tients. Therefore, an early detection of bone related and cardiovascular problems in this patient group will help to improve the therapeutic approach. Secondary hyperparathyroidism due to CKD is possibly one of the most important and most frequent comorbidities which is associated with CKD and a multifactorial dysregulation of bone and mineral metabolism. The respective systemic disorder has been named chronic kidney disease-mineral bone disorder (CKD-MBD), associated with increased cardio- and cerebrovascular calcification in this group of patients. Disturbances of mineral metabolism including parathyroid hormone (PTH), calcium, phosphorus, vitamin D, acidosis, and alkaline phosphatase (AP) are increasing during CKD, abnormalities in bone turnover, mineralization, volume, linear growth, or strength and vascular or other soft tissue calcifications contribute to the clinical outcomes. Bone biomarkers, e.g. PTH and isoforms of AP are increasingly important to generate diagnostic information independently of kidney function to predict underlying bone turnover and fracture risk, as well as diagnostic bone biopsies, which are underutilized. The KDIGO Clinical Practice Guideline for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease–Mineral and Bone Disorder (CKD–MBD) has focussed on the specific problems in CKD patients with regard to their mineral metabolism first in 2009. Since then, not only the attention of clinical doctors and scientists for CKD-MBD patients has increased, there is a number of new insights into bone regulation and its importance via therapy options. Hemodialysis systems, kidney transplantation as well as nutrition and hydratation balance have a large impact on mineral and bone metabolism, but also a large number of medications e.g. phosphate binders and vitamin D supplements, or calcimimetic drugs, based on allosteric activation of the calcium-sensing receptor expressed in various human tissues. Future developments include more sensitive biomarkers to define disease risks in CKG patients and new therapeutic options, e.g. via molecular modulations of new metabolic targets.

KEYWORDS: hyperparathyroidism, chronic kidney disease, diagnosis.

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ACROMEGALY AND MULTIPLE TUMORS

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Acromegaly is associated with increased growth hormone (GH) and insulin-like growth factor-1 (IGF-1) levels which, in addition to the characteristic signs, symptoms and complications of acromegaly, may support tumor development and growth. In 1993 a 52 year old female patient was operated due to a medulla oblongata tumor (no histopathology available). In 2012, aged 71 years she was diagnosed with acromegaly due to typical clinical and hormonal characteristics (IGF-1 586 ng/mL, GH in OGTT 2.38, 3.48, 1.96 ng/mL). However, contrast-enhanced MRI did not reveal a pituitary adenoma. The rest of the pituitary function was normal. We have started to search for ectopic source of GH/GHRH. Firstly, we made abdominal and chest CT (June 2012), which revealed three tumors: solid stomach tumor located on the border of the gastric cardia and corpus, right adrenal gland tumor and right lung tumor, communicating with pleura and lymphatic nodes up to 1.5 cm, located in the mediastinum. The CT also showed hypodense lesion in liver (segment IV b, 1.6 cm in diameter) and heterogeneous echostructure of thyroid gland with right lobe enlargement and left lobe solid-cystic tumor (2.6 cm in diameter). Somatostatin receptor scintigraphy revealed increased tracer accumulation in the right thyroid lobe. No tracer accumulation was noted in the location of the lungs and stomach. Circulating GHRH levels were assessed 3 times with normal values. All tumors were radically resected. The histopathological examination of these neoplasms did not reveal GH secretion. The repeated MRI pituitary gland revealed hypodense lesion 5 mm in diameter, could represent microadenoma. Revision of first MRI pituitary gland showed also this small adenoma on first pituitary MRI imaging. We also made control abdominal CT which showed left kidney tumor: 1.7×1.6×2.0 cm, with clear border, showing a strong contrast enhancement. Patient refused pituitary and kidney surgery. Acromegaly is well-controlled with monthly somatostatin analogue therapy (Octreotide LAR 30 mg i.m.). Despite of numerous further tests, the cause of the disease remains unknown. AIP and MEN1 mutations were excluded. Next-generation cancer panel containing 99 cancer genes did not identify possible unifying gene abnormality in her germline DNA. **Conclusions.** Coexistence of acromegaly and occurring tumours suggests a common aetiology of these disorders. To this time, no genetic abnormality could be identified with the tests that have performed. Whole exome or genome sequencing using germline and tumor sample DNA might further help the identification of a tumour-predisposing genetic alteration.

KEYWORDS: acromegaly, growth hormone, somatostatin, tumors.

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ALCOHOL-INDUCED PSEUDO-CUSHING SYNDROME WITH CHRONIC HYPOKALEMIA CAUSED BY DIURETIC ABUSE: CLINICAL CASE REPORT

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Background. Diagnosis of Cushing's syndrome often remains a challenge, as well as distinction between Cush-

ing's syndrome and pseudo-Cushing. In case of pseudo-Cushing, glucocorticoid excess may be due visceral obesity, anorexia nervosa, depression or alcohol abuse. Here we present a patient with pseudo-Cushing syndrome caused by excessive alcohol consumption and chronic hypocalemia due to diuretic abuse. **Case report.** 39-year-old woman attended neuroendocrinology and bone diseases department with complaints of cramping in the feet, nausea and vomiting, fatigue, weight loss (7 kg for the last 6 months) and lower back pain. Previous medical history included hypertension, low trauma left hip fracture (osteosynthesis was made after) and bleeding gastric ulcer. Cramps presented after the hip fracture. BMD was assessed by hip DEXA: Z-score −1.5SD neck, −1.0 whole hip. In 2015, patient attended general medicine department, where following laboratory investigations were made: calcium 0,97 mmol/l, repeated measurements showed hypokalemia from 2.6 to 3.5 mmol/l. From this point, patient was prescribed with intravenous potassium chloride injections, but potassium blood level remained low (up to 3.0 mmol/l). By the time of admission to neuroendocrinology and bone diseases department, the patient took 12 tablets of potassium chloride (7200 mg) daily, spironolactone 100 mg and calcium carbonate 500 mg twice a day. Physical examination showed no distinct cushingoid signs. Laboratory investigations showed following evidence: ASAT 312 U/l (5—34), ALAT 88 U/l (0—55), bilirubin 32.6 umol/l (3.4—20.5), gamma GT 842 U/l (9—36), potassium 3.2 umol/l (3.5—5.1). Levels for sodium, chloride, calcium, creatinine, alkaline phosphatase (AF) and PTH were normal. Circadian rhythm for ACTH and cortisol was preserved. Levels for plasma ACTH were normal, serum cortisol levels were elevated: morning cortisol 1750 umol/L (123—626), late evening cortisol 1233 umol/L (42—270). Late evening salivary free cortisol was also elevated (15.55 umol/L; reference 0.5—9.4) and urinary free cortisol level was within normal range. A short dexamethasone suppression test showed inadequate suppression of a morning plasma cortisol (355.2 umol/L; reference value <50 umol/L). During the ward round, the patient had breath-alcohol odor, but denied alcohol abuse. Toxicology screen could not be performed due to technical issues. In addition, nurse reported seeing the patient taking unprescribed tablets. Taking all these results into consideration, the diagnosis of Pseudo-Cushing was made. To exclude diuretic abuse, the patient was moved to another ward under strict supervision. Subsequently repeated laboratory investigations did not revealed hypokalemia. **Conclusion.** In cases of clinical and laboratory data mismatch, careful observation is important to exclude drug and alcohol abuse. Setting the diagnosis is troublesome without having technical capability to perform urine diuretic test and toxicology screen.

KEYWORDS: pseudo-Cushing syndrome, hypokalemia, diuretics abuse.

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TYPE 2 POLYGLANDULAR AUTOIMMUNE SYNDROME «SCHMIDT SYNDROME». CASE REPORT

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Introduction. Schimdt Syndrome (Type 2 Polyendocrine autoimmune syndrome) is defined as the co-existence of Addison's disease (insufficiency of surrenalis) associated with autoimmune thyroid disease or Type 1 Diabetis. In our study case we have co-existence of Addison's disease with autoimmune thyroid disease. Over time these patients have a development of other autoimmune disorders in various organs. These include hypergonadotropic hypogonadism, vitiligo, chronic atrophic gastritis, pernicious anemia, autoimmune chronic hepatitis and celiac disease. Antibodies detected are 21 hydroxylase antibodies (21OH antibodies) against adrenal cortex, thyroperoxidase antibody (TPO antibodies). **Clinical case.** Patient *B.T* 28 years old presented in emergency with weakness, fatigue, nausea, vomiting, profuse diarrhea, hypotension (TA=90/60 mm Hg), tachycardia (heart rate =110 beats/min) and widespread hyperpigmentation of the skin and oral mucosa. The patient had an anamnesis approximately 3 years ago that occasionally showed signs of weakness, nausea, diarrhea but not the hyperpigmentation of skin.The patient has made a previous consultation to the infectious disease doctor.

Technique: Cutting 5mm with oral and IV contrast and reconstruction multiplanar.

Data: Thorax parenchymal lesions inferior-free, without the liquid freely;

Liver: normal size, without evident parenchymal lesions without dilatation of bile intra hepatic roads;

Cholecystis has no obvious lesions, without dilatation of bile roads extra hepatic;

Pancreas without obvious lesions;

Spleen, slightly enlarged without visible lesions, the kidneys has no obvious lesions;

Kidneys, without evident lesions;

Glandula surrenalis no obvious lesions;

No stomach lesions evident;

Gout intestine without evident lesions;

Colon without evident lesions;

Retroperitoneal space without adenopathy;

Blood vessels with normal dimensions;

Pelvis without evident lesions.

Treatment: treatment of Type 2 Polyglandular Autoimmune Syndrome is the same as that of the individual disorders. Treatment of primary hypothyroidism: physiologic thyroid hormone replacement with levothyroxine. Our patient's treatment is 50 m.c.g levothyroxine (1.6 m.c.g/kg body) and adjusted every 4—6 weeks to maintain TSH and thyroxine in mid normal range. Chronic treatment of Addison disease: glucocorticoid and mineralocorticoid replacement. The dose of hydrocortisone