Potassium Chloride Infusion as the Cause of Altered Bio Distribution of 18F-Fluorodeoxyglucose on Whole-Body Positron Emission Tomography-Computed Tomography Scan

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Abstract

18F-fluorodeoxyglucose (18F-FDG) positron emission tomography-computed tomography is a standard diagnostic imaging tool in many types of cancer. Its physiological in vivo distribution includes the brain, liver, heart, kidneys, and urinary tract at 1 h after tracer injection. Skeletal muscle is known to show variable amounts of 18F-FDG uptake because it has a relatively high-glucose metabolism. We report a case of a 20-year-old patient with gross 18F-FDG uptake involving multiple muscle groups and its likely correlation to potassium chloride infusion before 18F-FDG injection.

Keywords: Potassium chloride and insulin, potassium chloride and muscles, potassium chloride infusion and 18F-fluorodeoxyglucose positron emission tomography-computed tomography scan

Introduction

18F-fluorodeoxyglucose (18F-FDG) is the radiotracer most commonly used for positron emission tomography-computed tomography (PET-CT) imaging. 18F-FDG molecule acts like glucose during initial enzymatic reactions within cells, but the altered structure prevents further metabolism, and it accumulates in most tissues at a rate proportional to glycolysis. Malignant cells have increased glucose transporter 4 (GLUT4) proteins on their cell surface as well as enhanced rates of glycolysis which facilitates their detection utilizing 18F-FDG PET-CT imaging. Unfortunately, 18F-FDG is not a cancer-specific agent, and its uptake has also been described in a number of inflammatory lesions. There are many potential pitfalls and artifacts associated with 18F-FDG PET-CT imaging hence it is important to know these pitfalls and recognize the important areas of normal uptake of 18F-FDG or absence of uptake that may or may not be of significance. This is necessary so that patients can be optimally prepared for their scans and accurate interpretation can be made.

Case Report

We present a case of a 20-year-old boy with a history of kidney transplantation for medical renal disease 6 months ago, who presented with generalized weakness, weight loss, fever, and loose motions and was referred for an 18F-FDG PET-CT scan. His K+ was 2.8 mEq/l (N: 3.3–4.8), Na+ was 124 mEq/l (N: 135–147), Cl- was 114 mEq/l (N: 101–111), serum creatinine: 3.4 mg%
Because of frequent episodes of loose motions, he had developed hypokalemia for which he required potassium chloride (KCl) infusion (10 cc KCl in 0.9% NS at a rate of 100 ml/h) which was continued till the time of \( ^{18} \text{F-FDG} \) injection. The whole-body PET-CT scan demonstrated altered physiological distribution of \( ^{18} \text{F-FDG} \) with markedly increased uptake in almost all major muscle groups, including those of the neck, thorax, abdomen, pelvis, and the extremities [Figure 1] and reduced uptake in organs that physiologically show much better uptake for example, liver. There were no obvious morphologic changes in these muscles on CT scan. Hence, the study was not interpretable. The patient’s medication at the time of \( ^{18} \text{F-FDG} \) PET-CT scan included KCl infusion and steroids (Deflazacort). There was no history of incomplete fasting, hyperglycemia, insulin administration, or excess muscle activity before imaging. The whole-body \( ^{18} \text{F-FDG} \) PET-CT scan was repeated after stopping KCl infusion 12 h before \( ^{18} \text{F-FDG} \) injection. It showed normal biological distribution of \( ^{18} \text{F-FDG} \) [Figure 2] with abnormal increased diffuse heterogenous uptake in enlarged pancreas with adjacent stranding [Figure 3] raising possibility of pancreatitis, and there was a minimal uptake in muscles. Pancreatitis was subsequently confirmed by serum amylase and lipase levels which were raised (220 u/l and 513 u/l, respectively).

**Discussion**

\( ^{18} \text{F-FDG} \) PET-CT scan has been growing as a standard diagnostic tool in patients with oncological and nononcological conditions such as pyrexia of unknown origin. This imaging modality takes advantage of the overconsumption of glucose by tumor and inflammatory cells.\(^{1,2}\) A detailed knowledge of these conditions as well as normal physiological uptake at various sites is therefore important to avoid a mistaken diagnosis or missing a diagnosis. \( ^{18} \text{F-FDG} \) activity in muscle is frequently encountered on \( ^{18} \text{F-FDG} \) PET-CT scans. Normal muscles accumulate little \( ^{18} \text{F-FDG} \), but muscles exercised just before or around the time of \( ^{18} \text{F-FDG} \) injection can exhibit intense \( ^{18} \text{F-FDG} \) uptake which is usually localized. Diffuse whole body muscle uptake can be caused by administration of insulin in hyperglycemic patients, recent food intake, and strenuous exercise that involves many muscle groups.

Elevated concentration of glucose in blood stimulates the release of insulin, and insulin acts on cells throughout the body to stimulate uptake, utilization, and storage of glucose. Insulin mediates the entry of glucose as well as \( ^{18} \text{F-FDG} \) into muscle, adipose tissue, and several other tissues and it also stimulates the liver to store glucose in the form of glycogen. Insulin increases blood flow and glucose extraction in skeletal muscles by stimulating glucose transport and phosphorylation via translocation of the insulin-sensitive GLUT4\(^{3}\) and an increase in hexokinase II activity\(^{5}\) resulting in increased \( ^{18} \text{F-FDG} \) uptake in muscles. Insulin also increases the permeability of many cells to potassium, magnesium, and phosphate ions. Insulin causes potassium to shift into the cells thereby decreasing the extracellular K\(^{+}\) level.

The effect of potassium ion on insulin release is clinically important. When there is more free K\(^{+}\) concentration in blood, it causes closure of the ATP-sensitive
K⁺ channel (KATP channel). This event ends up depolarizing the plasma membrane and opening the voltage-sensitive Ca²⁺ channels, which in turn increases the intracellular concentration of calcium and causes the exocytosis of insulin granules into the bloodstream (Macdonald et al., 2005).

In this patient, the typical distribution of ¹⁸F-FDG uptake and information from correlative CT scan leaves little doubt that the F18 FDG uptake corresponds to the muscular uptake. Furthermore, the patient was so ill that he had not undergone any strenuous physical activity before scanning. He also had no history of shivering, convulsions, or insulin administration around the time of examination. However, the patient had received KCl infusion for hypokalemia just before the scan.

**Conclusion**

Although the intensity and extent of ¹⁸F-FDG muscular uptake, in this case, are spectacular, the actual cause remains speculative. However, the overall evidence seems to be in favor of KCl infusion causing a metabolic disturbance, which in turn increases the ¹⁸F-FDG metabolism in all muscle groups. Such a mechanism would explain the global nature of the skeletal muscle uptake demonstrated in this patient as compared to the normal physiological distribution of ¹⁸F-FDG seen on the scan repeated a day later after stopping KCl infusion 12 h before the scan. To the best of our knowledge, this appears to be the only case report with not much data available in literature regarding possible effects of KCl infusion on biodistribution of ¹⁸F-FDG. Hence, more observation would be needed to support this for inclusion during patient preparation.

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**Conflicts of interest**

There are no conflicts of interest.

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