

## Assessment of the Effect of Blood Glucose Levels on Standardized Uptake Value (SUV) and $^{18}\text{F}$ -FDG Uptake at Tumors and Normal PET Examinations

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### Abstract

This study evaluated the relationship between the blood glucose level (BGL), maximal standardized uptake value (SUV<sub>max</sub>), and  $^{18}\text{F}$ -FDG uptake in tissues at normal and tumour organs with positron emission tomography/computed tomography (PET/CT) examinations. On the morning of the procedure, finger-stick fasting blood glucose is routinely assessed. The study looked at the SUV<sub>max</sub> in the liver and bone of 200 people with F-FDG PET/CT scans for tumors and healthy organs<sup>18</sup>. The study formed three groups of patients based on their serum glucose levels. The study retrospectively examined the relationship between glucose levels and standardized uptake values. The mean liver and bone SUV<sub>max</sub> gradually decreased as blood glucose levels increased, starting at 160 mg/dl. All the groups whose blood glucose levels were between 100 and 160 mg/dl had a slight but significant increase in the uptake of  $^{18}\text{F}$ -fluorodeoxyglucose (FDG) in bones and livers compared to the group whose BGLs were all normal. The study concludes that following a BGL of 160 mg/dl, hyperglycemia progressively lowers the absorption of  $^{18}\text{F}$ -FDG by the liver and bones. Studies using FDG-PET are especially inaccurate when BGLs are high. Therefore, individuals undergoing FDG-PET examinations should fast and consider their BGLs.

### Article Info.

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Blood Glucose,  $^{18}\text{F}$ -FDG, SUV<sub>max</sub>, Bone, Liver.

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### 1. Introduction

Positron emission tomography/computed tomography (PET/CT) is a commonly used imaging technique in cancer for the purposes of diagnosing, staging, and evaluating the effectiveness of treatment [1, 2]. The standard tracer used to assess neoplastic tissue is  $^{18}\text{F}$ -FDG, deoxy-2- $^{18}\text{F}$  fluoro-D-glucose, a glucose analogue tagged with radioactive  $^{18}\text{F}$ . The FDG-PET scan plays a vital role in cancer imaging due to its high sensitivity in detecting many forms of malignant tumors, which is attributed to their elevated glycolysis and metabolism rates compared to normal tissues [3, 4].

The efficacy of  $^{18}\text{F}$ -FDG PET imaging in cancer is contingent upon several factors that enhance glucose uptake within the tumor, including phosphorylation or dephosphorylation, and molecular shifts in glucose transporters [5]. As a result, several authors have documented that elevated blood glucose levels (BGL) impede the absorption of  $^{18}\text{F}$ -FDG in malignant lesions [4]. It has been reported that acute hyperglycemia, which is primarily observed in glucose-loading studies, substantially decreases the uptake of tumor tracers [6]. compared to healthy tissue, most cancerous cells absorb more glucose and  $^{18}\text{F}$ -FDG due to increased glycolysis and metabolism. In malignancies, the glucose transporter type (GLUT) (GLUT-1 and GLUT-3) transporters are significantly overexpressed [4]. It has also been shown that increased expression of tumoral hexokinase [7] may have an even greater impact on  $^{18}\text{F}$ -FDG uptake than GLUT upregulation. The differentiation in  $^{18}\text{F}$ -FDG absorption and subsequent positron emission across tissues enables the detection and diagnosis of tumor activity [8].



In addition, elevated BGLs stimulate the liver to increase the production of GLUT-2 [9]. Consequently, elevated BGLs can also result in heightened hepatic  $^{18}\text{F}$ -FDG absorption; BGLs will often elevate insulin levels. Insulin is recognized for its ability to enhance the absorption of glucose (and consequently  $^{18}\text{F}$ -FDG) into liver cells via the GLUT-2 transporter, despite the fact that the transporter itself is not influenced by insulin [10]. Insulin enhances glucose absorption in peripheral tissues by influencing the activity of the GLUT-4 transporter. More precisely, elevated levels of insulin prompt the recruitment of the GLUT-4 transporter to the outer membrane of cells, thereby enhancing the absorption of glucose in muscle and adipose tissue [11]. Thus, due to the zero-sum characteristic of  $^{18}\text{F}$ -FDG uptake, a reduction in tumor  $^{18}\text{F}$ -FDG uptake may occur [12].

Due to the significant impact that glucose levels have on the uptake of  $^{18}\text{F}$ -FDG, the majority of positron emission tomography (PET) centers endeavor to regulate and observe the glucose levels of patients prior to the PET scan [13]. A few days prior to the scan, a low-carbohydrate diet is recommended by a number of PET centers. It has been established that basal  $^{18}\text{F}$ -FDG uptake in tissues including the liver [14], myocardium, and brown fat [15] can be influenced by low-carbohydrate diets prior to PET scanning. A fasting regimen is implemented prior to the examination in order to mitigate the immediate competitive effects of glucose. A finger stick glucose measurement is conducted promptly prior to the examination, and in the event of an excessively elevated BGL, the scan may be canceled. Although glycemic control measures are frequently used on the examination day, prior research has yielded contradictory findings concerning their ability to improve the diagnostic accuracy of PET [16].

This study aimed to assess the impact of BGLs on the maximum standardized uptake value ( $\text{SUV}_{\text{max}}$ ) and  $^{18}\text{F}$ -FDG activity in both tumors and healthy organs during PET/CT exams.

## 2. Materials and Methods

The study comprised a total of 200 subjects (116 females and 84 males). The age range of the patients was 20 to 88 years, with a mean age of  $59.79 \pm 15.29$  years. The main diagnoses were bone cancer (50), liver cancer ( $n=50$ ), normal bone ( $n=50$ ), and normal liver ( $n=50$ ). Participants were to AL-Safeer Hospital, Baghdad, patients from March 2024 to April 2024. This study does not require ethical approval. Patients who had undergone chemotherapy including radiotherapy within 4 weeks before the examination were excluded from the study.

The nurse at the imaging center performed finger stick blood glucose measurements before the PET/CT examination. All subjects in the study were categorized into three groups according to their glucose levels: group I with glucose level  $<100$  mg/dl, group II with 100–160 mg/dl glucose level, and group III with 160–184 mg/dl glucose level.

Patients with fasting BGLs exceeding 200 mg/dl were excluded from participating in any part of the study. Each patient abstained from eating for a minimum of four to six hours prior to getting an injection of  $^{18}\text{F}$ -FDG. An intravenous injection of  $^{18}\text{F}$ -FDG was administered, with a mean dosage of 7.37 mCi for normal cases and 7.36 mCi for patients. The initial assessment was evaluating the connection between BGLs and  $\text{SUV}_{\text{max}}$  and  $^{18}\text{F}$ -FDG in various tissues of all patients.

## Procedure

The first step was to choose patients with different body weights from the department's database. The patients' weight ranged from 33 to 130 kg. On each of the chosen patients, a scan was done according to a set protocol. And after fasting for at least 4-6 hours but well hydrated, the patient is intravenously injected with  $^{18}\text{F}$ -FDG before the PET/CT scan. During the injection and the following uptake phase of one hour preceding

the scan, the individual has to rest in a quiet room, comfortable, warm and relaxed. The patient must try to avoid muscular or regional cerebral activity because this will increase FDG uptake in those areas. Since it is impossible to avoid breathing and/or swallowing, the glucose uptake cannot be controlled in this case.

$SUV_{max}$  is defined as the tissue activity concentration (MBq/ml) as determined in a chosen region of interest (ROI) or volume of interest (VOI) relative to the injected activity ID (MBq) corrected for decay of the radionuclide multiplied by the body weight (g) can be calculated by the Eq. (1) [17].

$$SUV (G/ML) = \frac{\text{AVERAGE ACTIVITY IN ROI (MBQ/ML)}}{\text{INJECTED DOSE (MBQ)}} \times \text{BODY WEIGHT(G)} \quad (1)$$

## 2. 1. Image Acquisition and Reconstruction

All studies were performed with a GEMINI TF PET/CT system (Philips Medical Systems, Cleveland, OH, USA). The Gemini TF is a fully three-dimensional (3D) PET scanner coupled to a 16-slice Brilliance CT scanner. The patient bore is 716 mm in diameter, with an active transverse field of view of 675mm. The axial field of view per bed position is 180mm. PET and CT are performed sequentially, with PET acquisition performed in the step-and-shoot mode using an acquisition time of 1.45 s for each bed position. All data were acquired in list mode and reconstructed using a 3D row action maximum likelihood algorithm. The data were corrected for random coincidences, dead time losses, and scatter, and a whole-body low-dose CT scan (120 kVp, 30 mAs/slice) was used for attenuation correction. After positioning, a CT scout image was taken to define the scan range. All patients underwent imaging in the arms-up position from the skull to the upper thighs region. After the completion of CT imaging, the PET scan was performed in the caudal–cranial direction. The scan duration differed according to patient body size and typically consisted of 12 bed positions. Patients were instructed to breathe shallowly during imaging. The scanner calibration factor is used to convert reconstructed images of patients from scanner units to radioactivity concentrations. To reduce variability, the calibration is performed quarterly, and a validation procedure of SUV is performed bimonthly as the manufacturer recommendation.

## 2. 2. Statistical Analysis

The mean and standard deviation (SD) were used to represent all results. Microsoft Office Excel 2016 was used for all statistical analysis. In order to compare data between variables, a paired and unpaired Student's t-test was used. The result was considered statistically significant if  $P < 0.05$ .

## 3. Results

The whole-body  $^{18}\text{F}$ -FDG PET/CT images of 100 normal cases (64 females and 36 males) were analyzed. The age of the subjects was with a range of 20–88 years; of a mean age of  $58.36 \pm 15.96$  years. The BGL was in the range of 67–184 mg/dl, and a mean of  $109.76 \pm 28.25$  mg/dl, as shown in Table 1.

**Table 1: Demographic data and indication for normal organ cases.**

Normal cases			
Age (years)	58.36±15.86		
Gender	Female (64), male (36)		
Serum glucose (mg/dl)	109.76±28.25		
<sup>18</sup> F-FDG dose (MBq)	7.37±0.96		
Tissue	Low blood glucose (<100mg/dl)	Medium blood glucose (100–160 mg/dl)	High blood glucose (160–184 mg/dl)
Diagnosis (number)			
Bone sample size	25	20	5
Liver sample size	25	20	5

The BGLs were measured immediately before the administration of the <sup>18</sup>F-FDG injection. Subsequently, the patients were divided into three groups based on their serum glucose levels. For the liver the number of instances as per the BGL: The first group had a low mean BGL of 100mg/dl, with a sample size of 25 individuals; The second group had a BGL ranging from 100 to 160mg/dl, with a sample size of 22 individuals; The third group had a BGL ranging from 160 to 184mg/dl, with a sample size of 3 individuals. For the bone: the first group had a low average BGL 100 mg/dl, with a sample size of 31 individuals; The second group had an average BGL ranging from 100 to 160 mg/dl, with a sample size of 16 individuals. The third group had an average BGL ranging from 160 to 184 mg/dl, with a sample size of 3 individuals. This information can be seen in Table 2.

**Table 2: Clinical diagnoses of patients undergoing PET/CT.**

Tumor cases			
Age (years)	61.22 ±14.43		
Gender	Female (53), male (47)		
Serum glucose (mg/dl)	103.64 ± 21.18		
<sup>18</sup> F-FDG dose (MBq)	7.36 ±2.04		
Tissue	Low blood glucose (<100mg/dl)	Medium blood glucose (100–160 mg/dl)	High blood glucose (160–184 mg/dl)
Diagnosis (number)			
Bone sample size	31	16	3
Liver sample size	25	22	3

The average SUV<sub>max</sub> range for bone and liver of the three groups of BGLs is shown in Table 3. Mean bone SUV<sub>max</sub> for normal group decreased progressively with increasing BGL. The value for the three groups were 3, 3.53, and 2.87; for liver. Mean liver SUV<sub>max</sub> increased with BGLs, for group I the mean value was 2.57; for groups II and III, it was 2.88, 2.46, respectively. Overall, BGL was related with tumor (bone and liver) SUV<sub>max</sub>. The bone SUV<sub>max</sub> somewhat drops as BGL increases (Table 3). A correlation existed between liver and BGLs as well, although it increased with BGL.

Table 4 shows the average <sup>18</sup>F-FDG uptake of the studied normal and tumor tissues for each of the three BGLs. The results showed that the average <sup>18</sup>F-FDG uptake of the liver decreased with greater BGLs (high BGL) and increased with BGLs between 100 and 160 mg/dl. The mean injection dose in the bone, 7.73 mCi, was larger in medium blood glucose than in low and high blood glucose. In tumor patients, BGL increased <sup>18</sup>F-FDG absorption in the liver but decreased it in the bone.

**Table 3: Mean  $\pm$  SD for  $SUV_{max}$  normal and tumor tissue for the three groups of BGLs.**

Tissue	Low blood glucose ( $<100\text{mg/dl}$ )	Medium blood glucose ( $100\text{--}160\text{ mg/dl}$ )	High blood glucose ( $160\text{--}184\text{ mg/dl}$ )	p-value
Normal cases				
Bone	$3.00 \pm 0.30$	$3.53 \pm 0.17$	$2.87 \pm 0.07$	$<0.001$
Liver	$2.57 \pm 0.12$	$2.88 \pm 0.23$	$2.46 \pm 0.02$	$<0.001$
Tumor cases				
Bone	$6.32 \pm 3.51$	$6.07 \pm 2.58$	$5.66 \pm 2.07$	$<0.001$
Liver	$5.66 \pm 2.21$	$6.4 \pm 3.18$	$5.3 \pm 1.055$	$<0.001$

**Table 4: Average  $^{18}\text{F}$ -FDG uptake of the tested tissues for the three groups of BGLs.**

Tissue	Low blood glucose ( $<100\text{mg/dl}$ )	Medium blood glucose ( $100\text{--}160\text{ mg/dl}$ )	High blood glucose ( $160\text{--}184\text{ mg/dl}$ )	p-value
Normal cases				
Bone	$7.47 \pm 0.52$	$7.73 \pm 0.59$	$6.78 \pm 0.84$	$<0.001$
Liver	$7.48 \pm 0.34$	$7.64 \pm 0.37$	$6.78 \pm 0.84$	$<0.001$
Tumor cases				
Bone	$6.23 \pm 2.31$	$7.88 \pm 0.89$	$7.52 \pm 0.21$	$<0.001$
Liver	$7.43 \pm 2.24$	$8.36 \pm 1.43$	$8.74 \pm 1.15$	$<0.001$

#### 4. Discussion

The PET scans are commonly employed to evaluate various types of cancers.  $^{18}\text{F}$ -FDG imaging of cancer is based on a change in glucose transporters in cancerous cells. The uptake of glucose by these cells is a complex process influenced by various factors, both insulin-dependent and noninsulin-dependent. The transfer of FDG into the cells is facilitated by glucose transporters (GLUT) 1 to 7 and sodium-glucose linked transporters, leading to an increased uptake of glucose within the tumor [18].

Studies have shown that hyperglycemia affects how well tumor cells absorb  $^{18}\text{F}$ -FDG. As such, we advise our patients to fast for six hours while keeping their serum glucose levels within reasonable bounds. The investigation was carried out to assess how fasting BGLs affected the biodistribution of  $^{18}\text{F}$ -FDG in normal and malignant tissues (bone, liver). Due to heightened glycolysis and metabolism, the majority of cancer cells exhibit a greater uptake of glucose and  $^{18}\text{F}$ -FDG in comparison to normal tissue. Tumors exhibit a significant increase in the expression of GLUT-1 and GLUT-3 transporters [19]. Elevated levels of hexokinase expression in tumors have also been confirmed, which may have a more potent impact on  $^{18}\text{F}$ -FDG uptake than the overexpression of GLUT [20]. The variations in tissue absorption of  $^{18}\text{F}$ -FDG and subsequent emission of positrons enable the detection of tumor activity. Fluctuations in BGLs have been observed to impact the absorption of  $^{18}\text{F}$ -FDG. High glucose levels promptly result in reduced absorption of  $^{18}\text{F}$ -FDG in various types of cells [21].

The results of previous investigations into this relationship have been inconsistent. A 2011 study conducted in Japan discovered a statistically significant direct relationship between elevated glucose and hepatic background uptake, particularly on delayed images (90–100 min) [22]. In 2013, a Swedish study on normal tissues discovered that there was no significant relationship between BGLs and hepatic parenchymal uptake while hyperglycemia substantially increased the uptake of  $^{18}\text{F}$ -FDG in skeletal muscle, with the exception of the other tissue types that were examined [12][23]. The study also suggested that the chronicity of hyperglycemia may help explain some of the conflicting results of various studies.  $^{18}\text{F}$ -FDG uptake may be more substantially influenced by acute or experimentally induced hyperglycemia than by chronic hyperglycemia. Additionally, the results of studies that examine the correlation between BGLs and tumor  $^{18}\text{F}$ -FDG uptake are inconsistent. The relationship between decreased tumor  $^{18}\text{F}$ -FDG absorption and

detection and increased glucose levels at scan time has been documented in numerous studies [23]. This observation may be attributable to competitive inhibition of the GLUT receptors by glucose [24].

Studies on the liver have shown a variety of outcomes. Although some studies suggest that hyperglycemia enhances  $^{18}\text{F}$ -FDG uptake in the liver, others found no significant difference from normal [25]. In situations of hyperglycemia, tumors show either unchanged/unaffected or reduced  $^{18}\text{F}$ -FDG uptake. Chronic hyperglycemia is thought to upregulate GLUT1 and GLUT3 in tumors, making it a risk factor for cancer progression. According to Hara et al., acute hyperglycemia causes reduced tumor  $^{18}\text{F}$ -FDG uptake, whereas chronic hyperglycemia has no significant effect on tumor  $^{18}\text{F}$ -FDG uptake [26].

Like previous studies, ours showed that hyperglycemia lowers bone  $^{18}\text{F}$ -FDG absorption. As in the literature, reduction of  $^{18}\text{F}$ -FDG absorption in the bone began at a BGL of 160 mg/dl and progressively became more noticeable with rising BGLs [24]. The results of this work demonstrate a clear correlation between nonpathologic  $^{18}\text{F}$ -FDG biodistribution in the liver and BGLs. When reading PET scans, one should take into account how blood glucose affects anticipated biodistribution patterns at comparatively normal glucose levels, especially in the liver. These data strengthen our conviction that a PET scan can be performed even in the case of pretest hyperglycemia less than 200 mg/dl; yet, when determining liver SUVs, BGLs must be taken into account.

Tracer accumulation in tissues is measured using a semiquantitative parameter called the  $\text{SUV}_{\text{max}}$  [27]. Due to compete with blood glucose, hyperglycemia reduces the tumor's ability to absorb FDG. Although there is some disagreement over the true impact of glycemia on  $^{18}\text{F}$ -FDG uptake in relation to the effects of BGLs on  $\text{SUV}_{\text{max}}$  in various organs, plasma glucose level has a significant impact on SUVs. The background tissues that are frequently utilized to determine treatment response are the liver and mediastinal blood pool. Glycemic variations can cause an inaccurate assessment of therapeutic response [28].

## 5. Conclusions

Chronically elevated serum glucose levels moderately affect  $^{18}\text{F}$ -FDG uptake in both normal and malignant human tissues and degrade PET images. Thus, patients enrolling in PET-FDG investigations should ideally be fasting, and their BGLs must be considered. These data reinforce our belief that we can perform a PET scan even when the pretest hyperglycemia is less than 200 mg/dl; however, it is crucial to consider BGLs when determining  $\text{SUV}_{\text{max}}$ .

## Conflict of interest

Authors declare that they have no conflict of interest.

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## تقييم تأثير مستويات الجلوكوز في الدم على قيمة الامتصاص المعيارية (SUV) وامتصاص $^{18}\text{F}$ -FDG في الأورام وفحوصات PET الطبيعية

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### الخلاصة

كان الهدف من هذه الدراسة هو تقييم العلاقة بين مستوى السكر في الدم، وقيمة الامتصاص المعيارية القصوى  $\text{SUV}_{\text{max}}$ ، وامتصاص  $^{18}\text{F}$ -FDG في أنسجة الأعضاء السليمة والمصابة باستخدام فحوصات التصوير المقطعي بالإصدار البوزيتروني/التصوير المقطعي المحوسب PET/CT. يتم قياس نسبة الجلوكوز في الدم الصائم من خلال الإصبع بشكل روتيني في صباح يوم الفحص. تم قياس الحد الأقصى لقيمة الامتصاص المعيارية  $\text{SUV}_{\text{max}}$  في الكبد والعظام في 200 مشارك خضعوا لفحص  $^{18}\text{F}$ -FDG PET/CT التصوير المقطعي المحوسب للورم والأعضاء السليمة. تم تقسيم المرضى إلى ثلاث مجموعات اعتماداً على مستويات الجلوكوز في الدم لديهم. تم إجراء تحليل بأثر رجعي للعلاقة بين مستويات الجلوكوز وقيمة الامتصاص المعيارية. انخفض متوسط معدل قيمة الامتصاص المعيارية للكبد والعظام تدريجياً مع زيادة مستويات الجلوكوز في الدم، بدءاً من 160 ملجم/ديسيلتر. أظهرت المجموعة بأكملها التي لديها مستويات الجلوكوز في الدم (100-160 ملجم /ديسيلتر) زيادة طفيفة ولكن ملحوظة في امتصاص FDG للعظام والكبد مقارنة بالمجموعة المتطابقة لجميع مستويات الجلوكوز في الدم الطبيعية. وخلصت الدراسة إلى أنه بعد وصول مستوى السكر في الدم إلى 160 ملجم/ديسيلتر، يؤدي ارتفاع السكر في الدم إلى انخفاض تدريجي في امتصاص الكبد والعظام لـ  $^{18}\text{F}$ -FDG. تعتبر الدراسات التي تستخدم PET-FDG غير دقيقة بشكل خاص عندما تكون مستويات الجلوكوز في الدم مرتفعة. ونتيجة لذلك، يجب على الأفراد الذين يخضعون لفحوصات FDG-PET أن يصوموا، ويجب أخذ مستويات السكر في الدم في الاعتبار.

**الكلمات المفتاحية:** سكر الدم، فلورين 18 فلوديوكسيكليكوز، قيمة الامتصاص المعيارية، العظام، الكبد.