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

	BEFORE	After	Normal Range
Blood test	Treatment may 2016	Treatment October 2016	
WBC	5.81×109/L	4.5×109/L	4.0—12.0
RBC	5.51×1012/L	5.9×1012/L	3.85.80
HGB	16.9 g/dL	16.4 g/dL	11.0—16.5
HCT	35%	51.6%	30.0—50.0
PLT	216×109/L	133×109/L	100—300
MCV	68.5 fL	87 fL	80.0—99.0
MCH	30.7 pg	27.6 pg	26.5—33.5
MCHC	483 g/L	31.7 g/L	320—360
GLYCEMIA	72 mg/dL	79 mg/dL	70—110
UREA	40 mg/dL	30 mg/dL	15—40
CREATINEMIA	1.1 mg/dL	1.0 mg/dL	0.7—1.2
URIC ACID	4.1 mg/dL	5.8 mg/dl	4.0—8.5
CHOLESTEROL	170 mg/dL	147 mg/dl	150—200
TRIGLYCERIDES	108 mg/dL	64 mg/dl	50—150
TOTAL PROTEIN	6.4 g/dL	6.8 g/dL	6.4—8.3
Na+	127 mmol/l	134 mmol/l	135.37—145.00
Cl-	102 mmol/l	114 mmol/l	96.00—106.00
Ca++	1.8 mmol/l	2.3 mmol/l	2.2—2.7
K+	8.3 mmol/l	5.1 mmol/l	3.48—5.50
Albumin	3.6 g/dL	3.8 g/dL	3.5—5.5
CORTISOLI 8.00 A.M	0.34μg/dL	2.41 μg/dL	5—25
ACTH 8.00 A.M	712.9	522.3	6—80 pg/Ml
TSH	6	3.63	0,4—4 μIU/ml
ANTI TPO	381	105	3—45 μIU/ml
ANTI 21-HYDROXILAZA	48.5	40.1	<0.1

used was 15—25 mg per day given as $^{2}/_{3}$ in the morning (20 mg) and $^{1}/_{3}$ in the evening (10 mg). Usual mineralocorticoid regimen is fludorcortisone 0.1 mg/day with monitoring of blood pressure, volume status, weight, plasma rein activity, sodium and potassium. **Discussion.** Type 2 Polyglandulare Syndrome typically occurs in early adulthood with a peak during the third or fourth decades and is three times more common in females than in males. This patient with, autoimmune thyroid disease and Addison disease has two major components of Schmidt's Syndrome. 10% of Schimidt's Syndrome patients have all three major DM, Addison and Hashimoto. Patients with autoimmune thyroiditis disease are prone to develope other autoimmune disease .In this case the patient with Addison is associated with Hashimoto thyroiditis.

KEYWORDS: polyglandular autoimmune syndrome, Schmidt syndrome, case report.

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A CASE OF HYPOPITUITARISM AND A SPONTANEOUS REGRESSION OF MASSIVE LESION OF HYPOTHALAMIC AREA

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Introduction. Hypopituitarism is a complex of one or more pituitary hormone deficiency due to diversity of underlying etiology, including rare poor studied forms. Clinical case presentation: we present a case of 57 years old woman with intracranial and extracranial mass lesions localized in medial part of the middle cranial fossa, skull base, third ventricle walls, chiasma opticum, sphenoid sinus and panhypopituitarism. The disease debut was associated with reactivation of chronic polypous sinusitis and left side otitis. It was suggested as a neoplasm due to aggressive progress of neurologic signs such as diplopia, vertigo, facial numbness and typical visualization feature with high contrast accumulation by MRI and positron emission tomography. But histological examination of sphenoid sinus mass, cytological liquor assessment did not reveal any tumor cells and alpha fetoprotein level was normal in contradiction to malignant lesion. Compensation of vital functions by substitution therapy by hydrocortisone 10—15 mg per day, L-thyroxine 75 mkg and desmopressin 0.1 mg twice a day improved overall health of the patient. Spontaneous regression of the vast majority of mass lesion within 6 months confirmed inflammatory process as a probable cause of this accident. **Conclusions.** Infection process could mimic tumor. Clear understanding of etiology of pathologic process in each case is necessary for accurate prognosis and treatment individualization.

KEYWORDS: hypopituitarism, case report.

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