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**Introduction.** During the natural evolution of prostate cancer in patients treated with prostatectomy many of them develop biochemical relapse and need radiation treatment.

**Objective.** To find potential predictor factors to response to radiotherapy in prostate cancer patients with biochemical relapse after prostatectomy.

**Methods.** We have selected the 59 prostate cancer patients treated with prostatectomy from 2006 to 2008 that were submitted after biochemical relapse. We have analyzed the following variables to see their impact on the response to the radiotherapy: pN, Gleason [ $\leq 7$  (3+4) vs.  $\geq 7$  (4+3)], irradiated volume, dose delivered (<70 Gy vs. 70 Gy), hormonal treatment (yes/no), and PSA level before radiotherapy. Follow up: mean 49 months (range 30–69).

**Results.** Two patients (3.39%) died (one for prostate cancer). 57 patients (96.61%) are still alive, of which 39 (66.10%) remain free of disease; 13 (22.03%) developed biochemical relapse and 6 (10.20%) distant metastases. PSA pre-radiotherapy was the only variable analyzed that showed a statistically significant incidence  $p=0.0002$  (HR 1254, 95% CI 0.107, 0.345). These results indicate that for every unit that PSA pre-radiotherapy raises, the risk of developing biochemical relapse is 25% higher. In our series, patients with PSA > 1.65 ng/ml had a risk of recurrence 3.95 times higher than those with a PSA  $\leq 1.65$  ( $p=0.0049$ ). Gleason, hormonal treatment and dose delivered: no showed statistically significant p values but their survival curves show a clear positive trend in patients with Gleason  $\leq 7$  (3+4), who received hormonal treatment and in which dose was  $\geq 70$  Gy. Volume treated and, pN: showed no statistical significance.

**Conclusions.** In our series the only variable that has demonstrated statistically significance as a predictor factor was PSA pre-radiotherapy ( $p=0.0002$ ). Patients with PSA pre-radiotherapy >1.6 ng/ml had 3.95 times more risk to developing biochemical relapse.

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#### PSA nadir and PSA bounce in low risk prostate cancer

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**Background.** The aim was to obtain the time to nadir of PSA (nPSA) in patients with disease free of recurrence in low risk carcinoma of prostate cancer, treated with radical external beam radiotherapy (EBRT). And to study the bounce PSA.

**Materials and methods.** A group of 45 patients, with low risk prostate cancer, (it was considered low risk prostate cancer, those patients with PSA  $\leq 10$  ng/ml, Gleason = 6 or stage T1–T2), who were treated in the years 2007–2009 either using external-beam RT (78 Gy) to prostate volume, without blockage androgen. The treatment was carried out in a lineal accelerator using photons of 18 MV, with standard technique and fractionation. After baseline prostate-specific antigen determination (PSAprERT), PSA was assessed 3 months after the start RTE, and after it was measured every six months. The median follow-up after RT was 27 months with a rate (21–51 months). A bounce was defined by a minimum rise in PSA of 0.4 ng/ml over a 6-month period, followed by a decrease without therapeutic intervention.

**Results.** The median PSAprERT was 7.35. Median nPSA value was 0.51 ng/ml (limits: 0–1.52) and the median time elapsed between PSAprERT and nPSA has been of 22 months (limits: 9–45). PSA bounce >0.5 ng/ml was noted in 18% of patients. After 6 months the values return close to nPSA values. The median length of time until the first PSA bounce was 24 months. None patient had relapse biochemical by the Phoenix definition.

**Conclusion.** In patients with low risk prostate cancer, when EBRT is used as monotherapy, time to nadir is very long. In our cohort were found benign PSA bounces. Our study reinforces the need for adequate follow-up to ensure accurate estimates of treatment efficacy and to avoid unnecessary secondary interventions.

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#### Quantification of changes in adipose tissue related to prostate androgenic blockade

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**Introduction.** Hypogonadism is a well known factor related to the adipose tissue increase, cardiovascular risk and skeletal myopathy. **Aim.** Quantitative CT scans have shown a fat redistribution and a decrease in bone density secondary to hypogonadism. Androgenic blockade (AB) induces an hypogonadism status and thus prostate cancer patients are expected to show a fat redistribution