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FDG PET in Prostate Cancer

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The role of positron emission tomography (PET) with [F-18]-fluorodeoxyglucose (FDG) for the imaging evaluation of patients with cancer has expanded rapidly around the world. The development of hybrid PET-CT imaging systems that can precisely localize metabolic abnormalities and characterize the metabolic activity of normal and abnormal structures, commercial regional distribution of FDG, growing clinical experience, recently spearheaded by National Oncologic PET Registry (NOPR; http://www.cancerPETregistry.org/), and improved understanding of clinical indications and reimbursement have all facilitated the increasing use of FDG PET and PET-CT in cancer imaging. FDG PET has been employed for diagnosis, initial staging, restaging, prediction and monitoring of treatment response, surveillance and prognostication in a variety of cancers and leads to improved clinical decision-making and cost-effective management changes in considerable number of patients (1–3).

The exact clinical use of PET and PET-CT in prostate cancer is currently being explored. The recent publications summarizing the findings of NOPR demonstrated that prostate cancer was one of the most common cancers with patient enrollment (4,5). Here we first review the available relatively limited data on the molecular biology basis of FDG accumulation in prostate cancer followed by a brief review of the current clinical experience with the use of PET with FDG in the imaging evaluation of prostate cancer.

Glucose Metabolism in Prostate Cancer

Several hallmarks of cancer have been described that include self-sufficiency in growth signals, insensitivity to anti-growth signals, evasion of apoptosis, limitless replicative potential, sustained angiogenesis, tissue invasion and launch of metastasis envoys, evasion of tumors from the immune system and increased glucose metabolism (6,7). The ability of FDG PET to detect cancer is based on the latter hallmark (Warburg effect). The relationship between tumor growth and the inefficient energy production by glucose metabolism is not well understood but may be explained in terms of adaptation to hypoxia through upregulation of glucose transporters and increased enzymatic activity of hexokinase (8,9). Glucose transporter - the currently approved gene symbol is “SLC2Ax” but here we will use the familiar symbol “GLUTx” - is the first rate-limiting step for glucose metabolism that allows energy-independent glucose transport across the cell membrane down the concentration gradient while hexokinase-II phosphorylates glucose to glucose-6-phosphate. Similarly, FDG is phosphorylated to FDG-6-phosphate but contrary to glucose-6-phosphate, it cannot be metabolized further in the glycolytic pathway and becomes trapped in the cell due to its negative

charge and the very low activity of the reverse enzyme, glucose-6-phophatase, in most cancers (10–15).

The GLUT1 mRNA expression has been assessed in the androgen-independent cell lines, DU145 and PC-3, and the androgen-sensitive cell line LNCaP (16). The poorly differentiated androgen-independent cell lines showed higher mRNA expression than the well-differentiated androgen-sensitive cell line suggesting that GLUT1 expression directly related to the malignancy grade. One study evaluated the expression of a number of hypoxia-associated genes within benign prostatic hyperplasia (BPH) and prostate cancer (Gleason score 5 to 10) human tissue specimen (17). GLUT1 gene expression was not only significantly higher in the tumor than in BPH but also was correlated directly with Gleason score (R=0.274, p=0.026), corroborating the direct relationship between GLUT1 expression and tumor grade. Other in-vitro studies have also shown that FDG uptake increases with hypoxic condition in both androgen-sensitive and androgen-independent prostate cancer cell lines (18). Moreover, hypoxia resulted in upregulation of hypoxia-inducible factors HIF-1alpha and HIF-2alpha that could be inhibited with the nonsteroidal anti-inflammatory drug Ibuprofen leading to cyclooxygenase-2 independent down-regulation of HIF-regulated proteins vascular endothelial growth factor (VEGF) and GLUT1 (19). The glucose metabolism of prostate cancer may not be limited to GLUT1 as another investigation showed that GLUT12 may also be involved (20). The modulatory effect of androgen on tumor glucose metabolism has also been investigated. Higher FDG accumulation has been observed in androgen-independent tumors than in androgen-sensitive tumors (21). Accordingly, it has been observed that castration reduces glucose metabolism in the prostate tumor (21–23). In summary, despite very limited data, it appears that glucose metabolism in prostate cancer is GLUT-mediated but the relationship is complex and may be affected by many factors such as hypoxia and androgen level.

Clinical Utility of FDG PET in Prostate Cancer

There is considerable heterogeneity in the current clinical experience with FDG PET inprostate cancer. This is due to variability in disease states, validation criteria, and end points among studies (24–26). Additional complicating factors include the inherent biological and clinical heterogeneity of the disease as well as technical and image processing factors (e.g. filtered-back projection in comparison to iterative reconstruction with segmented attenuation) irrespective of the underlying biological heterogeneity (18,27–29).

Normal Prostate

We recently reported on the FDG accumulation in the normal prostate gland in relation to age and prostate size on PET-CT studies that were performed on 145 men (age range 22-97 years) with no clinical and laboratory evidence of prostate pathology (30). The population average and range of the normal prostate size were 4.3 +/− 0.5 cm (mean +/− SD) and 2.9–5.5 cm, respectively. The population average of mean and maximum SUV (standardized uptake value as semi-quantitative measure of the amount of tracer accumulation in a region of interest normalized to body weight and injected tracer dose) was 1.3 +/− 0.4 (range 0.1–2.7) and 1.6 +/− 0.4 (range 1.1–3.7), respectively. Mean SUV tended to decrease as the prostate size increased ($r = -0.16$, $P = 0.058$) while the prostate tended to get larger as age increased ($r = 0.32$, $P < 0.001$). Despite the generally low FDG uptake by the normal prostate gland, however, other studies have shown that there can be significant overlap among FDG accumulation in normal prostate, BPH and prostate cancer (25,26,31–34).

Diagnosis and Initial Staging

FDG PET may not be useful in diagnosis and staging of clinically organ-confined disease and in detection of locally recurrent disease due to overlap of tumor uptake with scar tissue and also because of the obscuring that can occur due to intense urine activity in the adjacent urinary bladder (32,35). FDG is also not specific for cancer and false positives may occur with inflammatory condition such as prostatitis (36). However, despite these less enthusiastic observations, several animal-based translational and human-based clinical studies have shown that FDG PET can be useful in specific clinical circumstances in prostate cancer. FDG uptake in prostate cancer increases directly with increasing Gleason grade, clinical stage and serum PSA level (37,38). In an early experience with 34 men with prostate cancer and known or suspected metastatic disease, FDG PET demonstrated a SUV range of 2.1 to 5.7 for the metastatic lesions with the notation that PET was less sensitive than bone scintigraphy and that detection of pelvic lymph node metastases was limited due to intense bladder urine activity (39). However in patients with known osseous metastatic disease, FDG PET may distinguish the metabolically active lesions from the metabolically quiescent lesions (40–42). Also, recent data from our laboratory suggest that FDG PET concordance rate with other imaging studies appears to be higher in the castrate-resistant disease in comparison to castrate-sensitive disease and also higher for lymph nodes and visceral lesions than for osseous lesions (43).

PSA Relapse Only

A large group of men present with “PSA relapse only” (biochemical failure) at some point after definitive therapy in whom by definition there is no standard imaging evidence of disease. In this group of men, “non-standard” imaging detection of disease can direct appropriate treatment such as salvage radiation therapy for local recurrence in the prostate bed or systemic therapy for metastatic disease (Fig. 1, Fig. 2). In one study of 24 patients with a rising serum PSA level after treatment for localized prostate cancer, FDG PET was performed before pelvic lymph node dissection (44). In accordance with “PSA relapse only” disease, all men had negative findings on whole body bone scan and equivocal pelvic CT results. Validation was by histology of the pelvic lymph nodes harvested at surgery in 67% of patients. The authors reported a sensitivity of 75%, specificity of 100%, accuracy of 83.3%, positive predictive value of 100%, and negative predictive value of 67.7% in detecting metastatic pelvic lymph nodes in this relatively homogeneous state of disease. In a similar investigation of 91 patients with PSA relapse following prostatectomy and validation by biopsy or clinical and imaging follow-up, the researchers reported that mean PSA level was higher in PET-positive than in PET-negative patients (9.5 +/− 2.2 vs. 2.1 +/− 3.3 ng/mL) (45). Using a receiver operating characteristic (ROC) curve analysis, the best tradeoff between sensitivity and specificity was found at a PSA level of 2.4 ng/mL (80%, 73%, respectively) and PSA velocity of 1.3 ng/mL/y (71%, 77%, respectively). The authors reported that FDG PET detected local or systemic disease in 31% of patients with PSA relapse. However, confidence in the accuracy and relevance of this figure is guarded in view of the definition of PSA relapse and the heterogeneity of the validation criteria, frequent with other similar studies (46). Other studies have shown that FDG PET may be particularly useful in men with rising PSA level despite treatment and that in the clinical setting of PSA relapse, it is more sensitive than In-111 capromab pendetide scintigraphy (47, 48).

Evaluation of Treatment Response

FDG PET has also been investigated for its utility in the assessment of response to treatment. An earlier rat prostate cancer study was disappointing in showing that the global FDG uptake was unchanged after treatment with gemcitabine (49). However a later investigation demonstrated that FDG accumulation in the primary prostate cancer and metastatic sites

decreased 1–5 months after initiation of androgen deprivation therapy similar in principle to the results of the animal xenograft studies (21,50,51). Our preliminary results with both androgen deprivation and various chemotherapy regimens have shown that the tumor accumulation of FDG uptake decreases with successful treatment in concordance with other measures of response such as decline in serum PSA level (Fig. 3).

Prognostication

It has also been shown that the level and extent of FDG accumulation in metastatic lesions may also provide information on prognosis. One investigation reported that >33% increase in average SUVmax of up to 5 lesions or appearance of new lesions could dichotomize castrate metastatic prostate cancer patients treated with antimicrotubule chemotherapy as progressors or non-progressors (52). Similar prognostic utility was demonstrated in that men with highly FDG-avid primary prostate tumors had a poorer prognosis in comparison to those with low SUV (53). Since FDG uptake in the prostate tumor appears to be initially dependent on androgen, FDG PET may also be useful in the prediction of the time to androgen refractory state (e.g. early increase in tumor FDG uptake during castrate state) that may allow an early intervention in order to avert or delay this clinical state and improve overall patient outcome (21).

NOPR and Prostate Cancer

The NOPR was established in May 2006 to collect and analyze data on the clinical utility of FDG PET to provide evidence for reimbursement coverage by the Centers for Medicare & Medicaid Services (CMS)(4). The results for the first 2 years of data from 40,863 PET scans for staging, restaging, or detection of suspected recurrence in patients with pathologically-proven cancers were recently reported (5). The most number of FDG PET scans (5,309 scans equivalent to 13% of all scans) was performed in patients with prostate cancer. PET findings changed clinical management in 35.1% of prostate cancer cases (95% CI: 33.8%−36.4%) although the odds ratio for change in management compared with that for other cancers in the NOPR trial was less than 1 at 0.86 (95% CI: 0.81–0.92) implying that chance of changing management was lower for prostate cancer than that for all the other cancer types. The change in management was from non-treatment to treatment in 25.3% and from treatment to non-treatment in 9.7% of cases with a major change in type of treatment relative to the plan before PET in 8.5% of cases. The PET-directed change in management was nearly equal in relation to the testing indications with 32.0% (30.0–34.1%) for initial staging (n=2042 cases), 34.0% (95% CI: 31.6–36.4%) for restaging (n=1477 cases), and 39.4% (95% CI: 37.2–41.7%) for detection of suspected recurrence (n=1790 cases).

Conclusion

The current clinical experience with FDG PET in prostate cancer is afflicted by considerable variability in disease states, validation criteria, and end points. Despite this significant limitation, however, it appears that FDG PET may be useful for diagnosis and staging of known or suspected high Gleason score primary tumors, in detection of locally recurrent and/or metastatic disease in a portion of patients with “PSA relapse only” with scan sensitivity that increases with increasing PSA level, in assessment of the extent of metabolically active castrate-resistant disease, in monitoring response to androgen deprivation and other therapies, and in prognostication As suggested by NOPR trial, FDG PET can impact the clinical management of men with prostate cancer although this impact may be lower than that for other cancers. FDG PET may be limited in diagnosis and staging of clinically organ-confined disease and can be falsely negative due to overlap of tumor uptake with normal, BPH, and scar tissue as well as be falsely positive with inflammatory conditions. More extensive experience

obtained through well-designed clinical trials based on well-defined clinical states of disease and hard end-points can help to determine the exact role of FDG PET in prostate cancer. A prospective clinical trial is currently underway to help define the role of FDG PET-CT in therapy assessment and in outcome prediction (time to hormone refractory state and survival) in men with androgen-naïve and androgen refractory metastatic prostate cancer (54).

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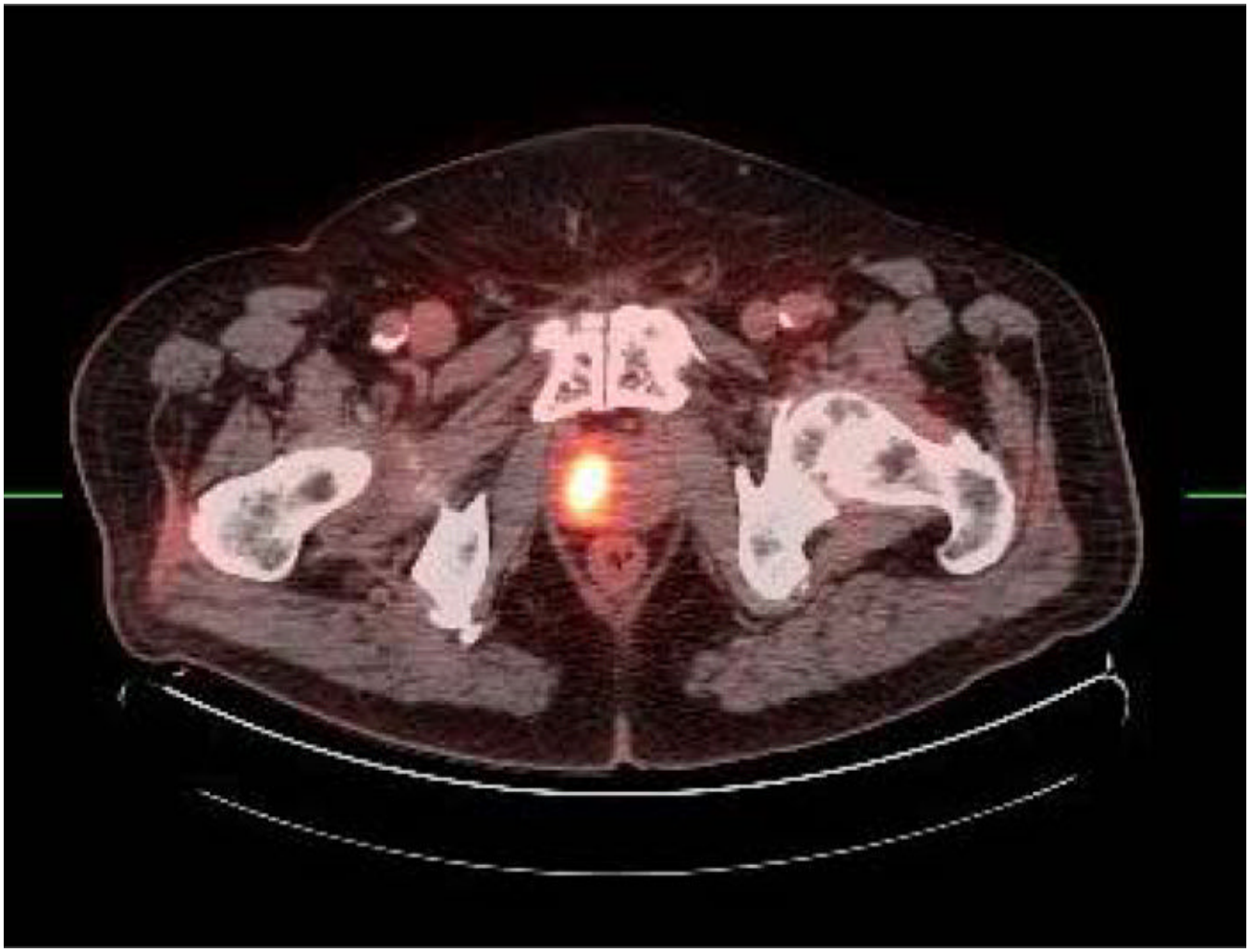

Fig. 1.
Axial fused FDG PET-CT of locally recurrent disease in right prostate lobe 11 years after radiation therapy for primary moderately differentiated prostate cancer with Gleason sum score of 4.

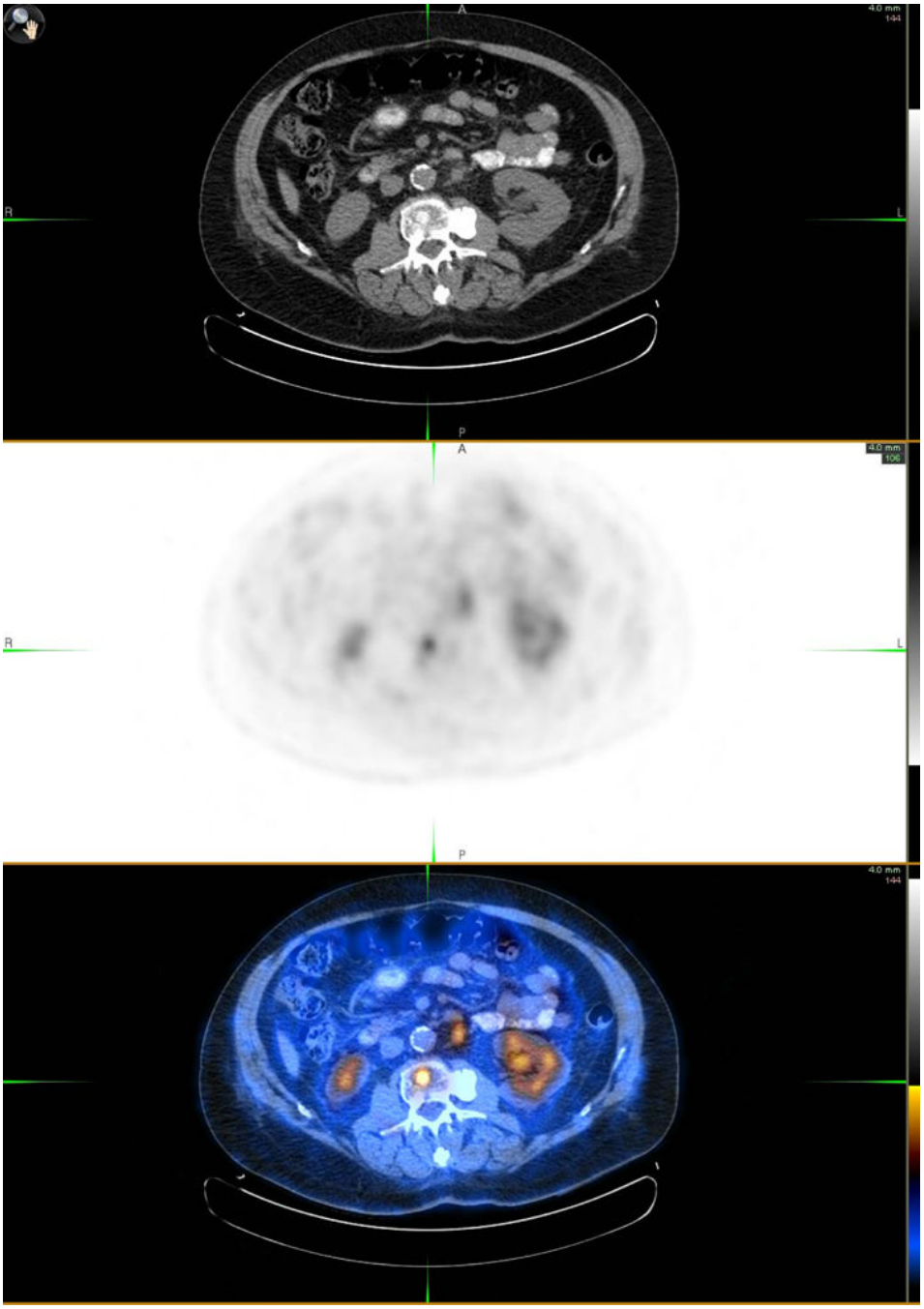

Fig. 2.
Axial CT (top panel), FDG PET (middle panel) and fused PET-CT (bottom panel) of metastatic prostate cancer involving L2 vertebral body (SUVmax = 4.8) and a conglomerate of subcentimeter left paraaortic nodes (SUVmax = 3.3, largest single node 9 mm).

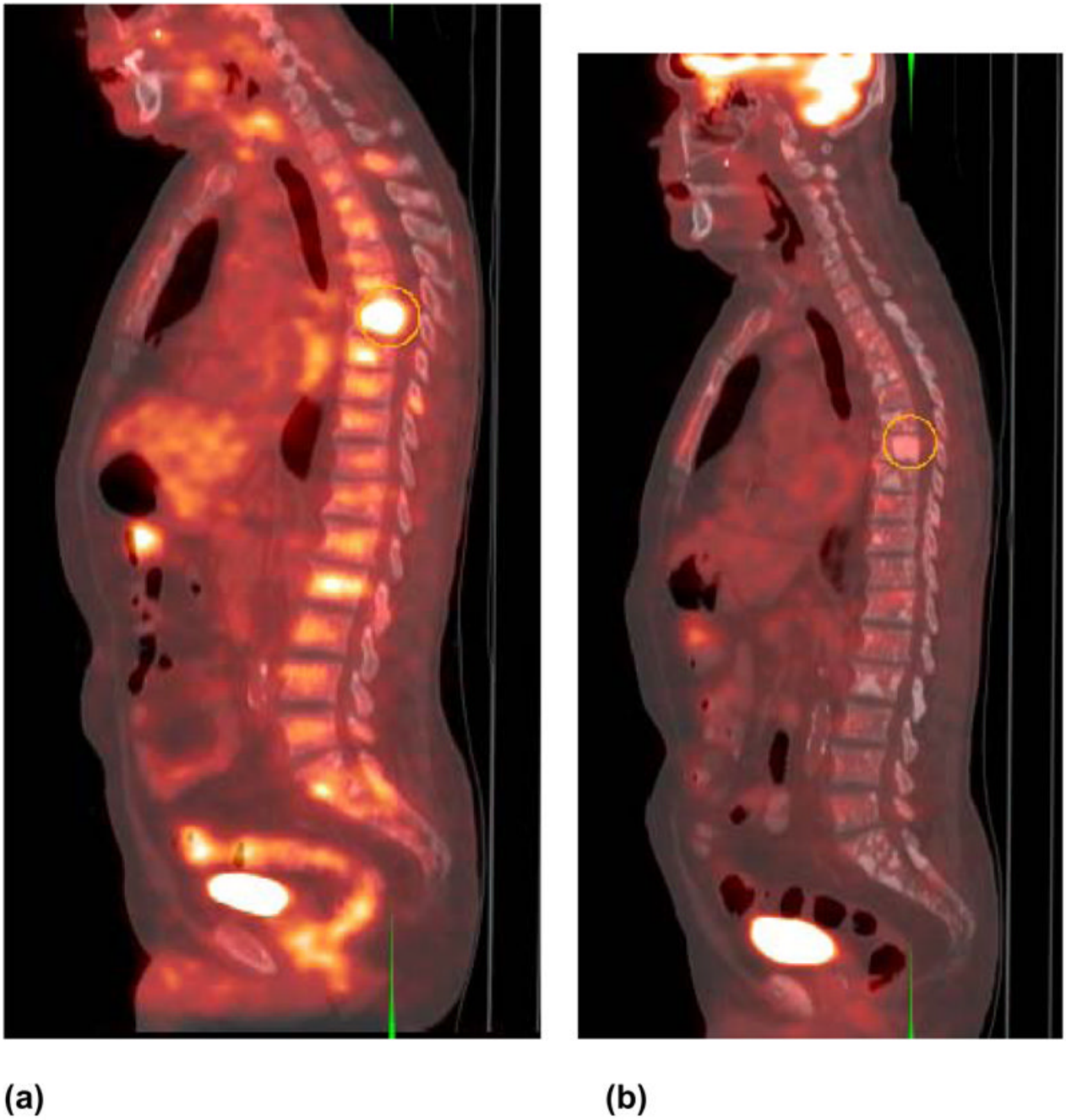

Fig. 3.
Sagittal fused FDG PET-CT in a man with castrate-sensitive metastatic prostate cancer **(a)** before and **(b)** five months after androgen deprivation therapy. Note the decline in the hypermetabolic activity of the sclerotic mid thoracic spine vertebral body in response to therapy. Lesion maximum SUV declined from 5.0 before therapy to 2.7 after therapy corresponding to serum PSA decline from 98 to 21 ng/mL, respectively.