Geriatric UI: A Case-Based Approach

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UI: The Problem

Prevalence in elderly ≥ 33%
Morbidity substantial
Costs >$76 billion annually\(^1\)
Yet ignored, although ↑ treatable

\(^1\) Koyne KS, et al. *J Manag Care Pharm.* 2014; 20: 130-40

Disclosures

• None

What’s Wrong with This Picture?

<table>
<thead>
<tr>
<th>Cause</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ Detrusor (DO)</td>
<td>Bladder relaxant</td>
</tr>
<tr>
<td>↓ Detrusor (DU)</td>
<td>Bethanechol</td>
</tr>
<tr>
<td>↓ Outlet (SI)</td>
<td>α adrenergic; BNS</td>
</tr>
<tr>
<td>↑ Outlet (obstruction)</td>
<td>x'azosin</td>
</tr>
</tbody>
</table>

It Doesn’t Account for the Changes of Normal Aging

LUT in *Continent* Elderly

<table>
<thead>
<tr>
<th>Condition</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Detrusor Overactivity</td>
<td>48</td>
</tr>
<tr>
<td>Obstruction (men)</td>
<td>53</td>
</tr>
<tr>
<td>Underactive Detrusor</td>
<td>13</td>
</tr>
<tr>
<td>Normal</td>
<td>18</td>
</tr>
</tbody>
</table>

Resnick, AJP 1995
Thus

Geriatric continence results **not** from **normal** lower urinary tract (LUT) function but **despite abnormal** LUT function

<table>
<thead>
<tr>
<th>Disease Severity</th>
<th>Compensatory Mechanisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic</td>
<td>Asymptomatic</td>
</tr>
</tbody>
</table>

Resnick, JAMA 1996

Rationale for a Different Paradigm

**Young**
- **Single** cause: LUT
- Invasive testing to determine diagnosis
- Rx LUT “diagnosis”

**Elderly**
- **Multiple** causes: LUT, drugs, diz, and ↓ function
- Clinical eval’n first step
- Rx all “contributors”
- If fails, consider testing

LUT Abnormality vs. UI

<table>
<thead>
<tr>
<th># Leaks/Day</th>
<th>Young</th>
<th>Old</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drugs</td>
<td>DO</td>
<td>DO</td>
</tr>
<tr>
<td>Diabetes, Ca^2+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHF/Edema</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mobility</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impaction</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Resnick, Lancet 1997

Case 1

89 yo F with cough-associated leakage.
- No precipitancy
- PMH: L MCA stroke, RA, UGI bleed, HTN
- Meds: ACEI, HCTZ, atenolol, Benadryl, trazodone, oxycodone, iron, enema
- Exam: ↓ sphincter; impxn; +ST, R h’paresis
- Void: 450 ml; PVR = 150 ml

<table>
<thead>
<tr>
<th>Time</th>
<th>Wet/Dry</th>
<th>Void</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>08:00</td>
<td>D</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>10:00</td>
<td>W</td>
<td>400</td>
<td>Cough → leak</td>
</tr>
<tr>
<td>12:00</td>
<td>D</td>
<td>125</td>
<td></td>
</tr>
<tr>
<td>14:00</td>
<td>D</td>
<td>40</td>
<td>Cough; no leak</td>
</tr>
</tbody>
</table>

Voiding Diary

Resnick, Lancet 1997
Case 1: Rx

- Despite stroke, not urge UI but stress UI
- Rx: \( \alpha \) adrenergic, BNS? Or…
- Stress UI at high bladder volumes often responds to keeping bladder less full
- Rx strategy (\( \downarrow \) load on sphincter):
  - \( \downarrow \) BV: disimpact; d/c Benadryl, \( \downarrow \) oxycodone;
    Rx RA/add PT to \( \uparrow \) toileting, activity & sleep
  - Stop cough: Discontinue ACEI \( \rightarrow \) continent
- Did not fix sphincter, but \( \downarrow \) challenge
- Continence achieved without UDS

Case 2

83 yo woman with nocturia and urge UI

PMH: Dementia, HTN, arthritis, falls, frx
Meds: doxazosin, CCB, NSAID, imipramine

PE: Orthostasis, edema, antalgic gait,
    - ST, void 80 ml @ 5 ml/s; PVR=10
VD: Voids = 150 ml (day), 250 ml (night)
Output = 600 ml/day, 1200 ml/night

Case 2: Rx

- Likely DO, worse b/c \( \downarrow \) compensatory mech:
  - \( \downarrow \) cognition, \( \downarrow \) mobility, and nocturia. Rx all!
- Treatment plan:
  - Cognition: Stop imipramine (anticholinergic)
  - Mobility: Eliminate meds \( \rightarrow \) orthostasis, edema
    (doxazosin, imipramine, CCB); add PT, etc.
  - Rx nocturia, if still present \textit{and} necessary
- Dry!

Case 4

75 yo man with urge UI; no UDS-BOO

Voiding diary reveals:
  - Voids every 1 - 2.5 hr
  - Largest void = 300 ml
  - PVR = 150 ml
  - 24\(^\circ\) volume = ~3000 ml
Dx: DHIC, normal TBV, \( \uparrow \) output
Rx: \( \downarrow \) fluid intake \( \rightarrow \) asymptomatic w/o Rx!
Detrusor Hyperactivity with Impaired Contractility (DHIC)

- Bladder is “spastic” but weak
- Most common cause of geriatric UI
- Implications:
  - Predisposes to urinary retention
  - Mimics stress incontinence/obstruction
  - Implications for treatment
  - Implications about the cause?

Resnick & Yalla, JAMA 1987

Case 5

67 yo woman with UI, only when tired.
Occurs with activity. No precipitancy.

PMH: Viral T2-4 tr'verse myelitis w/AUR

O/E: PFM lax. Neuro ± normal. Mildly +ST

VD: 6-7 voids/day, 0/noc, mild leak w/ex

Flow: Weak, but continuous and ↑ w/strain

Void: Voided 300 ml; PVR = 50 ml

Case 5: Rx

- Transverse myelitis ↑ risk DO and DSD but no evidence of either:
  - DO: 6-7 voids/24h, no precipitancy, N TBV, sleeps 7 hrs without voiding or leaking despite output >500 ml/night, and no gush even when PFM weak enough to allow SI.
  - DSD: no DO sx, normal proprioception, stream is continuous and ↑ with strain.
- Dx = SI due to residual neuropathy
- Pessary restored continence; no UDS

Case 6

80 yo F with freq; no urgency; occ UI at night

Voids q 2-3 hrs (day), 1-1.5 hrs (night)

No other symptoms. Bladder relaxant failed

PMH: Mild dementia

Meds: amlodipine

VD: Voids 60-120 ml day and night
Vol = 700 ml (day), 840 ml (night)

Case 6: Exam

Overweight, walks slowly with a cane
Mild dementia
No CHF or edema
Normal sacral sensation, tone, reflexes
Stocking neuropathy
Stress Test negative @ 70 ml; PVR=8 ml

Case 6: Discussion

Frequency likely not due to DO:
- No urgency or day UI, despite ↓mobility
Though neuropathy, not a factor:
- Reliable urge at <100 ml
- Small bladder capacity and ↑ residual
Exacerbated by CCB (via nocturia)?
Functional bladder contraction d/t habit
**Case 6: Rx**

Progressively ↑ voided vol to 250 ml

Treated nocturia
- Substituted a thiazide
- No furosemide (no edema/CHF)
- No DDAVP

Continent and normal voiding intervals

**Problems with “Functional UI”**

In functionally-impaired patients:
- UI not inevitable even if ↓mentat’n/mobility
- UI without LUT dysfunction is rare
- Obstrxn/SI common, potentially treatable
- Factors causing transient UI more likely

**Case 7**

90 yo M with Parkinsons developed urosepsis complicated by MI. Several days later, agitated, confused, UI.

PMH: CHF, moderate cognitive impairment

Meds: haloperidol, furosemide, antibx

**Case 7**

O/E: Delirious, Parkinsonian, CHF, bladder distention, and impaction

Labs: Unremarkable

ECG: Unchanged, CXR: CHF

PVR: 800 ml

Likely causes of his UI? Eval’n?

**Case 7**

- Catheter placed; haloperidol weaned
- Sinemet restarted
- Disimpacted, CHF treated
- Discharged home
- UI much improved
- Returned to the office in 2 weeks

**Case 7**

Voiding diary
- 1 leak/day, including 4-hr nap in WC
- UI q1-2 hourly at night, despite ↓fluids

Why still UI?
Why when asleep at night but not when asleep in the afternoon?
Case 7

- Recurrent CHF diuresed
- Became completely continent
- PVR = 30 ml

What further testing required?
Does he need UDS?

The End