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Case report

Accumulation of FDG in axillary sweat glands in hyperhidrosis: a pitfall in whole-body PET examination

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Abstract. A diabetic male with severe autonomic neuropathy and recently discovered Hodgkin's disease demonstrated bilateral uptake of [2-^{18}F]-2-fluoro-2-deoxy-D-glucose (FDG) in the axillary sweat glands during profuse sweating caused by hypoglycaemia at positron emission tomography examination. It is not yet clear whether the sweating interfered with the distribution of the radiopharmaceutical. Regardless of the cause or mechanism for the uptake, the finding is clinically relevant. A bilateral symmetrical accumulation of FDG in the axillae of a tumour patient does not necessarily indicate malignant involvement of the lymph nodes.

Key words: Hodgkin's disease – Positron emission tomography – Sweat glands – Fluoro deoxyglucose

Introduction

[2-^{18}F]-2-fluoro-2-deoxy-D-glucose (FDG) is a metabolic substrate which traces the first step of glucose metabolism [1, 2]. Although its distribution is non-specific, aberrant accumulation may contribute considerable information to the evaluation of tumour patients [3, 4]. FDG is thus an established agent for detecting and staging of tumours [5], particularly since sensitive positron emission tomography (PET) cameras for whole-body scanning are becoming available. Consequently, the normal anatomical distribution of FDG must be established if the detection and evaluation of neoplastic lesions is to be as accurate as possible. FDG normally accumulates in various tissues and organs [5–8], whereas its uptake in sweat glands has not been previously reported.

Here we report that a diabetic male with severe autonomic neuropathy and recently discovered Hodgkin's disease demonstrated uptake of FDG in the axillary apocrine sweat glands during profuse sweating in a hypoglycaemic condition.

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Case report

A 42-year-old male immigrant from Lebanon was admitted to Karolinska Hospital for severe insulin dependent brittle diabetes mellitus and recurrent diarrhoea. He had had diabetes for 4 years and had been insulin dependent for 2 years. Stool cultures for common enteric pathogens were negative. Gastric emptying was delayed and a urinary bladder dysfunction was observed. Both these findings and the diarrhoea were considered to be caused by autonomic neuropathy related to his diabetes. Furthermore, he had well-controlled psoriasis and had had a silent myocardial infarction with persistent moderate heart failure.

At physical examination several lymph nodes in the left inguinal region were enlarged. Biopsy specimens showed Hodgkin's disease with a histological subtype of mixed cellularity. Further evaluations including bone marrow biopsies, chest radiography, CT examination of the abdomen and blood status were negative. To complete the tumour staging, a PET examination of the thorax and abdomen was performed. The patient had fasted for 4 h without changing his insulin medication. The examination was started 20 min after the intravenous administration of 300 MBq FDG. Seven consecutive 10-min scans with 10 cm field of view were obtained using an ECAT EXACT 31 camera (CTI, Knoxville, Tenn., distributed by Siemens Medical Systems, Iselin, N.J.). Transmission scans and scatter correction were not performed. Images were reconstructed using filtered back projection and a Hann filter with a cutoff frequency of 0.4 cycles/cm.

By the end of the 70-min PET procedure, the patient showed apparent signs of hypoglycaemia with profuse sweating and paleness. Immediate analysis revealed a

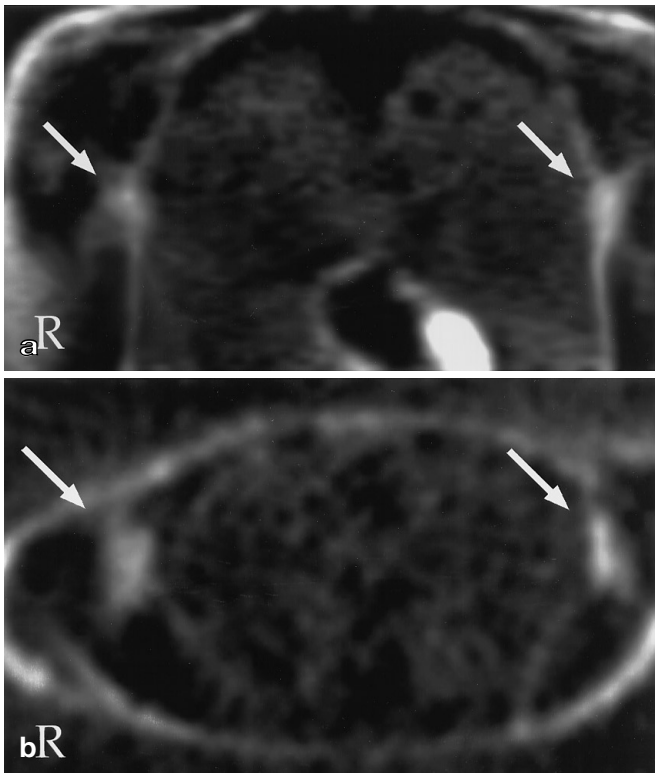

Fig. 1 a, b. Uptake of [^{18}F]-FDG in the axillary sweat glands *(arrows)* of a diabetic male during profuse sweating due to hypoglycaemia at PET examination. **a** Coronal section; **b** transverse section. Section level is indicated in the opposite image

markedly reduced blood glucose level (1.9 mmole/l, normal 4.0–7.0 mmole/l). The PET images revealed that in addition to a tumour uptake in the groin, there was symmetrical tracer accumulation in the axillae (Fig. 1). Careful physical examination performed on the same day as the PET scan revealed nothing pathological in the axillae. Complementary examinations were made prior to the institution of specific therapy. Neither US and CT 7 days nor MRI 15 days later revealed pathology in the axillae. The patient received no antimicrobial therapy during this time period. The Hodgkin's disease was classified as stage 1A (localised disease restricted to one lymphatic station). He was treated by local radiotherapy of the left groin and followed clinically for 5 months. He showed no signs of developing disease in the axillae during this period.

Discussion

Although the precise duration of the patient's hypoglycaemia could not be assessed due to difficulties in communication, he was seriously ill at the end of the PET study. This was apparently due to his unstable diabetes and the fact that his insulin dose was not reduced even though he had fasted for the examination. The last insulin was administered 3.5 h before the injection of FDG, and his blood glucose may well have been low at this time. The profuse sweating the patient experienced is characteristic of hypoglycaemia. In contrast, at normal blood glucose levels the autonomic dysfunction would be expected to impair sweating. The very symmetrical FDG uptake pattern and the negative complementary examinations indicate that the axillary uptake is due to normal anatomical structures not involved by the malignancy. In addition, in a previous series of patients we have found a normal uptake in apocrine axillary sweat glands at examination with ^{99m}Tc-hexakis-2-methoxyisobutyl isonitrile (^{99m}Tc-MIBI, ^{99m}Tc-Sestamibi, Cardiolite, DuPont Ltd., Stevenage, UK), which is also a metabolic tracer that may be used for tumour depiction [9]. Thus, we propose that the axillary uptake of FDG is in the sweat glands. No other areas with a high density of sweat glands showed increased uptake. This may be explained by the fact that the axillary sweat glands are *apocrine* glands, which are different histologically and functionally from other sweat glands, which are *holocrine*.

The exact reason for the FDG accumulation is unknown. However, FDG uptake reflects total metabolism. It can be expected that metabolic activity is required to make and to secrete sweat. Consequently, the uptake of FDG may be a normophysiological finding observed during increased metabolic activity of the apocrine sweat glands. Regardless of the exact mechanism, the finding is clinically relevant. Accumulation of FDG in the axillae of a tumour patient may be benign and does not necessarily indicate a malignant involvement of the lymph nodes.

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