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Blood Film Analysis after FDG-PET Imaging: Unveiling Hematological and Radiological Changes

Zainab A. Jihad[1*] and Baydaa T. Sih[1]
[1]*Department of Physics, College of Science, University of Baghdad, Baghdad, Iraq*
[*]Corresponding author: Zainab.abbas2304@sc.uobaghdad.edu.iq

Abstract

Positron emission tomography (PET) using ^{18}F-fluorodeoxyglucose (^{18}F-FDG) is a cornerstone in oncological imaging for tumor localization and therapeutic assessment. Despite its diagnostic utility, limited attention has been given to its transient effects on hematological parameters. This study evaluates hematologic and immunological alterations following FDG administration by analyzing changes in red blood cells (RBCs), white blood cells (WBCs), and platelets before and after injection. The experimental cohort included one healthy subject and three cancer patients (liver, breast, and uterine). Peripheral blood samples were obtained pre- and post-FDG injection, with subsequent blood film analysis assessing RBC aggregation, WBC distribution, platelet morphology, and indicators of blood viscosity. Findings revealed reversible hematological changes, notably increased RBC aggregation, suggesting transient hyper viscosity potentially driven by oxidative stress. Differential leukocyte analysis revealed neutrophilia, lymphopenia, and monocytosis, suggesting immune modulation. Additionally, a mild rise in platelet aggregation suggested a temporary prothrombotic state. The study concludes that FDG-PET elicits short-term hematological and immunological shifts. These outcomes highlight the importance of cautious interpretation of PET findings in oncology settings and emphasize the need for further investigations into oxidative and immunomodulatory effects to enhance patient safety and diagnostic accuracy.

Article Info.

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Positron Emission Tomography (PET), ^{18}F-Fluorodeoxyglucose, Blood Glucose, Blood Film, Hematological.

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1. Introduction

Positron emission tomography/computed tomography (PET/CT) has become integral to oncological diagnostics, playing a pivotal role in tumor detection, staging, and treatment evaluation [1]. The most commonly utilized radiotracer in clinical PET imaging is 18F-fluorodeoxyglucose (^{18}F-FDG), a glucose analogue labeled with the radioisotope fluorine-18 [2,3]. Its exceptional sensitivity in visualizing malignant tissues stems from the heightened glycolytic activity of cancer cells, which significantly exceeds that of normal cells [4]. Peripheral blood film (PBF) analysis, despite technological advancements in automated hematology systems, remains indispensable in clinical decision-making, contributing to nearly 70% of diagnoses [5]. It offers a cost-effective and direct method for examining blood cell morphology, identifying hematological abnormalities, and monitoring disease progression· Moreover, PBF can detect subtle cellular alterations often overlooked by automated analyzers [6].

In the context of PET imaging, PBF is particularly relevant because it enables detection of hematological changes potentially induced by both radiation exposure and FDG metabolism [7]. These changes may affect the function and integrity of red blood cells (RBCs), white blood cells (WBCs), and platelets, thereby influencing overall blood viscosity and immune response [8]. Evaluating such effects provides valuable insights into the systemic impacts of PET imaging and informs clinical management strategies [9,10].

This study aims to assess hematological alterations associated with FDG-PET by comparing blood morphology before and after tracer administration· By investigating peripheral blood parameters pre- and post-injection, we seek to elucidate the transient physiological

responses to radiation exposure and FDG metabolism, contributing to a broader understanding of PET-induced systemic effects in oncological settings.

2. Materials and Methods

This investigation included one healthy individual and three patients diagnosed with uterine, breast, and liver cancer, respectively. The study was conducted at the Nuclear Medicine Department, Medical City, Al-Amal Hospital, Baghdad, between December 2023 and June 2024.

Ethical approval was formally submitted to the institutional review board and is currently under evaluation· Verbal informed consent was obtained from all participants before enrollment, and the study was conducted in accordance with institutional protocols and the principles outlined in the Declaration of Helsinki. Patients who had undergone chemotherapy or radiotherapy within the previous four weeks were excluded· Before PET/CT scanning, capillary blood glucose levels were assessed by a trained nurse in the imaging unit· Individuals with fasting glucose levels exceeding 200 mg/dL were deemed ineligible· All participants adhered to a fasting period of 4–6 hours before intravenous administration of ^{18}F-FDG. The primary aim was to evaluate hematological effects induced by FDG-PET by analyzing peripheral blood morphology before and after radiotracer injection· In most cases, PET imaging was performed for routine clinical evaluation· Blood films were microscopically examined to detect alterations in RBC aggregation, WBC distribution, platelet morphology, and indicators of blood viscosity.

3. Results and Discussion

The effect of ^{18}F-FDG PET scan on peripheral blood morphology was examined in the present study by alterations in RBCs, WBCs, platelets (PLTs), and blood viscosity· To make a comprehensive evaluation, a normal subject was taken as a control (normal body) and compared with three cancer patients having uterine cancer, breast cancer, and liver cancer. Through a comparative evaluation of pre-FDG-PET and post-FDG-PET blood smears, this study aims to elucidate the impact of metabolic activity and radiation exposure on blood cell morphology, aggregation patterns, and immune processes. The following sections present the findings for each case individually, beginning with the healthy control and then proceeding to each cancer subject.

Case 1: Normal Body

1. Changes in Red Blood Cell (RBC) Morphology

Before FDG Injection: The RBCs are evenly distributed and remain in their biconcave form without clumping or deformity. Their suspension in plasma optimizes blood viscosity, delivery of oxygen, and microcirculation.

After the injection of FDG, the aggregation of RBCs was increased, which reflects a temporary increase in blood viscosity. Some are deformed by radiation-induced membrane instability and oxidative stress caused by positron annihilation. Such alterations implicate disturbances in microcirculation that can affect hemodynamics and diffusion of oxygen, though no generalized hemolysis is evident.

2. Changes in White Blood Cells (WBCs)

Before the injection of FDG, WBCs are distributed evenly and in proportion with a consistent leukocyte ratio. There were no signs of immune activation or suppression.

a. Neutrophilia: increased neutrophils represent a minor inflammatory response, presumably to the metabolism of FDG or radiation exposure, implying transient immune activation.
b. Lymphopenia: A moderate lymphocyte decrease implies temporary immunosuppression from radiation and metabolic stress.
c. Monocytosis: Increased monocytes suggest a heightened stress response and phagocytic activity for clearing damaged cells.

d. Eosinophils and Basophils Unchanged: The response is predominantly neutrophilic rather than eosinophilic or basophilic· as can be seen in Fig. 1a and b.

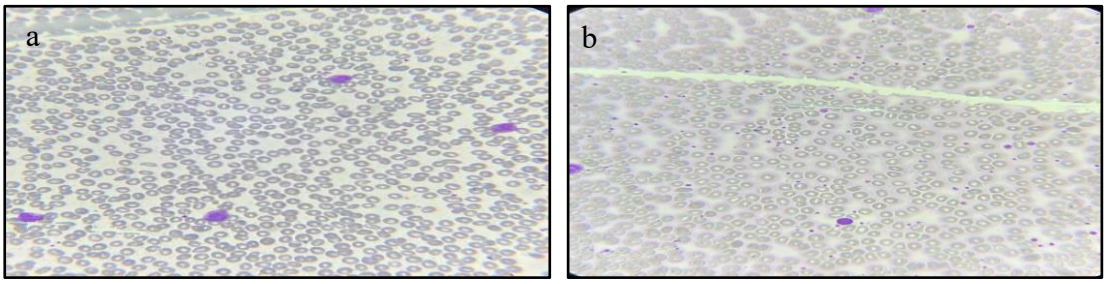

Figure 1: (a) After FDG Administration, (b) Before FDG Administration.

Before FDG injection, platelets are evenly distributed throughout the circulation, maintaining hemostatic balance and preserving vascular integrity without promoting excessive aggregation. Following FDG injection, however, subtle yet distinct changes occur. There is a mild and transient tendency toward platelet aggregation accompanied by localized clumping of red blood cells, suggesting a temporary prothrombotic effect. These changes influence microcirculatory dynamics, with blood flow and endothelial function being slightly impaired as a result of increased cellular aggregation. Nevertheless, no evidence of severe thrombocytosis is observed, indicating that these alterations are localized and reversible, reflecting transient modifications in endothelial contact and coagulation pathways. The mechanisms underlying these hematological changes can be attributed to several factors. First, the radiation emitted during FDG decay plays a central role· The β+ decay of ^{18}F-FDG produces high-energy 511 keV gamma photons, which generate oxidative stress within the blood, leading to lipid peroxidation in the membranes of red blood cells. This oxidative damage results in alterations of cell shape, increased aggregation, and compromised microvascular flow [11]. In addition, the metabolic and inflammatory responses triggered by FDG uptake contribute to these effects by inducing stress on white blood cells and stimulating acute-phase immune responses. This is manifested by elevated neutrophil and monocyte counts, reflecting a mild inflammatory reaction. The rheological properties of blood are also temporarily altered, as the aggregation of red blood cells increases blood viscosity and disrupts the efficiency of microcirculation, while mild platelet activation signifies a transient imbalance in coagulation homeostasis.

In the specific context of breast cancer, similar morphological and functional changes in blood cells are observed. Before FDG administration, red blood cells exhibit a uniform biconcave morphology and even distribution, supporting optimal oxygen delivery and circulatory efficiency. After FDG injection, however, increased aggregation of red blood cells is detected, causing a temporary rise in blood viscosity. Oxidative stress induced by radiation and membrane instability gives rise to abnormally shaped red cells, which may impair microcirculatory flow without causing generalized hemolysis.

White blood cell dynamics are also affected. Before FDG injection, leukocytes remain in a steady state, with normal differential counts and no evidence of immune activation. After FDG administration, there is a noticeable increase in neutrophil counts, consistent with a mild inflammatory response likely driven by FDG metabolism and radiation exposure. Conversely, lymphocyte levels decline, suggesting radiation-induced depletion. Monocytes increase modestly, indicating enhanced phagocytic activity and immune adaptation, while eosinophils and basophils remain unchanged, reinforcing that the observed response is predominantly neutrophilic rather than allergic. As illustrated in Fig. 2, these coordinated hematological changes reflect the complex interplay of radiation effects, metabolic stress, and immune modulation following FDG injection.

Before FDG administration, platelets remain evenly dispersed throughout the circulation, maintaining stable coagulation dynamics and ensuring vascular homeostasis. Blood viscosity is

within normal physiological limits, and red blood cells preserve their uniform distribution and morphology, facilitating smooth microcirculation. Following FDG injection, however, subtle but measurable changes emerge. There is a mild increase in platelet aggregation, which correlates with the observed tendency of red blood cells to cluster. This transient aggregation reflects a temporary shift in blood coagulability, subtly altering microcirculatory properties and potentially affecting endothelial interactions. Importantly, there is no evidence of overt thrombocytosis, suggesting that these changes are functional and reversible rather than pathological. The observed modifications likely reflect radiation-induced alterations in platelet-endothelial signaling, rather than an absolute increase in platelet count or sustained activation. Potential Mechanisms Underlying FDG-PET-Induced Hematological Changes Several physiological and biochemical mechanisms contribute to the hematological alterations observed after FDG administration.

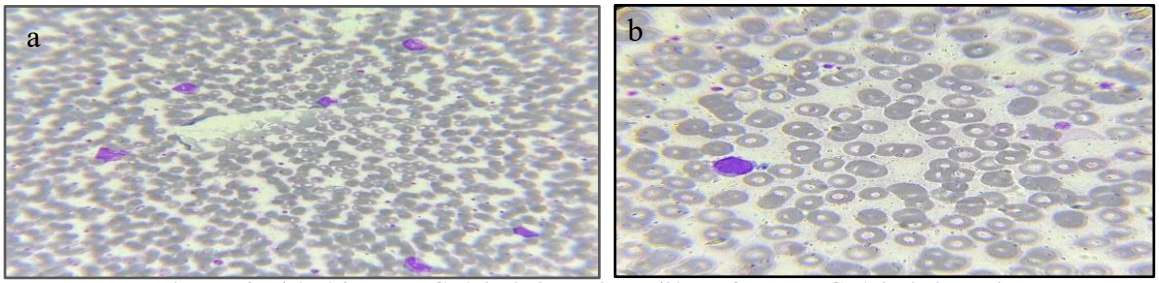

Figure 2: (a) After FDG Administration, (b) Before FDG Administration.

The β+ decay of ^{18}F-FDG emits 511 keV gamma photons, which generate oxidative stress within the blood. This oxidative stress promotes lipid peroxidation of cell membranes, particularly in red blood cells, leading to increased membrane instability, subtle morphological distortions, and an increased propensity to aggregate. Additionally, the metabolic impact of FDG uptake on glucose metabolism may disrupt cellular ion homeostasis and compromise membrane integrity, thereby facilitating cell clustering and altered rheology. In parallel, a modest inflammatory response appears to develop, likely triggered by metabolic and oxidative stress. Neutrophil and monocyte counts rise slightly, indicating transient immune activation and enhanced phagocytic readiness, while lymphocyte counts decline, consistent with the radiosensitivity of these cells. These immune changes reflect a mild acute-phase response rather than a sustained inflammatory state. The combined effect of these processes also leads to rheological alterations in blood properties. Red blood cell aggregation and shape changes temporarily increase blood viscosity, which can impact the efficiency of microcirculatory flow. Similarly, mild platelet activation suggests a transient, localized shift toward a more prothrombotic state, without evidence of systemic hypercoagulability.

Case 2: Uterine Cancer

In patients with uterine cancer, comparable hematological changes are observed following FDG administration. Before injection, red blood cells maintain a stable biconcave morphology and uniform distribution, ensuring low viscosity and effective oxygen transport throughout the microvasculature.

After FDG injection, red blood cells exhibit increased aggregation, temporarily raising blood viscosity. Minor morphological distortions are evident, likely due to oxidative stress and membrane destabilization induced by radiation exposure. While these changes may transiently impair microcirculatory flow and oxygen delivery, they do not appear to cause significant hemolysis or irreversible damage.

WBCs also display notable shifts. Before injection, leukocyte counts and distribution remain within normal limits, without evidence of immune activation or inflammation. Following FDG administration, neutrophil counts increase moderately, consistent with a mild inflammatory

response to metabolic and oxidative stress. Lymphocyte counts decline, reflecting their susceptibility to radiation-induced depletion· Monocyte numbers rise, suggesting enhanced immune adaptation and clearance of damaged cells. Eosinophil and basophil levels remain unchanged, indicating that the immune response is primarily neutrophilic and not allergic in nature, as shown in Fig. 3 a and b.

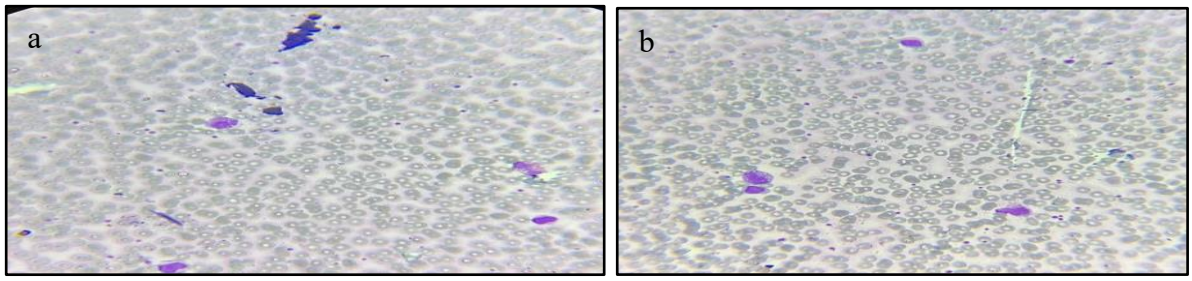

Figure 3: (a) After FDG Administration, (b) Before FDG Administration.

Before FDG injection, platelets are uniformly distributed in the bloodstream, maintaining normal coagulation balance and ensuring vascular integrity without evidence of abnormal clustering or hyperactivity. This physiological state preserves normal blood viscosity and stable microcirculatory flow, as shown in Fig. 4. Following FDG administration, however, subtle but significant hematological changes are observed. There is a mild increase in platelet aggregation, which appears to parallel the aggregation of red blood cells and slight increases in blood viscosity. These changes likely reflect a transient prothrombotic shift, affecting microcirculation and endothelial function through localized platelet reactivity. Notably, these effects are functional and reversible, with no evidence of pathological thrombocytosis or sustained platelet overproduction. Instead, the findings suggest that FDG-induced metabolic and radiophysiological stress transiently disrupts endothelial–platelet interactions and coagulation dynamics. The underlying mechanisms behind these hematological changes can be attributed to a combination of oxidative stress induced by positron annihilation, metabolic disruption, and rheological alterations· The β+ decay of ^{18}F-FDG produces 511 keV gamma photons, which generate reactive oxygen species, inducing lipid peroxidation and compromising the structural stability of red blood cell membranes [12,13]. This results in temporary morphological deformation and aggregation of erythrocytes, which impairs microvascular perfusion and increases blood viscosity [14,15]. Additionally, FDG uptake by metabolically active cells, including immune cells, alters their function and promotes mild immune activation. This is evidenced by elevated neutrophil and monocyte counts and reduced lymphocyte numbers, reflecting an acute-phase inflammatory response combined with the well-known radiosensitivity of lymphocytes. Such immune alterations are consistent with previous observations of radiation-induced immune modulation and are likely adaptive, facilitating clearance of damaged cells [16]. Analysis of platelet behavior further supports the notion of a localized, reversible prothrombotic state following FDG injection [17]. The mild increase in platelet aggregation observed is indicative of endothelial activation and transient coagulation imbalance, yet without progressing to overt thrombocytosis [18]. These findings align with earlier studies documenting radiation-induced alterations in platelet function and thrombus formation. While these changes are unlikely to cause significant clinical issues in healthy individuals, they may have implications for patients with underlying hematological or cardiovascular disorders, highlighting the need for careful interpretation of PET imaging findings in such populations [19]. The mechanisms underlying these effects can thus be categorized into three primary processes: radiation-induced oxidative stress, which damages red blood cell membranes and disrupts microcirculation; metabolic stress, which perturbs immune cell function and activates inflammatory pathways; and hemorheological changes, which increase blood viscosity and transiently disturb endothelial and vascular stability. These observations reinforce the systemic

impact of FDG-PET imaging on blood cell physiology and underline the clinical relevance of understanding and mitigating these effects, particularly in vulnerable patients [20].

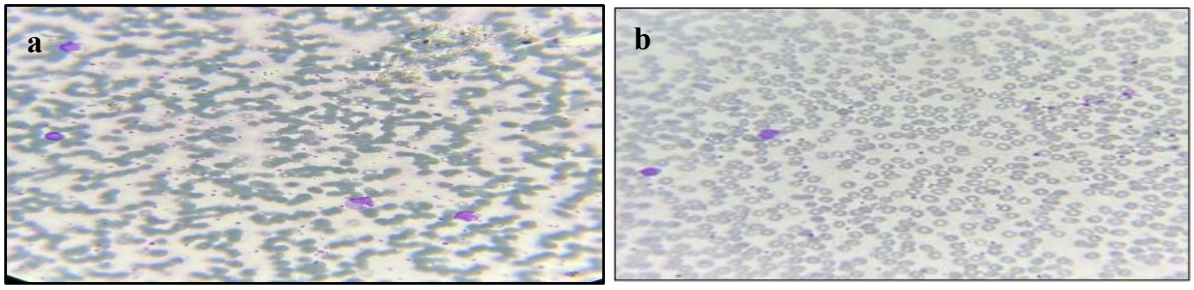

Figure 4: (a) After FDG Administration, (b) Before FDG Administration.

4. Conclusions

In summary, this study demonstrates that FDG-PET induces reversible but meaningful hematological changes, including mild platelet activation, red blood cell aggregation, increased viscosity, neutrophilia, lymphopenia, and monocytosis. These alterations are driven by oxidative and metabolic stress and reflect the complex interaction between radiation exposure and immune-vascular homeostasis. Although typically self-limiting, such changes warrant further investigation, particularly regarding their implications in patients with pre-existing disease and in the context of repeated PET imaging. Advances in radiopharmaceutical formulation, personalized dosimetry, and adjunctive therapies may help minimize these transient effects while preserving the diagnostic and therapeutic benefits of PET imaging. These findings contribute to a deeper understanding of the physiological responses elicited by FDG-PET and support ongoing research to enhance patient safety and optimize imaging outcomes.

Conflict of Interest

The authors declare that they have no conflict of interest.

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تحليل مسحة الدم بعد التصوير المقطعي بالإصدار البوزيتروني باستخدام فلوروديوكسي جلوكوز: الكشف عن التغيرات الدموية والإشعاعية

زينب عباس جهاد[1] وبيداء طاهر سيه[1]
[1]قسم الفيزياء، كلية العلوم، جامعة بغداد، بغداد، العراق

الخلاصة

يُعدّ التصوير المقطعي بالإصدار البوزيتروني (PET) باستخدام فلوروديوكسي جلوكوز-18 (FDG-18F) حجر الزاوية في التصوير التشخيصي للأورام لتحديد موقع الورم وتقييم العلاج. على الرغم من فائدته التشخيصية، إلا أن تأثيراته العابرة على المؤشرات الدموية لم تحظ بالاهتمام الكافي. تُقيّم هذه الدراسة التغيرات الدموية والمناعية التي تعقب حقن FDG من خلال تحليل التغيرات في خلايا الدم الحمراء (RBCs) وخلايا الدم البيضاء (WBCs) والصفائح الدموية قبل الحقن وبعده. شملت المجموعة التجريبية شخصًا سليمًا وثلاثة مرضى مصابين بالسرطان (سرطان الكبد، وسرطان الثدي، وسرطان الرحم). تم أخذ عينات من الدم المحيطي قبل حقن FDG وبعده، مع إجراء تحليل لاحق لشريحة الدم لتقييم تكتل خلايا الدم الحمراء، وتوزيع خلايا الدم البيضاء، وشكل الصفائح الدموية، ومؤشرات لزوجة الدم. كشفت النتائج عن تغيرات دموية قابلة للعكس، ولا سيما زيادة تكتل خلايا الدم الحمراء، مما يشير إلى فرط لزوجة عابر قد يكون ناتجًا عن الإجهاد التأكسدي. كشف تحليل تفريقي لخلايا الدم البيضاء عن زيادة في عدد العدلات، ونقص في عدد الخلايا اللمفاوية، وزيادة في عدد الخلايا الوحيدة، مما يشير إلى تعديل مناعي. يشير ارتفاع طفيف في تجمع الصفائح الدموية إلى حالة مؤقتة من فرط التخثر. وتخلص الدراسة إلى أن التصوير المقطعي بالإصدار البوزيتروني باستخدام فلوروديوكسي جلوكوز (FDG-PET) يُحدث تغيرات دموية ومناعية قصيرة الأمد. تُبرز هذه النتائج أهمية التفسير الدقيق لنتائج التصوير المقطعي بالإصدار البوزيتروني في سياقات الأورام، وتؤكد على ضرورة إجراء المزيد من الدراسات حول التأثيرات التأكسدية والمعدلة للمناعة لتعزيز سلامة المرضى ودقة التشخيص.

الكلمات المفتاحية: التصوير المقطعي بالأصدار البوزيتروني، فلوروديوكسي جلوكوز، جلوكوز الدم، فلم الدم، دموي.