



KUOS 뉴스레터

The Korean Urological Oncology Society

Vol. No **2015_2**

CONTENTS

회장 인사말 1

제28회 대한비뇨기종양학회 정기학술대회 안내 2

The 13th KUOS Multidisciplinary conference 3

- 발표자료 6
- 학술상내역 23

EAST ASIA azk MEETING 24

공지사항 25

🔍 회장 인사말



비뇨기종양학회 회원 여러분 안녕하십니까?

여름이 시작되지도 않았는데 벌써 더위가 성큼 다가왔습니다.

2015년 8월 29일 판교 테크노벨리에 있는 차의대 차바이오텍플렉스에서 개최되는 제28회 대한비뇨기종양학회 정기학술대회에 회원 여러분을 초대하게 되어 기쁘게 생각합니다.

최근 국내에서 발생한 메르스 확산으로 전 국민이 긴장하고 전국이 어수선한 상황입니다. 비뇨기종양학회의 자문위원, 원로회원과 젊은 회원을 모시고 6월 6일에 개최될 예정이었던 진료지침 워크숍을 메르스 확산으로 연기하게 되었습니다. 회원 여러분의 이해를 부탁드립니다. 적당한 시기에 새로운 일정을 정해서 진행하겠습니다.

우리 비뇨기종양학회의 정기 학술대회는 매년 새로운 지식과 정보, 연구 결과를 제공하고 공유하는 장으로서, 학문적 토의와 논쟁을 통한 발전의 공간으로서 그 역할을 충실히 해왔습니다. 지난 학술대회를 통해 회원들의 적극적인 참여와 노력은 비뇨기종양 연구 및 학문적 발전의 밑거름으로써 우리 학회의 가장 큰 자랑이자 역사라고 자부합니다.

이번 학술대회는 비뇨기종양 전반에 걸쳐 국내외에서 활발히 활동하고 연구하시는 분들을 좌장 및 연자, 패널로 모시고 비뇨기종양 분야의 이슈를 토론하고 정리할 수 있는 시간이 되실 것이라고 생각합니다. 특히 해외연자로 프랑스의 Jacques Irani 교수가 초대되어 고위험 전립선암의 최신지견에 대해 발표를 해 주실 예정입니다. 또한 Podium Session은 지난 1년 동안 회원 여러분들이 비뇨기종양 분야에 불철주야 연구하신 성과를 발표하고 서로 토의할 수 있는 유익한 자리가 되리라 생각합니다.

이번 학술대회가 유익한 정보를 서로 공유하며 학문적 발전을 도모하고, 회원 여러분들의 소통과 교제의 장이 될 수 있기를 바라며 회원 여러분의 적극적인 성원과 참여를 부탁드립니다.

아울러 무더운 여름철에 회원 여러분의 가정에 건강과 행복이 함께하시길 기원합니다.

감사합니다.

2015년 6월
대한비뇨기종양학회 회장 김형진 배상

함께하는 시간만큼 함께 할 이야기가 있습니다

1995

/ 최초의 SARI* 프로스카 국내 출시^{1,2,3}

1998

/ PLESS⁴ 연구 발표⁴
BPH[†] 환자를 대상으로 5ARI*를 평가한
최초의 장기간 위양대조군 연구^{4,5}

2003

/ MTOPS⁶ 연구 발표^{6,8}
BPH[†] 의 임상적 진행에 대해 위약, doxazosin, 프로스카,
프로스카/doxazosin 병용요법을 비교한 장기적 이중맹검 연구⁶

2011

/ 대한비뇨기학회 주관
블루애를 캠페인 후원

2014

/ 2007년 12월 대비 약 46% 약가인하
(2014년 1월 시행 기준)^{7,8}

2015

/ 프로스카 국내 출시 20주년⁹

전립샘비대증 치료를 위한
믿음직한 동반자!
최초의 5ARI* 제제, 프로스카^{1,2}



* SARI : 5- α -reductase inhibitors † BPH : Benign Prostatic Hyperplasia ‡ AUR : Acute Urinary Retention § PLESS : Proscar Long-term Efficacy and Safety Study ¶ MTOPS : Medical Therapy of Prostatic Symptoms

Study design

a.PLESS : PLESS was a 4-year, randomized, double-blind, placebo-controlled trial. A total of 3,043 men with BPH, diagnosed on the basis of moderate to severe symptoms of urinary obstruction, decreased maximum urinary flow rate, and an enlarged prostate were randomly assigned to receive PROSCAR 5mg (n=1,524) or placebo (n=1,516) daily. The primary endpoint was the change in symptom score. Predicted secondary endpoints included surgery for BPH and occurrence of AUR during the study. ¶.MTOPS : MTOPS was a double-blind, placebo-controlled, multicenter, randomized study with a mean follow-up of 4.5 years. A total of 3,047 patients were randomized to PROSCAR 5mg (n=768), doxazosin 8mg (n=756), PROSCAR and doxazosin (n=763), or placebo (n=757). Entry criteria included: men aged > 50 years; AUA symptom score 8-35, maximum flow rate(Qmax) 4-15 mL/sec, and voided volume \geq 125mL. The primary outcome was overall clinical progression, defined as the first occurrence of a \geq 4 point increase over baseline of at least 4 points in the AUA symptom score, acute urinary retention, renal insufficiency, recurrent urinary tract infection or urinary incontinence. Secondary outcomes included changes in AUA symptom score and maximal urinary flow rate.⁶

Reference 1. Proscar[®] 제품설명서, 한국 MSD 2. Marks LS. 5- α -reductase: history and clinical importance. Rev Urol. 2004;6(suppl 9):S11-S21. 3. Data on file, MSD Korea. 4. McConnell JD, Bruskewitz R, Walsh P, et al. Finasteride Long-Term Efficacy and Safety Study Group. The effect of finasteride on the risk of acute urinary retention and the need for surgical treatment among men with benign prostatic hyperplasia. N Engl J Med. 1998;338(9):559-563. 5. Nickel JC. Comparison of clinical trials with finasteride and dutasteride. Rev Urol. 2004;6(suppl 9):S31-S39. 6. McConnell JD, Roehrborn CG, Bautista OM, et al. The long-term effect of doxazosin, finasteride, and combination therapy on the clinical progression of benign prostatic hyperplasia. N Engl J Med. 2003;349(25):2387-2398. 7. 보건복지부 고시 제2006-98호 별시 8. 보건복지부 고시 제2012-27호 약제 급여 목록 및 급여 상한금액표 별지 62014년 1월 1일 시행

프로스카 주요 안전성 정보

1. 효능 효과 양성 전립샘 비대증의 치료: 양성전립샘 비대증 증상의 개선, 비후된 전립샘의 퇴행 및 유류 개선, 골성 요폐의 발생빈도 감소, 전립샘 경요도 절제술 및 전립샘 절제수술 등을 포함한 수술의 필요성 감소 **2. 용법 용량** 1회 1회 1정(5mg) 복용. 복용시 증상이 개선된다더라도 최소 6개월간의 치료가 필요하다. 프로스카정은 여러 종류의 심부전 환자 또는 노인에게 있어 용량을 조절할 필요는 없다. **3. 경고** 프로스카정은 소아 또는 여성에게 투여하지 않는다. 임부 또는 임신하고 있을 가능성이 있는 여성은 피나스테리드의 흡수 및 그 이후 남성태아에 대한 잠재적 위험의 가능성이 있으므로 프로스카정의 부사지거나 개진 조사를 면해서는 안 된다. 프로스카정은 코담뱃피가 있기 때문에 개진되거나 흡입되거나 흡입될 수 있다. **4. 금기** 이 약 또는 이 약의 구성성분에 과민반응 환자, 여성 또는 소아, 임부 또는 임신하고 있을 가능성이 있는 여성, 갈락토오스 분해효소, Lapp 유전변태 효소 결핍증 또는 포도당-갈락토오스 흡수장애 등의 유전적인 문제가 있는 환자 **5. 신중투여** 간기능에 이상이 있는 환자 **6. 이상반응** 프로스카정은 내약성이 우수하고 이상반응은 일반적으로 경미하고 일시적이다. 외국의 임상: 4년간의 위양대조 임상시험(PLESS)에서 프로스카정으로 치료한 3.7%(57명), 위약으로 치료한 2.1%(32명)의 환자가 심각한 이상반응으로 치료를 중단하였다. 프로스카정에서의 발현율이 1% 이상이었으며 임상시험 연구자에 의해 약물과 관련되어 있을 가능성이 있거나, 아마도 관련되어 있거나, 분명히 관련되어 있다고 간주된 이상반응으로는 치료 1년째 발기부전(프로스카정 8.1% vs 위약 3.7%), 성욕감퇴(6.4% vs 3.4%), 사정장애(3.7% vs 0.8%), 사정장애(0.8% vs 0.1%), 유방비대(0.5% vs 0.1%), 유방염통(0.4% vs 0.1%), 발진(0.5% vs 0.2%)이 있었다. 임상시험 2-4년에는 부야르간 발기부전, 성욕감퇴, 사정장애 발현율이 유의한 차이가 없었다. 외국의 시판 후 조사: 통 제제 그리고/또는 피나스테리드 복용량에서 보고된 이상반응으로는 기력, 두드러기 및 혈관부종(인술, 혀, 목구멍 및 얼굴의 종창을 포함)과 같은 과민반응, 우울증, 고원혈, 투여 중단 후 지속되는 성욕 감퇴, 성기능 장애(발기부전, 사정장애), 남성 불임 그리고/또는 정액의 질 저하(피나스테리드 투여 중단 후 정액의 질 정상화 혹은 개선), 남성유방암, 위장장애(속쓰림), 어지러움, 두통 등이 있었다. 국내의 시판 후 조사: 6년동안 3,675명을 대상으로 실시한 시판후조사 결과 프로스카정과 인과관계가 있을 수 있는 것으로 평가된 것은 위장장애(속쓰림), 발기부전, 성욕감퇴, 사정장애(정액량 감소), 발진, 기력감, 과민반응, 두통, 어지러움이었다. **7. 임상검사치에의 영향** 프로스카정으로 치료를 받은 전립샘암 환자에서 임상적 효능은 입증되지 않고 있다. 프로스카정은 전립샘암 존재 하에서도 양성전립샘비대증 환자의 혈청 PSA 농도를 대략 50%정도 감소시킨다. 프로스카정으로 치료를 받는 환자의 지속적인 PSA 수치 증가는 프로스카정의 치료에 대한 비효율 문제를 고려하는 것을 비롯하여 주의깊게 평가해야 한다. 유리 PSA 수치(비효율성 PSA)에 대한 유리 PSA의 비율은 프로스카정으로 인해 유의하게 감소하지 않는다. ※자세한 내용은 제품설명서 전문을 참조하시기 바랍니다.

제28회 대한비뇨기종양학회 정기학술대회



08:30-09:00	Registration
09:00-09:05	President's Welcome
09:05-09:10	Congratulatory Remarks
09:10-09:50	Podium Session I: Prostate Cancer
09:50-10:20	Special Lecture mTOR Inhibitors in Metastatic Renal Cancer - Do They Work and When Do They Work?
10:20-10:40	Coffee Break
10:40-11:50	Symposium I : Management of Advanced RCC 1) Integration of Molecular Diagnostics into Clinical Practice 2) The Role of Surgery in the Era of Targeted Therapy: Cytoreductive Nephrectomy and Metastasectomy 3) Immunotherapy in mRCC: From Cytokines to PD-1 Checkpoint Inhibition Panel Discussion: Case Based Approach
11:50-13:00	Lunch (공로패 증정 및 전체 사진 촬영, 이사회)
13:00-13:40	Project 2014 Report / 2015 Proposal
13:40-14:20	Podium Session II: Bladder and Renal Cancer
14:20-15:00	Invited Lecture
15:00-15:20	Coffee Break
15:20-16:00	Podium Session III: Prostate Cancer
16:00-17:10	Symposium II : Management of Localized Prostate Cancer 1) Optimizing Prostate Cancer Diagnostics: Transperineal, Transrectal and MRI-US Fusion Targeted Biopsy 2) Surveillance or Treatment: The 3Ms - Markers, Mapping, and MRI for Localized Prostate Cancer 3) Selection of Nerve Sparing Candidates - Nomograms, Imaging, or Frozen Section 4) Positive Resection Margin and/or Pathologic T3 with Undetectable Postoperative PSA after Radical Prostatectomy: to Irradiate or Not?
17:10-17:30	2015 KUOS Annual Business Meeting
17:30-17:40	학술상 시상 및 폐회사 (Adjourn)

Q The 13th KUOS Multidisciplinary conference

대한비뇨기종양학회

The 13th KUOS Multidisciplinary Conference

일시 | 2015년 3월 28일(토) 08:30-17:30 장소 | 서울아산병원 동관 6층 대강당

평점 | 대한의사협회 4점

초대의글

비뇨기종양학회 회원 여러분

안녕하십니까?

2015년 3월 28일(토) 서울아산병원 대강당에서 개최되는 13회 Multidisciplinary Conference에 여러분을 초대합니다.

Multidisciplinary Conference는 지난 10여년 간 비뇨기종양의 진단 및 치료에 대해 여러 과 선생님들을 모시고 깊이 있는 토론의 장으로서 발전해왔습니다. 올해에도 전립선암과 방광암을 주제로 최신지견과 생동감 있는 토론이 이루어지도록 준비하였습니다.

이번 Conference는 "Current issues in diagnosis and prognosis of MIBC", "Treatment of MIBC", "Updates of prostate cancer", 및 "Low-risk prostate cancer" 라는 주제로 전문지식을 넓히고 각 과 선생님들과 함께 실제 증례를 중심으로 열띤 학술 토론의 장이 될 것을 기대합니다. 특히 전립선암 분야에서 세계적 석학이신 University of Colorado의 David Crawford 교수의 강의도 마련되어 있습니다.

13회를 맞이하는 Multidisciplinary Conference에 여러 저명하신 교수님들을 초빙하여 우리 비뇨기종양학회회원들과 교류 및 공동연구의 방향을 모색하고자 합니다. 이번 학술대회를 계기로 비뇨기 종양을 연구하시는 여러 과 선생님들의 학술적 교류가 더욱 활발해지는 계기가 되기를 기원합니다. 경험이 많으신 회원님들의 적극적 참여와 지도를 부탁드립니다.

2015년 한해도 회원 여러분들의 건강과 발전이 함께 하시기를 기원합니다.

대한비뇨기종양학회 회장 김 형 진

학술대회 프로그램

08:30-09:00	Registration	
09:00-09:05	President's Welcome	대한비뇨기종양학회장 김형진
09:05-09:10	Congratulatory Remarks	대한비뇨기과학회장 주명수
09:10-10:10	Symposium (I): Current issues in diagnosis and prognosis of MIBC 1. Diffusion-weighted MR imaging for preoperative staging 2. Prognosis of histologic variants for bladder urothelial carcinoma 3. Systemic inflammatory responses (SIR) as prognostic factors 4. LN-dependent variables as prognosis markers	좌장: 천 준 (고려의대) 성득제 (고려의대 영상의학과) 문경철 (서울의대 병리학과) 구자현 (서울의대) 정인갑 (울산의대)
10:10-10:30	Memorial lecture The characteristics of prostate cancer in Korea men	좌장: 김형진 (전북의대) 안한중 (울산의대)
10:30-10:50	Coffee break	
10:50-12:10	Consensus meeting (I): Treatment of MIBC 1. Neoadjuvant chemotherapy: new standard of care 2. Adjuvant chemotherapy 3. Trimodality bladder-sparing approach Panel discussion 성득제 (고려의대 영상의학과), 문경철 (서울의대 병리과), 서호경 (국립암센터), 정승일 (전남의대), 강석호 (고려의대)	좌장: 이형래 (경희의대) 서호경 (국립암센터) 정승일 (전남의대) 강석호 (고려의대)
12:10-13:30	Special symposium 3-dimensional transperineal mapping biopsy and targeted focal therapy for the management of organ confined prostate cancer David Crawford (University of Colorado)	좌장: 최한용 (성균관의대)
13:30-14:30	Symposium (II): Updates of prostate cancer 1. Prostate imaging-reporting and data system (PIRADS) 2. Handling of RP specimen: emphasis on the evaluation of margin status 3. Role of surgery for advanced prostate cancer 4. Current status of intermittent androgen deprivation	좌장: 김홍섭 (건국의대) 박병관 (성균관의대 영상의학과) 조남훈 (연세의대 병리과) 홍성후 (가톨릭의대) 하홍구 (부산의대)
14:30-15:10	Invited Lecture Androgen annihilation as a new therapeutic paradigm in advanced prostate cancer David Crawford (University of Colorado)	좌장: 조진선 (한림의대)
15:10-15:30	Coffee break	
15:30-16:50	Consensus meeting (II): Low-risk prostate cancer 1. Comparison of active surveillance protocols 2. Novel biomarkers in the era of active surveillance 3. Various treatment options for low-risk prostate cancer Panel discussion 박병관 (성균관의대 영상의학과), 조남훈 (연세의대 병리과), 한준현 (한림의대), 김정현 (강원의대), 정병창 (성균관의대)	좌장: 박동수 (차의대) 한준현 (한림의대) 김정현 (강원의대) 정병창 (성균관의대)

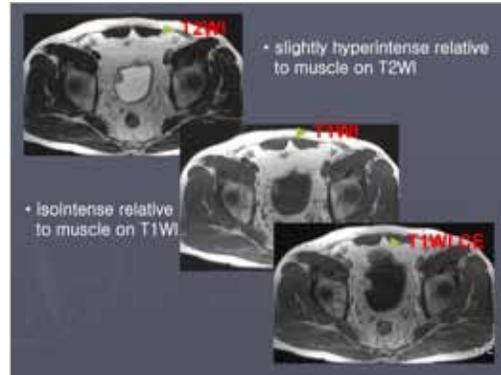


성득제

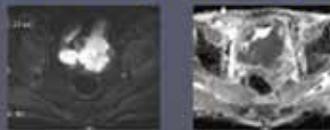
Diffusion-weighted MR imaging for preoperative staging of bladder cancer

Korea University, Anam Hospital, Department of Radiology

Deuk Jae Sung



Diffusion-weighted imaging (DWI)

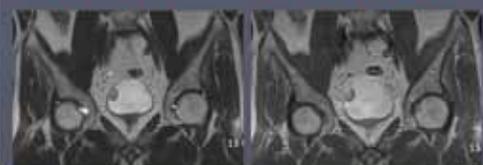


B value, 1000 s/mm²

ADC map

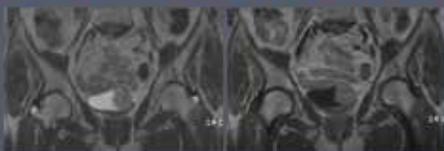
- aggregated **cancer** cells and fibrotic stroma
 - > inhibit the movement of water macromolecules
 - > resultant restriction of diffusion and reduction of apparent diffusion coefficient (ADC)
- bladder cancers
 - > high signal on raw DWI because of restricted diffusion
 - > low signal on ADC maps

Superficial Versus Infiltrating Tumor



- MRI cannot resolve the various bladder wall layers accurately.
- limiting the accuracy of T staging, particularly in distinguishing among T1 and T2 tumors.
- In one study, staging accuracy of DWI was 63.6% in differentiating superficial from invasive tumors

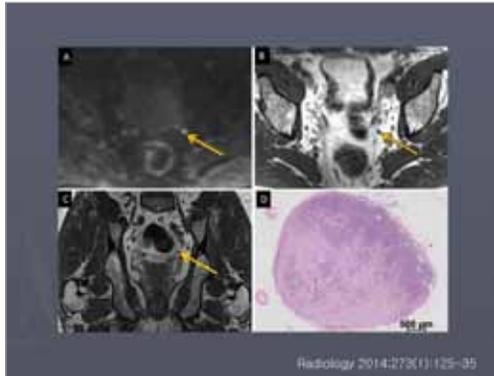
Localized Versus Locally Extensive Tumor



- Macroscopic extravesical extension (T3b)
 - : an irregular, ill-defined outer bladder wall
 - : soft-tissue nodules or fat stranding in the surrounding perivesical fat

Imaging Nodal Metastases

- A limitation of both CT and MR imaging is the detection of metastasis in normal-sized lymph nodes.
- Several studies describe significantly lower ADCs in malignant versus benign lymph nodes.
- Others reported that there were no statistically significant differences in the ADC of metastatic and non-metastatic nodes.



- DW imaging may be useful in treatment planning and in determining the prognosis in patients with bladder cancer.
- Further studies are needed to validate the utility of DWI as a biomarker for accurate T and N staging.

문경철

Prognosis of Histologic Variants of Urothelial Carcinoma

서울대학교 병원 병리과 문경철

Urothelial carcinoma

- Papillary urothelial lesion
 - Papilloma
 - Papillary urothelial neoplasm of low malignant potential (PUNLMP)
 - Papillary urothelial carcinoma, LG/HG
- Flat urothelial lesion
 - Urothelial carcinoma in situ
 - Invasive urothelial carcinoma

Incidence of variants

- 19.5% of 589 TURBs
(Urologic Oncology: Seminars and Original Investigations 31 (2013) 1650-1655)
 - 25% of 448 TURBs
(UROLOGY 70: 69-74, 2007.)
 - 40% of 108 pelvis high grade urothelial carcinomas
(Modern Pathology (2006) 19, 494-503.)
- Squamous diff (32%) > Small cell (16%) > Glandular diff (13%) > Micropapillary (12%) > Nested (8%) > Sarcomatoid (6%) > Lymphoepithelial (3%) > Plasmacytoid (1%)
(Urologic Oncology: Seminars and Original Investigations 31 (2013) 1650-1655)

Incidence according to stage

Table 4. Patient demographics and clinical and pathologic findings in pure UC and UC with mixed histologic type

Variable	Pure UC	UC with Mixed Histologic Type	P Value
TURBT			
Patients	336 (75)	112 (25)	
Median followup time (yr)	2.8	1.9	
Mean age (yr)	66.6	66.9	
Sex (percentage showing mixed)			
Male	75.1	24.9	
Female	74.6	25.5	
Tumor grade at TURBT			
Low	55 (16.4)	0	
High	281 (83.6)	112 (100)	
Clinical stage (for high-grade tumors)			
Ta/Ta	50 (17.6)	1 (0.9)	<0.001
T1	84 (29.9)	35 (33.4)	
T2	147 (52.5)	66 (65.7)	
Cystectomy			
Patients	205 (69.5)	90 (80.5)	
Pathologic stage (pT)			
T0	33 (16.1)	8 (8.9)	<0.001
Ta/Ta	30 (14.6)	3 (3.3)	
T1	30 (14.6)	2 (2.2)	
T2	42 (20.5)	19 (21.1)	
T3	47 (22.9)	48 (53.3)	
T4	23 (11.2)	10 (11.1)	

(UROLOGY 70: 69-74, 2007.)

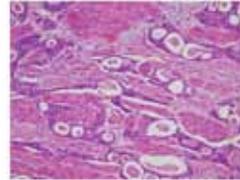
Prognosis of variants

- Most variants:
 - Higher stage
 - Aggressive behavior
 - Poor prognosis

- Lymphoepithelioma-like carcinoma
 - Relatively favorable prognosis

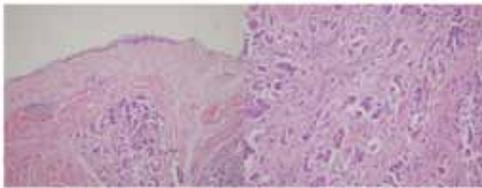
Microcystic variant

- Cystic change in tumor cell nests
- Diagnostic difficulties, especially in limited sample
- DDX from cystitis cystica / glandularis



Micropapillary variant

- 0.6-1% of urothelial carcinoma
- Micropapillary architecture
- Almost muscle invasive at presentation
- Frequent lymph node and distant metastasis



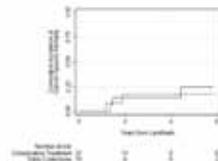
T1 micropapillary Uca of bladder

Clinical Outcome of Patients with T1 Micropapillary Urothelial Carcinoma of the Bladder

Masami Iwano, Spallivano, Guido Dall'Aglio, * Bernard H. Bachner, Bing Ying Poon, Hongping Huang, Wiktat A. Al-Ahmadia, Timothy F. Danaher, Jennifer M. Taylor, Joshua J. Moska, Daniel G. Sobberg, S. Manohar Desai, Victor J. Reuter and Marty W. Hear

Urology Service, Department of Urology, Dana-Farber Cancer Institute, Boston, MA; Harvard Medical School, Boston, MA; Department of Pathology and Microbiology, Dartmouth Medical School, Lebanon, NH; Department of Pathology, Brigham Young University, Provo, UT; Department of Pathology, University of Michigan, Ann Arbor, MI

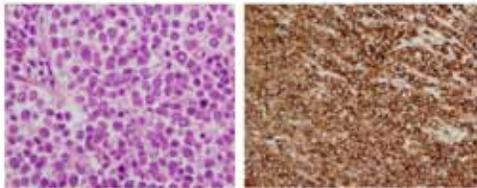
36 T1 MP Uca by TURB
 15 early RC within 3 mo
 21 conservative Tx or deferred RC
 → no significant prognostic difference



The Journal of urology 2014;192(3):702-707

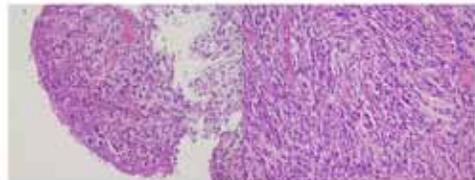
Plasmacytoid variant

- Tumor cells resembling plasma cells
- IHC: CK7/CK20 +, CD138 +/-
- Can be misdiagnosed as chronic inflammation



Sarcomatoid differentiation

- Sarcomatoid carcinoma: favored term
- Carcinosarcoma (overt carcinoma and sarcoma)



Neuroendocrine tumors

- Carcinoid
- Small cell carcinoma
- Large cell neuroendocrine carcinoma

Summary

- Not rare
- Most variants: aggressive behavior
- Frequently higher stage
- Some variants mimic benign lesion
- Prognosis: variable

구자현

Systemic Inflammatory Responses (SIR) as prognostic factors in MIBC

Ja Hyeon Ku, M.D., PhD
Associate Professor of Urology
Department of Urology,
Seoul National University School of Medicine

Prostate cancer – CRP

Systemic Inflammatory Response and Survival in Patients with Localised Prostate Cancer: 10-Year Follow-Up

Parameter	Univariate analysis	Multivariate analysis
Age	NS	NS
Confirmed Gleason score	NS	NS
Prostate treatment (surgery vs. radiation vs. androgen deprivation therapy)	NS	NS
Prior exposure to glucocorticoids (steroids) or anti-inflammatories at $t=0$	HR, 0.499, $p = .019$	HR, 0.513, $p = .024$
Prior exposure to antiandrogens at $t=0$	HR, 0.392, $p < .0001$	HR, 0.449, $p = .003$
Baseline PSA doubling time ≥ 2.7 mo	HR, 0.471, $p = .040$	HR, 0.449, $p = .037$
Baseline PSA level	NS	NS
Initial disease extent	HR, 0.491, $p = .0001$	NS
Interval of baseline PSA	HR, 0.581, $p = .009$	NS
Baseline alkaline phosphatase within the normal range	HR, 0.466, $p < .0001$	NS
Albumin	NS	NS
Prognosis, adjusted for (baseline PSA, age, T, N, M)	HR, 0.554, $p < .0001$	HR, 0.513, $p < .0001$

Abbreviations: HR, hazard ratio; PSA, prostate-specific antigen.

Prostate cancer – mGPS, NLR

Systemic inflammation and survival of patients with prostate cancer: evidence from the Glasgow Inflammation Outcome Study

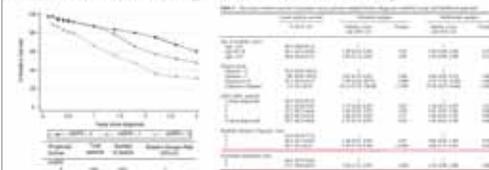


Figure 1. Five-year overall survival of high-risk prostate cancer patients treated with radical prostatectomy, stratified by GIOS score.

Stratigos, Prostate Cancer Prostate Dis. 2012

CRPC – NLR

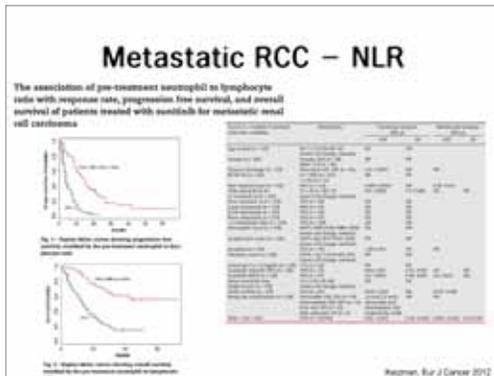
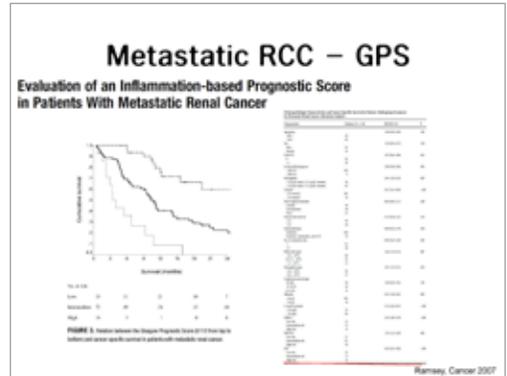
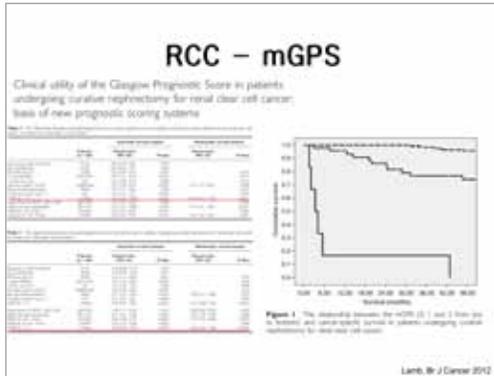
Persistent Neutrophil-to-Lymphocyte Ratio in Metastatic Castration-Resistant Prostate Cancer Patients Treated With Radiotherapy: Association with Outcome and Prognostic Nomogram

Table 2. Univariate and multivariate analysis of the association between inflammatory and prognostic factors and the progression-free survival period

Parameter	Univariate analysis	Multivariate analysis
Age	NS	NS
Confirmed Gleason score	NS	NS
Prostate treatment (surgery vs. radiation vs. androgen deprivation therapy)	NS	NS
Prior exposure to glucocorticoids (steroids) or anti-inflammatories at $t=0$	HR, 0.499, $p = .019$	HR, 0.513, $p = .024$
Prior exposure to antiandrogens at $t=0$	HR, 0.392, $p < .0001$	HR, 0.449, $p = .003$
Baseline PSA doubling time ≥ 2.7 mo	HR, 0.471, $p = .040$	HR, 0.449, $p = .037$
Baseline PSA level	NS	NS
Initial disease extent	HR, 0.491, $p = .0001$	NS
Interval of baseline PSA	HR, 0.581, $p = .009$	NS
Baseline alkaline phosphatase within the normal range	HR, 0.466, $p < .0001$	NS
Albumin	NS	NS
Prognosis, adjusted for (baseline PSA, age, T, N, M)	HR, 0.554, $p < .0001$	HR, 0.513, $p < .0001$

Abbreviations: HR, hazard ratio; PSA, prostate-specific antigen.

Kueman, Oncotarget 2012



- ### Summary
- Test in the routine clinical practice
 - Easily measured test
 - Reproducible test
 - Cost-effective test

안한종

The 13th KUOS Multidisciplinary conference
Memorial lecture

The Characteristic of Prostate Cancer in Korean Men

Hanjong Ahn, MD, Ph.D
University of Ulsan College of Medicine
Asan Medical Center, Seoul, Korea

DETECTION RATE OF PROSTATE CANCER ON BIOPSY ACCORDING TO SERUM PROSTATE-SPECIFIC ANTIGEN IN KOREAN MEN: A MULTICENTER STUDY

WON JAE YANG, DONG HYUN LEE, BYUNG HWA CHUNG, JIN SEON CHO, YOUNG DEUK CHOI, SE JOONG KIM, IN BAE CHO, HONG SUP KIM, CHUN IL KIM, SUNG JOON HONG, and MEMBERS OF THE SEVERANCE UROLOGIC ONCOLOGY GROUP

- 2,422 Korean men who had undergone prostate biopsy at 12 medical centers from 1993 to 2002 were analyzed.
- PSA > 4.0 ng/mL or abnormal DRE findings
- Of the 2,422 men, 962 (39.7%) were diagnosed with prostate cancer.

Yang et al. Urology 2006

Incidence and pathologic characteristics of prostate cancer in Korean men

- Prostate cancer is one of the most rapidly increasing cancer in Korean men along with colon cancer.
- Estimated incidence of prostate cancer was 36.9/100,000 in 2014, ranked as 4th most common cancer in Korean men.
- The detection rate of prostate cancer (PSA ; 4–10) has been increasing after mid-2000s, from 15.9-19.6% to 27.3-28.5%, which was comparable to the Western series.

- A distinguished characteristic of prostate cancer in Korean men is high prevalence of high grade cancer (GS \geq 7).
- The prevalence of high grade cancer among Korean men was remarkably high, even in men with low levels of PSA (< 4.0 ng/ml; 50.5%).
- This may be attributable to late detection or the nature of disease specific to Asian population.

High prevalence of high grade disease in Korean

- High prevalence of Gleason 7 or greater disease in the range of PSA 3-4 and in autopsy study (Russian vs. Japanese) supported that the predominance of high grade disease is the nature of disease specific to Asian.
- Comparative study between Korean and American and recent downward migration of disease stage and grade suggested that high prevalence of high grade disease in Korean men be due to late detection or selection bias.

Impact of PSA Screening on Grade & Stage Migration: AMC experience

- 12,503 men with TRUS-biopsy at AMC from 2000 to 2013 were reviewed retrospectively.
- Men who were newly diagnosed with PCa (n = 3,269) were included in the analysis.
- Before 2009, prostate biopsies were recommended in men with a serum PSA level \geq 4.0 ng/mL and/or if PCa was suspected clinically. After 2009 a PSA cutoff value of 3.0 ng/mL regardless of digital rectal examination (DRE) and/or TRUS findings.

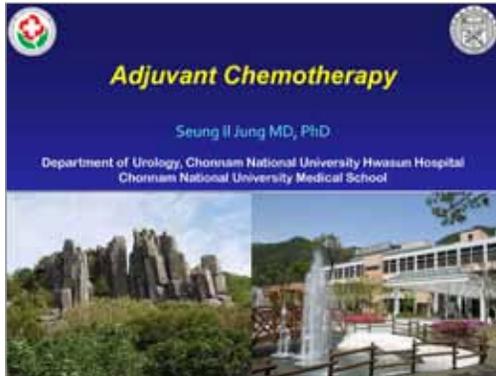
Shim et al. (in Submission) 2015

Characteristics of prostate cancer in Korean men

- Estimated incidence of prostate cancer in Korean men in 2014 was 36.9/100,000.
- High prevalence of high grade disease is a distinguished characteristic of prostate cancer in Korean men and actually increasing recently.
- The grade of disease is a major determinant of patients' survival after radical surgery + radiation therapy + hormonal treatment.

- While PSA screening rate is stationary, early detection of cancer at low PSA in clinical practice significantly decreases the metastatic disease at presentation.
- In the active surveillance era, do we need to recommend the prostate biopsy at low PSA? Yes, because high grade cancer prevails in Korea.

정승일



Adjuvant Chemotherapy

1) advantages

- optimal timing of surgery
: Avoiding delay in possibly curative surgery
- personalization of chemotherapy.
: based on pathologic staging

2) disadvantages

- limited amount of data
(no randomized comparisons of adequate sample size)
→ impossible to come to a clear recommendation

European Urology 48 (2005) 188-201

Neoadjuvant or adjuvant in MIBC

Meta-analysis of 11 RCT
→ DFS & survival benefit +

Neoadjuvant

Meta-analysis of 9 RCT
→ DFS & survival benefit +

adjuvant

The EAU has yet to recommend adjuvant chemotherapy
(this is purely because the data on adjuvant chemotherapy is insufficient)

Conclusion

- limited amount of data, which makes it impossible to come to a clear recommendation on adjuvant chemotherapy.

→ high-risk patients, such as those with extravesical and/or node-positive disease, will most likely benefit

- individual patient data meta-analysis including EORTC trial is required to confirm this.

panel.pptx consensus I

M/68

C/C: gross hematuria

P/H: hypertension medication (+)

P/I: 상기자 타병원에서 시행한 검사상 bladder tumor detect 되어 내원함

2010.09.27 Lab

BUN: 24.6mg/dL [8-23]

Creatinine: 1.3 mg/Dl [0.5-1.3]

U/A:

WBC: 10-15/HPF [0-4]

RBC: 1000/HPF [0-1]

Abdomen-pelvic CT

TUR-BT (2010.10.11)

↳ urethral orifice 주위 약 2.5cm 크기의 main nodular mass

posterior wall 과 dome 주위 small nodular mass 등 주변으로 multiple satellite lesion

• Q: immediate epirubicin or mitomycin instillation ?

TUR-BT (2010.10.11)

A. immediate intravesical epirubicin instillation was done

• Invasive urothelial carcinoma, high grade, (T1, high grade), micropapillary variant, no muscle invasion

Re-TURBT at Postop. 1 month (2nd TUR-BT, 2010-11-15)

• Pathology: T1, high grade, Urothelial Carcinoma, micropapillary pattern (+), muscle invasion negative

Q: what is your next Plan?

Postop. 6 months evaluation

Abdominopelvic CT

Renal scan

Lt vs Rt: 20.9: 79.1
(← Lt vs Rt 46.8: 53.2 (내원당시))

Radical cystectomy with orthotopic neobladder (2011.6.17)

1. urothelial carcinoma, micropapillary variants
: LN metastasis (28/51)
: presacral, Lt common, external iliac, obturator, internal iliac LN (+)

(pT3bN3M0)

2. Rt distal ureter: CIS but frozen margin negative

Post-radical cystectomy 1 month (2011.07)

• MAG-3 renal scan
→ Lt/Rt function = 16.5%/83.5%
• Lab: BUN 48.0, Cr 2.1

• pT3bN3M0 bladder cancer
• (micropapillary variants)
• F/U imaging: no metastasis

What will you do next?

1. Observation
2. Gemcitabine + Cisplatin
3. Gemcitabine + Carboplatin
4. M-VAC

Gemcitabine-Carboplatin adjuvant chemotherapy

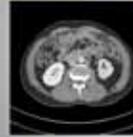
- 2011.8.3-2011.12.2 (6 cycle)
- Post op RFS: 12 months

Post salvage 6cycle GC chemo (post RC 21 months)

→ response : SD → chemo off

• chemo off 3 months
→ new aorticaval LAP

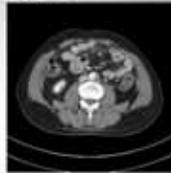
• chemo off : 8 months; retroperitoneal multiple conglomerate LAPs; Lt. hydronephrosis



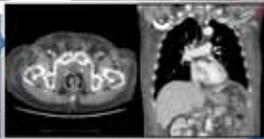
Q: what is your next Plan?

Salvage M-VAC

- 2nd Salvage M-VAC 6 cycle (2013.8-2014.3)
- response : PR
- Chemo off



- Post 2nd salvage M-VAC chemo off 7 months
- Carcinoma peritonitis, Rt hydronephrosis, neobladder recur (2014.11)
- 3rd Salvage M-VAC (2014.11 --)



M/73

C/C: gross hematuria

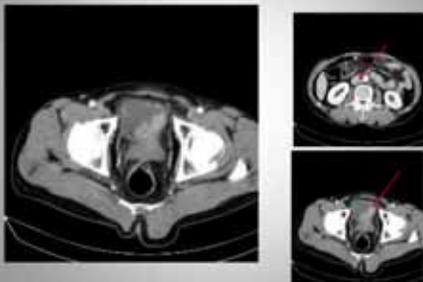
P/H: No known Hx of DM, Hepatitis, Pul TBC, HTN

P/I: 상기자 타병원에서 시행한 검사상 bladder tumor detect 되어 내원함

2010.04.29 Lab

BUN: 22.8 mg/dL [8-23]
Creatinine: 1.2 mg/dL [0.5-1.3]
U/A:
WBC: 30-39/HPF [0-4]
RBC: 1000#/HPF [0-1]

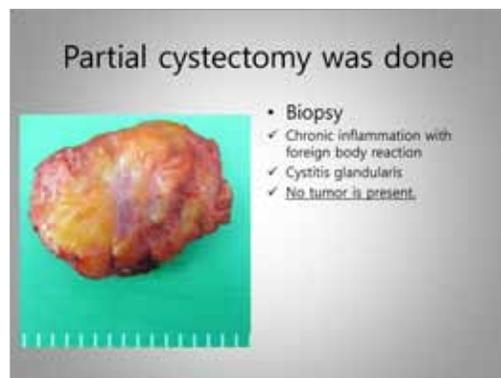
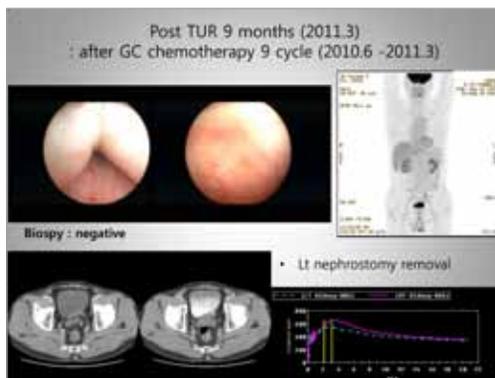
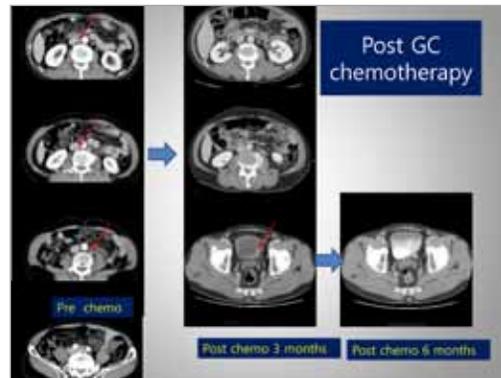
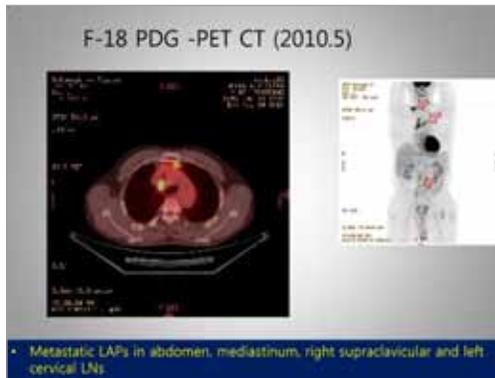
Abdomen-pelvic CT (2010.04)



1st TUR-BT (2010.05.14)



INFILTRATING PAPILLARY UROTHELIAL CARCINOMA, HIGH GRADE
Muscle invasion (+)



조남훈

Prostate Surgical Margin in RP

조남훈
연세의대 병리학교실

Yossepowitch et al. Collaborative Review-Eur Urol 2014; 65:303-13/ 2009; 55:87-99
Meeks & Eastham. Urol Oncol 2013; 31:974-9

Prostate Capsule

- **Definitely present, but not consistent**
 - Inner smooth muscle at transverse
 - Outer thin collagenous layer (intermingled in ant.)
 - Thickening fused to Denoviller's fascia
 - Huge large NVB entry: surgical incision
 - Terminal acini in TZ/PZ never reach surface!
- **Defect of capsule-** at the *apex, anterior* and *distal urethra*
 - Apical invasion-always hurdle to evaluate capsular invasion or true invasion?
 - Small NVB entry at lateral or posterolateral: inadvertent surgical margin (surgical incision: cut across the proper)

Prostate Margin

- Surgical Margin vs Anatomic capsule
- pT2+ vs pT3 stage criteria: absolutely depends on EPE, not surgical margin
 - Iatrogenic (capsular incision) vs non-iatrogenic
- PSM+ve clinical meaning?
 - Truly present?
 - Prognostic factor?
 - Adjunctive therapy?

PSM in clinical Px

- BCR-free survival
 - In major, "Maybe"
- PCa-specific survival
 - In major, "Not definite"
- Overall survival
 - In major, "Not definite"

홍성후

Role of surgery for high-risk and advanced prostate cancer

Sung-Hoo Hong, MD., PhD.
Department of Urology, College of Medicine,
The Catholic University of Korea

PCa with N+

- Indicating systemic disease
- Limited long-term survival
- Not considered surgical candidates
- Usually treated with hormonal therapy and/or radiotherapy
- Most urologists will abort surgery if nodal involvement is detected.

Cytoreductive prostatectomy

- Survival benefit and improved response to systemic therapy in kidney, colon, breast, and ovary cancers
- In contrast, the role of cytoreductive prostatectomy has not been rigorously evaluated.
- However, along with advances in surgical technique and staging, there has been some retrospective data suggesting a possible role for cytoreductive surgery in PCa.

RP in metastatic PCa

- Rats implanted with human PCa always develop lung metastases.
- Those in which the implanted tumor was removed lived longer and with few lung colonies. (Salmon et al., Urol 1980)
- Patients with metastatic PCa who had undergone previous RP had a better response to ADT and better survival than those with an untreated. (Swanson et al., Urol 2002; Thompson et al., Urol 2002)

Conclusions

- Good cancer-specific survival in high-risk PCa.
- More accurate risk assessment can guide additional effective therapy.
- Pathologically organ-confined disease in one third of patients

Conclusions

- Good survival rates in very high PSA levels
- Survival benefit in intraoperatively to be N+.
- Possible role of cytoreductive prostatectomy in advanced setting
- Salvage RP is most effective option with decreased side-effects in the most recent series.
- Nevertheless, multimodal therapy is needed to optimize cancer control.

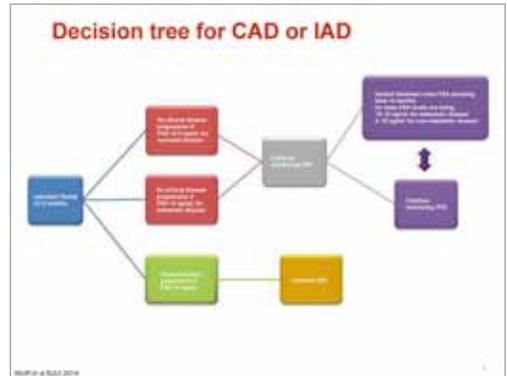
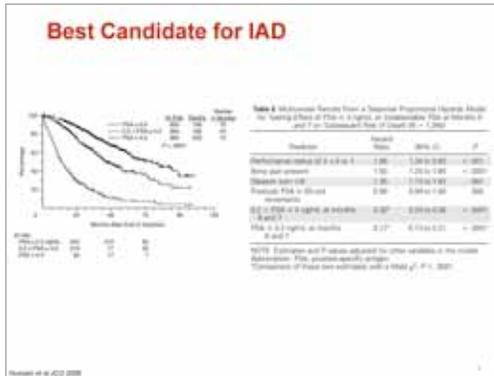
하흥구

ADT : Intermittent vs continuous

Hong Koo Ha, MD, PhD
Department of Urology
Pusan National University Hospital, Korea

IAD vs CAD in mPCA (3)

	Intermittent ADT (n = 27)	Continuous ADT (n = 27)	Total (combined) (n = 54)	Best combined (n = 54)
Age, years	68.7 ± 2.4	68.7 ± 2.1	68.7 ± 2.1	68.7 ± 2.1
Median PSA	10.5 ± 0.5	10.5 ± 0.5	10.5 ± 0.5	10.5 ± 0.5
PSA progression-free survival, n (%)	11 (40.7)	10 (37.0)	21 (38.9)	11 (20.2)
Median time to progression	13.2 ± 1.4	12.2 ± 1.4	12.7 ± 1.4	13.2 ± 1.4
OS, months	35.2 ± 3.5	35.2 ± 3.5	35.2 ± 3.5	35.2 ± 3.5
Median time to progression - overall	28.2 ± 3.1	27.2 ± 3.1	27.7 ± 3.1	28.2 ± 3.1
Median time to progression - prostate	27.2 ± 3.1	27.2 ± 3.1	27.2 ± 3.1	27.2 ± 3.1
Median time to progression - prostate + systemic	27.2 ± 3.1	27.2 ± 3.1	27.2 ± 3.1	27.2 ± 3.1
Median time to progression - prostate + systemic + overall	27.2 ± 3.1	27.2 ± 3.1	27.2 ± 3.1	27.2 ± 3.1
Median time to progression - prostate + systemic + overall (p-value)	0.001	0.001	0.001	0.001
Median time to progression - prostate + systemic + overall (p-value)	0.001	0.001	0.001	0.001



The Standard of Care

Disease state	Continuous ADT	Intermittent ADT
Metastatic	Yes	No
Non-metastatic, rising PSA	Yes	Yes

Presented at ASCO Annual Meeting, 2014. Presented by Gilbert W. Coombs.

- ### Conclusions
- Sexual activity scores were higher and the incidence of hot flushes was lower in patients treated with IAD
 - OS was similar between IAD and CAD in patients with locally advanced, recurrent or metastatic hormone-sensitive prostate cancer.
 - IAD might result in a modest increase on CSM in mPCa
 - Optimal candidate to IAD : patients with moderately elevated PSA, low tumor burden, and preferably non-metastatic

한준현

Comparison of active surveillance protocols

Hallym University Dongtan Sacred Heart Hospital
Han, Jun Hyun M.D., Ph.D.

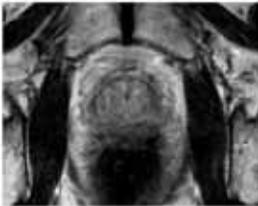
Follow-up criteria during active surveillance

Study	DRE	PSA	Biopsy	TRUS
Yoo et al	Every 3 mo for 2 yr, then every 6 mo	Year 1: monthly Year 2: every 3 mo Afterwards: every 6 mo	At 18-24 mo, then biannually	No mention
Dell'Isola et al	Every 3 mo	Every 3 mo	Every 12-24 mo	6-12 mo interval
Carver et al	Every 6 mo	Every 6 mo	Yearly	No mention
Klotz et al	Every 3 mo for 2 yr, then every 6 mo	Every 3 mo for 2 yr, then every 6 mo	At 18-24 mo	Optional
Favel et al	Every 3 mo for 1 yr, then every 6 mo	Every 3 mo for 1 yr, then every 6 mo	At 4 mo	At 4 mo
Silberny et al	Every 3 mo	Every 3 mo for 2 yr, then every 6 mo	At 6-12 mo, then individualized	No mention
Hosie et al	Every 3-6 mo for 2 yr, then every 6 mo	Every 3-6 mo for 2 yr, then every 6 mo	Not mention	Not mention

Bastian PJ, et al. Eur urology, 2009

2nd TRUS biopsy

- Adenocarcinoma, Gleason's score 3+3=6/10, tumor area up to 20%, 3 cores (+)/12 cores



Question 3

What's your treatment options?

- Radical prostatectomy
- Radiation therapy
- Continue active surveillance

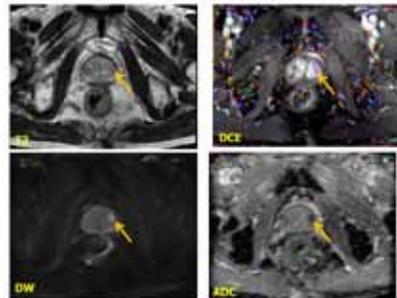
What's your trigger for Curative intervention in AS?

Radical perineal prostatectomy

CASE 2

- M/70
- Chief Complaint:
elevated PSA at other hospital
- PSA 3.76 ng/ml at SMC**
- TRUS Bx (12 cores, prostate size 28.3cc)
 - High grade prostatic intraepithelial neoplasm (1 core)
 - Gleason's score 3+3=6/10, tumor volume <5% (1 core)

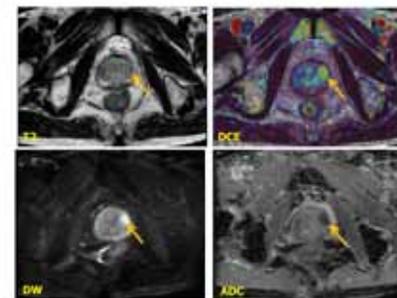
Prostate MRI



Active surveillance

- PSA follow up
 - 3 Mo later : PSA 3.75 ng/ml
 - 6 Mo later : PSA 4.21 ng/ml
 - 9 Mo later : PSA 7.46 ng/ml

What is your plan considering F/U PSA?



➔ Prostate MRI

- Prostate volume : 26 cc
- Interval increased size of prostate cancer (22mm) in the left transitional zone of midgland.
- No evidence of extracapsular extension, seminal vesicle invasion, and metastasis
- PI-RADS v2 : 4점

➔ 2nd TRUS biopsy

- Adenocarcinoma, Gleason's score 4+3=7/10, tumor area up to 75%, 5 cores (+)/13 cores
- Bone scan
: No definite evidence of bone metastasis
- A-P CT, Chest CT
: No evidence of metastasis

➔ Radical retropubic prostatectomy

- Adenocarcinomas, total tumor volume about 20 %
- 1) tumor : 2.0x1.9x1.3 cm, Gleason's score 4+5=9/10 (peripheral and transition zone of left lobe)
- 2) no extraprostatic extension
- 3) no involvement of bilateral seminal vesicles and negative vas
- 4) resection margin: apex, base, body (negative)
- 5) perineural invasion: not identified
- 6) lymphovascular invasion: present
- 7) high grade prostatic intraepithelial neoplasia : present

Pathology (pT2a R0)

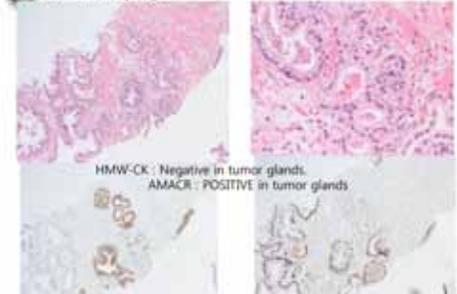
➔ CASE 3

- M/59
- Chief Complaint: elevated PSA at other hospital
- TRUS biopsy twice on Apr 2012, Nov 2013 at other hospital
- PSA: 5.1→5.3→7.2→5.4ng/ml at other hospital
- IPSS: 11/1 (urgency) IIEF-5: 22
- Ht 179cm; Wt 160Kg
BMI: 49.94 kg/m²
- DRE: >50g, right; firm to hard

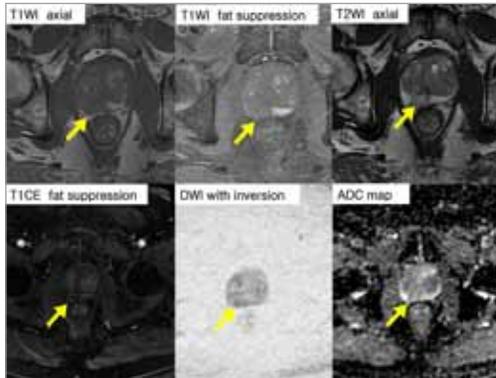
➔ MRI at other hospital



➔ 3rd biopsy



HMW-CK : Negative in tumor glands.
AMACR : POSITIVE in tumor glands



Question

What's your treatment option for prostate cancer?

- Radical prostatectomy
- Radiation therapy
- Brachytherapy
- Focal therapy
- Active surveillance

What's your treatment option for stone?

ESWL, URS, ureterolithotomy, observation

Brachytherapy

13th KUOS MDC conference 학술상

Korean J Urol Oncol 2014 Aug; 12(2): 65-70

Long Term Result of Modified 6+3 Bacillus Calmette-Guerin Maintenance Therapy for Non-muscle-invasive Bladder Cancer in Korea: Comparison of 6-week Therapy

Pil Moon Kang, Won Ik Seo, Jae Il Chung

Department of Urology, Busan Paik Hospital, InJe University, Busan, Korea



East Asia AZK Urologic Oncology Symposium

Date | March 27 (Fri), 2015

Venue | Best Western Gangnam, Seoul Diamond Room (B1)

Agenda

Time	Topic	Speaker
08:30-09:00	Introductory Remarks	Wu, Tony / Nakagawa, Masayuki / Ahn, Han Jong
09:00-11:00	Prostate Session 1	Chair: Ahn, Han Jong / Ogawa, Osamu
09:00-09:40	The role of adjuvant therapy for pT3 prostate cancer – a multicenter, retrospective study	Wu, Tony
09:40-10:20	Current status of the treatment of castration resistant prostate cancer	Ichikawa, Tomohiko
10:20-11:00	Peptide vaccination a promising therapeutic tool for CRPC – the final results of a randomized phase-II study	Uemura, Hirotsugu
11:00-11:10	Coffee Break	
11:10-13:00	Bladder Session	Chair: Lee, Hyung-Lae / Nishiyama, Hiroyuki
11:10-11:40	Panel Discussion: Radical cystectomy in Korea: multi-center retrospective database	Kim, Sun Il / Cho, Jin Seon
11:40-12:20	Aristolochic acid and urothelial carcinoma in Taiwan	Chen, Chung-Hsin
12:20-13:00	Advances in systemic therapy for urothelial cancer	Kitamura, Hiroshi
13:00-14:00	Lunch	
14:00-15:40	Prostate Session 2	Chair: Egawa, Shin / Pu, Yeong-Shiau
14:00-14:40	Brachytherapy and RALP in Japan	Nasu, Yasutomo
14:40-15:40	Panel Discussion: 1. Management of T3b prostate cancer 2. Chemotherapy in CRPC: early vs delayed	Ohyama, Chikara / Jeong, Chang Wook Jeong, In Gab / Hong, Sung Kyu
15:40-15:50	Coffee Break	
15:50-17:30	Kidney Session	Chair: Chung, Jin Soo / Tomita, Yoshihiko
15:50-16:20	Panel Discussion: – Characteristics and prognosis of Xp11.2 translocation renal cell carcinoma – Histological structures and dynamic CT patterns in renal cell carcinoma	Jeong, Chang Wook / Kimura, Go
16:20-17:00	Robotic-assisted partial nephrectomy: a Taiwanese single-institute experience	Pang ST Jacob
17:00-17:30	Panel Discussion: Can morphologic features of vena caval tumor thrombus be a prognostic factor after surgical treatment of RCC with IVC thrombus? – Korean Renal Cancer Study group multicenter study – Cytoreductive nephrectomy in the era of molecular targeting therapy	Seo, Seong Il / Eto, Masatoshi
17:30-19:00	Prostate Session 3	Chair: Kim, Choung Soo / Nakagawa, Masayuki
17:30-18:00	Panel Discussion: Salvage radiotherapy after radical prostatectomy	Jeon, Hwang Gyun / Takenaka, Atsushi
18:00-19:00	Case discussion: CRPC (2 cases)	Kwak, Cheol / Ozono, Seihiro / Cho, Jin Seon / Jeon, Seong Soo

Q 공지사항



1. 2015년 제28회 대한비뇨기종양학회 정기학술대회 및 총회

- 일시 : 2015년 8월 29일(토)
- 장소 : 차의대 차바이오컴플렉스

차의과학대학교 의학전문대학원(CHA BIO COMPLEX) 오시는길 (삼평동 689번지)

N(북)

↑ 주차장

GS주유소

판교역 방향 ↓

S(남)

상성테크빌

LIG네스원관고

광장지하 지도

→ 주차장 (입구)

후문

주차장

CHA BIO COMPLEX (삼평동 689)

버스정류소

입구

동성신대주 1단지아파트

삼평고등학교

아탑역 방향 →

구름다리

SK

KT NET

엠택IT

SK

상환아이텍스

IDE5

아이디스

상환아이텍스 B동

공화

We Made 엔터테이네

다산타워

휴먼시아 3단지아파트

휴먼시아 아파트

대중교통 이용 시

전철-신분당선 판교역 하차 - 1번 출구로 나와서 도보로 10분, 택시이용 시 기본요금, 버스 이용 812, 390번 버스노선(아탑역 3번 출구) : 812, 330, 350, 380, 390번 - 차병원 컨소시엄에서 하차

Watching one's grandchild grow
 can make a big difference
 in some lives.


Diphereline® P.R. 3.75mg
 triptorelin


Diphereline® P.R. 11.25mg
 triptorelin

Caring about men with prostate cancer

디페렐린피알3.75mg주, 디페렐린피알주11.25mg

[원료약품 및 그 분량] 디페렐린피알3.75mg주 1 바이알 중 주성분: 초산트립토헬렐린(별규) 3.75mg(트립토헬렐린으로서), 부형제 만니톨 85mg, 1 앵플 중 용제: 주사용수(EP) 2g, 등장화제 만니톨(EP) 16mg 디페렐린피알주11.25mg 1 바이알 중 주성분: 트립토헬렐린(별규) 14.58mg(트립토헬렐린으로서 11.25mg), 부형제 만니톨 63.75mg, 1 앵플 중 용제: 주사용수(EP) 2g, 등장화제 만니톨(EP) 16mg **[효능·효과]** 디페렐린피알 3.75mg주 형태 중 성스테로이드치 저하를 필요로 하는 호르몬의존성 전립선암 디페렐린피알주11.25mg 호르몬의존성 국소진행성 또는 전이성 전립선암 **[용법·용량]** 디페렐린피알3.75mg주 트립토헬렐린으로서 3.75mg을 매 4주 1회 근육주사한다. 디페렐린피알11.25mg주 트립토헬렐린으로서 11.25mg을 3개월마다 1회 근육주사한다. **[사용상의주의사항]** 1. 경고: 전이성 척추 손상(metastatic vertebral lesion)이나 요로폐색증을 수반한 환자는 치료 초기 몇 주 동안 세심하게 관찰하여야 한다. 2. 다음 환자에는 투여하지 말 것: 1) 호르몬 비의존성 전립선암 환자, 2) 양쪽 고환 절제술을 받은 후, 이 약에 의해 더 이상 테스토스테론의 감소를 기대할 수 없는 환자, 3) 임상적으로 명백한 골다공증 또는 골밀도의 저하와 같은 골다공증의 위험이 있는 환자, 4) 임부 및 수유부, 5) GnRH, 이의 유사체 또는 이 약의 첨가제에 과민반응의 병력이 있는 환자, 6) 진단된 뇌하수체 샘종 환자 3. 다음 환자에는 신중히 투여할 것: 1)고혈압 환자(혈압강하제를 투여한 환자는 고혈압 치료의 조치가 필요할 수 있다.

* 제품에 대한 자세한 사항은 제품설명서를 참조하시거나 입센코리아로 문의 하십시오.

[제조원] 수입·판매: 입센코리아㈜/ 서울시 송파구 중민로 10 8층 S-07호. Tel)02-512-6693, **제조외곽자:** Ipsen Pharma/65, Quai Georges Gorse - 92100 Boulogne - Billancourt, 프랑스, **제조자:** Ipsen Pharma Biotech/ Parc d'Activites de Plateau de Signes, Chemin Departmental no. 402, 83870, Signes, 프랑스