

In the clinical arena, Kaouk et al. (4) presented a series of single-port laparoscopic RP in 4 patients diagnosed with prostate cancer. They treated patients with localized disease, no previous pelvic surgery, and a body mass index < 35 kg/m². A single port device was placed transperitoneally through a 1.8-cm incision located at the umbilicus without any other instruments or ports needed to complete operations. Urethrovesical anastomosis was performed using free-hand interrupted suturing and extracorporeal knot tying. This work is an impressive publication verifying feasibility of this procedure with the use of single port and articulated instruments. Kaouk et al have also presented a previous experience in LESS-RARP using the R-port with adequate results.

We report to our the first clinical report of LESS-RARP. The procedure was successfully completed with the initial approach and a change in port triangulation was a key point to accomplish the task. Further evaluation of the technique is warranted.

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Re: The Influence of Statins on Prostate-Specific Antigen Levels

To the Editor,

The influence of statin medications on prostate specific antigen levels is somehow controversial. Recently, Hamilton RJ et al. analyzed data of men who were prescribed a statin for a long-term period. The authors reported a statistically significant decline in PSA levels in men without prostate cancer, after they were treated with statins (1). This finding is

in accordance with that of Cyrus-David et al., who also reported an important PSA decline in a small number of healthy men treated with statins for over 5 years (2). In contrast, Mills et al., who assessed the efficacy of statins in the treatment of lower urinary tract symptoms and prostate enlargement in a large, double-blind, placebo-controlled trial did not found

any difference between the effects of statins and placebo on the mean change from baseline in PSA levels after 26 wk of treatment (3). In our recently published study investigating the effects of statins on conventional medical treatment of lower urinary tract symptoms with finasteride, serum PSA values seemed to be generally lower in statin/finasteride arm compared to finasteride arm alone at the end of the study (4). The fact that the change in mean PSA from baseline to end point in patients treated with statins did not achieved statistical significance lead authors to conclude that statins do not seem to boost the finasteride's effect on PSA. However, under the light of the new evidence emerged from the study of Hamilton RJ et al., this could be attributed to the relatively low sample as well as to the relatively low duration of the study and an effect of statins on PSA would be probably detected if the study has been lasted over a longer period of time. In fact, effects of statins on prostate biology, as observed in large prospective cohort studies, are probably associated with higher doses and longer use (5). Although the specific mechanism by which statins influence PSA is not understood, it could be assumed that involves metabolic pathways. Since cholesterol is an important precursor for androgen formation, it is conceivable that by influencing cholesterol metabolism, statins may lower levels of intraprostatic androgens and in consequence they reduce PSA levels. An additional, non-cholesterol mediated effect of statins via anti-atherosclerotic action is not to be excluded also. Effects of statins in both prostate stromal and epithelial cells

have been attributed to the anti-oxidative properties of statins as well. Data suggesting that treatment with statins lower serum PSA with time may also indicate new possible drug mechanisms acting on prostate cells at the receptor level and may indicate a novel approach in both prostate cancer chemoprevention and benign prostate hyperplasia treatment. Therefore, further experimental studies are needed in order to investigate the exact mechanism by which statins impact on prostate cells.

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