expression cancers compared with mock counterparts (survival, P = 0.034). In animal models, HK2-overexpression cancers also induced morphologic change and CD133 activity.
Conclusion: High HK2 expression in bladder cancers induced oversecretion of lactate, which was associated with metastatic behaviors through the cancer stem cell formation, EMT promotion and nuclear translocation of phosphorylated NF-κB and Twist1. HK2 may be a novel oncoprotein and play as target for bladder cancer therapy.

PD11-6:
INHIBITION OF AUTOPHAGY ENHANCES EVEROLIMUS (RAD001)-INDUCED CELL DEATH IN HUMAN BLADDER CANCER CELLS

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Purpose: Mammalian target of rapamycin, mTOR, a downstream protein kinase of phosphoinositide 3-kinase (PI3K)/AKT signaling pathway, has been recognized to play a central role in controlling cancer cell growth. The PI3K/AKT/mTOR pathway promotes tumor growth and survival while suppressing autophagy, a catabolic process in cells to sustain energy homeostasis by collecting and recycling cellular components under stress condition. Conversely, inhibitors of the mTOR pathway such as Everolimus (RAD001), induce autophagy that promote tumor survival and thus, these agents potentially limit their own efficacy. We hypothesized that inhibition of autophagy in combination with mTOR inhibition would improve the cytotoxicity of mTOR inhibitors in bladder cancer.
Materials and Methods: The cytotoxicity of RT4 (grade I), 5637 (grade II), HT1376 (grade III) and T24 (grade III) human bladder cancer cells treated with RAD001 alone or combined with autophagy inhibitors (3-methyladenine (3-MA), bafilomycin A1 (BafA1), chloroquine (CQ) or hydroxychloroquine (HCQ)) was accessed by WST-8 cell viability kit. The autophagy status in cells was performed by the detection of microtubule-associated light chain 3 form II (LC3-II) using immunofluorescent staining and Western blot. Acidic organelles (AVOs) formation in treated cells was determined by acridine orange (AO) vital staining. Inhibition of mTOR pathway by RAD001 was monitored by home-made QPCR gene array and the detection of phospho-mTOR by Western blot. Induced apotposis was determined by measurement of caspase 3/7 activity and DNA fragmentation in cells after treatment.
Results: Advanced bladder cancer cells (5637, HT1376 and T24) were more resistant to RAD001 than RT4. Autophagy flux detected by the expression of LC3-II showed RAD001 induced autophagy. AVOs formation was detected in cells treated with RAD001 and inhibited by the addition of 3-MA or Baf A1. Co-treatment of RAD001 with autophagy inhibitors further reduced cell viability and induces apoptosis in bladder cancer cells.
Conclusion: Our data suggest that coordinate inhibition of the mTOR and autophagy pathway promotes apoptosis, and could be a new therapeutic paradigm for the treatment of bladder cancer.

Podium-12
LUTS

PD12-1:
THE ROLE OF HELICOBACTER PYLORI ANTIGEN IN CHRONIC PROSTATITIS

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Purpose: Epidemiological evidences had shown that patients with Helicobacter pylori (H.p.) infection had more urological prostatitic diseases. And chronic prostatitis patients had significantly higher positive anti-H.p. rate. Therefore, we investigated the possible role of H.p. in chronic prostatitis.
Materials and Methods: We set up chronic prostatitis model by introducing H.p. into male rats. Physiological bladder changes along with pain sensitivity in scrotum and tail base were evaluated with cystometrogram and Von Frey filament correspondently. Local prostatic inflammation was checked with Western blot and immunohistochemical stain. Systemic inflammation was evaluated by checking cytokines in spleen.
Results: Hypersensitivity in rat scrotum confirmed the development of chronic prostatitis. Tail base sensitivity showed less significant correspondence. There was significant difference caspase 1 expression in H.p. antigen stimulated prostate. Matured caspase 1 ratio also increased. However, local IL-1b did not seem to have significance. Systemic inflammation was confirmed by significantly increased TNF-alpha in spleen protein extraction.
Conclusion: H.p. antigen may induce local and systemic inflammation. Local effect in prostate induces chronic prostatitis-like response. Further investigation on the systemic effect of H.p. antigen may be necessary to confirm its immunological role on chronic prostatitis.

Female Urology & Urodynamics

PD12-2:
CELLULAR AUTOPHAGY OF HUMAN STEM CELLS IN THE PROCESS OF DIFFERENTIATION

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Purpose: The study is conducted to understand the cellular autophagy of human adipose derived stem cells during the process of smooth muscle cell (SMC) differentiation.
Materials and Methods: Human adipose derived stem cell (hADSC) were induced differentiation into SMC by the use of low-serum level smooth muscle induction medium (SMIM) during SMC differentiation. Real-time PCR analysis were used to check the mRNA expression of smooth muscle marker genes such as α-smooth muscle actin (SMA), SM22α, Calponin, Caldesmon and smooth muscle myosin heavy chain (MHC). We used western blot assay and immumofluorescence staining were also usedImmunofluorescence staining of the cellular actin cytoskeleton to testify the change at protein level.
Results: There was increased expression of smooth muscle marker genes such as SMA and smooth muscle MHC from hADSCs which were exposed to SMIM for 6 weeks. Increased cellular complexity and granularity in induced hADSC, suggesting the intracellular organelles might be increased during the process of SMC differentiation. The lysosome content is significantly increased but mitochondria and endoplasmic reticulum are not. The increased protein content of the lysosomal-associated membrane protein 1 (LAMP-1) confirmed the increase in lysosome content during SMC differentiation. On the other hand, conversion from LC3-I to LC3-II was increased during SMC differentiation and significant increase was observed at the 3w-differentiation.
Conclusion: These results suggest that autophagy appears to be up-regulated in the early stage of SMC differentiation. Autophagy might play an important role in SMC differentiation of ADSC which may be potential biomaterial for the treatment of urinary incontinence and for bladder reconstitution.

PD12-3:
RESULTS OF THE SURGICAL TREATMENTS OF ULCER TYPE INTERSTITIAL CYSTITIS

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Purpose: The pathophysiology of ulcer type interstitial cystitis (IC) is still unclear. Various medical and surgical therapies have been used without a

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common consensus. This study aimed to evaluate the surgical outcomes of ulcer type IC.

Materials and Methods: We retrospectively collected 14 ulcer type IC female patients, who were diagnosed during cystoscopic hydrodistension, and their medical records were reviewed. The severity and duration of symptoms, and self-assessed surgical responses were presented with descriptive statistics.

Results: The mean age on the development of symptoms and on the diagnosis were 56.0 ± 8.4 and 59.1 ± 7.0 years, respectively, with a mean VAS pain score 8.2 ± 1.4. Ten (71.4%) patients received intravesical Botox (botulinum toxin-A) injection with 50% (5 of 10) response rate of pain relief; however, in 4 of 5 patients, their pain increased again after 2.75 ± 0.38 months. All patients received electrocauterization with 85.7% (12 of 14) response rate; however, in 11 of 12 patients, their pain increased again after 3.09 ± 1.75 months. Five patients with poor electrocauterization outcome received simple partial cystectomy; however, four patients (80%) did not satisfy the surgical outcome due to rapid relapse of pain after 1.75 ± 0.75 months. Two of these 4 patients subsequently received extensive (supratrigonal) partial cystectomy concurrent with augmentation enterocystoplasty (AE). Finally, total 6 patients received extensive (supratrigonal) partial cystectomy concurrent with AE, and all patients (100%) satisfied with the surgeries due to the significant relief of pain and symptoms during a follow-up of 7.67 ± 3.22 months.

Conclusion: For ulcer type IC patients, electrocauteterization as a treatment option is effective but with short term efficacy and a high relapse rate. Simple partial cystectomy is not feasible in these patients. Extensive partial cystectomy concurrent with AE can effectively relieve pain and symptoms with high satisfaction.

PD12-4:

INTRAVESICAL BOTULINUM TOXIN-A INJECTIONS REDUCE BLADDER PAIN OF INTERSTITIAL CYSTITIS/ BLADDER PAIN SYNDROME REFRACTORY TO CONVENTIONAL TREATMENT – A PROSPECTIVE, MULTICENTER, RANDOMIZED, DOUBLE-BLIND, PLACEBO-CONTROLLED CLINICAL TRIAL

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Purpose: Intravesical onabotulinumtoxinA (BoNT-A) injection is a beneficial treatment for interstitial cystitis/bladder pain syndrome (IC/BPS), yet its therapeutic efficacy remains to be validated. This study tests efficacy and safety of intravesical BoNT-A injections for treatment of IC/BPS.

Materials and Methods: A multicenter, randomized, double-blind, placebo-controlled trial in patientswith IC/BPS refractory to conventional treatment was conducted. Patients were randomized in a 2:1 ratio to hydrodistention plus suburothelial injections of BoNT-A 100U (Botox group) or the equivalent amount of normal saline (N/S group). The primary endpoint was a decrease in pain assessed using a visual analog scale (VAS) at week 8 after treatment. Secondary endpoints included voiding diary and urodynamic variables. The Wilcoxon sign rank and rank sum tests were used for statistical analyses.

Results: A total of 60 patients (8 males, 52 females, age 50.8 ± 13.9 years) including 40 in the Botox and 20 in the N/S groups were enrolled. At week 8, a significantly greater reduction of pain was observed in the Botox group compared to the N/S group (-2.6 ± 2.8 VS. -0.9 ± 2.2, p = 0.021). The other variables did not differ significantly between groups except for cystometric bladder capacitu, which was increased significantly in the Botox group. The overall success rates were 63% (26/40) in the Botox group and 15% (3/20) in the N/S group (p = 0.028). Adverse events did not differ between the groups.

Conclusion: Intravesical Botox injection of 100U of BoNT-A effectively reduced bladder pain symptoms in patients with IC/BPS. The adverse events were acceptable.

PD12-5:

THE PREVALENCE OF NON-BLADDER COMORBIDITY BETWEEN PATIENTS WITH INTERSTITIAL CYSTITIS / BLADDER PAIN SYNDROME (IC/BPS) AND KETAMINE INDUCED UROPATHY (KIU)

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Purpose: Recreational ketamine abuse cause lower urinary tract symptoms including dysuria, urinary frequency, urgency, urge incontinence and hematuria. Several reports showed denuded epithelial inflammation of the bladder and petechial hemorrhage. As with similarly presenting interstitial cystitis, several studies investigated the pathophysiology as purinergic neurotransmission of ketamine induced uropathy. A recent study found that patients with IC/BPS often have other non-bladder conditions such as irritable bowel syndrome (IBS), fibromyalgia (FM), migraine headaches, and depression. The aim of this study is to investigate subjective symptom score, voiding diary parameters he findings of cystoscopic hydrodistension and presence of non-bladder condition compared with IC/BPS patients.

Materials and Methods: This was a retrospective study. Of 24 patients who were admitted due to severe lower urinary tract symptoms with recreational ketamine abuse history more than one year and 173 female patients who were compatible with AUA/SUFU criteria including unpleasant sensation (pain, pressure, discomfort) perceived to be related to bladder with duration >6 weeks were included as control group. All of patients with ketamine induced uropathy and IC/BPS patients were assessed by cystoscopic hydrodistension and all of them have different severity of glomerulations. These patients completed measures of pain severity (Visual Analog Scale) and bladder symptom severity (IC Symptom Index, IC Problem Index, The Pelvic Pain and Urinary/Frequency scale). Three day voiding diary was also collected and all patients completed non-bladder condition as medical history questionnaire including migraine, FM, IBS, and depression. These data were analyzed using point bi-serial correlation for association (ANOVA) and post-hoc analysis.

Results: The ketamine induced uropathy patients with a mean age of 26.58 ± 4.4 years were statistically significant younger than IC/PBS patients with a mean age of 44.26 ± 12.5 years (p<0.001). Patients with ketamine induced uropathy had more severe pain score (p = 0.01) and PUF score (p = 0.005) than IC/BPS patients. KIU have significant decrease of night-time volume (p = 0.02) as well as increased day-time and night-time frequency. Moreover, KIU have significant decrease of anesthetic bladder capacity during cystoscopic hydrodistension than IC/BPS patients (p<0.001). However, there were no differences in non-bladder condition between KIU and IC/BPS patients (Table 1). After adjusted age between IC/BPS and KIU group, there were still no differences in non-bladder condition (Table 2).

Conclusion: Subjective symptom scores, three day voiding diary parameters, and anesthetic bladder capacity in patients with KIC seem more severe compare to those with IC/BPS. Compare to age matched IC/BPS, there were no differences in non-bladder condition between KIU and IC/BPS patients.

PD12-6:

CASE REPORT-BLADDER VOIDING STIMULATION TECHNEQUE CAUSE VESICOURETERAL REFLUX

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Introduction: Neurogenic bladder is a dysfunction of the urinary bladder due to disease of the central nervous system or peripheral nerves involved in the control of micturition. Methods to improve bladder emptying consist of two main strategies: to increase intravesical pressure and to decrease outlet resistance. Textbook also mentioned that using trigger technique such as tapping or scratching the skin above the pubis or external genitalia, pulling the skin or hair of the pubis, scrotum, or thigh; squeezing the clitoris; or by digital rectal stimulation, could induce bladder reflexively to empty. Our patient had use this kind of technique but cause VU reflux and hydronephrosis.

Case report: A 62-year-old male who was falling down from a tree(3m height) developed T7 and T8 burst fracture. T7 complete paraplegia with neurogenic bladder and neurogenic bowel were noted after surgery. CISC was arranged for him. He had ICP 4 times/day plus suprapubic tapping and Crede manuvar. and decreased residual urine amount after TURP+TUI. He had regular OPD follow up and residual urine all around 100ml and renal echo found no hydronephrosis. We had suggested him not to tapping and had increase ICP frequency and add Anti-M drug.

However, Recurrent UTI and follow up renal echo found Bil. Hydronephrosis, and Left hydroureter recently. We arrange VUDS for