Obesity leads to a higher rate of positive surgical margins in the context of robot-assisted radical prostatectomy. Results of a prospective multicenter study

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Introduction

Current results concerning the effect of body mass index (BMI) on positive surgical margins (PSMs) after robot-assisted radical prostatectomy (RARP) in patients with localized prostate cancer are inconsistent. Therefore, the aim of this study was to further analyse the association between BMI and PSMs after RARP.

Material and methods

Between March 2017 and December 2017 a multicentre, prospective, randomised, single-blind series with a blinded outcome assessment of 232 RARP patients was performed. Multivariate logistical regression models were used to analyse the independent effect of obesity, with body-mass-index (BMI) dichotomised at 30 kg/m² (model-1) and at 90th percentile (model-2), on PSMs.

Results

Median BMI was 27.2 kg/m², PSMs were found in 15.5% (n = 36). In multivariate model-1, obesity did not have a significant effect on PSMs (OR 2.34, p = 0.061). However, if BMI was dichotomized at the 90th percentile (BMI ≥33.7 kg/m²), patients with a higher BMI showed PSMs four-times more frequently (OR 3.99, p = 0.013). In both models, preoperative prostate-specific antigen (PSA) levels and pathological tumour stage had a significant effect on PSMs. There was no significant correlation between BMI and the extent of PSMs, nor a significant difference between the BMI groups and the localisation of PSMs. There was a higher percentage of posteriolateral PSM localisation in obese patients compared to patients with a BMI of less than 30 kg/m² (58.3% and 25.3% of the localisations were posterolateral in obese and non-obese patients, respectively), however this effect was not statistically significant (p = 0.175).

Conclusions

In addition to a longer operation time and about twice as many complications, patients with a BMI of ≥33.7 kg/m² had a higher PSM rate after RARP. Differences in localization of PSMs in relation to obesity should be evaluated in future research.

Key Words: prostate cancer • radical prostatectomy • robotic surgery • body mass index • localisation of positive surgical margins • extent of positive surgical margins

INTRODUCTION

Robot-assisted radical prostatectomy (RARP) using the da Vinci Surgical System (Intuitive Surgical Inc., Sunnyvale, CA, USA) was performed for the first time in the year 2000 by Jochen Binder in Frankfurt/Main (Germany) [1]. Since then, RARP has been adopted by many centres as a standard surgical approach for localised prostate cancer (PCa) due to its good oncological and functional results and...
low perioperative morbidity. Furthermore, RARP is associated with lower blood loss, lower transfusion rates and a shorter inpatient stay compared to open radical prostatectomy [2, 3].

Obesity represents a growing health problem in industrial Western nations. According to the World Health Organisation (WHO), in the year 2016, 39% of men worldwide were overweight (body mass index (BMI) more than 25 kg/m²) and 11% of men were obese (BMI more than 30 kg/m²) [4]. When it comes to surgical interventions of obese patients, surgeons are faced with a challenge, even with regard to laparoscopic and robot-assisted surgery [5]. The Pasadena Consensus Panel recommends that patients with a BMI of more than 30 kg/m² should be managed by an experienced RARP-surgeon [6]. The current state of literature on this topic, however, is inconclusive. Several studies have explored perioperative parameters (such as estimated blood loss (EBL), operating time) and oncological outcomes (mainly defined as a positive surgical margin (PSM)) of RARP with regard to patients’ BMI [7–15]. Some of these studies showed a positive correlation between PSMs and BMI [7, 8, 9], although in one series this correlation could only be shown for apical PSMs [9]. Furthermore, BMI was shown to be a risk factor for higher EBL and longer operating time [7, 10, 11]. On the other hand, several other studies found no influence of patients’ BMI on PSMs [11–14]. Interestingly, one recently published paper by Porcaro et al. even showed a statistically significant inverse association for patients’ BMI and PSMs [15].

The aim of this prospective multicentre series of RARP patients was to examine the effect of BMI on PSMs. For this we analysed the independent influence of obesity on the rate of PSMs, and additionally, we investigated the relationship between BMI with localisation and the extent of PSMs.

MATERIAL AND METHODS

Study design

The data analysed for this publication was originally recorded within the context of the PIANOFORTE (Impact of peritoneal flap on outcome after robotic prostatectomy) study [16]. This study was designed as a multicentre, prospective, randomised, single-blind study, with a blinded outcome assessment and a follow up period of 90 days.

Study group and clinical criteria

Between March 2017 and December 2017, 404 RARPs (clinically organ-confined PCa and all M0) were performed in three German centres and one Austrian centre; of these, after the application of the inclusion and exclusion criteria, 232 patients (57.4%) could be included in the PIANOFORTE study [16]. Inclusion and exclusion criteria as well as obtained patient characteristics & follow up are summarized in Table 1. Further details of the study as well as its results concerning the effect of the peritoneal flap on different study endpoints were described in an earlier publication [16]. The PIANOFORTE study has an ethics committee’s positive vote and was registered in the clinical trials registry (DRKS-ID: DRKS00011115) [17]. RARP with a simultaneous bilateral pelvic lymph node dissection (PLND) was performed in all patients regardless of their preoperative risk classification. RARP was conducted in each study centre in a standardised manner via transperitoneal approach [16]. All surgeons had already completed their learning curve (>100 RARPs).

Histopathological criteria

The uropathologists in the four centres evaluated the histopathological samples according to a standardized protocol [18]. The Gleason grade was assessed according to the ISUP (International Society of Urological Pathology) classification [19]. The uropathologists assessed the linear expansion and location of the PSM. The surgical margins were then positive if cancer cells could be visualized on the inked surface of the histopathological samples [20]. According to the anatomical location, PSMs were classified as posteriolateral (left and right), posterior, anterior, bladder neck and apical. Lymph nodes were examined histopathologically after hematoxylin and eosin (HE) staining. In each case, the number and histopathological status of the removed lymph nodes was recorded. The histopathological specimens were classified according to the AJC staging system 2017 for PCa (pT and pN status) [21]. The weight of the prostate was also documented.

Statistical analysis

Continuous variables were reported as median and interquartile range (IQR), and categorical endpoints as absolute and relative frequencies. The Kruskal-Wallis-H-Test was used to differentiate the distribution of continuous criteria (endpoints) between the treatment groups. The distribution of categorical endpoints was analysed using the Chi-squared test (in case of 2 x 2 contingency tables: Fisher’s exact test). The correlation between BMI and extent (length) of PSMs was examined using Spearman’s correlation.
The independent effect of the dichotomised BMI on PSMs was analysed by means of multivariate logistical regression models (MLRM). The model prerequisites were previously defined as follows: a) no multi-collinearity, b) linearity of the logit, c) no outliers (analysed using the model’s standardised residuals), d) significance of the final model (examined using the Omnibus test), and e) a minimum of eight events per degree of freedom of the independent variables included in the final model. Due to the low event rate of PSMs, a stepwise backward elimination of the independent variables on the basis of the probability of the likelihood-ratio-statistics was chosen. The primary independent variables elected were: preoperative PSA-level (continuously in ng/ml), histopathological tumour stage (pT3-4 vs. pT2), Gleason-grading (ISUP-grade 3–5 vs. 1–2), nerve sparing (dichotomised) and prostate weight (continuously in g). The dichotomised BMI was analysed as an independent variable with regard to its effect on PSMs in
two models by adjustment for the factors mentioned above: model 1 with a dichotomisation in ≥30 kg/m² vs. <30 kg/m² and model 2 with a dichotomisation in ≥90th percentile vs. <90th percentile (33.7 kg/m²). Data analysis was carried out using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, N.Y., USA). All mentioned p-values are two-tailed, the significance level was defined as p<0.05.

RESULTS

A total of 232 RARP patients were analysed. A selection of clinical, histopathological and functional study criteria with a division of patients with and without obesity in relation to the BMI threshold value of 30 kg/m² is shown in Table 2 (data in relation to the BMI threshold of 33.7 kg/m² (90th percentile) are provided in Table 4). Median BMI was 27.2 kg/m² (IQR: 25.2–29.7 kg/m²). When compared to patients without obesity, obese patients had a longer operating time by 20 minutes (console time, 180 vs. 160 min, p = 0.013) and experienced more than twice as many complications (Clavien-Dindo grade ≥1) within the first 90 postoperative days (p = 0.013). It is worth

Table 4. Distribution of study criteria among patients with and without obesity in relation to the BMI threshold value of 33.7 kg/m² (90th percentile)

| Criteria                        | Study group (n = 232) | BMI <33.7 kg/m² (n = 209 [90.1%]) | BMI ≥33.7 kg/m² (n = 23 [9.9%]) | p     |
|---------------------------------|-----------------------|-----------------------------------|---------------------------------|-------|
| Perioperative – clinical and functional criteria |                        |                                   |                                  |       |
| Median age in years (IQR)       | 65 (60–70)            | 66 (60–70)                        | 65 (61–69)                      | 0.935 |
| BMI in kg/m² (IQR)              | 27.2 (25.2–29.7)      | 26.6 (25–28.6)                    | 36.3 (35.4–37.3)                | <0.001|
| PSA in ng/ml (IQR)              | 8.2 (6–12.9)          | 8 (6–12.5)                        | 9.5 (6.9–15.5)                  | 0.156 |
| Prostate weight in g (IQR)      | 50 (40–60)            | 50 (40–67)                        | 50 (40–71)                      | 0.487 |
| Operating time at the console in min (IQR) | 167 (129–217)       | 165 (126–214)                     | 183 (160–250)                   | 0.029 |
| Number of removed LNs (IQR)     | 16 (11–21)            | 15 (10–20)                        | 22 (13–29)                      | 0.002 |
| Nerve sparing (%)               | 122 (52.6%)           | 109 (52.2%)                       | 13 (56.5%)                      | 0.827 |
| Clavien-Dindo grade ≥1 at time of d90 (%) | 43 (18.5%)           | 35 (16.7%)                        | 8 (34.8%)                       | 0.047 |
| SUI grade 2–3 at time of d90 (%) | 55 (23.7%)            | 46 (22.0%)                        | 9 (39.1%)                       | 0.075 |
| Histopathological criteria      |                        |                                   |                                  |       |
| Tumour stage >pT2 (%)           | 67 (28.9%)            | 61 (29.2%)                        | 6 (26.1%)                       | 0.999 |
| pN1 (%)                         | 16 (6.9%)             | 14 (6.7%)                         | 2 (8.7%)                        | 0.664 |
| ISUP–GGG 1                      | 17 (7.3%)             | 16 (7.7%)                         | 1 (4.3%)                        | 0.883 |
| ISUP–GGG 2                      | 120 (51.7%)           | 106 (50.7%)                       | 14 (60.9%)                      |       |
| ISUP–GGG 3                      | 56 (24.1%)            | 51 (24.4%)                        | 5 (21.7%)                       |       |
| ISUP–GGG 4                      | 21 (9.1%)             | 19 (9.1%)                         | 2 (8.7%)                        |       |
| ISUP–GGG 5                      | 18 (7.8%)             | 17 (8.1%)                         | 1 (4.3%)                        |       |
| ISUP–GGG 3–5 (%)                | 95 (40.9%)            | 87 (41.6%)                        | 8 (34.8%)                       | 0.657 |
| PSM (%)                         | 36 (15.5%)            | 28 (13.4%)                        | 8 (34.8%)                       | 0.013 |

BMI – body mass index; IQR – interquartile range; LNs – lymph nodes; ISUP–GGG – International Society of Urological Pathology-Gleason grading groups; OR – odds ratio; PSA – prostate-specific antigen; PSM – positive surgical margins

Table 3. Multivariate models with backward elimination for the endpoint PSMs (model 1 including the BMI dichotomised at the obesity cut-off (30 kg/m²), model 2 including the BMI dichotomised at the 90th percentile of the study group (33.7 kg/m²))

| Independent variable | OR (95% CI) | p     |
|----------------------|-------------|-------|
| Model 1              |             |       |
| PSA (continuously in ng/ml) | 1.06 (1.03–1.10) | 0.001 |
| Tumour stage (>pT2 vs. pT2) | 4.36 (1.91–9.94) | <0.001|
| ISUP–GGG (3–5 vs. 1–2) | –          | –     |
| Nerve sparing (yes vs. no) | –         | –     |
| Prostate weight (continuously in g) | – | –     |
| BMI (≥30 kg/m² vs. <30 kg/m²) | 2.34 (0.96–5.71) | 0.061 |
| Model 2              |             |       |
| PSA (continuously in ng/ml) | 1.06 (1.03–1.10) | 0.001 |
| Tumour stage (>pT2 vs. pT2) | 4.41 (1.92–10.12) | <0.001|
| ISUP–GGG (3–5 vs. 1–2) | –          | –     |
| Nerve sparing (yes vs. no) | –         | –     |
| Prostate weight (continuously in g) | – | –     |
| BMI (≥90th percentile vs. <90th percentile) | 3.99 (1.34–11.89) | 0.013 |

BMI – body mass index; CI – confidence interval; ISUP–GGG – International Society of Urological Pathology-Gleason grading groups; OR – odds ratio; PSA – prostate-specific antigen; PSM – positive surgical margins
| Author                | Trial design                     | Study period | Number of patients | Median PSA (ng/ml) | pT3/4 (%) | PSM (%) | BMI categories | Median BMI (kg/m²) | Influence of BMI on PSM frequency | Influence of BMI on PSM localization |
|-----------------------|---------------------------------|--------------|--------------------|--------------------|-----------|----------|-----------------|---------------------|-----------------------------------|-------------------------------------|
| Castle (2008) [10]    | Retrospective, single centre    | 2003–2006    | 140                | 7.3 vs. 7.1 (mean) | 17.9%     | 17.9%    | <30 kg/m², ≥30 kg/m² | 24.9 vs. 32.6 (mean) | Yes (p = 0.009)                    | Not reported                        |
| Coelho (2010) [9]     | Prospective, single centre      | 2008–2009    | 876                | 4.9                | 19%       | 11.5%    | Analysed as continuous variable | 28                                | No (p = 0.746)                     | Yes, high BMI → more PSM-apical (p = 0.0119), 38.6% apical, 34.6% PL, 15.8% MF, 10.4% BN |
| Moskovic (2010) [13]  | Retrospective analysis of prospective data, single centre | 2003–2009 | 1112               | 5.0 vs. 5.0 vs. 5.2 | 19.3%     | 17.1%    | <25 kg/m², 25–29.9 kg/m², ≥30 kg/m² | 23.5 vs. 27.3 vs. 32.1 | No (p = 0.94)                     | No                                  |
| Patel (2011) [8]      | Retrospective, multicentre      | 2002–2009    | 8418               | Not reported        | 22.7%     | 15.7%    | Analyzed as continuous variable | Not reported | Yes (p <0.001)                    | Not reported                        |
| Zilberman (2012) [14] | Retrospective, single-centre    | 2003–2009    | 577                | 5.3                | 18.2%     | 23.1%    | 23.1% (10.2% apical, 3.6% base, 14.2% peripheral) | 28.2                                | No (p = 0.35)                     | Yes, but not significant; trend towards more PSM-basal for higher BMI (BMI ≥30 kg/m²: 4.6%, BMI 25–29.9 kg/m²: 3.3%, BMI <25 kg/m²: 2.9%, p = 0.71) |
| Abdul-Mushin (2014) [7]| Retrospective, single centre    | 2008–2012    | 88                 | 5.3 vs. 5.0        | 27.3%     | 18.2%    | <40 kg/m², ≥40 kg/m² | 42.0 vs. 28.8 | Yes (p = 0.097)                    | Not reported                        |
| Albisinni (2018) [12] | Retrospective analysis of prospective data, multicentre | 2005–2015    | 539 (347 RARP, 192 LRP) | 6.5 None, excluded | 24%       |         | <25 kg/m², 25–29.9 kg/m², ≥30 kg/m² | 26.1                                | No (p = 0.14)                     | No                                  |
| Porcaro (2020) [15]   | Retrospective analysis of prospective data, single centre | 2013–2017    | 732                | 6.3                | 21.9%     | 26.3%    | Analysed as continuous variable | 25.8                                | Inverse (odds ratio, OR = 0.936; p = 0.021) | Not reported                        |
| Our series            | Prospective, multicentre        | 2017         | 232                | 8.2                | 28.9%     | 15.5%    | Model 1: <30 kg/m², ≥30 kg/m² Model 2: <90th percentile, ≥90th percentile | 27.2                                | Yes (p = 0.061 for cut-off at BMI 30 kg/m², p = 0.013 for cut-off at 90th percentile) | Higher rate of PSM-PL in BMI >30 kg/m² (BMI ≥30 kg/m²: 58.3%, BMI <30 kg/m²: 33.3%, p = 0.175) [case number too low to reach statistical significance] |

BMI – body mass index; BN – bladder neck; IQR – interquartile range; LRP – laparoscopic radical prostatectomy; MF – multifocal; OR – odds ratio; PL – posterolateral; PSA – prostate-specific antigen; PSM – positive surgical margin; RARP – robot-assisted radical prostatectomy
noting that there were no grade 4 or 5 complications. In terms of functional outcome no differences regarding grade 2 or 3 stress urinary incontinence were noticed 90 days following RARP (22.1% vs. 29.4%, \( p = 0.351 \)).

Histopathological criteria revealed no significant differences between the two groups, although patients with obesity did show about an 10% higher PSM rate (23.5% vs. 13.3%, \( p = 0.082 \)). BMI in the 90th percentile was 33.7 kg/m\(^2\). Eight out of 23 patients (34.8%) with a BMI ≥90th percentile had PSMs, compared to 13.4% (28/209 patients) with a BMI below the 90th percentile (\( p = 0.013 \)). It is worth noting that the original PIANOFORTE study randomised patients into the groups ‘peritoneal flap’ vs. ‘no peritoneal flap’, although this grouping had no influence on the PSM rate (\( p = 0.718 \)) [16].

When BMI was dichotomised at 30 kg/m\(^2\) for the multivariate model to predict PSMs (model 1), it affected the model’s quality (when applying backward elimination, it remained in the model until the last step). There was, however, no significant effect on the endpoint PSM (OR 2.34, \( p = 0.061 \)). However, if BMI dichotomisation was applied at the 90th percentile (model 2), patients with a higher BMI showed PSMs about four times more frequently (OR 3.99, \( p = 0.013 \)). In both models, preoperative PSA-levels and the histopathological tumour stage had a significant effect on PSMs (Table 3).

Among the 36 patients with PSMs, median BMI was 27.4 kg/m\(^2\) (IQR: 25.4–32.4 kg/m\(^2\)), and median PSM extent was 7.5 mm (IQR: 3.1–11 mm). There was no significant correlation between BMI (continuously and dichotomised at 30 kg/m\(^2\), respectively) and PSM extent (continuously) (\( r = 0.04 \); \( p = 0.980 \) and \( r = 0.117 \); \( p = 0.497 \), respectively). Notably, PSMs were more frequently found in the area of the neurovascular bundles (posteriolateral) in patients with a BMI ≥30 kg/m\(^2\). Obese patients showed a trend towards posteriolateral PSM localisation (seven PSM locations were posteriolateral from a total of 12 patients with PSM, 58.3%), compared to 8/24 (33.3%) in patients with a BMI <30 kg/m\(^2\) (\( p = 0.175 \)). Further trends when comparing other PSM localisations to the dichotomised BMI categories could not be found (results not shown). All in all, there were no significant differences between PSM localisation and BMI (dichotomised or continuously).

The proportion of nerve-sparing operations between BMI groups (<30 vs. ≥30 kg/m\(^2\)) did not reveal significant differences (54.7% vs. 45.1%; \( p = 0.267 \)). In patients with a BMI <30 kg/m\(^2\), the PSM rate did not differ between nerve-sparing and non-nerve-sparing surgery (13.1% vs. 13.4%). However, looking at patients with a BMI ≥30 kg/m\(^2\), there are at least descriptively relevant differences in the PSM rates between nerve-sparing and non-nerve-sparing surgery (30.4% vs. 17.9%, no significance calculations due to the small sample size).

Centre effects did not impact the study results (data not shown).

**DISCUSSION**

Obesity represents a growing health problem. According to the WHO, obesity has nearly tripled worldwide since 1975 [4]. This has an impact on PCa, being the most frequent malignant tumour disease among men, as well as on surgical PCa therapy. Over the last years RARP has emerged as the new standard of care in the surgical treatment of localised PCa. Obesity has been identified as a risk factor for tumorigenesis, progression and mortality in various malignancies. However, data investigating the influence of BMI on oncological parameters and perioperative outcome in PCa following RARP are inconsistent [22–27]. Several studies have demonstrated that a higher BMI is associated with more advanced tumour stages, more aggressive tumour biology as well as impaired functional outcome [24, 25, 28, 29, 30]. This might be explained by the fact that diagnosis of PCa by prostate biopsy may be delayed in obese patients due to relatively lower PSA levels (in relation to tumour volume) caused by haemodilution [30, 31, 32]. According to the recommendations of the Pasadena Consensus Panel, obese patients undergoing RARP may be best operated by experienced surgeons as these procedures are considered to be challenging [6].

Several studies have investigated the influence of BMI on oncological and perioperative outcome in recent years – and have shown that this point is still unclear from a scientific point of view (Table 5) [7–10, 12–15]. Our prospective multicentre cohort consisted of 232 RARP patients with a median BMI of 27.2 kg/m\(^2\), which compares to most of the other studies examining this topic [7–10, 12–15]. Besides a pathological tumour stage (TNM) and ISUP group, PSM has been identified as an independent predictor for impaired oncological outcome with an increased risk of biochemical recurrence [2]. Hence a PSM represents a key factor for the initiation of adjuvant radiotherapy following RARP. Contemporary RARP series report overall PSM rates ranging from 11.5% to 26.3% [7–12, 14, 15]. PSM rates of the present study (15.5%) are within this range. The risk of PSMs following RARP has been associated with pathological and clinical factors. PSM rates primarily depend on pathological tumour stage, surgeon’s expertise as well as the nerve sparing technique used.
The positive association of BMI and PSMs might be related to both reduced vision as well as limited angle movement during RARP in obese patients [8]. However, higher BMI was reported as an independent factor that is associated with a reduced risk of focal PSMs in one recent study [15]. The authors hypothesized this effect might be related to peri-prostatic fat thickness which is more present in obese patients. Interestingly enough, this series by Porcaro reported the lowest median BMI of all published series [15]. In addition, a second manuscript from this Italian series showed that a low BMI only increases the rate of focal PSMs (≤1 mm), while the probability of non-focal PSMs (>1 mm) remains unaffected [35]. Nevertheless, with regard to the influence of patients’ BMI on PSMs, the current state of literature is still under debate.

Therefore, two multivariate regression models with inclusion of different BMI cut-offs have been analysed in the present study. In model 1, patients were dichotomised at a BMI cut-off of 30 kg/m² (according to the WHO definition). Obese patients showed higher PSM rates, although this effect was, by a narrow margin, not statistically significant (p = 0.061). To further examine this finding, a second BMI dichotomisation at the 90th percentile (the BMI cut-off of 33.7 kg/m²) was applied for multivariate analysis (model 2). Using this model, a significant influence of patients’ BMI on PSMs was found (OR 3.99, p = 0.013). Our results are thus in accordance with the aforementioned publications showing a positive correlation between PSMs and BMI [7–10]. A similar approach with a dichotomisation of the BMI above the WHO obesity cut-off has also been chosen by Abdul-Mushin et al. [7]. In their series they expressly examined morbidly obese patients with a BMI cut-off of 40 kg/m². However, in contrast to our findings, differences with regards to PSMs failed to reach statistical significance in this rather small single-centre cohort [7].

Considering the localisation and extent of PSMs in RARP, the current state of literature is sparse. In one RARP series, a higher BMI was identified as an independent predictive factor for PSMs located at the prostatic apex [9], while only one of the aforementioned studies examined the extent of PSMs [12]. In our series a higher amount of PSMs were found to be localised in the area of the neurovascular bundles (posteriolateral) among patients with a BMI ≥30 kg/m² (58.3% vs. 33.3%, although not statistically significant, p = 0.175), whereas no significant correlation was found with regards to PSM extent. This first point should be examined in the future by larger studies and should be taken into account in the case of nerve-sparing RARP until these results are available.

The work has some limitations that need to be considered when interpreting the results. There is a relatively low number of patients, which is due to the biometric case planning of the prospective-randomized PIANOFORTE study [16]. There is also differences in sample size between the groups (obese vs. non-obese) are a result of the original PIANOFORTE randomisation process (‘peritoneal flap’ vs. ‘no peritoneal flap’). Based on this, the number of PSMs is low (n = 12 out of 51 patients with BMI > 30 kg/m², n = 8 out of 23 patients with BMI ≥ 33.7 kg/m²), which had to be taken into account when designing the multivariate models. Only univariate statistical tests were therefore possible for the comparative analysis of the PSM localizations. For reasons of case load, the relationship between surgeon and PSMs was not evaluated, although all surgeons had clearly exceeded their personal learning curve (> 100 RARP). In addition, it is not a series of consecutive patients, as not all RARP patients in the four centres agreed to participate in the PIANOFORTE study. However, the exclusion criteria of the PIANOFORTE study were not based on the patient’s BMI [16].

CONCLUSIONS

In addition to a longer operating time and about twice as many complications, patients with a BMI of ≥ 33.7 kg/m² had a higher PSM rate after RARP. The trend observed in our prospective study towards more posteriolateral PSMs in patients with obesity should be evaluated in larger studies in terms of sample size, since a difference here would have a direct influence on the intraoperative preservation of the neurovascular bundles.

CONFLICTS OF INTEREST

All authors declare that they have no conflicts of interest. No author has direct or indirect commercial financial incentive associated with publishing the article.

ETHICAL APPROVAL

The study was approved by local IRB and conducted according to 1964 Helsinki good clinical practice guidelines.

INFORMED CONSENT:

Informed consent was obtained from all participants.

CLINICAL TRIALS REGISTRY (DRKS-ID)

DRKS-00011115
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