

Received: 2025.12.14
Accepted: 2026.06.01
Available online: 2026.06.08
Published: 2026.XX.XX

Septic Shock Associated With Cystostomy: A Fatal Case Due to Delayed Infection Recognition

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Financial support: None declared
Conflict of interest: None declared

Patient: Male, 76-year-old
Final Diagnosis: Septic shock
Symptoms: Hypoxemia • tachycardia and low blood pressure
Clinical Procedure: —
Specialty: Urology

Objective: Diagnostic/therapeutic accidents
Background: Septic shock is highly prevalent in clinical practice. Some cases present insidiously and have poor prognoses. We report a case of a patient with a dislodged catheter who developed septic shock during a cystostomy procedure.

Case Report: Our patient was a 76-year-old man with a history of hypertension, type 2 diabetes mellitus, and cerebral infarction. He had been confined to bed for a long time following a cerebral infarction and had impaired consciousness. One year prior, he developed urinary retention due to benign prostatic hyperplasia. As he did not undergo surgical treatment, a long-term indwelling urinary catheter was subsequently placed. He initially presented to the emergency department of our hospital at 8: 30 PM on November 1, 2025, 8 hours after accidental urethral catheter dislodgement. A cystostomy was recommended, but he refused and left against medical advice. At approximately 8: 00 AM on November 2, 2025, he returned to the hospital and consented to cystostomy. During the procedure, he developed septic shock. Postoperatively, he was transferred to the intensive care unit for further treatment. Three days later, the family decided to discontinue active treatment and took the patient home. Follow-up revealed that the patient died 2 days after discharge.

Conclusions: This case highlights the critical importance of early infection screening and prompt sepsis recognition prior to surgical procedures in elderly, frail, and catheter-dependent patients. It also highlights the importance of keeping the catheter unobstructed and ensuring proper home care.

Keywords: case reports • cystostomy • septic shock

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Introduction

Sepsis is defined as a “life-threatening organ dysfunction caused by a dysregulated host response to infection” [1]. Its pathophysiology involves complex mechanisms such as systemic vasodilation and reduced effective circulatory volume. This results in global tissue hypoperfusion and hypoxia, which progresses to multiple organ dysfunction syndrome (MODS) and potentially fatal outcomes. According to a recent WHO publication, there were 48.9 million cases and 11 million sepsis-related deaths worldwide, representing 20% of all global deaths [2]. Septic shock is showing an increasing global trend in incidence, although mortality rates have declined in regions such as Europe and North America [2,3].

The first consensus definition of septic shock was established in 1992, which included the following key components: body temperature above 38 °C or below 36 °C, heart rate greater than 90 beats per minute, respiratory rate greater than 20 beats per minute or carbon dioxide partial pressure below 4.3 kPa, and neutrophilia above 12000/mm³ or neutropenia below 4000/mm³ with 10% or more of non-segmented peripheral blood neutrophils. The diagnosis of sepsis was based on the presence of a suspected infection and clinical or microbiological evidence of infection in the presence of at least 2 of the 4 systemic inflammatory response criteria (SIRS) [4]. With the iterative updates to the Surviving Sepsis Campaign guidelines, assessment tools such as NEWS, MEWS, and qSOFA have been subsequently incorporated to facilitate the rapid identification of septic shock. Early identification and appropriate management of septic shock is a critical determinant of improved survival rates [5]. Studies have demonstrated that prehospital recognition of septic shock, coupled with interventions such as antibiotic administration, effectively reduces mortality [6,7].

Advanced age and long-term indwelling urinary catheter are important risk factors for urinary tract infection. Older adults frequently present with atypical, non-specific symptoms of infection; classic signs such as fever and leukocytosis may be absent or attenuated, body temperature may be normal or even low, and white blood cell count may not be elevated. This can delay recognition of sepsis and septic shock in this population. Altered mental status, elevated lactate, and decreased urine output are important early indicators of sepsis and septic shock, particularly when typical inflammatory signs are absent [5]. The EAU guidelines show that the incidence of bacteriuria associated with indwelling catheterization is 3% to 8% per day. The duration of catheterization is the most important risk factor for the development of a catheter-related urinary tract infection (CAUTI). Meanwhile, the guidelines point out that catheter-associated UTIs are the leading cause of secondary healthcare-associated bacteremia [8].

Septic shock can develop at any time. We present a case of insidious-onset septic shock that occurred during a cystostomy procedure and was characterized by rapid clinical deterioration.

Case Report

A 76-year-old man with a history of neurogenic bladder requiring long-term indwelling catheterization was brought to the emergency department by his family 8 hours after accidental catheter dislodgement, requesting re-catheterization due to an inability to void spontaneously. He was asymptomatic at presentation. His past medical history included hypertension and type 2 diabetes mellitus, with an unclear medication history. He had a cerebral infarction 10 years prior, resulting in cognitive impairment and long-term confinement to bed. No significant family history was reported.

Attempts at transurethral catheterization by the emergency physician were unsuccessful. To relieve the urinary obstruction, urethroscopy or suprapubic cystostomy was advised; however, the family declined and took the patient home. He returned to the hospital 12 hours later, reporting lower abdominal distension and urinary incontinence. On physical examination, the patient refused palpation of the bladder area. The family consented to a suprapubic cystostomy. At that time, his body temperature was 36.7 °C, blood pressure was 132/76 mm Hg, heart rate was 108 beats per minute, and oxygen saturation was 98% without oxygen therapy. Due to his cognitive impairment, he was unable to cooperate under local anesthesia alone. Following evaluation by the anesthesiologist, the procedure was planned under intravenous sedation with local anesthesia.

Vital signs before the start of anesthesia were as follows: blood pressure 156/85 mm Hg, heart rate 98 bpm, temperature 36.8 °C, and oxygen saturation 97%. He was placed in the supine position. The anesthesiologist administered 15 mg of intravenous propofol for sedation, along with 5 mg of lidocaine for local anesthesia. Upon confirmation of adequate anesthesia by the anesthesiologist, we proceeded with the suprapubic cystostomy.

During puncture needle insertion into the bladder, the anesthesiologist detected a sudden clinical deterioration. The patient's blood pressure dropped precipitously to a systolic pressure of 68 mm Hg, accompanied by tachycardia (165 beats per minute), hypoxemia with an oxygen saturation of 66%, and hyperthermia (39.5 °C). Recognizing the presentation of septic shock, the anesthesiologist promptly initiated advanced airway management (tracheal intubation), administered an intravenous bolus of norepinephrine, and commenced rapid fluid resuscitation.

Table 1. Laboratory test results.

Item	On day 1	On day 2	On day 3
RBC (10⁹/l)	4.12	4.12	3.88
WBC	4.1	11.6	14.2
PLT	36	16	11
Hb	115	118	111
Liver function			
AST (U/L)	205	315	489
ALT (U/L)	81	230	479
TBIL (μmol/l)	57.6	83.3	107.2
DBIL (μmol/l)	39.6	56.0	79.2
IBIL (μmol/l)	18	27.3	28.0
Renal function			
Cr (μmol/l)	244	172	128
BUN (nmol/l)	16.07	16.46	18.44
BNP (pg/ml)	1235.5	1548.8	2520.2
cTnl (ng/ml)	0.264	/	0.253
Coagulation function			
PT (s)	17.7	20.1	14.7
APTT (s)	49.9	47.3	39.7
TT (s)	25.5	18.2	16.6
Fibrinogen (g/l)	2.652	5.747	7.396
D-Dimer (μg/l)	3010	3220	30300
Procalcitonin (ng/ml)	> 100.00	> 100.00	> 100.00
Interleukin-6 (ng/ml)	> 5000.0	3338.3	1282.7
CRP (mg/l)	163.99	266.49	279.38
Lactate (mmol/l)	7.3	10.3	5.6
PO₂ (mm Hg)	143.0	97.3	103.0
PCO₂ (mm Hg)	31.5	41.5	44.1

Table 2. ICU-monitored parameters.

Item	On day 1	On day 2	On day 3
Systolic blood pressure (mm Hg)	100	117	127
Heart rate (/min)	160	157	140
Respiratory rate (/min)	15	15	15
Body temperature (°C)	39.5	40.0	38.8
ScvO₂ (%)	97	98.7	98.2
Level of consciousness	Sedated state	Sedated state	Sedated state
Urine output (mL)	120	5000	2114

The cystostomy was completed within 5 minutes. At that time, the patient's systolic pressure was 81 mm Hg, with a heart rate of 158 bpm. On ventilator support, the oxygen saturation was 92%, and body temperature remained elevated at 39.5 °C. Subsequently, he was transferred to the intensive care unit for further management.

In the intensive care unit, he continued to receive advanced airway management and norepinephrine infusion propofol sedation for blood pressure support, and was initiated on broad-spectrum antibiotic therapy (imipenem-cilastatin 1 g every 8 hours) within 30 minutes. Additional interventions included vasoactive support with metaraminol, corticosteroid therapy (dexamethasone), and blood product transfusions along with intravenous fluid resuscitation. Following a 3-day course of intensive treatment and monitoring, diagnostic evaluations were performed. A bedside chest radiograph revealed evidence of bilateral lower lobe pneumonia. Lower extremity venous Doppler ultrasound identified an active thrombus in the right common femoral vein and thrombi within the muscular veins of the right calf. Subsequent ultrasound examinations of the liver, kidneys, heart, gallbladder, and pancreas did not reveal significant abnormalities. The results of pertinent laboratory investigations and clinical parameters are summarized in **Tables 1 and 2**. Hematologic and urine culture results identified *Escherichia coli* and *Pseudomonas aeruginosa*, both of which are hospital-acquired multidrug-resistant organisms.

On the third day, the patient's family decided to discontinue active treatment, and he was discharged home. During follow-up, it was ascertained that the patient had died 2 days after discharge.

Discussion

Our patient presented with a body temperature exceeding 38.0 °C, a heart rate over 90 beats per minute, carbon dioxide partial pressure below 4.3 kPa, and positive blood cultures, accompanied by liver and kidney failure. These findings collectively indicated the presence of septic shock. Additionally, his platelet count was $< 50 \times 10^9/L$, D-dimer was > 7 times the upper limit of normal, and prothrombin time was > 6 seconds. The hematological test results are highly compatible with disseminated intravascular coagulation (DIC) according to the 2025 international criteria (ISTH). The elevation of fibrinogen suggests a particular phase of the process. Although in advanced DIC (consumption phase) fibrinogen is usually low, a level of 7.396 g/L does not rule out DIC. Fibrinogen is an acute phase reactant, which means that it increases significantly in response to severe inflammation, infections, or tumors. In the early stages or in cases of DIC associated with intense inflammatory processes (such as sepsis or cancer), the body produces

fibrinogen faster than it consumes it. Serum lactate serves as a critical marker of tissue hypoxia and dysfunction. Current guidelines explicitly specify that elevated lactate levels are integral to the definition of septic shock [5].

Septic shock can occur in individuals of all demographics. The occurrence of sepsis is closely associated with underlying health conditions. Risk factors are older and very young age, pregnancy, malignant tumors, organ transplantation, AIDS, radiation therapy, burns, sores, polytrauma, diabetes mellitus, hepatic failure, renal failure, malnutrition, catheters or other invasive devices, urinary catheters, and other health problems [2,9,10]. In this case, in addition to the patient's poor baseline condition, the anesthesia status and the surgical procedure itself were significant contributing factors. Although propofol can attenuate the release of pro-inflammatory cytokines, anesthesia-induced hypothermia and hemodynamic instability can impair the immune system [11]. Our patient showed no significant changes immediately following intravenous anesthesia. However, during the cystostomy procedure, there was a rapid onset of hypotension and decreased oxygen saturation. We hypothesize that the induction of hemodynamic instability during anesthesia damages the immune system and that the insertion of the trocar led to a sudden increase in intravesical pressure, which may have been a key factor in exacerbating the underlying infection. Early administration of antibiotics is crucial for lowering mortality rates [12]. Current guidelines recommend administering antibiotics within the first hour for patients with septic shock [4]. Recommendations for antibiotic selection suggest that empiric antibiotics should prioritize broad coverage against aerobic gram-negative bacilli, including *Enterobacterales*, with consideration of local antibiotic resistance patterns [5]. Evidence indicates that beyond 3 hours, mortality increases by approximately 35% for each additional hour of delay [13]. For patients with long-term indwelling catheters in whom trauma is suspected, the prophylactic use of antibiotics is recommended. Since preoperative infection was not confirmed, and to curb hospital antibiotic resistance rates, prophylactic antibiotics were withheld. This case serves as a reminder that we should strengthen the clinical management of prophylactic antibiotic use.

Effective clinician-patient communication is instrumental in the treatment process [14-16]. The patient initially refused the recommended cystostomy upon arrival at the hospital and left for 12 hours. The emergency physician failed to recognize the underlying infection, likely due to the patient's chief concerns and refusal of further examination. Nevertheless, the failure to recognize the potentially lethal urinary retention was a significant clinical oversight. Although the risks were explained, if there had been more intensive communication to secure patient cooperation, early relief of the urinary tract obstruction could have been achieved, potentially improving the prognosis.

Catheter-associated urinary tract infection (CAUTI) is very common and largely preventable [17]. Effective home care is essential to reducing its incidence, requiring active participation from community healthcare providers and physicians, as well as from patients and their families [18,19]. The dark yellow, foul-smelling urine drained after the procedure indicated poor long-term care of the patient's urinary catheter.

Infection is the fundamental prerequisite for septic shock. Rapid identification of infection can effectively enhance vigilance for the prevention of septic shock. The clinical manifestations of septic shock vary across its progressive stages. Early-stage manifestations include abnormal body temperature, tachycardia, tachypnea, restlessness, mild confusion, pallor, cyanosis, oliguria, nausea, and vomiting. Mid-stage manifestations typically involve hypotension, drowsiness, dyspnea, further increased heart rate, and progressive reduction in urine output. Late-stage manifestations can progress to coma, refractory hypotension, multiple organ failure, and severe metabolic disturbances. The disease progression in this case was very rapid, with the early and mid-stages being virtually indistinguishable. The qSOFA, NEWS, and MEWS scoring systems are recommended by guidelines to aid in the early recognition of septic shock [4]. The often subtle or nonspecific signs of infection in the geriatric population require clinicians to actively gather corroborating evidence from laboratory and imaging studies. Hyperlactatemia is frequently indicative of shock and is associated with clinical outcomes [20]. Capillary refill time is associated with tissue hypoperfusion, which is valuable for the identification of septic shock [5]. Imaging modalities such as ultrasound and CT often reveal findings including vesicoureteral reflux, structural abnormalities, intraluminal debris, and tissue thickening, which necessitate the exclusion of infection in the differential diagnosis. Cross-sectional imaging (CT/MRI) can demonstrate varied radiographic presentations among different categories of urinary tract infection, which can aid in their differentiation [21].

Our patient subsequently developed pulmonary inflammation and intramuscular venous thrombosis of the lower extremity. A close bidirectional causal relationship exists between pulmonary inflammation and septic shock. On one hand, severe community-acquired and hospital-acquired pneumonia are among the most common causes of septic shock; pulmonary pathogens and their toxins enter the systemic circulation, triggering an intense systemic inflammatory response that leads to vasodilation, circulatory disturbance, and tissue hypoperfusion, thereby rapidly progressing to septic shock. On the other hand, septic shock can induce systemic immunoparalysis,

altered mental status, increased risk of aspiration, and mechanical ventilation-related complications, all of which significantly increase the incidence of pneumonia. These 2 conditions mutually reinforce each other, forming a vicious cycle. Septic shock is an independent and strong risk factor for lower extremity venous thrombosis. It triggers a systemic inflammatory response that leads to vascular endothelial injury, and the associated hypercoagulable state causes venous stasis [22]. Concurrently, peripheral hypoperfusion further contributes to these abnormalities, and together these factors constitute the necessary prerequisites for thrombus formation.

In older patients or those with impaired consciousness, clinical manifestations of infection are often subtle or atypical, requiring heightened vigilance and proactive screening in these populations to prevent the development of septic shock. In the present case, factors such as the timing of antibiotic administration and the delayed relief of obstruction were critical factors contributing to the adverse outcome.

Conclusions

This case report illustrates the insidious onset and rapid progression of septic shock, highlighting the critical importance of infection prevention and early clinical recognition. For older patients who are catheter-dependent and have cognitive impairment, we recommend establishing an emergency department/urology department/infectious diseases department rapid consultation process. Under this model, the emergency department rapidly completes preoperative infection screening, the infectious diseases department formulates a rational anti-infection regimen, and the urology department promptly intervenes to relieve urinary tract obstruction. Patients with long-term indwelling urinary catheters must adhere to a strict home care regimen to minimize risk, such as regular catheter changes and appropriate disinfection protocols. Finally, we will further examine whether prophylactic antibiotics provide clinical benefit to infection-prone patients following failed catheter placement.

Department and Institution Where Work Was Done

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Patient Permission/Consent Declarations

Patient consent was obtained.

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