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-