P331

Thyroid papillary microcarcinoma presenting as parotid metastasis: a case report

Juan Antonio García-Arnés, Cristina Maldonado, Rosario Fernández & Montserrat Gonzalo
Hospital Carlos Haya, Málaga, Spain.

Differentiated thyroid carcinoma is the most common endocrine malignant tumor. Papillary carcinoma frequency in our country is 80% of thyroid neoplasia. Papillary thyroid carcinoma usually metastasizes to cervical lymph. Metastases to distant organs are rare and most often affect the lungs, liver and bones. Despite of its anatomical proximity, metastasis parotid have not been described in the existing papillary carcinomas literature. We report a case of parotid gland metastasis as the first manifestation of an occult papillary microcarcinoma.
A 30 year old male is derived to maxillofacial surgery because of the appearance of a painless tumor at the right parotid level. An ultrasound report reveals a right parotid increased in size, with a lesion in its hypoechoic interior, with lobulated margins that measure 23×22×27 mm. With these data, he is intervened, being the parotid tumor resected. Pathologic diagnosis of serous salivary gland (parotid) with papillary adenocarcinoma infiltrating intraparotid lymph nodes and does not affect resection margins. The patient is sent to endocrinology consultation for further study. Exploration of the thyroid is normal showing no nodules or lymphadenopathy. Thyroid ultrasound shows normal size of right and left lobe. A left lobe level there is a well defined solid nodule, hypoechoic of 4×6×6 mm. A cytology by FNA result suggestive of papillary carcinoma. A complete thyroidectomy with central and bilateral lymph node dissection was performed. The pathological report is: Right lobe without alterations. Left lobe with two focus of papillary carcinoma of 0.4 and 0.1 cm which do not infiltrate the capsule. We present the first case of parotid metastasis from an occult thyroid microcarcinoma.

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P332

Medullary and papillary thyroid cancer metastasis in the same lymph node: case report

Merve Yilmaz[1], Arzu Gedik[2], Ahmet Omer Ikiz[3], Tugba Gumus[4], Mehmet Calan[4], Ulku Aybüke Tunc[4], Serafettin Canda[5], Ahmet Alacacioglu[6], Abdurrahman Cömlekci[4] & Tevfik Demir[4]
[1]Endocrinology Department, Gazi Government Hospital, Samsun, Turkey; [2]Endocrinology Department, Agri Government Hospital, Agri, Turkey; [3]KBB Department, Medical Faculty Dokuz Eylul University, Izmir, Turkey; [4]Endocrinology Department, Medical Faculty Dokuz Eylul University, Izmir, Turkey; [5]Pathology Department, Medical Faculty Dokuz Eylul University, Izmir, Turkey; [6]Medical Onkology Department, Izmir Katip Celebi University, Izmir, Turkey.

Introduction
Two tumors originating from different cells in the thyroid to metastasis same lymph nodes is very rare. We aimed to present that the same lymph node metastasis of medullary and papillary thyroid in a case.
Case
A 4-year-old male presented with backache without a formerly known disease, has no other property in the patient's history. Vertebra BT was taken and T_5, T_4, T_6 vertebral metastasis have been detected. Malignant cytology was seen in the biopsy from T_4 vertebrae. Thyroid ultrasound was performed to determine primary tumor foci. Malignant nodule (10×23×23 mm) in the left lobe of the thyroid gland and bilateral multiple pathological lymph nodes in the neck were shown. Fine needle aspiration (FNA) biopsy of the thyroid has been reported as malign. Also medullary thyroid cancer (MTC) was thought because of the height of CEA and calcitonin (Calcitonin >2000 pg/ml, CEA 538.3 ng/ml). Malignant involvement was detected in the left lobe of the thyroid gland and increased FDG uptake was determined in the cervical, mediastinal, hilar, abdominal lymph nodes and multiple bones using PET–CT. The patient underwent total thyroidectomy and neck dissection. Multifocal papillary thyroid carcinoma (PTC) in the right lobe, multifocal MTC in the left lobe and isthmus, MTC metastasis in the multiple lymph nodes, PTC and MTC metastasis association in the central area were reported as a result of pathologic. The patient underwent with external radiotherapy for bone metastases. Then systemic chemotherapy and sorafenib was given by medical oncology.
Conclusion
The MTC originating from parafollicular C cells of the thyroid and PTC originating from follicular cells of the thyroid to metastasis to same lymph nodes is very rare. This situation can be explained by the activation of a common tumorigenic pathway for both follicular and parafollicular cells or coincidental.

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Developmental Endocrinology

P333

Effect of glycemia on sleep indicators for patients with type 1 diabetes mellitus

Ina Darashkevich[1,3], Tatyana Mokhort[2], Sergey Tishkovsky[1,3], Lola Nikonova[1] & Valery Lyschik[3]
[1]Grodno State Medical University, Grodno, Belarus; [2]Belarusian State Medical University, Minsk, Belarus; [3]Grodno Regional Clinical Hospital, Grodno, Belarus.

Study evaluates the impact of glycemia on the parameters of night's sleep for patients with decompensated type 1 diabetes mellitus (T1DM).
Materials and methods
A total of 21 cases with T1DM (HbA1c 8.5%). The monitoring of the average daily glycemia was performed through 'SGMSGold Medtronik' (USA) with estimation of average level of glucose (ALG). Patients divided into groups: group A – 10 patients with ALG ≤7.75 (6.5–8.3) mmol/l, group B – 11 patients with ALG 11.6 (8.9–16.6) mmol/l. All patients underwent polysomnographic monitoring by diagnostic complex 'SOMNOlab2'.

Comparative results ($P<0.05$).

Indicators	Group A	Group B
ALG (mmol/l)	7.75 (6.5–8.3)	11.6 (8.9–16.6)
Duration of hyperglycemia/day (%)	16.5 (6.0–34.0)	68 (21–86)
Duration of normoglycemia/day (%)	80 (61–92)	32 (14–79)
Sleep onset latency (SOL; min)	56.5 (20–133)	14 (12–80)
Efficiency of sleep phase3 (%)	45.6 (30.5–74.8)	35.8 (15.9–50.9)

Results
The analysis revealed a significant correlation between ALG and SOL ($r=0.71$); the duration of hyperglycemia during a day (%) and latency of REM-sleep phase ($r=0.76$); negative correlation between the duration of euglycemia during the day before sleep study (%) and the latency of REM-sleep phase ($r=-0.79$); between HbA1c Sleep latency and ($r=-0.89$) in group A. There was no correlations between factors listed above, but a correlation between ALG and total sleep time ($r=0.83$), efficiency of sleep phase1 ($r=0.85$) and efficiency of sleep phase 2 ($r=0.85$; all $P<0.05$) in group B.
Conclusions
ALG affects on dates of the upcoming night sleep in decompensated T1DM patients. ALG >8.3 mmol/l increases total sleep time, the efficiency of sleep phase 1 and 2. The increasing of ALG extends SOL, latency of REM-sleep phase. Duration of euglycemia during the day reduces latency of REM-sleep phase.

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P334

Physiological expression of thyroid hormone receptors during zebrafish development and effects of their molecular disruption

Federica Marelli[1] & Luca Persani[1,2]
[1]Laboratory of Endocrine and Metabolic Research, IRCCS Istituto Auxologico Italiano, Milan, Italy; [2]Department of Medical Science, University of Milan, Milan, Italy.

Thyroid hormone action defects (THADs) are caused by the defective action of thyroid hormones (THs) through their receptors (TRs). TRs variants (TRα1 or TRβ) are associated with several defects, which depend mainly on the tissue-specific expression of the defective-receptor. One of the most striking manifestations is the deregulation of the hypothalamus–pituitary–thyroid axis (HPT); patients with TRα1-mutations show normal thyroid volume, low T4/T3 and high T3/rT3 ratios. Conversely, patients carrying TRβ-mutations exhibit goitre, high T4 and T3 production with unsuppressed TSH. In this study we take advantage of the zebrafish model to further understand the role TRs during development and in the HPT axis regulation. The tissue-specific expression of TRs are analysed by RQ-PCR and whole mount *in situ* hybridization at several developmental-stages. We than create two different mutant-lines (MOs_TRα1 and MOs_TRβ) using specific morpholinos, and we studied the of the HPT axis by several techniques. In zebrafish the expression of TRs is temporally and tissue-specific regulated. During the embryonic development, TRs are expressed during the first 4 h-post-fertilization (hpf), followed by a down-regulation window until 24 hpf, when the expression is again up-regulated in tissue-specific manner: TRα1 is prevalently expressed in brain, heart, thyroid, and gastrointestinal tract; TRβ is prevalently expressed in the pituitary, eyes and otic vesicles. The generation of different 'heterozygous-like' mutant-lines by specific antisense-RNAs shows that