

patients underwent retroperitoneal RALPN from January 2014 to October 2016. The patients included 30 males and 22 females, with an average age of (55.2±9.5) years old. There were 32 tumors in the left and 20 in the right. The mean lesion diameter was 4.5±0.7 cm.

Results: All cases were performed successfully. The operation was completed in a mean of 80.5±12.3 min, the mean warm ischemia time was 17.4±5.1 min, the mean blood loss was 38.2±7.4 mL and the mean extubation time was 2.1±0.7 d. Postoperative pathology reported malignant tumor in all cases, including renal clear cell carcinoma in 45 cases, papillary renal cell carcinoma in 5 cases and chromophobe cell carcinoma in 2 cases. The surgical margin of all cases were negative.

Conclusions: Retroperitoneal RALPN is an effective, safe and minimally invasive surgical management for T1b renal carcinoma.

Keywords: Retroperitoneal approach; renal carcinoma; partial nephrectomy; robotics

doi: 10.21037/tau.2017.s062

Cite this abstract as: Tang H, Zhang ZY, Zhou WQ, Wei W, Xue S, Zhou ZK, Li P, Ge GP. Clinical analysis of retroperitoneal robotic-assisted laparoscopic partial nephrectomy in management of T1b renal carcinoma. *Transl Androl Urol* 2017;6(Suppl 3):AB062. doi: 10.21037/tau.2017.s062

AB063. A prostaglandin E (PGE) receptor EP4 is involved in the cell growth and invasion of prostate cancer via the cAMP-PKA/PI3K-AKT signaling pathway

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Background: Prostate cancer (PCa) is one of the most prevalent diagnosed malignancies in the world. Previous studies have proved that prostaglandin E₂ (PGE₂) was closely related to the tumorigenesis and progression of PCa. However, the molecular mechanisms are not very clear, and it is necessary to be further studied. MMPs, RANKL and RUNX2, involved in the cell growth and bone metastasis, are activated or overexpressed in many cancers, including PCa.

Methods: This study was designed to demonstrate the internal relationship and molecular mechanisms among the PGE₂, EP4 and MMPs, RANKL and RUNX2 in PCa, and to define their roles in PCa cell proliferation and invasion.

Results: The results in this study showed that the protein and the mRNA expression levels of the MMP-2, MMP-9, RANKL and RUNX2 in PC3 cells were significantly upregulated by treated with PGE₂, and knockdown of these proteins blocked PGE₂-induced cell proliferation and invasion in PC3 cells. The effect of PGE₂ on the protein and mRNA expression levels was mainly regulated via the EP4 receptor. EP4 receptor signaling activated cAMP-PKA signaling pathway, and forskolin, the activator of the adenylate cyclase (AC), has similar effects of the EP4 receptor agonist on the protein expression, while SQ22536, the inhibitor of AC, inhibited the protein expression. These results confirmed the AC/cAMP pathway was involved in EP4 receptor-mediated protein expression upregulation. By using specific inhibitor of PKA, it is demonstrated that cAMP/PKA was also related to the upregulation of the EP4 receptor-mediated protein expression. In addition to the signaling pathway of the PKA, the EP4 receptor also exerts activities through activating Akt kinase. The results in present study confirmed the hypothesis that the EP4 receptor-mediated protein expression of PCa cells, which were pretreated with the specific inhibitor of PI3K, was significantly inhibited.

Conclusions: In summary, it is revealed that PGE₂ significantly upregulates the mRNA and protein expression levels of the MMPs, RANKL and RUNX2, and the EP4 receptor was involved in the cell proliferation and invasion of PCa via the cAMP-PKA/PI3K-AKT signaling pathway in this study. The present study may provide a new way of thinking and therapeutic strategy for the prevention and treatment of PCa.

Keywords: Prostaglandin E; Prostate cancer (PCa); AKT; PGE₂

doi: 10.21037/tau.2017.s063

Cite this abstract as: Xu S, Ge JP, Zhang ZY, Zhou WQ. A prostaglandin E (PGE) receptor EP4 is involved in the cell growth and invasion of prostate cancer via the cAMP-PKA/PI3K-AKT signaling pathway. *Transl Androl Urol* 2017;6(Suppl 3):AB063. doi: 10.21037/tau.2017.s063

AB064. Downregulation of miR-129 in peripheral blood mononuclear cells is a diagnostic and prognostic biomarker in prostate cancer

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Background: The present study was designed to explore the clinical values of microRNA-129 (miR-129) expression in peripheral blood mononuclear cells for prostate cancer patients and the role of miR-129 in the proliferation of prostate cancer.

Methods: The peripheral blood mononuclear cells were isolated from blood sample from 98 patients confirmed with prostate cancer and 56 matched healthy volunteers. Reverse transcription quantitative real-time polymerase chain reaction (qRT-PCR) was employed to determine the expression level of miR-129 in peripheral blood mononuclear cells. Cox proportional hazards regression models and Kaplan-Meier analysis were used to evaluate the association of miR-129 expression with clinical and pathological characteristics of prostate cancer patients. The effect of miR-129 on the proliferation of prostate cancer cells *in vitro* was also determined.

Results: Reverse transcription quantitative real-time polymerase chain reaction (qRT-PCR) results showed that the expression of miR-129 was dramatically down-regulated in peripheral blood mononuclear cells for prostate cancer patients in comparison with healthy controls ($P < 0.05$).

The decrease in miR-129 expression in peripheral blood mononuclear cells was significantly associated with aggressive clinical pathological features such as histological grade ($P = 0.010$), high preoperative PSA level ($P = 0.002$), pathological stage ($P = 0.011$), high Gleason score ($P = 0.005$), lymph node metastasis ($P = 0.002$), angiolymphatic invasion ($P = 0.004$), biochemical recurrence ($P = 0.001$). The prostate cancer patients with a low miR-129 expression in peripheral blood mononuclear cells had an obviously shorter BCR-free survival compared with high miR-129 expression ($P < 0.001$). The Cox multivariate analysis established that the miR-129 expression may be an independent prognostic factor for biochemical recurrence (BCR)-free survival prostate cancer patients ($P = 0.000$). The results of *in vitro* CCK-8 assays, as well as proliferating cell nuclear antigen phosphorylated histone-3 (P-H3) (markers of proliferation) indicated that miR-129 overexpression markedly retarded the proliferation of PC-3 and DU-145 cells.

Conclusions: Our results provide the first evidence that the miR-129 is significantly downregulated in prostate cancer patients and multivariate analysis confirmed that miR-129 is a novel independent prognostic factor for prostate cancer. Overexpression of miR-129 exerts tumor suppressive functions and abrogates prostate cancer growth.

Keywords: microRNA-129 (miR-129); prostate cancer; prognostic biomarker

doi: 10.21037/tau.2017.s064

Cite this abstract as: Xu S, Ge JP, Zhang ZY, Zhou WQ. Downregulation of miR-129 in peripheral blood mononuclear cells is a diagnostic and prognostic biomarker in prostate cancer. *Transl Androl Urol* 2017;6(Suppl 3):AB064. doi: 10.21037/tau.2017.s064

AB065. EP4 antagonist suppresses bone metastasis in prostate cancer

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