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Glucose profiles in healthy volonteers assessed by CGMS (Guardian®)

Author/Address of institution

Krummenacher Irène, Fischli Stefan, and Henzen Christoph
Division of Endocrinology and Diabetology, Luzerner Kantonsspital, CH-6000 Luzern 16

Background/Introduction

Continuous measuring of subcutaneous glucose levels is feasible with the Guardian® REAL-Time CGMS, providing additional information about glucose fluctuations in diabetic patients. However, only few data are available describing normal glucose fluctuations, i.e. continuous glucose profiles in healthy people, and there are no data published yet for caucasians.

Methods

Inclusion of healthy volonteers, 18 to 50 years old, with normal BMI and normal oGTT, who had the instruction and placement of the Medtronic® Guardian® REAL-Time CGMS for 48 hours, and who were leading a protocol of carbohydrate intake and physical activity.

Results

A total of 15 probands (7 male/8 female) were included: mean age (SD) 39.8 (9.3) years, mean BMI 21.3 (1.9), mean daily carbohydrate intake 251 (98) g with a minimal intake of 100 g and a maximum intake of 510 g, and a median daily physical activity of 9.5 min (minimum 0 min, maximum 110 min). There were no problems with the Guardian® REAL-Time CGMS, and complete data set were created for all probands with a total of 10.329 glucose readings. The mean 24h glucose concentration was 5.31 (0.87) mmol/l, the minimal glucose level was 3.44 mmol/l, the maximum glucose level 7.55 mmol/l. Glucose readings of the CGMS and the capillary blood glucose levels (for calibration) correlated well (R=0.22, p=0.011).

Conclusion

In healthy volonteers there is a very thight control of glucose fluctutations as assessed with the Guardian® REAL-Time CGMS. Although, there was a great difference in carbohydrate intake and physical activity, glucose concentrations almost always remained in the normoglycemic range.

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Red head, Horner`s syndrome and syncope – where is the link?

Author/Address of institution

R. Locher, I. Krull, M. Brändle

Division of Endocrinology and Diabetes, Department of Internal Medicine, Kantonsspital St. Gallen

Background/Introduction

While thyroid nodular goiter is a common condition, it rarely causes local complications such as superior cava syndrome, carotid sinus syndrome or Horner`s syndrome. We report on a patient with an uncommon cause of a cystic thyroid mass leading to these compressive symptoms.

Case Report

A 68-year-old man presented at the emergency room with a syncope. The clinical examination revealed redness of the head, right sided Horner`s syndrome and a 10x10 cm large, indurated, fixed mass at the right thyroid lobe without local redness or tenderness. CRP and blood leucocytes were grossly elevated, indicating systemic inflammation. Computed tomography of head and neck revealed a spacious cystic nodule of the right thyroid lobe. A purulent appearing fluid was drained by fine needle aspiration and the cytologic and microbiological workup included a bacterial or tuberculous abscess, malignant tumor including anaplastic thyroid carcinoma and metastasis as differential diagnoses. Computed tomography of the chest showed multiple pulmonary tumorous lesions equivocal for lung cancer. Bronchoscopy-guided fine needle aspiration cytology yielded a non-small-cell lung carcinoma, which was compatible with the results obtained from the thyroid nodule. Palliative drainage of the thyroid mass was attempted with partial resolution of the compressive symptoms. During follow-up the patient died from septic shock. Autopsy confirmed a non-small-cell carcinoma of the lung with multiple lymph node metastases infiltrating both thyroid lobes. The red head reflecting a positive Pemberton sign, Horner`s syndrome (miosis, ptosis) and syncope were explained by thrombosis of the vena cava superior, tumor compression of the sympathetic nerve system and carotid sinus.

Conclusion

Local tumor infiltration or metastatic involvement of the thyroid gland by a malignant tumor is a rare, but important differential diagnosis of thyroid nodules. Although a cystic thyroid lesion is not per se suspicious for malignancy, the distinct symptoms caused by local compression led to the likely diagnosis of a malignant disease.

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Familial non-autoimmune hyperthyroidism due to an activating TSH receptor mutation

Author/Address of institution

R. Locher, S. Bilz, M. Brändle

Division of Endocrinology and Diabetes, Department of Internal Medicine, Kantonsspital St. Gallen

Background/Introduction

The most frequent causes for hyperthyroidism are Graves' disease and thyroid nodules with autonomous function which account for more than 90% of thyrotoxicosis.

Case Report

A 63-year-old man with a history of hyperthyroidism first described in childhood was referred. Despite negative TSH-receptor antibodies and absence of thyroid eye disease hyperthyroidism had been supposed to be secondary to Graves' disease. He had never attained an euthyroid state despite thionamide therapy. Clinical examination and thyroid ultrasound showed a large multinodular goiter (~ 60 ml), and subclinical hyperthyroidism persisted despite daily therapy with 7.5 mg carbimazole.
The further diagnostic work-up revealed cardiomyopathy with atrial fibrillation and decreased left ventricular ejection fraction and severe osteopenia, both presumably due to long-standing hyperthyroidism. Remarkably, his family history was positive for multinodular goiter and hyperthyroidism in both his father and grandfather and his son and daughter, suggesting autosomal-dominant, familial non-autoimmune hyperthyroidism. Indeed, an activating mutation (Gly431Ser) in the first membrane spanning domain of the TSH-receptor (TSHR) was found in the patient's son, confirming the diagnosis. Whereas both children underwent thyroid ablation with 131J, our patient has denied thyroidectomy or treatment with radioiodine.

Conclusion

TSHR germline mutations occur as activating mutations in familial non-autoimmune hyperthyroidism (FNAH) or sporadic non-autoimmune hyperthyroidism (SNAH). The diagnosis of FNAH should be considered in cases with a positive family history, early onset of hyperthyroidism, goiter, absence of clinical stigmata of autoimmunity and recurrent hyperthyroidism or resistance to medical therapy, whereas SNAH is characterized by absence of a family history. Although those mutations are rare, their prevalence may be underestimated and should be considered as an important differential diagnosis of hyperthyroidism, particular in patients with any of the described stigmata. The persistent excess of thyroid hormones leads to several organ damages including cardiopathy and osteoporosis. Thus, optimal management of hyperthyroidism due to activating TSHR gene mutation includes total thyroidectomy alone or in combination with radioiodine to destroy all thyroid tissue and prevent recurrent disease.

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Association between circulating cytokine levels, diabetes and insulin resistance in a population-based sample (CoLaus study).

Author/Address of institution

Pedro Marques-Vidal 1; François Bastardot 2; Roland von Känel 3; Fred Paccaud 1; Martin Preisig 4; Gérard Waeber 2 and Peter Vollenweider 2

1 Institute of Social and Preventive Medicine (IUMSP), CHUV and Faculty of biology and medicine, Lausanne; 2 Department of Medicine, Internal Medicine, CHUV and Faculty of biology and medicine, Lausanne; 3 Division of Psychosomatic Medicine, Bern University Hospital Inselspital, and University of Bern; 4 Department of Psychiatry, CHUV, Lausanne, Switzerland

Background/Introduction

The objective of this study was to assess the associations between diabetes, insulin resistance (assessed by HOMA), metabolic syndrome and cytokine (interleukin-1beta - IL-1β; interleukin-6 – IL-6 and tumor necrosis factor-α – TNF-α) and high sensitivity C-reactive protein (hs-CRP) in a healthy Caucasian population.

Methods

Population sample of 2884 men and 3201 women aged 35 to 75. CRP was assessed by immunoassay; the other cytokines were assessed by multiplexed particle-based flow cytometric assay. An oral glucose tolerance test was performed in a subgroup of 532 randomly selected participants to screen for impaired glucose tolerance (IGT).

Results

IL-6, TNF-α and hs-CRP were significantly and positively correlated with fasting plasma glucose, insulin and HOMA-IR. Participants with diabetes had higher IL-6, TNF-α and hs-CRP levels than non-diabetics; after multivariate adjustment this difference persisted for hs-CRP only. Participants with metabolic syndrome had higher IL-6, TNF-α and hs-CRP levels and these differences persisted after multivariate adjustment. Participants in the highest quartile of HOMA-IR had higher IL-6, TNF-α and hs-CRP levels, but these differences persisted for TNF-α and hs-CRP only after multivariate adjustment. No association was found between IL-1β levels and all diabetes and insulin resistance markers studied. Finally, participants with IGT had higher hs-CRP levels than participants with a normal OGTT, but this difference disappeared after adjusting on body mass index.

Conclusion

Subjects with diabetes, metabolic syndrome and increased insulin resistance present with higher levels of IL6, TNF-α and hs-CRP, while no association was found with IL-1β. The increased inflammatory state of subjects with IGT appears to be mediated by BMI.

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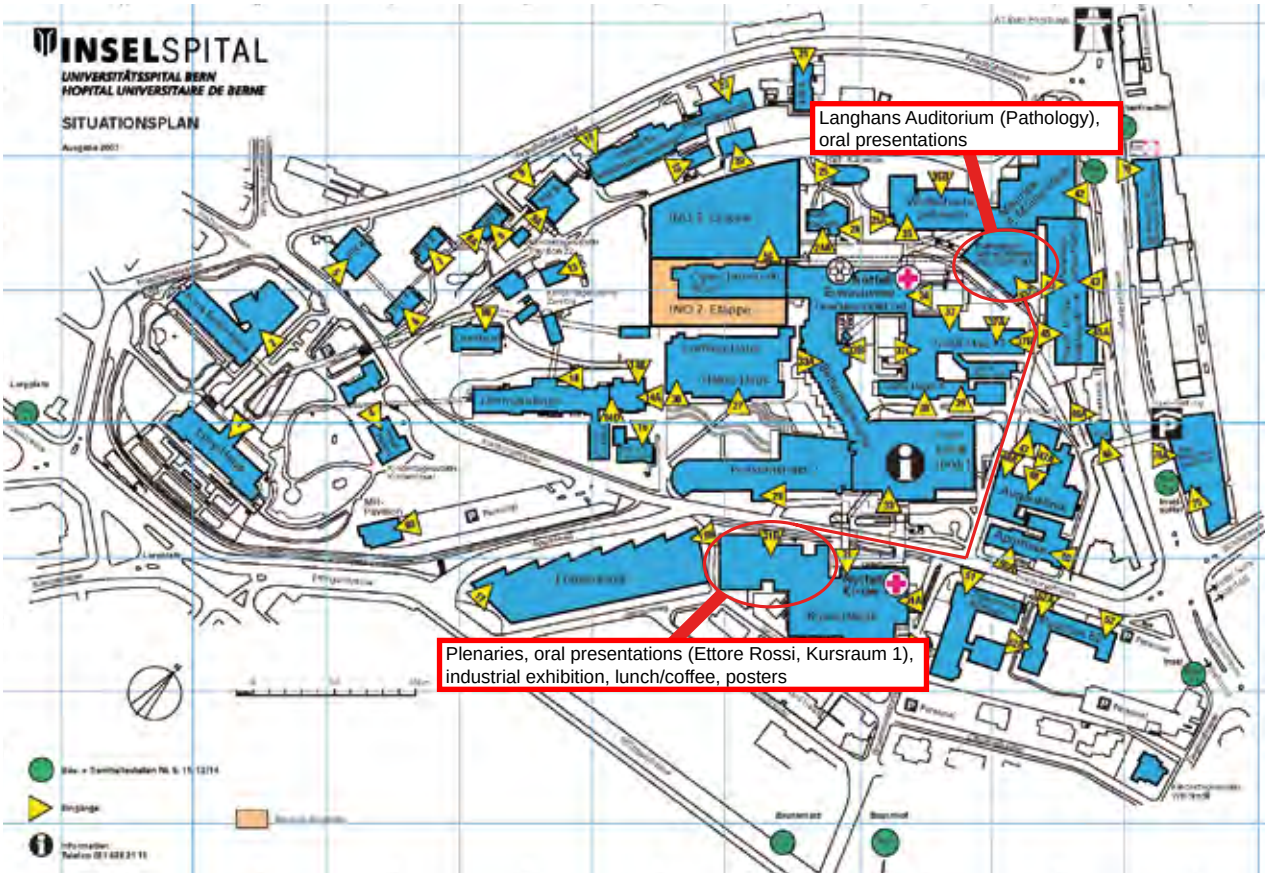

Kontaktadresse:

Schweizerische Gesellschaft für
Endokrinologie und Diabetologie
Rütistrasse 3a
CH-5400 Baden
Tel. 056 200 17 50, Fax 056 200 17 95
office@sgedssed.ch, www.sgedssed.ch