Prostate gland disorder

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โรงพยาบาลต้าววาก
สำนักงานต้าววากแห่งชาติ
• Prostate cancer
  • Overview
  • Diagnosis
  • Treatment

• Lower urinary tract symptoms
EPIDEMIOLOGY

- The most common noncutaneous malignancy in U.S.
  - 27% of all such cancers
- Incidence varies by race/ethnicity
- Incidence rose dramatically from 1989 to 1992 after the introduction of a prostate-specific antigen (PSA) screening test
- The second most common cancer and the sixth leading cause of cancer deaths worldwide
- The forth most common cancer in Thai male
## EPIDEMIOLOGY

<table>
<thead>
<tr>
<th></th>
<th>INCIDENCE*</th>
<th>MORTALITY*</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>138.6</td>
<td>21.3</td>
</tr>
<tr>
<td>African-American</td>
<td>220</td>
<td>50.9</td>
</tr>
<tr>
<td>Hispanic/Latino</td>
<td>124.2</td>
<td>19.2</td>
</tr>
<tr>
<td>Asian-American and Pacific Islander</td>
<td>75</td>
<td>10.1</td>
</tr>
<tr>
<td>American Indian and Alaska Native</td>
<td>104.1</td>
<td>20.7</td>
</tr>
</tbody>
</table>

Age at Diagnosis

• Prostate cancer is rarely diagnosed in men less than 50 years of age
  • 2% of all cases (Jani et al, 2008)
  • 63% diagnosed after age 65 (Ries et al, 2008)

• Shift in diagnosis to an increasingly younger population after the introduction of PSA screening

• The average age of death from prostate cancer is 77 years
  • remained stable over the last three decades (Epstein et al, 2012)
Stage at Diagnosis

• Since the introduction of PSA testing
  • 81% of newly diagnosed men have localized disease
  • incidence of metastatic disease has decreased by 75% (Newcomer et al, 1997)
• The use of PSA screening has also resulted in...
  • Substantial downward pathologic stage migration
RISK FACTORS

• Considerable evidence suggests that both genetics and environment play a role in the origin and evolution of prostate cancer
## RISK FACTORS

<table>
<thead>
<tr>
<th>FAMILY HISTORY</th>
<th>RELATIVE RISK</th>
<th>95% CONFIDENCE INTERVAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>1</td>
<td>1.90-2.49</td>
</tr>
<tr>
<td>Father affected</td>
<td>2.17</td>
<td>1.90-2.49</td>
</tr>
<tr>
<td>Brother affected</td>
<td>3.37</td>
<td>2.97-3.83</td>
</tr>
<tr>
<td>First-degree family member affected, age &lt;65 yr at diagnosis</td>
<td>3.34</td>
<td>2.64-4.23</td>
</tr>
<tr>
<td>&gt;2 first-degree relatives affected</td>
<td>5.08</td>
<td>3.31-7.79</td>
</tr>
<tr>
<td>Second-degree relative affected</td>
<td>1.68</td>
<td>1.07-2.64</td>
</tr>
</tbody>
</table>
RISK FACTORS

• Infections cause about 16% of all cancers worldwide
• Hormone
  • Androgen, estrogen
• Insulin like growth factor
• vitaminD
Other influences

• Sexual Activity/Sexually Transmitted Infections
• Vasectomy
• Smoking
  • there is clear evidence that current smokers are at higher risk of biochemical recurrence, metastasis, and prostate cancer–specific mortality
• Diet
  • lower levels of serum cholesterol and use of cholesterol-lowering agents (statins) reduce the risk of aggressive and advanced-stage disease (Platz et al, 2006, 2009)
Other influences

• Obesity
  • associated with a lower risk of low-grade disease, but a greater risk of high-grade disease (Gong et al, 2006; Rodriguez et al, 2007; Wright et al, 2007)

• Alcohol consumption
Prostate Cancer Tumor Markers

• PSA is widely accepted as a prostate cancer tumor marker
  • it is organ specific and not cancer specific
  • There is significant overlap in serum PSA levels among men with cancer and men with benign disease

• At the present time, serum PSA levels of 2.6 ng/mL are used

• 40% of men presenting with an elevated PSA (serum PSA 4 to 10 ng/mL)
  • false-negative rate of 20%
Prostate Cancer Tumor Markers

• Currently FDA-approved biomarkers include
  • PSA, free-to-total PSA ratio (%fPSA), Prostate Health Index(PHI), and PCA3

• PSA (Prostate specific antigen)
  • primarily produced by prostatic luminal epithelial cell
  • expressed to a greater degree in noncancerous relative to cancerous prostate tissue
  • 70% to 80% of PSA in serum is protein-bound
  • Elevated serum PSA levels are probably a product of disruption of cellular architecture within the prostate gland
Prostate Cancer Tumor Markers

- In men 50 years old and older, ejaculation can lead to a transient increase in PSA.
  - return to baseline within 24 hours (Rajaei et al, 2013)
- Long distance (>55km) cycling is another potential cause of false PSA elevation
- Prostate cancer, BPH, and prostatitis are the most important factor affecting serum PSA
- The 5α-reductase inhibitors (5ARI) have been shown to lower PSA levels
  - roughly 50% after 12 months of treatment
Prostate Cancer Tumor Markers

• Free Prostate-Specific Antigen
  • The %fPSA is most useful in the setting of PSA levels less than 10 ng/mL
  • Numerous studies evaluated %fPSA cut points
  • Christensson and coworkers (1993) measured fPSA and tPSA fractions in men with and without prostate cancer
    • found that a fPSA/tPSA cutoff of 0.18 (18% fPSA)
    • sensitivity rates of 70% to 95%
  • %fPSA cutoff of 25% detected 95% of cancers (sensitivity), (Catalona et al, 1998; Gretzer et al, 2002)
  • Proposed cut points generally range from 15% to 25%
Prostate Cancer Tumor Markers

- Prostate-Specific Membrane Antigen
  - On going trial

- Human Kallikrein 2
  - may be used in conjunction with PSA to improve the selection of patients for prostate needle biopsy

- Other tumor marker ; on going trial
  - Kallikrein Tumor Markers, Endoglin, Circulating Tumor Cells
Prostate Cancer Tumor Markers

• URINE-BASED BIOMARKERS
  • Prostate Cancer Antigen 3 (FDA approved)
  • Gene fusion
  • Metabolomics
  • Annexin A3
  • MicroRNA
Prostate Cancer Tumor Markers

- Digital rectal exam
- Urine specimen
- PCA-3 and PSA mRNA concentrations measured in separate tubes

Quantitative ratio of PCA-3/PSA mRNA = PCA-3 score

- PCA-3 score < cutoff: Lower risk of positive biopsy
- PCA-3 score ≥ cutoff: Higher risk of positive biopsy
Key points of tumor markers

• Most prostate cancer arises as clinically nonpalpable (stage T1c) disease with PSA between 2.5 and 10 ng/mL
• PSA is organ specific and not disease specific
• Men with prostate cancer have lower percentage of free/total PSA compared with men without prostate cancer
• 5α-reductase inhibitors used for treatment of BPH have been shown to lower PSA levels
http://deb.uthscsa.edu/URORiskCalc/Pages/calcs.jsp

- University of Texas Health Science Center at San Antonio
Prostate Biopsy

• Transrectal ultrasonography of the prostate (TRUS) has become a standard tool in the practice of urology
• Commercially available endorectal probes are available
• Some newer biplane probes provide simultaneous sagittal and transverse imaging modes
Indications for Prostate Biopsy

• Data from the Prostate Cancer Prevention Trial demonstrated that
  • there is no safe PSA threshold that can rule out prostate cancer in any age
    range (Thompson et al, 2005)

• Most organizations have abandoned absolute PSA level cutoff values
  for prostate biopsy
Indications for Prostate Biopsy

- Men at elevated risk for having prostate cancer are... (Heidenreich et al, 2014)
  - older than 50 years of age
  - have a family history of prostate cancer and are older than 45 years
  - African-Americans
  - PSA level greater than 1 ng/mL at 40 years and greater than 2 ng/mL at 60 years
Indications for Prostate Biopsy

• Adjuncts to serum PSA testing have been advocated
  • free-to-total PSA ratio
  • PSA velocity
  • PSAD
  • but are **not** uniformly reliable
Indications for Prostate Biopsy

• The National Comprehensive Cancer Network (NCCN) (2012) advocates
  • positive digital rectal examination regardless of PSA level
  • PSA 4 to 10 ng/mL based on patient risk benefit
  • PSA level 2.5 ng/mL or less and PSA velocity 0.35 ng/mL or greater per year
  • PSA level 2.6 to 4.0 ng/mL
  • PSA level 4.0 ng/mL or greater, especially if the free PSA level is 10% or less
Contraindication to prostate biopsy

- Significant coagulopathy
- Severe immunosuppression
- Acute prostatitis
- Painful anorectal conditions
  - biopsy under general or regional anesthesia should be considered
Prostate Biopsy

• Preparing Patients for Biopsy

  • Anticoagulant therapy should be stopped 7 to 10 days before prostate biopsy
  • Prostatic biopsy should not be performed until the international normalized ratio has been corrected below 1.5
    • Bridging anticoagulation with unfractionated heparin or low-molecularweight heparin is suggested
  • Antibiotic prophylaxis
  • Cleansing enema
    • effect on reducing infections is debatable
Prostate Biopsy
Prostate Biopsy
Prostate Biopsy

• Today, MRI is often used to detect and guide biopsies of anterior tumors that may escape standard TRUS prostate biopsy (Volkin et al, 2014)

• Repeat and Saturation Prostate Biopsy
Prostate Biopsy

• Risks and Complications of Prostate Biopsy
  • The incidence of serious complications requiring hospitalization is relatively low (<1%)
  • The majority of hospital admissions (72%) were related to bacterial infections
  • Common complications include
    • hematospermia, hematuria, rectal bleeding, prostatitis, fever, epididymitis, urinary retention, and other complications requiring hospitalization
  • Risk factors for prostate biopsy–related infection include
    • Increased number of comorbidities, diabetes mellitus, prostate enlargement, and recent antibiotic use
Prostate Biopsy

• ADVANCED AND INVESTIGATIONAL TECHNIQUES FOR PROSTATE BIOPSY
  • Color and Power Doppler Transrectal Ultrasonography
  • Prostate HistoScanning
  • The TargetScan
  • TRUS/MRI fusion
Prostate Biopsy

• Key points for prostate biopsy
  • TRUS and/or MRI alone cannot diagnose prostate cancer without a tissue biopsy
  • Patients undergoing TRUS-guided prostate biopsy require oral antibiotic prophylaxis for up to 24 hours perioperatively
  • Systematic biopsy procedures should include a minimum 12 cores
  • Advanced US techniques can improve cancer detection but do not reliably identify all malignant foci
  • TRUS/MRI fusion is a promising technique that takes advantage of the strengths of each modality when performing prostate biopsy
Management of Localized Prostate Cancer

<table>
<thead>
<tr>
<th>RISK GROUP</th>
<th>CLINICAL STAGE</th>
<th>PSA (ng/mL)</th>
<th>GLEASON SCORE</th>
<th>BIOPSY CRITERIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>T1a or T1c</td>
<td>&lt;10</td>
<td>2-6</td>
<td>Unilateral or &lt;50% of core involved</td>
</tr>
<tr>
<td>Intermediate</td>
<td>T1b, T1c, or T2a</td>
<td>&lt;10</td>
<td>3 + 4 = 7</td>
<td>Bilateral</td>
</tr>
<tr>
<td>High</td>
<td>T1b, T1c, T2b, or T3</td>
<td>10-20</td>
<td>4 + 3 = 7</td>
<td>&gt;50% of core involved or perineural invasion or ductal differentiation</td>
</tr>
<tr>
<td>Very high</td>
<td>T4</td>
<td>&gt;20</td>
<td>8-10</td>
<td>Lymphovascular invasion or neuroendocrine differentiation</td>
</tr>
</tbody>
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Management of Localized Prostate Cancer

<table>
<thead>
<tr>
<th>RISK GROUP</th>
<th>LIFE EXPECTANCY (YEARS)</th>
<th>RECOMMENDED TREATMENT</th>
</tr>
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<tbody>
<tr>
<td>Low</td>
<td>0-5</td>
<td>AS, HT</td>
</tr>
<tr>
<td></td>
<td>5-10</td>
<td>AS, RT, HT, O</td>
</tr>
<tr>
<td></td>
<td>&gt;10</td>
<td>RP, RT, AS, O</td>
</tr>
<tr>
<td>Intermediate*</td>
<td>0-5</td>
<td>AS, HT, RT, O</td>
</tr>
<tr>
<td></td>
<td>5-10</td>
<td>RT, HT, RP, O</td>
</tr>
<tr>
<td></td>
<td>&gt;10</td>
<td>RP, RT, O, HT</td>
</tr>
<tr>
<td>High*</td>
<td>0-5</td>
<td>AS, RT + HT, O</td>
</tr>
<tr>
<td></td>
<td>5-10</td>
<td>RT + HT, HT, RP, O</td>
</tr>
<tr>
<td></td>
<td>&gt;10</td>
<td>RT + HT, RP + RT + HT, HT</td>
</tr>
<tr>
<td>Very high*</td>
<td>0-5</td>
<td>AS, RT + HT, O</td>
</tr>
<tr>
<td></td>
<td>5-10</td>
<td>H, RT + HT, ST</td>
</tr>
<tr>
<td></td>
<td>&gt;10</td>
<td>RT + HT, RP + RT + HT, HT, ST, IT</td>
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### Active Surveillance of Prostate Cancer

<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>PSA ≤10 ng/mL</td>
<td>PSA ≤10 ng/mL</td>
<td>PSA ≤10 ng/mL</td>
<td>PSA ≤10 ng/mL</td>
<td>PSA ≤10 ng/mL</td>
<td>PSA ≤10 ng/mL</td>
<td>PSA ≤15 ng/mL</td>
<td>PSA ≤10 ng/mL</td>
</tr>
<tr>
<td>PSA density ≤0.15 ng/mL per gram</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage ≤T2a</td>
<td></td>
<td>Stage ≤T2a</td>
<td>Stage ≤T2a</td>
<td>Stage ≤T2a</td>
<td>Stage ≤T2a</td>
<td>Stage ≤T2a</td>
<td>Stage ≤T2a</td>
</tr>
<tr>
<td>Grade ≤3+3</td>
<td></td>
<td>Grade ≤3+3</td>
<td>Grade ≤3+3</td>
<td>Grade ≤3+3</td>
<td>Grade ≤3+3</td>
<td>Grade ≤3+4</td>
<td>Grade ≤3+3</td>
</tr>
<tr>
<td>No. cores positive ≤2</td>
<td></td>
<td>% Cores positive ≤1/3</td>
<td>% Cores positive ≤2</td>
<td>% Cores positive ≤2</td>
<td>% Cores positive ≤50</td>
<td>% Cores positive ≤50</td>
<td>% Cores positive ≤3</td>
</tr>
<tr>
<td>Single-core positivity ≤50%</td>
<td></td>
<td>Single-core positivity ≤50%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median follow-up</td>
<td>2.7 years</td>
<td>4.5 years</td>
<td>1.6 years</td>
<td>6.8 years</td>
<td>1.8 years</td>
<td>5.7 years</td>
<td>2.1 years</td>
</tr>
<tr>
<td>% free of treatment</td>
<td>54.4%</td>
<td>70%</td>
<td>75.6%</td>
<td>70%</td>
<td>95%</td>
<td>69%</td>
<td>80%</td>
</tr>
<tr>
<td>Disease-specific survival</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>99%</td>
<td>100%</td>
<td>99%</td>
<td>Not recorded</td>
</tr>
<tr>
<td>Overall survival</td>
<td>98%</td>
<td>97%</td>
<td>99.3%</td>
<td>78.4%</td>
<td>98%</td>
<td>94%</td>
<td>Not recorded</td>
</tr>
</tbody>
</table>
## Treatment of Locally Advanced Prostate Cancer

<table>
<thead>
<tr>
<th>AUTHOR (yr)</th>
<th>NO. PATIENTS</th>
<th>ADJUVANT TREATMENT</th>
<th>OVERALL SURVIVAL</th>
<th>CANCER-SPECIFIC SURVIVAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morgan et al (1991)</td>
<td>232</td>
<td>54%</td>
<td>85%</td>
<td>89%</td>
</tr>
<tr>
<td>van den Ouden et al (1994)</td>
<td>59</td>
<td>—</td>
<td>83%</td>
<td>90%</td>
</tr>
<tr>
<td>Lerner et al (1995)</td>
<td>812</td>
<td>60%</td>
<td>86%</td>
<td>90%</td>
</tr>
<tr>
<td>Gerber et al (1997)</td>
<td>242</td>
<td>NR</td>
<td>—</td>
<td>90%</td>
</tr>
<tr>
<td>van den Ouden et al (1998)</td>
<td>83</td>
<td>0%</td>
<td>75%</td>
<td>88%</td>
</tr>
<tr>
<td>Pound et al (1999)*</td>
<td>55</td>
<td>0%</td>
<td>—</td>
<td>85%</td>
</tr>
<tr>
<td>Ward et al (2005)</td>
<td>842</td>
<td>62%</td>
<td>90%</td>
<td>60%†</td>
</tr>
<tr>
<td>Carver et al (2006)</td>
<td>176</td>
<td>36%†</td>
<td>88%</td>
<td>95%</td>
</tr>
<tr>
<td>Loeb et al (2007)</td>
<td>288</td>
<td>15%</td>
<td>91%§</td>
<td>94%</td>
</tr>
<tr>
<td>Freedland et al (2007)</td>
<td>56</td>
<td>0%</td>
<td>—</td>
<td>92%§</td>
</tr>
<tr>
<td>Xylinas et al (2009)</td>
<td>100</td>
<td>25%</td>
<td>85%</td>
<td>98%</td>
</tr>
</tbody>
</table>

Note: ¥ = 5 yr; † = 10 yr; ‡ = 15 yr; § = 20 yr; 95% (79%) = 95% at 15 yr, 79% at 20 yr
Metastasis prostate cancer

Overall survival in patients undergoing intermittent androgen deprivation therapy compared to those undergoing continuous androgen deprivation therapy

Benign prostatic hyperplasia

- Lower urinary tract symptoms (LUTS)
- Previously held notions that the clinical symptoms of male LUTS
  - a mass-related increase in urethral resistance are too simplistic
- It is now clear that a significant portion of male LUTS is due to age-related detrusor dysfunction and other conditions
Benign prostatic hyperplasia

• The transition zone also enlarges with age, unrelated to the development of nodules

• One of the unique features of the human prostate
  • the prostatic capsule, which plays an important role in the development of LUTS
  • Presumably the capsule transmits the “pressure”
Benign prostatic hyperplasia

- Importance of Prostatic Smooth Muscle
  - both passive and active forces in prostatic tissue play a major role in the pathophysiology of BPH
  - Blockade of this stimulation clearly diminishes this response
  - Active smooth muscle tone in the human prostate is regulated by the adrenergic nervous system
Benign prostatic hyperplasia

- The Bladder’s Response to Obstruction
  - It is also clear that LUTS in men with BPH or prostate enlargement are related to obstruction-induced changes in bladder function
  - Detrusor instability or decreased compliance
    - Frequency and urgency
  - Changes associated with decreased detrusor contractility
    - force of the urinary stream, hesitancy, intermittency, increased residual urine, and (in a minority of cases) detrusor failure
Evaluation and Nonsurgical Management of Benign Prostatic Hyperplasia

• Diagnosis
  • The complex of symptoms (LUTS) is not specific for BPH
  • LUTS is not sex, age, or disease specific

• Initial Evaluation
  • Medical history
  • Physical examination
    • The size of the prostate is not critical in deciding whether active treatment is required.
    • Size does not correlate precisely with symptom, severity, degree of urodynamic obstruction, or treatment outcomes
Evaluation and Nonsurgical Management of Benign Prostatic Hyperplasia

- **Urinalysis**
  - assists in distinguishing UTIs and bladder cancer from benign prostate disease
- **Serum Creatinine Measurement**
  - The AUA guidelines on BPH no longer recommend routine creatinine measurement
- **Serum PSA**

- **Symptom Assessment**
  - The identical International Prostate Symptom Score (IPSS) is recommended as the symptom scoring instrument
    - symptoms can be classified as mild (0 to 7), moderate (8 to 19), or severe (20 to 35)
    - used for the baseline assessment of symptom severity in men with LUTS
<table>
<thead>
<tr>
<th></th>
<th>not at all</th>
<th>less than 1 time in 5</th>
<th>less than half the time</th>
<th>about half the time</th>
<th>more than half the time</th>
<th>almost always</th>
</tr>
</thead>
</table>
| 1. Incomplete Emptying  
Over the last month, how often have you had a sensation of not emptying your bladder completely after you finish urinating? | 0 | 1 | 2 | 3 | 4 | 5 |
| 2. Frequency  
During the last month, how often have you had to urinate again less than two hours after you finished urinating | 0 | 1 | 2 | 3 | 4 | 5 |
| 3. Intermittency  
During the last month, how often have you stopped and started again several times when you urinate? | 0 | 1 | 2 | 3 | 4 | 5 |
| 4. Urgency  
During the last month, how often have you found it difficult to postpone urination? | 0 | 1 | 2 | 3 | 4 | 5 |
| 5. Weak Stream  
During the last month, how often have you had a weak urinary stream? | 0 | 1 | 2 | 3 | 4 | 5 |
| 6. Straining  
During the last month, how often have you had to push or strain to begin urination | 0 | 1 | 2 | 3 | 4 | 5 |
| 7. Nocturia  
During the last month, how many times did you most typically get up to urinate from the time you went to bed until the time you got up in the morning? | 0 | 1 | 2 | 3 | 4 | 5 |
BASIC MANAGEMENT OF LUTS IN MEN

LUTS cause little or no bother
- Reassurance and follow-up

Recommended tests:
- Relevant medical history
- Assessment of LUTS symptom severity and bother
- Physical examination including DRE
- Urinalysis
- Serum PSA
- Frequency-volume chart

Complicated LUTS:
- Suspicious DRE
- Hematuria
- Abnormal PSA
- Pain
- Infection
- Palpable bladder
- Neurologic disease

Predominant significant nocturia
- Frequency-volume chart
- No polyuria

Bothersome LUTS
- Polyuria

Polyuria
- 24-hour output ≥3 L
- Lifestyle and fluid intake is to be reduced
- Nocturnal polyuria ≥33% output at night
- Fluid intake to be reduced
- Consider desmopressin

Polyuria
- Failure
- Continue treatment

Standard treatment
- Alter modifiable factors
- Drugs
- Fluid and food intake
- Lifestyle advice
- Bladder training
- Drug treatment

SUCCESS
- Continue treatment

Specialized management

Failure
- Success in relieving bothersome LUTS:
- Continue treatment

1 When life expectancy is >10 years and if the diagnosis of prostate cancer can modify the management.
2 When significant nocturia is a predominant symptom.
3 Assess and start treatment before referral.
4 In practice, advise patients with symptoms to aim for a urine output of about 1 L/24 hr.
Initial evaluation
- History
- DRE and focused PE
- Urinalysis
- PSA in select patients

AUA/IPSS symptom index
Assessment of patient bother

Mild symptoms (AUA/IPSS 7) or no bothersome symptoms

Moderate/severe symptoms (AUA/IPSS 8)

Optional diagnostic tests
- Uroflow
- PVR

Discussion of treatment options

Patient chooses noninvasive therapy

Watchful waiting

Medical therapy

Patient chooses invasive therapy

Optimal diagnostic tests
- Pressure flow
- Urethrocystoscopy
- Prostate ultrasound

Minimally invasive therapies

Surgery
MEDICAL THERAPY FOR LOWER URINARY TRACT SYMPTOMS AND BENIGN PROSTATIC HYPERPLASIA
MEDICAL THERAPY FOR LOWER URINARY TRACT SYMPTOMS AND BENIGN PROSTATIC HYPERPLASIA

• α-Adrenergic Blockade
  • Intraoperative floppy iris syndrome (IFIS) complicates approximately 2% of cataract surgery cases

• ANDROGEN MANIPULATION
  • Finasteride, Dutasteride

• COMBINATION THERAPY WITH α-BLOCKER AND 5α-REDUCTASE INHIBITOR
  • Prostate volume 30ml or greater
MEDICAL THERAPY FOR LOWER URINARY TRACT SYMPTOMS AND BENIGN PROSTATIC HYPERPLASIA

• ANTICHOLINERGIC (ANTIMUSCARINIC) RECEPTOR BLOCKERS
  • Combination Therapy: α-Adrenergic Blockers and Anticholinergic (Antimuscarinic) Receptor Blockers

• β3 Agonist (Mirabegron)

• PHOSPHODIESTERASE INHIBITORS
  • Further data on safety and cost-effectiveness, especially for combination therapy, are needed
MEDICAL THERAPY FOR LOWER URINARY TRACT SYMPTOMS AND BENIGN PROSTATIC HYPERPLASIA

• PHYTOTHERAPY
  • Serenoa repens (Saw Palmetto Berry), Pygeum africanum (African Plum), Hypoxis rooperi (South African Star Grass)
  • Appropriate randomized placebo controlled clinical trials monitored by an outside agency are needed to ascertain and to confirm the efficacy of these products
  • β-sitosterol
KEY POINTS

• BPH is the commonest cause of LUTS in men beyond middle age
• Evaluation requires a history and symptom score (IPSS) and a careful physical examination including a DRE
• Either an α-blocker or (if the prostate is large) a 5α-reductase inhibitor is now the usual first-line management of uncomplicated LUTS
• Antimuscarinic agents and PDEIs are useful
Minimally Invasive and Endoscopic Management of Benign Prostatic Hyperplasia

• Indications for Treatment
  • Recurrent gross hematuria
  • Bladder calculi, Bladder diverticular
  • Recurrent urinary tract infection
  • Bilateral hydronephrosis with renal function impairment
  • Refractory urinary retention
Minimally Invasive and Endoscopic Management of Benign Prostatic Hyperplasia

Transurethral Resection of the Prostate (TURP)
Minimally Invasive and Endoscopic Management of Benign Prostatic Hyperplasia

• Laser option
  • Holmium
  • KTP
  • Thulium
Minimally Invasive and Endoscopic Management of Benign Prostatic Hyperplasia

- Complications (TUR-P)
  - Urethral stricture occur about 4%
  - Bladder neck contracture occur about 2%
  - Incontinence 0.6 – 5%
  - Significant hemorrhage
    - Delayed bleeding around 1 to 4 weeks postoperatively.
  - Postoperative urinary retention after any BOO procedure
    - incidence ranges from 6.5% to 7.1%
    - May require re-treatment
Minimally Invasive and Endoscopic Management of Benign Prostatic Hyperplasia

- Urinary storage symptoms
- Ejaculatory problems
What is the prostate function?
Thank you for your attention