

3. Our report describes the second case of galactorrhea in a male-to-female transsexual in the world. The first case was reported by *R. Flückiger et al.* in 1983.

4. These findings indicate that the mechanism of lactation is independent of chromosomal sex. The possibility of drug-induced galactorrhea in males does exist.

KEYWORDS: prolactinoma, cabergoline, bromocriptine intolerance, spontaneous pregnancy, drug-induced galactorrhea, transgender, lactation.

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ELEVATED T4 AND TSH, APPROACH TO DIFFERENTIAL DIAGNOSIS

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The ‘inappropriate secretion of thyrotropin (TSH)’ syndrome includes two types of central hyperthyroidism: TSH-secreting pituitary adenomas (TSH-omas) and thyroid hormone action resistance (RTH). Both types are characterized by high levels of FT4 and FT3 in the presence of unsuppressed TSH concentrations in contrast to primary hyperthyroidism, where TSH levels are always undetectable. Failure to diagnose these different disorders may result in improper thyroid ablation in patients with TSH-omas or unnecessary pituitary surgery in patients with RTH. Several diagnostic steps should be carried out to differentiate the two types of central hyperthyroidism: laboratory evaluation (alpha-subunit of glycoprotein pituitary hormones (α -GSU), sex hormone-binding globulin, C-terminal telopeptide (CTx); MRI visualization; functional tests should be performed (T3 suppression test and thyrotropin releasing hormone (TRH) stimulation test); genetic analysis. The presence of pituitary lesions on an MRI scan strongly supports the diagnosis of TSH-oma. However, the usefulness of such imaging is limited by the known prevalence of pituitary incidentalomas in healthy subjects. A partial inhibition of TSH secretion after T3 suppression test is seen only in RTH patients. The TSH response to TRH stimulation is usually preserved in RTH patients. The finding of a similar thyroid biochemical phenotype in other first-degree relatives is highly suggestive of RTH. Mutations in the thyroid hormone receptor beta gene are identified in ~ 75–80% of RTH. High α -GSU concentrations and/or high α -GSU/TSH molar ratios are typically present in patients with TSH-omas. Circulating sex hormone-binding globulin levels are usually high in patients with TSH-omas, whilst being of normal level in RTH. Chronic administration of long-acting somatostatin analogs caused a marked decrease of free T4 and free T3 levels in nearly all patients with TSH-omas, while patients with RTH did not respond at all. Echocardiologic examination was performed, no valve pathology was found.

KEYWORDS: TSH-secreting pituitary adenoma, thyroid hormone action resistance, differential diagnosis, somatostatin.

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DIABETES INSIPIDUS

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Diabetes insipidus is a disorder that dramatically interferes with a patient’s everyday life due to the need to constantly replenish the fluids lost in increased urination, which comes amid shortage of synthesis, secretion or action of pituitary hormone vasopressin. Differential diagnosis of types of diabetes insipidus in patients with polydipsia-polyuria syndrome is the main difficulty, for a correct diagnosis predetermines the safety and efficacy of further treatment. This session will present current concepts on the etiology, diagnosis and treatment of central diabetes insipidus (CDI). Comparative characteristics of various preparations of desmopressin for the treatment of the central form of the disease will be discussed, and features of the management of selected patient populations with CDI will be taken in consideration: during pregnancy and lactation, pathology of the thirst sensation, after traumatic brain injury and neurosurgery.

KEYWORDS: diabetes insipidus, differential diagnosis, safety, efficacy, treatment.

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PITUITARY CAUSES OF BONE LOSS

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Different hormonal disorders can influence bone metabolism and cause secondary osteoporosis. In childhood, pituitary diseases can hamper gaining of proper peak bone mass and skeletal size. In the adult life they can stimulate bone loss by increasing bone resorption and decreasing bone formation. The consequence of these processes are decreased bone mineral density (BMD) and trabecular bone score (TBS), deterioration of bone quality, diminished bone strength and finally increased bone fracture risk. Among pituitary disorders such effects are possible in patients with hyperprolactinemia, Cushing’s disease, acromegaly and hypopituitarism. Hyperprolactinemia increases bone resorption and loss of BMD, there is increased fracture risk in patients with prolactinoma. Hypercortisolism due to Cushing’s disease (ACTH-dependent Cushing’s syndrome) diminishes formation and increases resorption of bone, causing trabecular bone loss and increased fracture risk. Moreover, there are decreased calcium absorption and disturbances in sex steroids secretion. In acromegaly, GH excess stimulates bone formation, but concomitant hyperprolactinemia and hypogonadism caused by pituitary