Clinical note

Normal blood glucose level and $^{18}$F-FDG PET/CT

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**Abstract**

Patient preparation for FDG PET studies is perhaps more critical and more complex than for any other commonly performed imaging procedure. We report a patient with normal blood glucose level prior to the execution of a PET study in which FDG uptake was virtually zero in internal organs and was very extensive in large muscle groups. The patient recognizes ingestion several minutes before the test. Ten days later, a repeated PET scan with normal blood glucose level, showed a normal organs distribution of FDG.

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**Introduction**

There is a wide range of causes that are considered, as potential false positives because cellular uptake of FDG is not specific to malignant tumors. Also described are other causes of false negative result, such as hyperglycemia, since glucose and FDG compete for the same membrane transporter in cellular uptake. Several studies defined ideal blood glucose limit for the completion of a FDG PET-CT values below 150–180 mg/dl and such procedures and guidelines are described. Currently there are several different lines of work around the real influence of the values of hyperglycemia and cellular uptake of FDG, and depending on the type of hyperglycemia, chronic or acute. Skeletal muscle has no significant uptake of FDG at rest, but during exercise, myocytes use glucose as an energy source, so the usual protocols are recommended to avoid doing exercises on the day of the performance of the test. The distribution and characteristics of FDG uptake in the muscles can in most cases correct identification. However, sometimes this is a very intense hypermetabolism and does not affect the whole muscle leading to asymmetric or isolated deposits and thus make a study uninterpretable and findings misleading.

The predominant glucose transporter in muscle GLUT-4 is insulin-dependent.

Initial studies have shown that there are distinct proximal pathways that are responsible for the stimulation of GLUT4 translocation and glucose transport as well produced by exercise or by the insulin. We report a case where, despite maintaining glucose levels within acceptable limits for the performance of a PET, $^{18}$F-FDG uptake by the internal organs was very low.

**Case report**

A 46-year-old woman diagnosed with type B non-Hodgkin lymphoma diffuse large cell mediastinal in March 2006, treated with chemotherapy CHOP-Rituximab type scheme that ended in November 2006, achieving complete remission is reported. Studies are performed by $^{18}$F-FDG PET/CT annually for four years and found no evidence of residual disease or recurrence. The patient was referred to our unit for performing a PET/CT without intravenous contrast, according to standard protocol, including fasting for 6 h before the test and avoiding physical exertion during the previous day. On arrival, personal data and preparation for the test are verified with a questionnaire and the patient takes a peripheral intravenous catheterization venous, obtaining a blood glucose level of 120 mg/dl. 355 MBq of $^{18}$F-FDG was then administered and then 500 cc of saline given during the 50 min waiting time. The patient is lying on a stretcher, wrapped in a blanket, without auditory or light stimuli. Water is also used as a negative oral contrast before the images. PET/CT is performed at 60 min after

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Fig. 1. First study with $^{18}$F-FDG carried out when the patient had blood glucose level of 120 mg/dl. From left to right: coronal images of CT, PET, fused PET/CT and maximum intensity projection showing diffuse intense uptake in the large muscle groups.

Fig. 2. Second study, ten days after the first, with blood glucose level of 109 mg/dl. In coronal images, internal organs and low muscle uptake are displayed properly.
injection of the radiotracer, with 3 min per bed and low-dose CT, on a computer Discovery ST PET-CT (GE Healthcare).

The images obtained (Fig. 1) show intense FDG uptake in all major muscle groups of the axial and peripheral skeleton bilaterally and symmetrically, as well as heart muscle, and no uptake in other internal organs. Talking with the patient again, she admitted to having a small snack before arriving to the service. This study were assessed as non-diagnostic, and ten days later, she carried out a second study PET/CT with the same protocol, in this case with a preinjection blood glucose level of 109 mg/dl and getting the images at 55 min after injection. In this second study (Fig. 2) a physiological distribution of FDG is displayed in both internal organs and in other structures, not shown in macroscopic metabolically active malignant disease.

Discussion

Insulin in a diabetic person is produced at baseline and after eating. The secretion of insulin by the pancreatic beta cell is different in both situations. At baseline, the beta cell secretes very small amounts of insulin to keep the system stable and ensure that the liver does not make too much glucose in a tricky balance because the effect of insulin varies depending on the time of the day and the secretion of insulin takes place in pulsos. The secretion of insulin after eating program is produced by a cascade consisting of two phases. The first phase is fast and intense, lasts 15–30 min, giving a high peak of insulin, higher than that of glucose indicates. This discrepancy seems to be that the insulin takes time to become effective because the receptor has to stimulate cellular and allow changes in the role of cellular enzymes. This first phase will have two important effects. As it is secreted by the pancreas into the portal system, the first will cause the cessation of production of glucose in the liver. The second effect of this first phase is to “prepare” or “priming” the system in glucose uptake in other tissues giving preference to the muscular system that will capture 80% of glucose intake, opening the door of the GLUT transporters-4, and entry of glucose into cells. Insulin secretion in this first phase is amplified by the cephalic phase (vagus nerve that stimulates acetylcholine secreted by the islets) and the secretion of digestive hormones or local incretins. For when the food begins to be absorbed and enters the blood stream increasing blood glucose levels higher, the beta cell to secrete insulin has been at least 15 min before alerting the entire system.

The second phase of insulin secretion is less intense and takes about 2 h. The door of the transporters GLUT 4 is open, one can only keep it open, for the rest of the glucose continues to penetrate into cells with a slightly elevated insulin levels from baseline. When all the glucose goes into cells, blood glucose returns to normal and beta cell secretion passes “basal”.

In this context, PET/CT after a recent ingestion may be on the first phase of the cascade of insulin secretion that can vary from 15 to 30 min depending on the patient. In this situation, normoglycemia levels, even in ranges away from the upper margin of tolerance for it, would not be reliable for a proper distribution of FDG and should consider delaying the radiopharmaceutical injection 6 h after ingestion.

References