retrospectively evaluated. Results: Acute rectal toxicity was recorded as follows: G0 in 42% (25/60), G1 in 47% (28/60), G2 in 11% (7/60). No case of acute toxicity  $\geq$  G3 was registered. During treatment, median week onset of rectal toxicity was 3th (range=2th-5th). Late rectal toxicity was recorded as follows: G0 in 63% (38/60), G1 in 24% (14/60), G2 in 13% (8/60). No case of toxicity  $\ge$  G3 was registered. Using logistic regression analysis, significant correlations between rectal toxicity and the volume of rectal irradiation V50 Gy > 45% (p<0.03) and V60 Gy > 35% (p<0.001) were found. A planning rectal volume < 80 cc is related to acute toxicity rectal ≥ G1. Conclusion: As already described in the literature, our experience confirmed the optimal rectal tolerability to the moderate hypofractionation regimens in prostate cancer, related to favorable alpha/beta ratio of this organ at risk. At a 2-year median follow-up, no moderate-severe rectal effects are reported. Longer follow-up is needed to assess clinical outcomes.

## 18 VISCERAL FAT TISSUE ACTIVITY DOES NOT CORRELATE TO HIGH GRADE PROSTATE CANCER RISK AT BIOPSY

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Introduction/Aim: High-grade prostate cancer (PCa) has been reported in association with metabolic syndrome (MS). In a previous study we found no significant correlation between body mass index (BMI) and prevalence of high Gleason score at biopsy (1). BMI could be not an accurate marker of the endocrine activity of visceral adipose tissue responsible of high plasmatic levels of adipokines promoting PCa aggressiveness. We estimated visceral adiposity dysfunction by the visceral adiposity index (VAI) considering waist circumference (WC), BMI, triglycerides (TG) and high density lipoproteins (HDL)

plasma levels of each patient (2). The aim was to correlate VAI values with PCa detection rate and Gleason patterns 4 and 5 at biopsy. Patients and Methods: Patients who underwent prostate biopsy for suspicious digital rectal examination and/or elevated PSA levels were enrolled. After informed consent, a transrectal prostate biopsy, 12 cores at least (24 in case of re-biopsy), was performed. VAI was expressed as:  $WC/[39.68 + (1.88 \times BMI)] \times TG/1.03$ × 1.31/HDL, assuming VAI=1 in healthy, non obese men with TG and HDL levels within normal limits. PCa detection at biopsy, Gleason score patterns, VAI and BMI were statistically analyzed using the Mann Whitney *U*-test. Results: Ninety-five patients were entered with a median age of 67 years (range=47-79). The median BMI was 27 kg/m<sup>2</sup> (range=17.4-40) and the median VAI was 4.47 (range=1.3-15.6). Median PSA was 7.9 ng/ml (range=0.47-53). A prostate nodule was palpable in 27 (28.4%) patients. Ten patients (10.5%) had a previous negative biopsy. A prostate cancer was diagnosed in 43 (45.2%) patients, Gleason patterns 4 and 5 were detected in 18 (41.8%) patients. Median BMI and VAI were 27.4 Kg/m2 and 26.3  $Kg/m^2$  (p=0.53) and 4.25 and 4.66 (p=0.28) in patients with positive and negative biopsy, respectively. Median BMI and VAI resulted 27.7 Kg/m<sup>2</sup> and 27.3 Kg/m<sup>2</sup> and 4.78 and 3.98 in patients with and without Gleason patterns 4 or 5, respectively. No statistically significant difference was highlighted for VAI (p=0.37) or BMI (p=0.85). Discussion and Conclusion: The identification of patients harboring an aggressive PCa remains an important goal. To date the relation between MS and PCa remains contradictory. Moreover, an accurate marker of MS has not yet been determined (3). VAI might be a more accurate marker than BMI in indicating the activity of visceral fat. In spite of VAI adoption, our analysis does not reveal any statistically significant correlation between VAI, PCa detection rate and incidence of Gleason patterns 4 or 5 at biopsy. Diet, race and other environmental and genetic factors, playing a promoting or protective role in PCa, should be also considered in further studies.

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