The Influence Of Androgen Deprivation Therapy On ^{68}Ga-PSMA-PET/CT Uptake In Patients With Prostate Cancer: A Retrospective Study

Aram Avedesian (student number: 5726727), Supervisor: Y. Chahid, Referee: Prof. dr. J. Booij
Amsterdam UMC, location AMC, department of Radiology and Nuclear Medicine

ABSTRACT

INTRODUCTION ^{68}Ga-PSMA-PET/CT imaging is rapidly becoming the gold standard for imaging of PCa. Androgen deprivation therapy (ADT) and radiation therapy (RT) are frequently used as first-line treatment. The aim of this study is to investigate the effect of ADT on ^{68}Ga-PSMA-PET-CT uptake (SUV_{max}) in the prostate tumour lesion and normal liver tissue in patients with PCa.

METHODS A total of 235 ^{68}Ga-PSMA-PET/CT scans from the period 2016-2021 were examined. All PCa patients who received a scan during that period were included. Since literature suggests that AB can affect tumour uptake, patients who used AB and patients without AB were selected. All scans were evaluated on various characteristics. Differences in PSMA uptake (SUV_{max}) in the prostate tumour lesions and physiological uptake (SUV_{max}) in the liver were used to calculate the tumour-to-liver ratio to draw a result.

RESULTS ADT did not have a significant influence on ^{68}Ga-PSMA uptake. Furthermore, recent RT (<5 years ago) showed a significant influence on tumour uptake (0.54 ± 1.16; $p < 0.001$). In addition, recurrence after prostatectomy also showed a significant influence on tumour uptake (1.36 ± 1.15; $p = 0.034$).

CONCLUSION ADT did not significantly influence ^{68}Ga-PSMA tumour uptake. Patients who recently had undergone RT showed a statistically significant decrease in tumour uptake, while patients that had undergone prostatectomy followed with recurrence showed a statistically significant increase in tumour uptake. Further research is needed to conclude whether treatment or therapy can intervene with a planned ^{68}Ga-PSMA-PET/CT scan in patients with prostate cancer.

KEY WORDS: ^{68}Ga-PSMA; prostate cancer; PSMA; PET/CT; androgen blockers; GnRH agonists, bicalutamide

INTRODUCTION

Prostate cancer (PCa) is one of the most commonly diagnosed malignancy and fifth leading cause of cancer death worldwide. In 2018 there were 1.3 million new cases and 359,000 deaths. (1) Over 30% of patients progress from localized stage disease to advanced stage over 10 years. (2)

PCa mortality has decreased in many countries due to screening, early detection and improved treatment. (3) The prostate gland is located in the male pelvis, at the base of the penis. The prostate gland requires androgen (testosterone) to function in a good manner (1). For this reason, androgen blockers work well as a PCa treatment. Cancer begins with a mutation in normal prostate glandular cells, they grow and multiply while they spread to the surrounding prostate tissue forming a tumour nodule. It is common that PCa can metastasize to the bones and lymph nodes. (1)

Prostate-specific membrane antigen (PSMA) is a membrane protein receptor and possesses an oncogenic signaling role in the prostate cancer cell, by acting on glutamate receptors, through its catalytic activities (NAALDase and folate hydrolase) which both result in the release of glutamate and also by activation of the Pi3K-AKt growth pathways. (4) The fact that PSMA is strongly overexpressed in the prostate cancer cells makes it a valid marker that can be used for imaging as well as for therapy. PSMA based positron emission tomography (PET) together with computed tomography (CT) (5), also known as PSMA-PET/CT imaging, is rapidly becoming the gold standard for imaging of the staging of intermediate and advanced PCa. (6) ^{68}Ga-PSMA binds to the cells that express PSMA, including the PCa cells that overexpress PSMA.

Androgen deprivation therapy (ADT), like androgen blockers and gonadotropin-releasing hormone agonists (GnRH agonists) and also radiation therapy are frequently used as first-line treatment to manage relapse after progression and for patients with metastatic hormone sensitive prostate cancer (HSPC). (1) This treatment suppresses the growth of the cancer cells by depleting androgen in different ways. The cancer cells need androgen to survive. This treatment can be effective for several years, until resistance develops, at which point the stage of castrate resistant prostate cancer (CRPC) is almost unavoidable. (7, 8)

Several preclinical studies and some clinical studies also showed that there was a difference in tumour uptake of the ^{68}Ga-PSMA (9-14), comparing groups that received androgen blockers with groups that received no androgen blockers. More research is needed to conclude whether treatment with androgen blockers affects ^{68}Ga-PSMA-PET/CT uptake. If there is an relevant influence, it must be taken into account before a ^{68}Ga-PSMA-PET/CT scan is planned for a patient.

The aim of this study is to investigate the effect of androgen deprivation therapy on ^{68}Ga-PSMA-PET/CT uptake, which is indicated for the diagnosis of prostate cancer. The specific objective is to assess whether androgen blockers, GnRH agonists and radiation therapy cause a difference in ^{68}Ga-PSMA uptake, measured as the maximum standardized uptake value (SUV_{max}), in the prostate tumour lesions and normal liver tissue in PCa patients.

METHODS

Patient selection

The study was conducted at the Amsterdam UMC, location AMC, the Netherlands. This study was approved

by the Medical Ethics Assessment Committee. During the period 2016-2021, patients were included if they had received a ^{68}Ga-PSMA-PET/CT scan, active or earlier diagnosed and who were at least 18 years old at the time of the scan in the Amsterdam UMC, location AMC. The design of the study was to retrospectively evaluate all scans involved on various characteristics described later in the article. In addition, the tumour uptake was determined per scan and linked to the scan. Since the available literature suggests that androgen blockers can affect tumour uptake, in the available data patients who use androgen blockers and also patients who do not use androgen blockers were selected. Ultimately, in addition to the use of androgen blockers, other factors were also examined, such as undergoing radiotherapy, and the influence of the other variables on tumour uptake was examined. Differences in ^{68}Ga-PSMA SUV_{max} in the primary tumour, differences in SUV_{max} in liver and differences in tumour-to-liver ratio between the variables were reviewed to draw a result. The PSMA uptake in the liver was included to serve as physiological PSMA uptake, since it is known there are low levels of physiologic PSMA expression in the liver. (15) To correct for background, the SUV_{max} of the psoas major muscle is also calculated. (16) As described in the introduction, in addition to the use of androgen blockers, the use of GnRH agonists was also examined, as well as the undergoing of radiotherapy and prostatectomy to assess the differences in ^{68}Ga-PSMA uptake in patients with prostate cancer.

Data collection

The data of patients were retrospectively collected in the IBC-database of the Radiology and Nuclear Medicine department. The following data is available in the IBC-database for all patients: date of PET/CT scan, date of birth, length, weight, BMI, activity (MBq) and dosage (ng) of the administered radiopharmaceutical (^{68}Ga-PSMA-11). Data from Epic and Hermes software were manually added to the database. The following data was added from Epic: androgen blocker use (divided into two groups: <1 month ago and ≥1 month ago (14)), GnRH agonists use (divided into two groups: <3 months ago and ≥3 months ago (14)), radiation therapy (divided into two groups: <5 years ago and ≥5 years ago (17)), prostatectomy (17), date of treatment/last dose, type of PCa (hormone sensitive vs. castration resistant (9, 12)) , Gleason-score (divided into two groups: ≤7 and >7 (9)) , tumour grade (13) and PSA levels (9). The following data was added from the Hermes database: SUV_{max} in primary tumour, SUV_{max} in the liver and SUV_{max} in the psoas major muscle. The latter served as background PSMA uptake.

^{68}Ga-PSMA labeling procedure

First, 1.1 ml ^{68}Ga solution in 0.1 M HCl was generated using a Ti02 based ^{68}Ge /^{68}Ga generator (Galli Eo, IRE ELit, Belgium). Then, a sodium acetate buffer dilution was made from 25 ml European Pharmacopoeia (Ph. Eur.) sodium acetate buffer solution (Fischer Chemical, United Kingdom) and 75 ml NaCl 0.9%. Thereafter 30 µg PSMA-11 (ABX, Germany) was solved in 2 ml of this solution, added to the 1.1ml ^{68}Ga and reacted for 5 minutes at room temperature. Radiolabeling efficiency was controlled using a radio high-performance liquid chromatography, a radiolabeling efficiency of >95% was sufficient.

^{68}Ga-PSMA-PET/CT imaging

The patients were imaged using a PET/CT system. Up to October 1 2017 the PET/CT scans were performed with the Gemini TF (Philips Healthcare, Eindhoven, the Netherlands).). First, a diagnostic CT scan was performed from the thigh to the skull base (120kV, 150mAs, 16x1.5 collimation, 0.8013 pitch) during administration of 2ml/kg intravenous contrast agent (iopromide 300 mgI/ml, Ultravist 300; Bayer Healthcare Pharmaceuticals, Berlin, Germany) with 2ml/s flow and a 50s delay (portal phase). Then, the PET acquisition followed, with a scan time of 2,5 minutes per bed position and reconstructed in 5mm slices and 4x4mm pixels.

After October 1, 2017 a Biograph mCT Flow (Siemens Medical Solutions, Erlangen, Germany) equipped with enhanced axial field of view (TrueV) scanner was used. A diagnostic CT scan was performed with automatic modulation in current and voltage. Reference values were set on 120 kV and 160 mA, 128x0.6 collimation and 0.9 pitch. CT scan was performed after administration of intravenous iodinated contrast medium (90 ml Xenetix 350; Guerbet, Roissy CdG Cedex, France) with 3 ml/s flow and a 65s delay (portal phase). PET was performed with continuous bed motion at 1.5 mm/s in 3D acquisition mode. CT data was used for PET attenuation correction and PET data were reconstructed with TrueX algorithm (three dimensions ordered subsets expectation maximization iterative reconstruction with time of flight and point spread function compensation, 21 subsets, 2 iterations, and a 5 mm Gaussian post-filter) in 4 mm × 4 mm × 5 mm voxels.

PET/CT image acquisition was performed 60 to 75 minutes after intravenously injection of approximately 1.5 MBq/Kg of ^{68}Ga-PSMA-11. The patients were injected were injected with 10mg of Furosemide (Centrafarm, Breda, the Netherlands) one hour before the scan and instructed to drink at least 1000mL of water to stimulate ^{68}Ga-PSMA-11 excretion from the renal calyces. Patients were asked to urinate before the scan. Patients were scanned head first in supine position and instructed to breath normally during the scan.

The analysis of the PET/CT images and calculation of SUV_{max}

The PET/CT images were all analyzed during the period 2016 up to and including 2021. To calculate the SUV_{max} of the prostate tumour, the liver and the psoas major muscle, the PET/CT images were analysed using Hermes Hybrid Viewer software (version 5.1.0). With the help of certain tools in this program, the areas in the prostate, liver and muscle could be drawn in order to calculate the SUV_{max}. The SUV_{max} was calculated with amendment for body weight, using a spherical volume of interest (VOI) tool with lengths of 4.00 cm for the prostate and liver and 2.00 cm for the psoas major muscle. The latter was done because the psoas major muscle serves as background, so you do not want to accidentally include tumour uptake from another part of the body in the calculation. This is because if multiple tumour lesions were present, the most tumour with the highest SUV_{max} was chosen for the calculation. As described above, during the period 2016 – 2021 two different scanner were used to perform PET/CT scans. To compensate for a possible difference in sensitivity between these two scanners it was decided to use the SUV_{max} of the liver or tumour-to-background ratio to draw results (SUV_{max} of tumour or liver divided by the SUV_{max} of the muscle). For the liver it was drawn in the

TABLE 1

Results of multivariable linear regression analysis for the influence of variables on ^{68}Ga-PSMA-PET/CT uptake in patients with prostate cancer

Characteristics	Number	Univariate linear regression		Multivariate linear regression	
		B† ± SD	*P*-value	B† ± SD	*P*-value††
PSA	235 (100%)	1.00 ± 1.00	0.036	1.00 ± 1.00	0.059
Tumour stage					
T1 and T2	91 (38.7%)	1		1	
T3 and T4	135 (57.5%)	1.18 ± 1.14	0.199	1.25 ± 1.12	0.062
Unknown	9 (3.8%)				
Recurrence after prostatectomy					
Yes	76 (32.3%)	1.33 ± 1.17	0.065	1.36 ± 1.15	0.034
No	159 (67.7%)	1		1	
Radiation therapy					
No	125 (52.2%)	1		1	
≥5 years ago	43 (18.3%)	0.56 ± 1.18	< 0.001	0.79 ± 1.19	0.174
<5 years ago	67 (28.5%)	0.43 ± 1.14	0.001	0.54 ± 1.15	< 0.001
GnRH agonists					
No	136 (57.9%)	1		1	
≥ 3 months ago	65 (27.6%)	0.59 ± 1.15	< 0.001	0.80 ± 1.18	0.179
< 3 months ago	34 (14.5%)	1.08 ± 1.01	0.659	1.01 ± 1.18	0.955
Androgen blockers					
No	112 (47.7%)	1		1	
≥ 1 month ago	79 (33.6%)	0.78 ± 1.15	0.076	0.81 ± 1.18	0.197
< 1 month ago	44 (18.7%)	1.32 ± 1.18	0.099	1.21 ± 1.16	0.206
Number of ^{68}Ga-PSMA scans					
1	174 (74.0%)	1		1	
≥ 2	61 (26.0%)	0.69 ± 1.16	0.014	0.79 ± 1.16	0.118
^{68}Ga-PSMA-11					
activity	235 (100%)	1.16 ± 1.22	0.464		
peptide	235 (100%)	1.00 ± 1.00	0.434		
Age	235 (100%)	1.01 ± 1.01	0.495		
BMI	235 (100%)	1.00 ± 1.01	0.985		
Prostate cancer type					
HS	174 (74.0%)	1			
CR	42 (17.9%)	1.07 ± 1.17	0.682		
Unknown	19 (8.1%)				
Gleason score					
≤ 7	91 (38.7%)	1			
> 7	130 (55.3%)	1.14 ± 1.15	0.324		
Unknown	14 (6.0%)				

Data are displayed as B ± SD. Multivariate linear regression was used.

† B = regression coefficient (slope).

†† *P*-value < 0.05 was considered as statistically significant in the multivariate linear regression analysis.

right lower lobe for all patients, as long as there were no present metastases. In this case the region of interest was drawn elsewhere in the liver. Some patients had undergone a prostatectomy, meaning their prostate had been removed. In these patients, the patients with recurrence were included in the SUV_{max} of the tumour. In the patients without recurrence after prostatectomy only the SUV_{max} of the liver and the psoas major muscle were calculated, since it was not possible to calculate a SUV_{max} of the prostate.

Statistical analysis

SPSS software (version 28, IBM, USA) was used to perform the statistical analysis. The data was transformed using natural logarithm to achieve normal distribution. However, the results are reported according to the original values. Descriptive statistics of variables focused on frequencies. Univariate logistic regression models were used to examine the relationships between the different characteristics and SUV_{max} in tumour-to-liver ratio. This was done to see which variables showed a trend thus could possibly influence the PSMA tumour uptake. All variables with a *P* value below 20% in the univariate analysis were included for the multivariate linear regression analysis. Multivariate linear regression was performed to predict the correlation between the patient characteristics, treatment and tumour characteristics with ^{68}Ga-PSMA tumour uptake. This analysis was two-tailed and a *P*-value of < 0.05 will be considered as statistically significant.

RESULTS

Study population

A total of 174 patients who in total underwent 235 ^{68}Ga-PSMA PET/CT scans were enrolled in this study. Mean patient age was 73.2 ± 7.5 years and the mean BMI was 26.9 ± 4.7 years. A total of 174 patients were retrospectively reviewed in this study. This amounted to 235 PET/CT scans, whereas some patients had undergone multiple ^{68}Ga-PSMA PET/CT scans (from 1 scan to 6 scans). Each scan was considered an individual scan, as all information associated with that specific scan was collected from scratch.

Univariate linear regression

Table 1 presents the results of the univariate linear regression and of the multivariate linear regression. All variables with a *P*-value below 20% in the univariate analysis were included for the multivariate linear regression analysis. The univariate linear regression analysis showed that the variables ^{68}Ga-PSMA-11 activity (p = 0.464), ^{68}Ga-PSMA-11 peptide (p = 0.434), age (p = 0.495), BMI (p = 0.985), prostate cancer type (p = 0.682) and Gleason score (p = 0.324) did not have a significant influence on PSMA tumour uptake, hence were not included. The univariate linear regression analysis also showed that the variables (PSA (1.00 ± 1.00, p = 0.036), tumour stage (1.18 ± 1.14, p = 0.199), prostatectomy (1.33 ± 1.17, p = 0.065), radiation therapy (>5 years ago, 0.56 ± 1.18, p < 0.001; <5 years ago, 0.43 ± 1.14, p = 0.001), GnRH agonists (>3 months ago, 0.59 ± 1.15, p < 0.001), androgen blockers (>1 month ago, 0.78 ± 1.15, p = 0.076; <1 month ago, 1.32 ± 1.18, p = 0.099) and the number of ^{68}Ga-PSMA PET/CT scans (0.69 ± 1.16, p = 0.014) independently appeared to have a potentially significant influence on ^{68}Ga-PSMA tumour uptake and thus were included in the multivariate linear regression analysis.

Multivariate linear regression

As can be seen in table 1, the multivariate linear regression analysis showed that the variables PSA (1.00 ± 1.00, p = 0.059), tumour stage (1.25 ± 1.12, p = 0.062), GnRH agonists (>3 months ago, 0.80 ± 1.18, p = 0.179; <3 months ago, 1.01 ± 1.18, p = 0.955), androgen blockers (>1 month ago, 0.81 ± 1.18, p = 0.197; <1 month ago, 1.21 ± 1.16, p = 0.206) and the number of ^{68}Ga-PSMA PET/CT scans (0.79 ± 1.16, p = 0.118) did not have a significant influence on ^{68}Ga-PSMA tumour uptake in the multivariate model.

The multivariate linear regression analysis also showed that the variables prostatectomy (1.36 ± 1.15, p = 0.034) and recent radiation therapy (<5 years ago, 0.54 ± 1.15, p < 0.001) do have significant influence on ^{68}Ga-PSMA tumour uptake in the multivariate model. Figure 1 shows a visual display of two patients who have undergone prostatectomy, of which one patient had a recurrence and the other patient did not.

DISCUSSION

This is the first study that has looked at the effect of androgen blockers and GnRH agonists on SUV_{max} of the tumour-to-liver ratio in patients with this relatively large amount of scans. Our study showed a significant higher SUV_{max} of the tumour-to-liver ratio after prostatectomy and a significant lower SUV_{max} of the tumour-to-liver ratio after recent radiation therapy (under 5 years ago). This means that after a patient has undergone radiation therapy recently, the ^{68}Ga-PSMA tumour uptake is significantly lower. Also this means that after a patient has undergone prostatectomy the ^{68}Ga-PSMA tumour uptake is significantly higher. The tumour uptake after recent radiation therapy seemed to be 46% lower (p < 0.001), while the tumour uptake after prostatectomy seemed to be 36% higher (p = 0.034).

A recent study with five patients showed an possible enhancing effect on PSMA-imaging in patients with recurrence after radical prostatectomy. (18) This corresponds to our results for the group recurrence after prostatectomy. It is indeed possible for prostate cancer to return after a prostatectomy, a study in 2013 suggested that prostate cancer recurs in 20-40 percent of men within 10 years of having a radical prostatectomy. This can be patients with an aggressive type of prostate cancer. (19)

A study of Vlachostergios *et al.* examined the association of PSMA uptake with the overall survival in

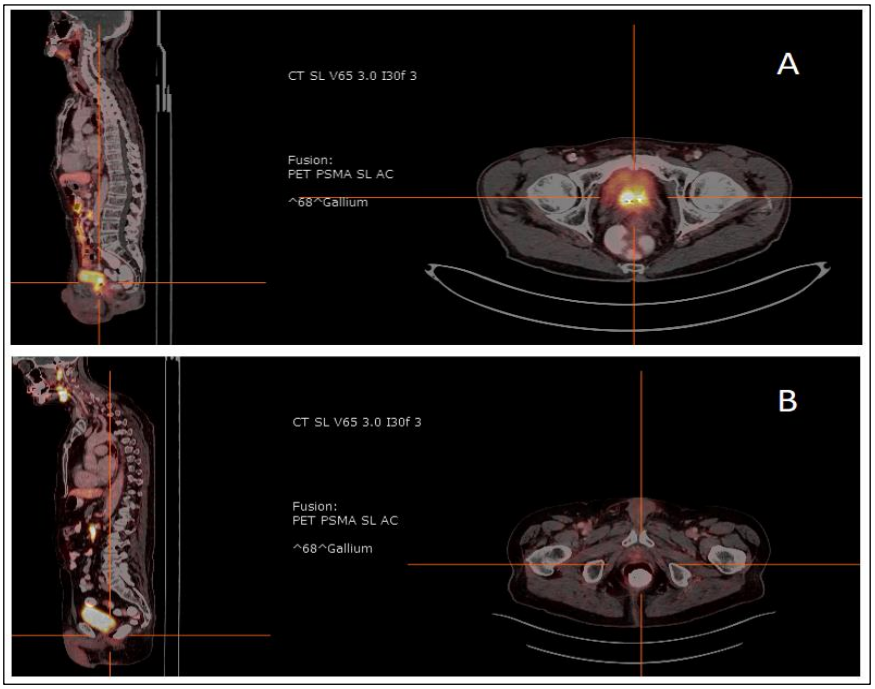

FIGURE 1. The figure shows two different PET/CT scans (Hermes Hybrid Viewer software, version 5.1.0). The top scan (A) shows a patient who has had a recurrence after a prostatectomy. This can be seen in the highly illuminated tumour lesion. The bottom scan (B) shows a patient who has not had a recurrence after a prostatectomy. Although this is known in the data, this can also be seen in the scan itself.

238 progressed patients. (20) They found a significant correlation of high PSMA uptake with shorted median overall survival compared to low or absent PSMA uptake. This means that a lower PSMA uptake can predict a longer median overall survival. Another study of 635 patients, conducted in 2018 at the John Hopkins Hospital, compared radiation therapy against observation. (17) The patients who had undergone radiation therapy showed an statistically significant threefold increase in prostate cancer specific survival rate after a follow-up of six years compared with observation. However PSMA uptake was not measured, so we couldn't say that an statistically significant increase in prostate cancer specific survival after radiation therapy is associated with a decrease in PSMA uptake. But a decrease in PSMA uptake seems to have an effect on the survival rate of the patients with prostate cancer. So with the findings of our study we could say that the decrease in PSMA uptake after radiation therapy seems to have a positive effect on survival.

The membrane antigen PSMA is a transmembrane glycoprotein and is moderately expressed in several tissues, including normal prostate cells. It is however known that prostate cancer cells typically show increased PSMA expression. (21) Androgen blockers act by directly binding to androgen receptors and competitively dislocating androgens like testosterone from the androgen receptor. In this way they prevent androgens from activating the androgen receptor and thus also the biological effects. (22) The prostate gland requires androgen to function in a good manner. Blocking the androgen receptors appears to lead to upregulation of PSMA. (14) Besides the androgen blockers, there is also the GnRH agonists which bind to the GnRH receptor. Activation of the GnRH receptor, expressed on the pituitary gland and prostate cells, leads to release of follicle-stimulating hormone and luteinizing hormone. Prolonged activation of this receptor leads to desensitization of the pituitary gland, through downregulation of the GnRH receptor, which in turn leads to less secretion of follicle-stimulating hormone and luteinizing hormone, what leads to less production of androgens by the testicles. (23) Cell line and mouse models demonstrated that the PSMA receptor is manipulable when it its influenced by androgen blockers. (9)

Some clinical studies showed an increase in PSMA uptake after androgen deprivation therapy, including mostly androgen blockers and some GnRH agonists (10, 18, 24), while another study showed a decrease in PSMA uptake after androgen deprivation therapy, including androgen blockers and GnRH agonists. (25) Also one study showed decrease in uptake in the HSPC cohort and increase in uptake in the CRPC cohort. (9) However there is still a deficiency of information on the effect of androgen deprivation therapy on PSMA-PET/CT imaging in vivo and the impact of this potential effect. Our results showed no influence of ADT on tumour uptake. The studies we referred to had a small sample size (from n=1 to n=15) relative to ours. This gives our results more power.

For the time being, the fact that factors such as androgen deprivation therapy are potentially able to influence the PSMA receptor and with that also the tumour uptake, can have a significant impact on the value of ^{68}Ga-PSMA PET/CT imaging as a diagnostic tool and also its ability to monitor response on treatment. In our study we did not see an significant influence of the use of androgen blockers on the tumour uptake, both in the univariate regression as in the multivariate regression. So we could not confirm in vivo the same results as the clinical studies. Our study also did not find a significant influence of the use of GnRH agonists on the tumour uptake, both in the univariate regression as in the multivariate regression. As in the case with the androgen blockers, for the GnRH agonists we also cannot confirm in vivo the same results as the previous studies.

In the study of Onal *et al.* a significant moderate correlation was observed between PSA level and median tumour SUV_{max}. Patients with serum PSA > 10 ng/ml showed a significantly higher tracer uptake in the primary tumour. (13) Our study showed a trend (1.00 ± 1.00 with $p = 0.059$) but this prediction was not significant, although it was close to a *P*-value of < 0.05. In the same study also the tumour stage was assessed and they found that the tumour stage was significantly associated with lymph node metastasis, but no influence on prostate tumour uptake was shown. (13) In our study the multivariate analysis showed a trend that the tumour stage led to a higher tumour uptake, but this result was not significant (1.25 ± 1.12 with $p = 0.062$). This means we can confirm the absence of statistically significant influence of tumour stage on tumour uptake.

Our study is not devoid of limitations. Firstly, this study was of retrospective nature, which could have led to some patient characteristics to be missing. Secondly, the sample size could also be seen as a limitation. This study had a relatively large sample size, but a bigger sample size could have further strengthen our results nevertheless, although this was not possible due to time limitation. Furthermore, in the period of 2016-2021 two different scanners were used for the PET/CT scans and SUV_{max} could fluctuate across different PET/CT scanner due to intrinsic variability of SUV (26). To compensate for this, we drew result with the use of the tumour-to-background and liver-to-background ratio. Although we have compensated for the variability, the use of two different scanners can still be seen as a limitation. In addition, we did not compare patients intra-individually.

CONCLUSION

This study showed that androgen deprivation therapy did not significantly influence ^{68}Ga-PSMA tumour uptake, which possibly could indicate that androgen deprivation therapy does not intervene with ^{68}Ga-PSMA-PET/CT imaging. Furthermore, patients who recently had undergone radiation therapy showed a statistically significant decrease in uptake and thus radiation therapy may possibly interfere with imaging. On the contrary, a patient that had undergone a prostatectomy followed with a recurrence, showed a statistically significant increase in uptake and thus recurrence after prostatectomy can also have an influence on ^{68}Ga-PSMA-PET/CT imaging. Further research is needed to conclude whether treatment or therapy can intervene with a planned ^{68}Ga-PSMA-PET/CT scan in patients with prostate cancer.

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