POSTRENAL ACUTE KIDNEY INJURY SECONDARY TO URINARY RETENTION FROM BENIGN PROSTATIC HYPERPLASIA COMPLICATED BY MULTIDRUG-RESISTANT ACINETOBACTER INFECTION AND CHRONIC PULMONARY THROMBOEMBOLISM: A CASE REPORT

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Abstract

Introduction: Acute kidney injury (AKI) is a frequent and serious condition in elderly patients, often multifactorial and associated with increased morbidity. Postrenal causes, particularly urinary obstruction due to benign prostatic hyperplasia (BPH), represent a reversible etiology if promptly recognized. The coexistence of infection and thromboembolic disease may further complicate the clinical course.
Case Presentation: We report an 87-year-old male with multiple comorbidities, including cardiovascular disease and recently diagnosed chronic pulmonary thromboembolism, who presented with acute urinary retention, abdominal pain, fatigue, hemoptysis, and dyspnea. His condition developed in the postoperative setting following intestinal surgery.
Diagnostic Assessment: Laboratory findings revealed AKI (creatinine 240 µmol/L, urea 17.5 mmol/L), marked systemic inflammation (CRP 89.7 mg/L), electrolyte disturbances, normocytic anemia, and significantly elevated D-dimer levels. Urinalysis showed severe infection, and urine culture identified multidrug-resistant (MDR) Acinetobacter spp., sensitive only to fosfomycin. Imaging suggested prostatic enlargement and chronic pulmonary thromboembolism.
Differential Diagnosis: Considerations included prerenal AKI due to dehydration, intrinsic renal injury, postrenal obstruction, complicated urinary tract infection, pulmonary embolism, and postoperative abdominal complications.
Final Diagnosis: Postrenal AKI secondary to urinary retention from BPH, complicated by MDR Acinetobacter urinary tract infection and coexisting chronic pulmonary thromboembolism.
Treatment and Management: Immediate bladder decompression via Foley catheterization led to improved urine output. Management included targeted antibiotic therapy based on susceptibility testing, electrolyte correction, hydration, and initiation of tamsulosin for BPH.
Outcome and Follow-up: The patient showed clinical improvement with stabilization of renal function and resolution of infection symptoms. Follow-up included monitoring renal parameters, urological evaluation, and assessment of thromboembolic risk.
Discussion and Conclusion: This case highlights the importance of early identification of reversible causes of AKI, particularly postrenal obstruction in elderly patients. It underscores the growing challenge of MDR infections and the diagnostic complexity of elevated D-dimer in inflammatory states. A multidisciplinary, systematic approach is essential for optimal management of patients with overlapping renal, infectious, and thromboembolic conditions.

Keywords: Postrenal acute kidney injury; Benign prostatic hyperplasia; Urinary retention; Multidrug-resistant Acinetobacter; Complicated urinary tract infection; Pulmonary thromboembolism.

Introduction

Acute kidney injury (AKI) is a common and serious complication in hospitalized patients, particularly in older adults, and is associated with increased morbidity and mortality. Even

minor increases in serum creatinine may reflect clinically significant renal dysfunction, underlining the importance of early recognition and timely intervention [1].
Among the different etiologies, obstructive nephropathy represents a frequent and potentially reversible cause of AKI. In elderly patients, postoperative urinary retention and lower urinary tract obstruction, often related to benign prostatic hyperplasia, may rapidly lead to postrenal AKI if not promptly recognized and treated. Although outcomes may be more favorable than in other AKI forms, obstructive AKI can still result in prolonged hospitalization and incomplete renal recovery [2].
Complicated urinary tract infections may further worsen the clinical course, especially when caused by multidrug-resistant organisms. Acinetobacter spp., including Acinetobacter baumannii, are important nosocomial pathogens due to their ability to develop multidrug resistance, which frequently limits treatment options and necessitates targeted therapy based on susceptibility testing [3].
In postoperative patients, systemic inflammation, infection, and reduced mobility may also contribute to a hypercoagulable state. Markedly elevated D-dimer levels are clinically relevant because the likelihood of pulmonary embolism increases with rising D-dimer concentrations, particularly when respiratory symptoms coexist [4].
We present the case of a frail elderly postoperative patient in whom chronic pulmonary thromboembolism, in conjunction with postrenal acute kidney injury and a multidrug-resistant Acinetobacter urinary tract infection, resulted in a complex and clinically challenging presentation.

Case Presentation

History of Present Illness

An 87-year-old retired male with a history of cardiovascular disease, arterial hypertension, hypercholesterolemia, chronic pulmonary thromboembolism (diagnosed approximately one month earlier), partial intestinal obstruction (subileus) treated surgically about six weeks prior, ischemic stroke (~20 years earlier) with residual speech impairment, and intestinal surgery (~40 years ago), presented with difficulty in urination, generalized body pain, fatigue, hemoptysis, and shortness of breath (dyspnea).
One month prior to the current admission, the patient was diagnosed with chronic pulmonary thromboembolism. He presented with acute urinary retention, accompanied by abdominal pain and generalized fatigue. The abdominal pain began approximately two days before admission, initially intermittent and later becoming persistent. He also reported dark-colored urine, episodes of hemoptysis, and dyspnea. Approximately six weeks before admission, he had undergone surgical treatment for partial intestinal obstruction (subileus). Postoperatively, he developed constipation and recurrent vomiting, which persisted and were still present on the day of admission.
He has a known allergy to peaches. Social history reveals that he is a former smoker, does not consume alcohol, has limited physical activity due to fatigue, and reports good sleep.
Physical Examination
On admission, the patient appeared ill and fatigued. Signs of urinary retention were present. Respiratory examination revealed dyspnea. The abdomen was tender in the postoperative

setting. No new focal neurological deficits were identified apart from chronic speech impairment.

Diagnostic Assessment

Timeline (Chronological Summary) [5-12]

Date	Event/Investigation	Findings	Clinical Significance
October 2025	Abdominal X-ray (native)	Air–fluid levels in bowel loops; no free subdiaphragmatic air	Suggestive of subileus / partial intestinal obstruction
October 2025	Abdominal X-ray (second report)	Small bowel air–fluid levels compatible with subileus; mild right pleural effusion	Confirms subileus + incidental pleural fluid
November 2025	Chest X-ray	Right pleural effusion with compressive atelectasis; bilateral reticulonodular changes; cardiomegaly	Suggests pneumonia/effusion, chronic lung changes
November 2025	ICD-10 diagnosis codes	R06, J18, J90	Breathing abnormality + pneumonia + pleural effusion
November 2025	Outpatient therapy documented	Edemid forte, spironolactone, Lebel 500, Tricef 200, famotidine	Diuretic + antibiotic/supportive therapy
December 2025	Hospital admission	Urinary retention, abdominal pain, fatigue, hemoptysis, dyspnea	Suspected postrenal AKI + infection, evaluate respiratory symptoms

December 2025	D-dimer	18,295 ng FEU/mL	Severe inflammation/hypercoagulability
December 2025	D-dimer	35,260 ng/mL	Persistent extreme elevation
During hospitalization (date unavailable)	CT abdomen with contrast incidentally noted findings suggestive of chronic pulmonary thromboembolism (as stated in the radiology report) Suggestive changes requiring further pulmonary imaging.	Dilated small bowel loops, likely adhesions; enlarged prostate; renal microlithiasis; suggested chronic pulmonary thromboembolism	Supports GI adhesions + possible chronic PTE

Laboratory Findings

Hemostasis Analysis and Clinical Interpretation

Coagulation profile demonstrated preserved global coagulation function, with PT, aPTT, and TT values within reference ranges, without evidence of intrinsic or extrinsic pathway impairment.
In contrast, serial D-dimer levels were markedly elevated, indicating pronounced activation of coagulation and fibrinolysis in the setting of systemic inflammation. This pattern is consistent with a secondary, inflammation-driven hypercoagulable state, likely related to the postoperative condition, acute infection, reduced mobility, and concomitant acute kidney injury.
Overall, the findings support reactive activation of the coagulation system rather than a primary coagulopathy, in keeping with the clinical context. [10-11].

Urinalysis Analysis and Clinical Interpretation

Urinalysis demonstrated marked inflammatory changes, with significant leukocyturia (500 leukocytes/µL) and hematuria (300 erythrocytes/µL), accompanied by abundant bacteriuria, consistent with an active urinary tract infection. The overall pattern supports a severe, complicated infection in the given clinical context.
The absence of pathological casts argues against prominent intrinsic renal involvement, although concomitant acute kidney injury, particularly of postrenal origin, remains clinically plausible. [11].

Laboratory Findings and Clinical Interpretation

Laboratory investigations demonstrated preserved hepatic and muscular function, accompanied by significant electrolyte disturbances, marked systemic inflammation, secondary activation of coagulation, and mild metabolic alterations.

Analysis of liver and muscle enzymes revealed values within reference ranges, including alkaline phosphatase (ALP 56 U/L), aspartate aminotransferase (AST 30 U/L), alanine aminotransferase (ALT 13 U/L), gamma-glutamyl transferase (GGT 31 U/L), lactate dehydrogenase (LDH 226 U/L), creatine kinase (CK 48 U/L), and CK-MB (9 U/L), indicating preserved hepatic integrity and absence of clinically significant muscle or myocardial injury.

Electrolyte assessment identified multiple abnormalities, including hypocalcemia (2.09 mmol/L), hypomagnesemia (0.70 mmol/L), and hypophosphatemia (1.20 mmol/L). Serum sodium (138 mmol/L) and chloride (98 mmol/L) were within normal limits, while potassium was at the lower limit of normal (3.50 mmol/L). Serum iron was reduced (9.60 µmol/L). These findings are consistent with acute kidney injury in the context of postoperative metabolic stress, systemic inflammation, and ongoing diuretic therapy, and may contribute to fatigue, neuromuscular weakness, and increased risk of cardiac arrhythmias.

Lipid profile analysis demonstrated controlled total cholesterol (3.30 mmol/L) and triglycerides (1.03 mmol/L), with acceptable low-density lipoprotein levels (LDL 2.15 mmol/L). Reduced high-density lipoprotein cholesterol (HDL 0.68 mmol/L) was noted, representing a chronic cardiovascular risk factor rather than an acute pathological abnormality.

Inflammatory markers were markedly elevated, with C-reactive protein (CRP 89.7 mg/L), indicating a pronounced systemic inflammatory response consistent with active infection and postoperative status.

Hemostasis evaluation revealed markedly elevated D-dimer levels on serial measurements, reflecting activation of coagulation and fibrinolysis. In this clinical context, these findings are most consistent with secondary, inflammation-associated hypercoagulability related to infection, postoperative state, and reduced mobility, rather than primary thrombotic disease, although thromboembolic processes cannot be excluded without definitive imaging.

Urine chemical analysis showed negative bilirubin, normal urobilinogen, and absence of glucosuria or ascorbic acid. Mild ketonuria (0.5 mmol/L) was present, likely reflecting a postoperative catabolic state and reduced oral intake, without evidence of diabetes mellitus or hepatobiliary dysfunction.

Overall, the laboratory profile is consistent with a frail postoperative elderly patient presenting with acute kidney injury, severe systemic inflammatory response, secondary hypercoagulability, and significant electrolyte imbalance in the setting of a complicated multidrug-resistant urinary tract infection, while hepatic and muscular functions remain preserved. [11].

Hematological and Biochemical Laboratory Findings

Hematological evaluation revealed a normocytic anemia, characterized by reduced red blood cell count (RBC 3.54 $\times 10^{12}$/L; reference 4.2–5.5 $\times 10^{12}$/L), decreased hemoglobin concentration (6.83 mmol/L; reference 7.7–10.6 mmol/L), and low hematocrit (34.4%; reference 37–54%). Mean corpuscular volume remained within normal limits (MCV 97.4 fL; reference 80–99 fL), supporting the classification of anemia as normocytic in nature. White blood cell count was

within the normal range (WBC 6.70 ×10⁹/L; reference 4–9 ×10⁹/L), and platelet count was preserved (PLT 152 ×10⁹/L; reference 140–340 ×10⁹/L), indicating the absence of significant leukocytosis or thrombocytopenia.
Glycemic parameters were within normal limits, with fasting glucose (5.50 mmol/L; reference 3.5–6.1 mmol/L) and glycated hemoglobin (HbA1c 5.06%; reference <5.7%), effectively excluding diabetes mellitus and stress-induced hyperglycemia as contributing factors.
Renal function tests demonstrated clear evidence of acute kidney injury, with markedly elevated serum urea (17.50 mmol/L; reference 2.5–6.4 mmol/L) and creatinine (240 µmol/L; reference 62–115 µmol/L). Serum uric acid was also increased (556 µmol/L; reference 208–428 µmol/L), consistent with impaired renal excretion and an enhanced catabolic state.
Assessment of protein status revealed hypoproteinemia and pronounced hypoalbuminemia, with total serum proteins (55.0 g/L; reference 64–82 g/L) and albumin (25.0 g/L; reference 34–50 g/L) both significantly reduced. These findings are most consistent with a combination of systemic inflammation, postoperative physiological stress, and possible nutritional compromise.
Overall, the laboratory profile is indicative of acute kidney injury, inflammation-associated normocytic anemia, and significant hypoalbuminemia in the context of preserved leukocyte and platelet counts, consistent with a frail postoperative elderly patient presenting with systemic inflammatory response and a complicated infectious process. [11].

Renal Function and KDIGO Staging

Acute kidney injury (AKI) was diagnosed based on a markedly elevated serum creatinine level on admission (240 µmol/L), in accordance with the KDIGO criteria. Although blood urea was also elevated (17.5 mmol/L), precise staging of AKI (KDIGO Stage 1–3) could not be established due to the absence of documented baseline creatinine values and incomplete pre-admission urine output data.
From a clinical perspective, the AKI was considered significant and likely multifactorial in origin, with a predominant postrenal component related to acute urinary retention, and a possible prerenal contribution associated with reduced oral intake, postoperative vomiting, and potential volume depletion. The rapid clinical response following bladder decompression via Foley catheterization, evidenced by a marked increase in urine output, strongly supports the diagnosis of postrenal AKI secondary to urinary obstruction.
Overall, the clinical and laboratory findings are consistent with obstructive nephropathy as the primary driver of renal impairment, with additional contributing factors related to the patient's postoperative state and systemic condition. [11].

Microbiological Findings

Urine culture demonstrated growth of multidrug-resistant (MDR) Acinetobacter species at a bacterial load of 10^4 CFU/mL, which was considered clinically significant in the presence of urinary symptoms and systemic inflammatory response. In symptomatic patients, particularly elderly males, colony counts at this threshold may represent true infection rather than contamination.
Antimicrobial susceptibility testing revealed extensive resistance to multiple antibiotic classes, including penicillins, β-lactam/β-lactamase inhibitor combinations, cephalosporins,

aminoglycosides, fluoroquinolones, trimethoprim–sulfamethoxazole, and nitrofurantoin. The isolate demonstrated susceptibility exclusively to fosfomycin, thereby necessitating targeted, culture-guided antimicrobial therapy rather than empirical treatment.
Overall, the microbiological findings are consistent with a complicated urinary tract infection caused by a multidrug-resistant nosocomial pathogen, highlighting the importance of microbiological confirmation and susceptibility testing in guiding appropriate therapeutic management. [12].

Summary of Clinically Significant Abnormal Findings

Hematology
Hemoglobin: 6.83 mmol/L ↓
Hematocrit: 34.4% ↓
RBC count: 3.54 ×10¹²/L ↓
Platelets: 152 ×10⁹/L

Findings consistent with normocytic anemia [11].

Renal Function
Urea: 17.5 mmol/L ↑
Creatinine: 240 μmol/L ↑
Uric acid: 556 μmol/L ↑

Consistent with acute kidney injury, likely multifactorial (postrenal + prerenal components) [11].

Inflammatory and Coagulation Markers
CRP: 89.7 mg/L ↑
D-dimer: 18,295 ng FEU/mL (11.12.2025) ↑↑ [11]
D-dimer: 35,260 ng/mL (12.12.2025) ↑↑ [10]
D-dimer results were reported using different assays/units; therefore, direct numeric comparison is limited, but both values were markedly elevated above the reference range.

Indicating severe systemic inflammation and hypercoagulability [10-11].
Electrolytes
Calcium: 2.09 mmol/L ↓
Magnesium: 0.70 mmol/L ↓
Phosphate: 1.20 mmol/L ↓

Significant electrolyte imbalance requiring correction [11].

Urinalysis
Turbid, amber-colored urine
Leukocytes: 500/μL ↑

Erythrocytes: 300/µL ↑
Bacteria: 118.8/HPF ↑
Proteinuria present

Suggestive of severe urinary tract infection [11].

Microbiological Findings
Urine culture revealed Acinetobacter species (10^4 CFU/mL).

Antibiotic susceptibility testing:
Resistant to: ampicillin, amoxicillin, amoxicillin–clavulanate, cefuroxime, cefixime, ceftazidime, ceftriaxone, cefepime, gentamicin, ciprofloxacin, levofloxacin, nitrofurantoin, trimethoprim–sulfamethoxazole.
Sensitive only to fosfomycin.
Diagnosis of multidrug-resistant urinary tract infection [12].

Table. Summary of Clinically Significant Abnormal Findings [11-12]

Category	Parameter	Result	Interpretation / Clinical significance	Ref.
Hematology	Hemoglobin	6.83 mmol/L ↓	Normocytic anemia (reduced oxygen-carrying capacity)	[11]
Hematocrit	34.4% ↓	Supports anemia		[11]
RBC count	3.54 ×10¹²/L ↓	Supports anemia		[11]
Platelets	152 ×10⁹/L	Within/low-normal range (no thrombocytopenia)		[11]
Renal function	Urea	17.5 mmol/L ↑	Renal impairment / azotemia	[11]
Creatinine	240 µmol/L ↑	Acute kidney injury (AKI)		[11]
Uric acid	556 µmol/L ↑	Hyperuricemia, may reflect renal dysfunction/catabolism		[11]

Inflammation & coagulation	CRP	89.7 mg/L ↑	Severe systemic inflammation	[11]
D-dimer	18,295 ng FEU/mL ↑↑ (11.12.2025)	Marked hypercoagulability; high thrombotic risk		[11]
D-dimer	35,260 ng/mL ↑↑ (12.12.2025)	Further increase, strongly abnormal		[10]
Electrolytes	Calcium	2.09 mmol/L ↓	Hypocalcemia (requires correction/monitoring)	[11]
Magnesium	0.70 mmol/L ↓	Hypomagnesemia (arrhythmia risk, worsens hypocalcemia)		[11]
Phosphate	1.20 mmol/L ↓	Hypophosphatemia (weakness, impaired recovery)		[11]
Urinalysis	Appearance	Turbid, amber-colored	Suggestive of infection/dehydration	[11]
Leukocytes	500/µL ↑	Significant pyuria → UTI		[11]
Erythrocytes	300/µL ↑	Hematuria, often in severe UTI/inflammation		[11]
Bacteria	118.8/HPF ↑	Marked bacteriuria		[11]
Proteinuria	Present	Inflammatory renal/urinary involvement		[11]

Microbiology	Urine culture	Acinetobacter spp. (10^4 CFU/mL)	Clinically significant growth in symptomatic patient	[12]
Susceptibility	MDR profile	Resistant to multiple antibiotics; sensitive only to fosfomycin		[12]

Differential Diagnosis

Several conditions were considered in the differential diagnosis based on the patient's presenting symptoms, laboratory findings, and imaging results.

• Prerenal acute kidney injury related to dehydration, reduced oral intake, vomiting, or diuretic therapy.

• Intrinsic renal injury, including acute tubular necrosis or infection-related renal involvement.

• Postrenal obstruction due to benign prostatic hyperplasia, which was later confirmed as the primary cause of acute kidney injury.

• Complicated urinary tract infection or urosepsis, particularly in the context of urinary retention and significant pyuria.

• Pulmonary embolism, considered due to dyspnea, hemoptysis, and markedly elevated D-dimer levels.

• Postoperative abdominal complications, such as recurrent intestinal obstruction or intra-abdominal inflammation following previous surgery.

Final Diagnosis

Postrenal acute kidney injury (AKI) secondary to acute urinary retention, most likely related to benign prostatic hyperplasia (BPH).

Complicated urinary tract infection (UTI) caused by multidrug-resistant (MDR) Acinetobacter spp. (urine culture 10^4 CFU/mL; susceptible only to fosfomycin).

Postoperative status following intestinal surgery for subileus/partial intestinal obstruction (~6 weeks prior).

Markedly elevated D-dimer with radiologically suggested chronic pulmonary thromboembolism (CT finding).

Additional findings: normocytic anemia, severe hypoalbuminemia, and electrolyte disturbances (hypocalcemia, hypomagnesemia, hypophosphatemia).

Treatment and Management

Initial Management

The patient was admitted to the hospital on 09 December 2025 due to acute urinary retention associated with abdominal pain, fatigue, hemoptysis, and dyspnea. Initial management focused

on stabilization and rapid identification of reversible causes of acute kidney injury (AKI). Intravenous access was established and laboratory investigations were obtained, including complete blood count, renal function tests, electrolytes, inflammatory markers, coagulation profile with D-dimer, and urinalysis.
Given the strong suspicion of urinary obstruction, prompt bladder decompression was performed via Foley catheterization, followed by close monitoring of urine output and fluid balance. Nephrotoxic medications were avoided whenever possible, and supportive therapy was initiated with careful hydration and correction of electrolyte disturbances. A urine culture was collected to guide antimicrobial therapy in the setting of suspected complicated urinary tract infection.

Pharmacological Treatment

Two months prior (October 2025) to the current hospitalization (December 2025), the patient had undergone intestinal surgery for ileus, without respiratory symptoms in the immediate postoperative period.
In November 2025, the patient presented with newly developed dyspnea. Chest radiography demonstrated a right-sided pleural effusion with compressive subsegmental atelectasis and bilateral reticulonodular opacities. Cardiomegaly was additionally observed. Hospital admission was advised but declined.
Based on the outpatient assessment, the following therapy was prescribed:
Furosemide (Edemid forte) – diuretic (loop diuretic)
Spironolactone – diuretic (aldosterone antagonist / potassium-sparing)
Levofloxacin (Lebel) – antibiotic (fluoroquinolone)
Cefixime (Tricef) – antibiotic (third-generation cephalosporin)
Famotidine – antiacid (H2 receptor blocker)
In December 2025, the patient was evaluated and managed for benign prostatic hyperplasia (BPH). Due to urinary catheterization, regular replacement of the Foley catheter (CH 18–20) was performed. Additionally, antibiotic therapy was administered based on urine culture and antibiotic susceptibility testing results. Doreta was given as needed for pain management.
During follow-up evaluation, the patient continued to be managed for BPH. Pharmacological therapy was adjusted to include tamsulosin 0.4 mg once daily as an alpha-adrenergic blocker to improve urinary symptoms.

Outcome and Follow-Up

After initiation of treatment, including bladder decompression with Foley catheterization, the patient’s urinary obstruction was significantly relieved, leading to improved urine output and reduction of abdominal discomfort.
The patient showed overall clinical improvement with resolution of urinary tract infection symptoms and better control of lower urinary tract symptoms.
Renal function and inflammatory markers were monitored during hospitalization, showing gradual stabilization. The patient remained under observation for respiratory complaints and markedly elevated D-dimer levels, with radiological findings suggestive of chronic pulmonary thromboembolism.

At discharge, follow-up was recommended, including repeat renal function tests and urinalysis/urine culture if symptoms recurred. Further evaluation by urology was advised for management of the underlying obstructive uropathy, along with pulmonary assessment to evaluate thromboembolic risk.

Discussion

The present case illustrates the diagnostic and therapeutic complexity that may arise in elderly postoperative patients presenting with acute kidney injury and multiple overlapping clinical conditions. In this patient, postrenal acute kidney injury caused by urinary retention occurred in the context of recent abdominal surgery, systemic inflammation, and severe urinary tract infection caused by multidrug-resistant Acinetobacter species.
Taken together, this case emphasizes how the coexistence of renal, infectious, and thromboembolic conditions in a short time period may create a complex clinical picture that requires careful evaluation and coordinated management across multiple medical specialties.

Conclusion

In conclusion, this case highlights the importance of promptly identifying postrenal causes of acute kidney injury in elderly postoperative patients. It underscores the growing clinical challenge posed by multidrug-resistant urinary pathogens and the diagnostic complexity associated with markedly elevated D-dimer levels in inflammatory states. A multidisciplinary and systematic approach is essential to optimize outcomes in frail patients with overlapping renal, infectious, and thromboembolic conditions.

Key Learning Points

- Postrenal acute kidney injury should always be considered in elderly patients presenting with acute urinary retention, as early bladder decompression can rapidly improve renal function.
- Multidrug-resistant urinary pathogens represent an increasing challenge in hospitalized and postoperative patients, making microbiological culture and susceptibility testing essential for appropriate therapy.
- Markedly elevated D-dimer levels in elderly patients may reflect multiple concurrent processes, including infection, inflammation, postoperative status, and thromboembolic disease.
- Elderly patients with multiple comorbidities frequently present with overlapping pathologies, requiring a systematic and multidisciplinary diagnostic approach.
- Early recognition of reversible causes of acute kidney injury combined with targeted antimicrobial therapy can significantly improve clinical outcomes.

Declarations

Conflict of Interest: The authors declare no conflict of interest.
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