of acute G2 GU toxicity was about 3 times if the prostate volume is ≥ 80 cc (p-value 0.004; 95% CI: 1.05 - 9.5). In the adjusted prediction model using the logistic regression, the probability of acute G2 GU toxicity was about 60% with the same prostate volume cut-off (p-value 0.001; 95% CI: 0.13 - 0.46), with an attitude to develop a moderate toxicity in the first 3 weeks from the beginning of treatment. In the late setting, a trend to significance (p=0.076) to develop an acute GU toxicity ≥ G1 was found for bladder volume of 60 Gy ≥ 15%.

Conclusion: In moderate hypofractionation in 30 fractions for prostate cancer, a prostate gland volume greater than 80 cc resulted as predictor of moderate acute GU toxicity.

Results: Median follow-up was 4.3 years. At the time of median follow up the median PSA was 0.19. PSA bounce was seen in 32.6% (n=31). Magnitude of PSA bounce was <1ng/ml in 55% (n=17), 1-2ng/ml in 13% (n=4), >2ng/ml in 32% (N=10). In 16 out of 17 patients with a PSA bounce of <1ng/ml was due to a benign bounce. 50% of patients with a PSA bounce between 1-2ng/ml had a benign bounce and the remaining 50% developed biochemical failure. In 9 out of 10 patients who had a PSA bounce of >2ng/ml subsequently developed a biochemical failure. Most common time for benign PSA bounce was between 6 and 18 months.

Conclusion: PSA bounce is a common phenomenon which occurs in about a third of men who were treated with short term ADT in combination with HDR boost and EBRT. Benign PSA bounce tends to have a smaller magnitude of rise in PSA <1ng/ml. However patients who developed biochemical failure had PSA bounce of larger magnitude >2ng/ml. Investigators at the time of submission of the abstract are examining variables which predict PSA bounce.

Purpose or Objective: We have created and implemented in our department a new scheme of hypofractionated salvage volume modulated arc therapy with simultaneous integrated boost for patients with recurrence of prostate cancer (PCa) after radical prostatectomy (RP). The aims of our research are to evaluate toxicities and biochemical response rate.

Material and Methods: Patients with recurrence of PCa after RP have been treated by hypofractionated (HF) salvage radiotherapy (SRT). Characteristics of HF radiotherapy were as follows: the prescribed dose to the regional lymphatic nodes was 46.8 Gy of 1.8 Gy per fraction, to the prostate bed - 61.1 Gy of 2.35 Gy per fraction in case of biochemical recurrence (BR) and if region of clinical recurrence (CR) was identified - 65 Gy of 2.5 Gy each, in 26 fractions with pretreatment imaging; VMAT (two arcs: CW (185°-175°), CCW (175°-185°) technology with SIB was used. Toxicities were scored using RTOG/EORTC Radiation Toxicity Grading.

Results: 41 patients were treated by the HF SRT. Median follow-up was 22 months (10 - 30). Biochemical control rate 37 (90.2%) patients, locoregional control rate - 41 (100 %) patients. No grade 3 or greater acute toxicities were observed.

Conclusion: We would like to suggest a new scheme of HF SRT with SIB in 26 fractions for patients with recurrence of PCa after RP. The toxicities and early biochemical response rates were comparable with conventional fractionation SRT.

Purpose or Objective: The Aim of this study is to evaluate PSA kinetics in men with intermediate and high risk prostate cancer treated with HDR brachytherapy boost in combination with external beam radiotherapy (EBRT) and short term androgen deprivation therapy (ADT).

Material and Methods: Data from 134 consecutive patients treated with HDR brachytherapy boost in combination with external beam radiotherapy was extracted from a prospectively maintained database. All the patients had a minimum follow up of 4 years. Patients who were on androgen deprivation therapy for over 12 months were excluded from the analysis. After exclusion we had 95 evaluable patients. All patients received either 17 Gy in 2 fractions or 15 Gy in single fraction of HDR brachytherapy boost followed by external beam radiotherapy 37.5 Gy in 15 fractions. 70% of patients received Androgen deprivation therapy (ADT) for less than or equal to 6 months, 15% received for 6-12 months, and 15% received no hormones. 3-6 months of ADT was given neoadjuvantly. Date of HDR boost was considered as time=0. Benign PSA bounce was defined as PSA rise of >0.2ng/ml followed by subsequent decline to pre bounce level.

Results: Median follow-up was 4.3 years. At the time of median follow up the median PSA was 0.19. PSA bounce was seen in 32.6% (n=31). Magnitude of PSA bounce was <1ng/ml in 55% (n=17), 1-2ng/ml in 13% (n=4), >2ng/ml in 32% (N=10). In 16 out of 17 patients with a PSA bounce of <1ng/ml was due to a benign bounce. 50% of patients with a PSA bounce between 1-2ng/ml had a benign bounce and the remaining 50% developed biochemical failure. In 9 out of 10 patients who had a PSA bounce of >2ng/ml subsequently developed a biochemical failure. Most common time for benign PSA bounce was between 6 and 18 months.

Conclusion: PSA bounce is a common phenomenon which occurs in about a third of men who were treated with short term ADT in combination with HDR boost and EBRT. Benign PSA bounce tends to have a smaller magnitude of rise in PSA <1ng/ml. However patients who developed biochemical failure had PSA bounce of larger magnitude >2ng/ml. Investigators at the time of submission of the abstract are examining variables which predict PSA bounce.