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Review Article

Beyond the Mortality Paradox: Redesigning UTI Care for People Living with Dementia through Diagnostic Stewardship

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Abstract: Managing urinary tract infections (UTI) in people living with dementia presents a mortality paradox: clinicians must balance the risks of antibiotic overuse against the dangers of delayed treatment in high-risk phenotypes. Standard diagnostic criteria often rely on classic, localizing markers like fever and dysuria. However, these gatekeeper signs are frequently absent or blunted in dementia care due to age-associated immune dysregulation and immunosenescence. Consequently, infections often manifest as nonspecific neurobehavioral changes, leading to predictable misclassification and harmful diagnostic delays. This paper reframes UTI management in dementia as a clinical pathway and measurement architecture problem. We propose a verification-first service pathway that replaces symptom-first heuristics with objective diagnostic certainty and closed-loop follow-up. The model incorporates physiologic stability triage, risk stratification for vulnerable phenotypes, and a standardized confirmation bundle. To ensure operational reliability, the pathway is linked to four auditable key performance indicators focused on verification completion, avoidable antibiotic starts, time-to-action, and stewardship follow-up. By treating diagnostic infrastructure as a system property, this approach aims to reduce "sticky empiricism" and improve safety in the face of clinical ambiguity.

Keywords: Dementia, Urinary Tract Infection, Diagnostic Stewardship, Immunosenescence, Delirium.

INTRODUCTION

Urinary tract infections (UTI) are a major healthcare consideration [1-7]. And UTIs are even more concerning among elderly patients living with dementia [8, 9]. Managing UTIs in people living with dementia is a high-stakes clinical problem because uncertainty cuts both ways: it prevents antibiotic overuse, yet it can also delay treatment when infection is real. Clinicians therefore operate within a *mortality paradox*, where treating low-certainty cases increases avoidable antimicrobial exposure and adverse events, while delaying treatment in high-risk phenotypes increases the likelihood of sepsis and death [10]. This paradox is sustained by operational policies and guideline-derived workflows that assume infection will announce itself through classic, localizing markers. Standard definitions such as the McGeer criteria [11], and Loeb criteria [12]. foreground dysuria, urgency, and fever as prerequisites for diagnosis and treatment initiation. Yet in older adults, and especially in dementia care contexts, those gatekeeper signs are frequently absent, delayed, or poorly reported, creating predictable misclassification: antibiotics started under low certainty in stable episodes, or harmful delay when infection is present.

The mismatch is not only behavioral; it is biological. Age-associated immune dysregulation can blunt the physiologic signals that conventional UTI pathways rely on. A blunted febrile response in older adults has been attributed to reduced pyrogen sensitivity and impaired thermoregulation, with a meaningful proportion of infections presenting without fever [13]. When fever is operationalized as a hard prerequisite, genuinely infected patients can be misclassified as asymptomatic until physiologic instability develops, at which point the cost of delay is materially higher. More broadly, immunosenescence and inflammaging reduce physiologic reserve and amplify systemic inflammatory responses, increasing the likelihood that infection manifests as delirium, malaise, or functional decline rather than local urinary symptoms [14, 15]. In dementia, this becomes a practical signal problem: peripheral infection can translate into neuropsychiatric

deterioration rather than focal genitourinary complaints, complicating bedside interpretation and increasing the probability of both overtreatment and delay [16].

Although multiple lenses explain UTI mismanagement in dementia (impaired symptom reporting, guideline non-adherence, and organizational pressures), they converge on a single operational requirement: health systems must build diagnostic certainty and follow-up reliability into service delivery rather than relying on symptom-first heuristics. When nonspecific signals coexist with high baseline bacteriuria, uncertainty becomes structurally predictable; the design question is not whether ambiguity will occur, but how the system will respond to it.

Accordingly, this paper reframes UTI management in dementia as a clinical pathway plus a measurement architecture problem. We propose a verification-first service pathway that differentiates unstable presentations requiring immediate treatment from stable presentations where confirmation and structured reassessment should govern antibiotic decisions. The pathway incorporates risk stratification to identify phenotypes with a higher penalty for delay (for example, dementia plus diabetes), stewardship rules that vary by diagnostic certainty, and closed-loop follow-up across transitions of care. To keep the model operational rather than conceptual, we translate each pathway link (certainty, timing, follow-up) into auditable key performance indicators (KPIs), aligning the approach with the premise that improvement must be measurable at the point of delivery [17].

Why Symptom-First UTI Criteria Underperform in Dementia Care

Symptom-first UTI criteria underperform in dementia care because they assume the host can reliably generate, perceive, and report localizing infection signals. In practice, widely used minimum-criteria frameworks prioritize dysuria, urgency, and fever as prerequisites for labeling infection and initiating treatment [11, 12]. In dementia contexts, those prerequisites collide with two population-level realities: (a) altered host physiology dampening classic inflammatory markers and (b) altered symptom expression and reporting shifting infection presentation toward nonspecific neurobehavioral change. The result is a predictable error pattern: false negatives early in true infection (delay until instability) and false positives when nonspecific deterioration is reflexively labeled as UTI (overtreatment).

Physiologic signal attenuation is the first mechanism. Fever is often treated as a gatekeeper sign, yet older adults frequently show a blunted febrile response due to reduced pyrogen sensitivity and impaired thermoregulation [13]. When fever is made a required trigger, the absence of fever becomes an erroneous reassurance signal and disadvantages the most vulnerable patients: infection may be dismissed until hemodynamic instability emerges. The same logic applies to other “classic” symptoms. The physiologic capacity to generate focal discomfort or the cognitive capacity to interpret and communicate it may be compromised, making the absence of dysuria or urgency weak negative evidence in dementia care.

Centralization of the clinical signal is the second mechanism. Atypical presentations in dementia are often downstream of immunosenescence and inflammaging, which reduce physiologic reserve and amplify systemic inflammatory responses [15]. Infection-driven inflammation may manifest as delirium, agitation, or functional decline rather than urinary complaints. This pattern aligns with neuroinflammation accounts of delirium and dementia progression, where systemic inflammation can accelerate vulnerability and symptom expression [14]. Operationally, the dominant signal at the point of care is often mental status change rather than a urinary symptom cluster. When symptom-first criteria are applied rigidly, clinicians face a harmful binary: dismiss the episode as non-UTI because urinary symptoms are absent, or over-attribute the nonspecific change to UTI because UTI becomes the default explanation for delirium in older adults.

Workflow dependence on unreliable follow-up is the third mechanism. When urinalysis or culture collection is delayed or logistically difficult, empiric prescribing becomes the path of least resistance, particularly when clinicians anticipate limited capacity to reassess after results return (). In nursing home workflows, antibiotics are often initiated primarily on nonspecific presentations (including mental status change), with low concordance to minimum-criteria frameworks and low rates of discontinuation once therapy starts [18]. This is a system property: the organization rewards immediate action more than delayed confirmation under staffing and time pressure, and it often lacks accountable ownership for culture review, de-escalation, and stop decisions across transitions.

Finally, symptom-first criteria underperform because documentation and care context vary. Dementia-oriented care frameworks emphasize the role of care-context variability (community vs institutional care), protocol gaps, and caregiver involvement in downstream utilization and risk [11]. When documentation is incomplete, the system cannot reliably distinguish “low-certainty empiricism” from “high-risk justified urgency,” and improvement efforts drift toward education campaigns that do not repair structural conditions producing the errors.

Accordingly, symptom-first checklists are not merely imperfect; they are systematically misaligned with dementia care. The core failure is not that criteria exist, but that they are treated as sufficient decision rules

in a population where biological signal generation, symptom reporting, and follow-up reliability are compromised. This motivates a pathway shift: stable patients should be governed by verification and structured reassessment, while unstable patients require time-critical treatment in parallel with confirmatory testing.

A Verification-First Clinical Pathway for Suspected UTI in Dementia

A verification-first pathway is grounded in a service-delivery premise: early dementia-context presentations often contain high noise and low specificity, so the health system must treat diagnostic certainty and closed-loop follow-up as infrastructure, not optional clinician effort. Instead of allowing nonspecific change to trigger antibiotics by default, the pathway begins with (1) physiologic stability triage, (2) risk stratification for delay harm, and (3) role-assigned, time-bounded verification and follow-up.

Step 1: Triage by Physiologic Stability

The first fork separates unstable from stable presentations because the penalty for delay is asymmetric. Unstable presentations (for example, clear systemic deterioration) should trigger sepsis-oriented management while diagnostic work proceeds in parallel. Empiric antibiotics may be appropriate, but they are framed as time-critical stabilization rather than an implicit diagnosis of UTI. Stable presentations are managed differently: the default action is verification and structured reassessment, not antibiotics. This prevents "antibiotics as reassurance" from becoming the system's automatic response when nonspecific deterioration is present and classic urinary markers are absent.

Step 2: Risk Stratification for Delay Harm

Within stable presentations, the pathway applies a high-penalty phenotype screen to identify patients for whom delayed recognition is more dangerous (for example, dementia with diabetes and related vulnerability features). This screen does not convert nonspecific symptoms into a diagnosis; it modifies tempo: faster sampling, tighter reassessment windows, and lower tolerance for "wait-and-see" without objective data. Risk stratification clarifies what the pathway is preventing in each subgroup: overtreatment harms in lower-penalty cases and delay harms in higher-penalty cases. This structure directly targets the mortality paradox by reducing low-certainty prescribing while tightening timelines for vulnerable phenotypes.

Step 3: Confirmation Bundle and Certainty Thresholds

For stable patients, verification-first care requires a confirmation bundle that is fast, standardized, and auditable, because ambiguity grows when sample collection is delayed or inconsistent. At minimum:

1. Obtain objective testing early (urinalysis as a screen and culture as the confirmation anchor).
2. Assess competing causes of delirium or functional decline in parallel, rather than treating UTI as the default explanation for neurobehavioral change, consistent with the neuroinflammation account of systemic-to-central symptom translation [14].
3. Define a time-bounded reassessment point (for example, 24–72 hours depending on phenotype and turnaround), at which antibiotics are started, withheld, or stopped based on objective evidence and trajectory.

Where available, adjunctive biomarkers can function as certainty enhancers, but they should not replace confirmation, particularly where stewardship and stop decisions depend on organism-level evidence.

Step 4: Antibiotic Initiation Rules Depend on Certainty, Not Anxiety

Verification-first pathways reduce unnecessary antibiotic starts by tying initiation to explicit certainty thresholds and stability status rather than nonspecific triggers. Operationally, the pathway uses three prescribing stances:

1. Immediate empiric therapy for unstable presentations (parallel confirmation; reassess when objective data returns).
2. Deferred therapy pending verification for stable presentations (especially when objective evidence is absent and the signal is nonspecific).
3. Targeted therapy with de-escalation when objective evidence supports infection, with stewardship actions triggered by culture review and clinical response.

This preserves clinical discretion while making decisions legible and auditable: antibiotics are started because pathway conditions were met, not because uncertainty was uncomfortable.

Step 5: Closed-Loop Culture Review and Transition-of-Care Reliability

In dementia care, the largest operational leak is often not diagnosis but failure to complete the information loop once results return. Without accountable ownership for culture review and stop/de-escalate decisions, empiric therapy becomes sticky and stewardship becomes aspirational. The pathway therefore treats follow-up as a reliability requirement:

1. Role assignment: designate an accountable reviewer (RN, pharmacist, or covering clinician).
2. Time-bound action: culture review within a defined interval (commonly 48–72 hours) with explicit outcomes (continue, narrow, switch, stop).
3. Transition reliability: when patients move across settings, handoffs must include pending cultures and ownership of follow-up.

This directly targets the real-world constraint identified in long-term care: when follow-up is uncertain, clinicians anticipate uncertainty and prescribe "just in case" [19].

Step 6: Documentation as an Auditable Interface

Because improvement depends on measurement, verification-first pathways require minimal documentation elements that distinguish justified urgency from avoidable empiricism: stability classification, risk phenotype, objective testing and timing, rationale for antibiotics (if started), and culture review outcome. This documentation discipline enables measurement and aligns clinical operations with value-based accountability expectations that care quality must be demonstrable and trackable [17].

Measurement Architecture: Auditable KPIs for Verification, Timing, and Follow-Up

A verification-first pathway becomes a health services delivery intervention only when it can be audited as reliably as it can be described. In dementia-context UTI care, measurement should capture three aims: verification (did the system build diagnostic certainty), timing (did it accelerate action when risk and evidence warranted), and follow-up reliability (did it close the loop rather than letting empiric therapy persist). The KPI set is intentionally limited and high-signal so it can be computed from routine EHR/lab/pharmacy fields and interpreted by clinicians and administrators.

Episode-of-Care Definition

KPIs should be computed at the level of an episode of suspected UTI rather than a single lab result or antibiotic order because failure modes unfold over time. A workable episode definition includes:

- t0: first documentation of suspected UTI or a defined trigger (e.g., delirium/functional decline prompting evaluation)
- tV: verification time (UA and/or culture collected; culture anchors when obtained)
- tA: antibiotic start time
- tR: culture final (or UA result time if culture not ordered)
- tC: closure time (documented reassessment decision to continue, narrow, change, stop)

Minimum Dataset

At minimum, each episode should capture stability classification, risk phenotype flag, verification actions with timestamps, antibiotic decision with timestamp, and follow-up action with timestamp. The goal is not a burdensome model; it is to make the pathway auditable and improvable.

Four Core KPIs

1. Verification completion rate: proportion of suspected UTI episodes where objective verification is completed within a defined window from t0 (culture preferred; UA secondary).
2. Avoidable antibiotic starts in stable, low-certainty episodes: among stable episodes, proportion with antibiotics initiated before verification (tA < tV) and without documented instability.
3. Time-to-action once objective support exists in high-penalty phenotypes: among high-penalty episodes with objective support, elapsed time from objective support (tV or tR) to appropriate action (initiate if not started; adjust/escalate if started).
4. Closed-loop culture review and stewardship action rate: among episodes with finalized culture, proportion reviewed with an explicit action documented within a specified window (e.g., 48–72 hours), including continue, narrow, switch, stop, with a guardrail of discontinuation/de-escalation when cultures do not support infection.

Expected Clinical and System Outcomes of a Verification-First Pathway

Success should appear first in leading indicators because they reflect the pathway's mechanisms and should move before distal outcomes stabilize in routine dashboards. Reduced avoidable antibiotics in stable, low-certainty episodes. The most immediate effect should be fewer antibiotic initiations in stable episodes before objective verification, particularly those triggered by nonspecific delirium or functional decline. This directly addresses dementia-context misclassification where classic signs are unreliable [20, 21], and where empiric antibiotics commonly persist when follow-up is unreliable [19]. Evidence of success includes a declining KPI 2 and increased evidence-based stop decisions (KPI 4), with overall reductions in antibiotic burden.

Faster appropriate action in high-penalty phenotypes once objective support exists. The pathway should reduce delay harm by tightening verification and reassessment tempo for vulnerable phenotypes and by accelerating action once objective support exists. This is particularly important where infection signals may be centrally expressed and fever attenuated [22]. Improvement should be reflected in shorter KPI 3 intervals and fewer episodes that "declare late" with rapid deterioration after prolonged ambiguity.

Higher closed-loop reliability and stewardship action. A major operational aim is that culture results change management rather than arriving after decisions are fixed. Higher KPI 4 values and higher discontinuation/de-escalation when cultures do not support infection indicate that the system is correcting course and preventing "sticky empiricism" [19].

Improved documentation integrity and episode legibility. Because measurement requires identifiable episode anchors, success includes improved documentation of stability, verification timing, and closure decisions. Without this, episodes become analytically invisible and improvement collapses back to education. Distal outcomes as lagging validation. Once KPI movement stabilizes, distal outcomes such as sepsis progression, transfers, and readmissions can be evaluated as longer-horizon validation endpoints. These outcomes matter most but are confounded in dementia populations by baseline frailty and comorbidity, so they should not be the first proof point.

Limitations

This work is a service-design and clinical operations proposal rather than a prospective validation study. Although the logic is supported by biologically grounded accounts of atypical infection signaling in older adults and by observed workflow patterns in long-term care, it does not estimate causal effects under controlled implementation conditions. Measurement depends on feasible and accurate episode-level data capture. Incomplete documentation, free-text dependence, and fragmented data across transitions can cause misclassification and distort KPI values independent of true practice. The pathway also operates within persistent ambiguity created by asymptomatic bacteriuria and nonspecific symptoms in older adults; verification-first reduces antibiotic defaulting but does not eliminate uncertainty. Symptom-first criteria were designed to reduce inappropriate treatment, and dementia-context atypical presentation complicates their application. Implementation feasibility varies by setting, particularly culture turnaround, staffing for reassessment, and ownership of culture review. Sites with weak follow-up infrastructure may struggle to achieve closed-loop reliability, which can reintroduce empiric prescribing as a hedge. Finally, KPIs can be misused if treated as punitive targets rather than learning signals; governance should emphasize interpretability and improvement-oriented feedback loops.

CONCLUSION

UTI management in people living with dementia remains difficult because dementia-context infection frequently presents with attenuated or atypical signals and unfolds inside workflows that reward rapid action more than diagnostic certainty. Symptom-first criteria that prioritize fever and localizing urinary symptoms can misclassify early infection when classic signs are blunted, while nonspecific delirium and functional decline can be incorrectly treated as UTI in the setting of common bacteriuria. The resulting mortality paradox is structurally predictable: avoidable antibiotics in stable low-certainty episodes coexist with dangerous delay in high-penalty phenotypes.

A verification-first pathway reframes the problem as a health services delivery design challenge. By separating unstable from stable presentations, accelerating objective verification, committing to time-bounded reassessment, and enforcing closed-loop culture review, the pathway prevents uncertainty from being resolved by default antibiotics or passive delay. Coupled with a small auditable KPI set, it makes pathway fidelity visible through verification completion, avoidable empiric starts, time-to-action once objective support exists in high-penalty phenotypes, and closed-loop follow-up with stewardship action. In this framing, diagnostic certainty and follow-up reliability become infrastructure. The system remains imperfect, but it becomes measurable, corrigible, and safer under predictable ambiguity.

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