 months (10.5-70.5). After PRRT1 all 20 patients progressed. From the 20 patients included, 2 were lost to follow-up. The median PFS after PRRTR was 17.5 months (0-62). At the time of analysis, after PRRTR 15/18 patients progressed and 3/18 had stable disease. For the patients that progressed, the median time to progression was 9 months (0-36). The median overall survival from the time of first cycle of PRRT1 was 66 months. Decrease in platelets count after PRRTR was statistically significant (*p*=0.03). No significant renal or liver toxicity was reported, as well as hemoglobin drop. Radiotherapy previous PRRTR was associated with a longer PFS (*p*=0.017) and the presence of metastases pre-PRRTR was associated with shorter time to progression following PRRTR (*p*=0.04).
Conclusion: Patients who progressed after PRRT1 can achieve good PFS with PRRTR and minor toxicity. Radiotherapy pre-PRRTR associates with a longer PFS.

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EP07. ACROMEGALY: GLYCEMIC METABOLISM MAY PREDICT A MORE AGGRESSIVE TUMOR

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Introduction: Acromegaly is a disorder with excess growth hormone (GH) secretion. In the majority of cases, there is a pituitary adenoma responsible for this hypersecretion. It is also associated with the presence of several comorbidities, such as arterial hypertension (HT) and type 2 diabetes mellitus (DM2). However, the association between these comorbidities at diagnosis and the presence of a more aggressive growth hormone-secreting tumor remains to be elucidated.
Aim: To determine if comorbidities at diagnosis are predictors to a more aggressive tumor in patients with acromegaly.
Methods: Patients diagnosed with acromegaly in our center between January 2008 and November 2020 and with a follow-up period of at least 12 months were included. Patients without data regarding the age at diagnosis, tumor characteristics, clinical or biochemical parameters evaluated at presentation were excluded. We considered the clinical and biochemical predictors of a more aggressive tumor at diagnosis as the primary outcome.
Results: Seventy-three patients with acromegaly were included and 63% were females. The mean age at diagnosis was 50.3 ± 15.2 years old and only 1 patient had family history suggestive of familial pituitary adenoma (FIPA). At first evaluation, mean HbA1c levels were 6.6 ± 1.2 % and LDL cholesterol 112.1 ± 31.3 mg/dL, 65.3% of patients presented with a macroadenoma, 38.9% of the tumors had extrasellar extension and 18.1% of were locally invasive. Our results showed a positive association between levels of blood glucose and IGF-1 at diagnosis and the presence of an invasive tumor (OR=1.015, p=0.018; OR=1.002, p=0.009, respectively). There was also a positive correlation between levels of GH and maximum tumor diameter (b=0.037, p=0.019). Furthermore, we highlighted a negative correlation between tumor maximum diameter at diagnosis and levels of total cholesterol (b=-0.80; p=0.017) and LDL cholesterol (b=-0.096, p=0.024) at one-year follow-up.
Conclusion: Higher levels of blood glucose may be associated with a more aggressive tumor at diagnosis, specifically higher prevalence of more invasive adenomas, in patients with acromegaly.
Keywords: Acromegaly; Aggressive Tumor; Diabetes Mellitus; Glucose Metabolism, DM.

EP08. TSH-SECRETING PITUITARY ADENOMAS: A REPORT OF 5 CASES FROM A TERTIARY CENTRE

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Introduction: TSH-secreting pituitary adenomas are a rare cause of hyperthyroidism, accounting for 0.5 to 3% of all functioning pituitary tumors. During the last 10 years, only 5 patients with this disease have been followed in our tertiary centre.
Case Reports: The first patient is a 75-year-old female diagnosed in 2011 in the context of altered blood results. Magnetic resonance imaging (MRI) evidenced an 8 mm microadenoma (Hardy IA), that was submitted to transsphenoidal surgery. Due to disease recurrence 6 months after surgery, monthly Octreotide-LAR 10 mg was initiated, providing disease control to this date.
The second case, a 42-year-old female that presented symptoms and abnormal thyroid function since 2011, was initially medicated with thiamazole. She was correctly diagnosed in 2015, with the evidence of a 10 mm TSH-secreting adenoma. She underwent surgery in 2015 and then again in 2018 due to disease recurrence. The patient is currently medicated with monthly Octreotide-LAR 20 mg and well-controlled.
The third patient, a 46-year-old female diagnosed in 2015 with a TSH-secreting mass of 14 mm with suprasellar extension and abutting the right cavernous sinus (Hardy IIB). She was submitted to transsphenoidal surgery in 2015 and again in 2018 due to disease recurrence. The patient remained clinically and analytically euthyroid to this date (under no medical therapy).
The fourth patient, a 63-year-old woman presented an 8 mm TSH-secreting adenoma. The patient was submitted to transsphenoidal resection which resulted in normalization of thyroid function. Three months after surgery there was a relapse of the hyperthyroidism. She is currently under treatment with octreotide LAR 10 mg/month and well-controlled.
Finally, a 67-year-old man whose thyrotropin secreting adenoma was managed solely with octreotide, presented rapid response with tumoral volume reduction.
Conclusion: These five cases highlight the need for a high index of suspicion for this disease, that sometimes remains undiagnosed for several years. A timely diagnosis is crucial for an effective management plan. This work also stresses the efficacy of first-generation somatostatin analogs as a valuable therapeutic option in these patients.

EP09. LARGEST DIMENSION OF THE PHEOCHROMOCYTOMA IS ASSOCIATED WITH MORTALITY: RESULTS OF THE ANALYSIS OF 62 CASES

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Introduction: Pheochromocytomas are rare adrenomedullary neuroendocrine tumors arising from catecholamine-producing chromaffin cells. Their clinical presentation and anatomical features are variable, highlighting the importance of understanding its particular features to make an accurate diagnosis.
Methods: Cross-sectional study including patients submitted to pheochromocytoma resection surgery in our centre between January 2010 and December 2020.
Results: We included a total of 62 patients, being 57.4% females. The mean age at diagnosis was 50.7 ± 15.7 years. Thirty five percent of the cases were diagnosed as an incidentaloma and, between the ones who were symptomatic at the diagnosis, palpitations/tachycardia (30.6%), headaches (26.5%), profuse sweating (22.4%) and abdominal pain (14.3%) were the most reported symptoms. Hypertension was present at diagnosis in 71.2% of the cases. Most of the lesions were right-sided (56.9%) and only 3 patients (5.2%) had bilateral disease. The tumours measured 52.2±28.3 mm in the largest dimension. Four patients (7.4%) had malignant pheochromocytoma and one patient died from this cause. Around 16% of the individuals had a concomitant malignancy (mostly in colon, kidney and thyroid). There was no association between mortality and age, sex, surgical approach, incidentaloma at diagnosis; however, there was a positive correlation between the largest dimension of the tumour and mortality (OR 1.05, p=0.014). It is important to underline that this is an underpowered study due to the rarity of events (4 deaths). Genetic study was performed in 24 patients: 2 (8.3%) presented *RET* gene mutations, 3 (12.5%) VHL gene mutations, 1 *SDHD*, 1 *SDHB* and 1 *TSC2* gene mutations. Type 1 neurofibromatosis was diagnosed in 2 patients.
Conclusion: This casuistic analysis emphasizes the great heterogenicity in the clinical presentation and imagiological features among patients with pheochromocytoma. The knowledge of these findings, allied with a high index of suspicion, pave the way for an early diagnosis of this disease.

EP13. COVID-19 PANDEMIC IMPACT ON METABOLIC CONTROL OF PATIENTS WITH TYPE 1 DIABETES MELLITUS

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Introduction and Aims: The COVID-19 outbreak imposed changes in the daily life of people around the world. This study aims to evaluate the impact of the COVID-19 pandemic on the metabolic control of patients with type 1 diabetes (T1D).
Methods: We conducted a retrospective observational study in 169 patients with T1D followed in an adult insulin pump consultation. We collected data on anthropometric, blood pressure, continuous glucose monitoring (CGM) and laboratory parameters in three different moments: before, at 6 and at 12 months after the start of the lockdown.
Results: HbA1c decreased during the first year of the COVID-19 outbreak. At 6 months, the variation was -0.12±0.62% (p=0.03). At 12 months it was -0.19±0.86% (p=0.02). Weight increased in the first six months (0.80±3.2 kg, p=0.03), but not at 12 months. There were no other statistically significant variations.
Discussion: The first year of the COVID-19 pandemic did not negatively impact patients with T1D, suggesting that more regular routines can positively influence T1D management.
Conclusion: We observed a significant improvement of glycemic control in patients with T1D, a transitory increase in weight, without significant change in other parameters.
Keywords: COVID-19; Diabetes Management;, Glycemic Control; Lockdown; Type 1 Diabetes.

EP18. PAGET`S DISEASE OF THE BONE: A CASE SERIES OF 83 PATIENTS

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Introduction: Paget`s disease of the bone is the second most common metabolic bone disease. Despite this fact, it is one of the most overlooked diseases in the realm of Endocrinology.
Methods: Cross-sectional study including patients that underwent bone scintigraphy compatible with the diagnosis of Paget`s disease of the bone between January 2010 and December 2020 in our hospital centre.
Results: A total of 83 patients were included (49.4% females), with a mean age at diagnosis of 68.4 ± 12.8 years. The reason that led to diagnosis was an imagiological incidental finding in 35.6%, pain complaints in 28.6% and blood test results in 16.1% of patients; The disease was polyostotic in 60.8% of patients, affecting mainly the bones of pelvis (57.7%), sacrum (27,3%), cranium (27.3%), lumbar spine and femur (both with 19.2%). At presentation, the disease was asymptomatic in 24.6% of the individuals while 67.2% presented complaints of bone pain, 5.9% bone deformity, 7.4% hearing loss and 7.0% bone fractures. More than one third of the patients (35.6%) were not submitted to any treatment, 47.2% were medicated with zoledronic acid and 13.9% with pamidronate. Normalization of the alkaline phosphatase (AF) levels after treatment was achieved in 83.8% of patients (mean AF levels before treatment: 201.5 IU/L; three months after: 86.0 IU/L). A case of osteosarcoma was found. More than two thirds of these patients (67.1%) were followed by rheumatologists, 16.4% by internists and 4.1% by endocrinologists.
Conclusion: Paget`s disease of the bone is a disease with heterogeneous clinical presentation, which probably contributes to the late diagnosis. Despite this fact, even when the diagnosis is achieved, a relevant group of these patients is not submitted to any treatment.

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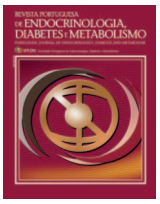

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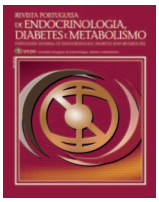

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O registo numa base de dados pública de ensaios clínicos é condição necessária para a publicação de dados de ensaios clínicos na Rev Port Endocrinol Diabetes Metab, de acordo com as recomendações do International Committee of Medical Journal Editors (ICMJE, http://www.icmje.org). Os ensaios devem ser registados anteriormente ou no início do período de recrutamento de doentes. Um ensaio clínico é definido como qualquer estudo de investigação que prospectivamente atribua a participantes humanos, individualmente ou em grupo, uma ou mais intervenções relacionadas com a saúde, com o objectivo de avaliar os seus resultados relacionados com a saúde. As intervenções relacionadas com a saúde incluem qualquer intervenção usada para modificar um resultado biomédico ou relacionado com a saúde (por exemplo, fármacos, procedimentos cirúrgicos, dispositivos médicos, tratamentos comportamentais, intervenções nutricionais e alterações do processo de prestação de cuidados). Os resultados relacionados com a saúde incluem qualquer medida biomédica ou relacionada com a saúde obtida em doentes ou participantes, incluindo medidas farmacocinéticas e eventos adversos. Os estudos puramente observacionais (aqueles em que a atribuição de uma intervenção médica não é do critério do investigador) não exigem registo.

O número de registo do ensaio clínico (TRN) bem como a data desse registo devem ser referidos no final do resumo do artigo.

Disponibilização dos Dados

A Rev Port Endocrinol Diabetes Metab sugere fortemente que todos os conjuntos de dados nos quais se baseiam as conclusões de um artigo sejam disponibilizados para os leitores. Sugere-se assim aos autores que assegurem que os seus dados ficam disponíveis em repositórios públicos (sempre que estes estejam disponíveis e sejam adequados), que sejam apresentados no manuscrito principal ou em arquivos adicionais, sempre que possível em formato tratável (por exemplo, em folha de cálculo e não em pdf).

A Rev Port Endocrinol Diabetes Metab exige uma declaração de disponibilização dos dados, presente no final de cada manuscrito. Para ensaios de fármacos ou dispositivos médicos, a declaração deve referir, pelo menos, que os dados relevantes de cada doente, devidamente anonimizados, estão disponíveis mediante pedido justificado aos autores.

Sugerem-se formulações para a referida declaração:
"Disponibilização dos dados: os dados individuais dos doentes [e/ou] o conjunto completo de dados [e/ou] o anexo técnico [e/ou] as especificações da análise estatística, estão disponíveis em [/doi] [com acesso livre/com as restrições] [do autor correspondente em]. Os participantes deram o seu consentimento informado para disponibilização de dados [ou... não foi obtido consentimento dos participantes, mas os dados apresentados estão anonimizados e o risco de identificação é reduzido... ou não foi obtido consentimento dos participantes, mas os benefícios potenciais da disponibilização destes dados justificam os prejuízos potenciais, uma vez que ...]"

Se os dados não estiverem disponíveis, deve ser referido o seguinte: "Disponibilização dos dados: não estão disponíveis dados adicionais."

Esta opção não se aplica a ensaios clínicos de fármacos ou dispositivos médicos.

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**