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Case Report

Spinal Intradural Hematoma Following Neuraxial Anesthesia in a Uroseptic Patient: A Case Report and Narrative Review

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Abstract: Spinal intradural hematoma (SIH) is a rare but potentially devastating complication of neuraxial anesthesia, particularly in critically ill patients with sepsis-associated thrombocytopenia and coagulation abnormalities. A 63-year-old woman presented with septic shock secondary to obstructive uropathy caused by ESBL-producing Klebsiella pneumoniae. Initial management included intravenous fluid resuscitation, vasopressor support, antimicrobial therapy, and urinary decompression. Following transient hemodynamic stabilization, she underwent urological intervention under spinal anesthesia. Retrospective review demonstrated thrombocytopenia (63 $\times 10^9$/L) on the day of the procedure, which had not yet been recognized at the time of anesthesia. Within 24 hours, the patient developed acute bilateral lower limb weakness and sensory deficits. Magnetic resonance imaging (MRI) confirmed spinal intradural hematoma at the L2–L3 level with significant neural compression. Emergency decompressive laminectomy was performed; however, neurological recovery remained incomplete. This case highlights the importance of continuous reassessment of coagulation parameters in critically ill septic patients undergoing neuraxial procedures. New neurological deficits following neuraxial anesthesia should be regarded as a medical emergency requiring immediate imaging and urgent neurosurgical evaluation.

Keywords: Septic shock; Urosepsis; ESBL; *Klebsiella pneumoniae*; Spinal intradural hematoma; Spinal anesthesia.

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1. Introduction

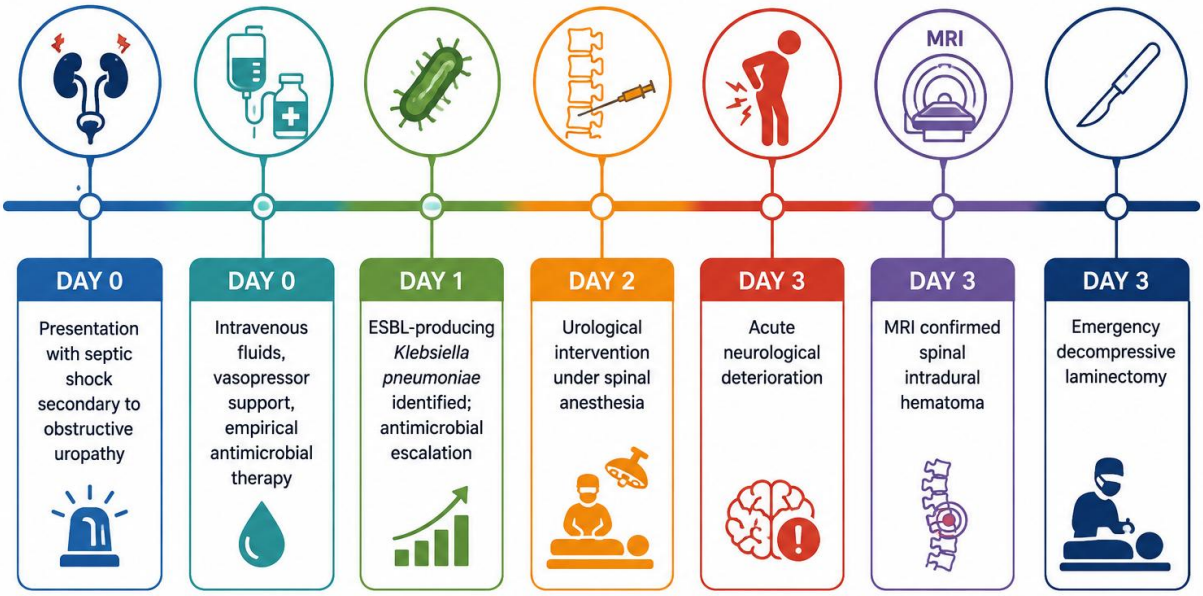
Obstructive uropathy in the presence of infection constitutes a urological emergency with substantial risk of progression to sepsis and septic shock [1–3]. Delayed decompression is associated with increased morbidity and mortality; therefore, early urinary drainage combined with effective antimicrobial therapy remains essential [1–7]. The increasing prevalence of antimicrobial resistance has further complicated the management of urosepsis. Infections caused by extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae are associated with delayed effective therapy and increased mortality [8–12]. Carbapenems remain the preferred treatment for severe ESBL-associated infections in critically ill patients [13, 14]. Neuraxial anesthesia is commonly used during urological procedures and is generally considered safe. However, spinal intradural hematoma (SIH) remains a rare but severe complication that may result in irreversible neurological injury

[15–18]. Reports involving septic patients with rapidly evolving thrombocytopenia remain limited.

2. Case Report

A 63-year-old woman with insulin-dependent diabetes mellitus and hypertension presented to the emergency department with fever, dysuria, and abdominal pain (Figure 1). Clinical evaluation demonstrated hypotension, tachycardia, and acute kidney injury consistent with septic shock secondary to urosepsis. Initial management included intravenous fluid resuscitation, vasopressor support with noradrenaline 2-3ug/kg/min, and empirical antimicrobial therapy with amoxicillin/clavulanic acid according to local institutional protocols.

Figure 1. Clinical timeline of spinal intradural hematoma following meuraxial anesthesia in a uroseptic patient.



Within 24 hours, due to persistent clinical deterioration and concern for resistant infection, antimicrobial therapy was escalated. Laboratory investigations demonstrated leukocytosis and thrombocytopenia consistent with sepsis-associated hematological abnormalities. Serum creatinine was 329 µmol/L (peak approximately 399 µmol/L), urea 16.6 mmol/L, and C-reactive protein (CRP) 29.6 mg/dL (Table 1).

Table 1. Laboratory Results Overview.

Parameter	Day 0	Day 2	Day 3	Day 6	Day 7	Day 8	Day 10	Day 15	Day 15
Kidney & Electrolytes									
Glucose (mmol/L)	12	6.7	3.5	2.7	–	3.4	–	–	–
BUN (mmol/L)	–	16.6	15.2	18.1	17.4	17.6	14.4	9.5	8.3
Creatinine (µmol/L)	233	339	297	322	306	294	239	185	168
Sodium (mmol/L)	120	117	131	133	132	135	130	132	130
Potassium (mmol/L)	4.3	4.5	3.1	4.0	4.0	4.4	4.3	4.0	5.0
Calcium (mmol/L)	–	–	–	1.63	–	1.77	1.61	–	1.78
Phosphate (mmol/L)	–	–	–	0.82	–	1.22	–	–	1.42
Magnesium (mmol/L)	–	–	–	–	–	0.62	–	–	–
Albumin (g/L)	–	–	–	19.1	–	–	–	–	–

Liver Panel									
Alkaline Phosphatase (U/L)	–	287	–	–	–	–	619	–	–
Total Bilirubin (μmol/L)	–	16.9	–	28.5	–	–	18.2	–	–
ASAT (U/L)	–	20	–	21	–	–	13	–	–
ALAT (U/L)	–	12	–	10	–	–	8	–	–
GGT (U/L)	–	131	–	–	–	–	424	–	–
LDH (U/L)	–	195	–	262	–	–	170	–	–
Amylase (U/L)	–	12	–	–	–	–	–	–	–
Iron (μmol/L)	–	–	–	11	–	–	–	–	–
Inflammation									
CRP (mg/dL)	11.3	29.6	28	24.1	16.4	–	8.7	5	1.9
Amylase (urine) (U/L)	–	11	–	–	–	–	–	–	–
Hematology									
Hemoglobin (mmol/L)	6.5	4.8	4.8	4.4	4.2	–	4.1	3.7	4.1
Hematocrit (L/L)	0.29	0.22	0.21	0.20	0.19	–	0.19	0.18	0.20
Leukocytes ($\times 10^9$/L)	21.9	23.3	13.7	18.5	13.9	–	9.6	9.3	6.9
Thrombocytes ($\times 10^9$/L)	323	150	63	23	59	–	182	353	511
Erythrocytes ($\times 10^{12}$/L)	3.5	2.7	2.7	2.5	2.4	–	2.3	2.1	–
MCV (fL)	83	81	80	81	80	–	82	85	–
Blood Gas (Arterial)									
pH	–	7.17	7.32	7.24	–	7.31	7.37	7.33	7.34
pCO_2 (mmHg)	–	38	33	42	–	38	30	32	35
pO_2 (mmHg)	–	32	50	33	–	33	44	40	25
HCO_3 (mmol/L)	–	13.1	17.2	16.7	–	18.6	18.6	17	18.4
Base Excess (mmol/L)	–	-13.6	-8.7	-8.8	–	-6.6	-7.0	-8.7	-6.5
Lactate (mmol/L)	–	3.3	–	–	–	1.2	–	1.4	1.2
Coagulation & Serology									
aPTT (sec)	32								
PTT (sec)	20.1								
INR	1.18								
HCV	Negative								
HBsAg	Negative								
Blood Group	O+								

Computed tomography demonstrated a distal ureteral stone with associated hydronephrosis, confirming obstructive uropathy. Urine cultures subsequently grew ESBL-producing Klebsiella pneumoniae, and antimicrobial therapy was adjusted according to susceptibility testing and current recommendations [13, 14]. On day 2 of admission, after transient hemodynamic stabilization and discontinuation of vasopressor support, the patient underwent urological intervention under spinal anesthesia. Spinal anesthesia was administered without documented immediate procedural complications. Laboratory values from the preceding day demonstrated a platelet count of 150 $\times 10^9$/L. However, retrospective review later demonstrated that the platelet count on the day of the procedure had decreased to 63 $\times 10^9$/L.

Approximately 24 hours after the procedure, the patient developed acute bilateral lower limb weakness and sensory deficits. MRI of the lumbar spine demonstrated spinal intradural hematoma at the L2–L3 level with significant neural compression (Figure 2 a 4) (15). Emergency decompressive laminectomy with evacuation of the hematoma was subsequently performed. Postoperatively, septic shock resolved with improvement of inflammatory markers and partial recovery of renal function. However, neurological recovery remained incomplete, requiring prolonged rehabilitation.

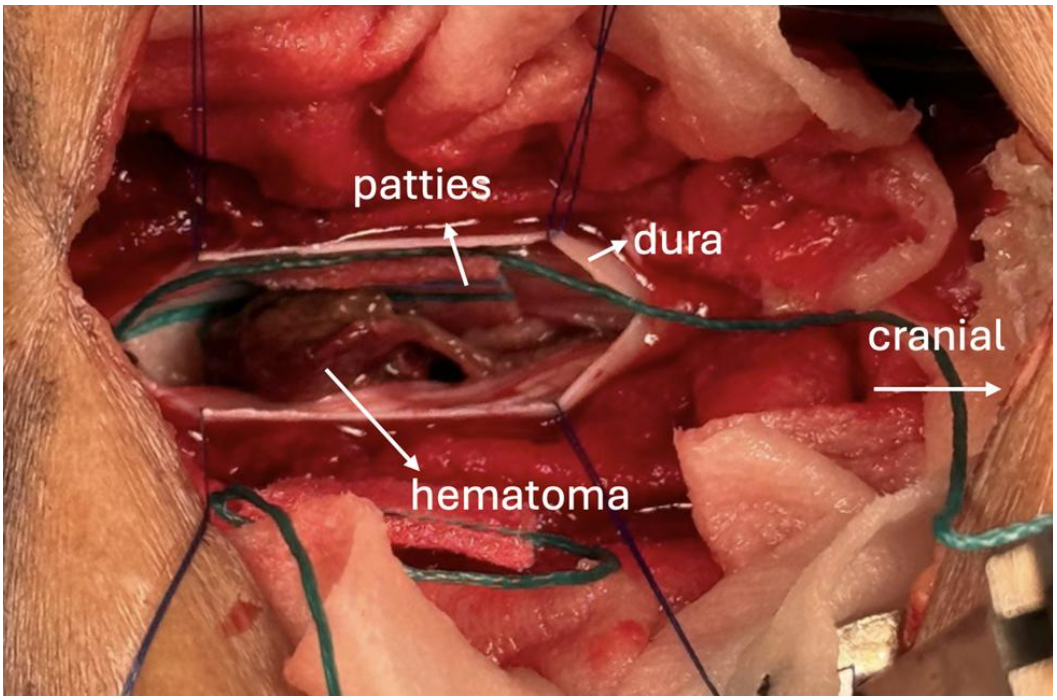


Figure 2. Intraoperative view after dural opening demonstrating evacuation of the spinal intradural hematoma.

Figure 3. Gross specimen of the evacuated spinal intradural hematoma obtained during surgical decompression.

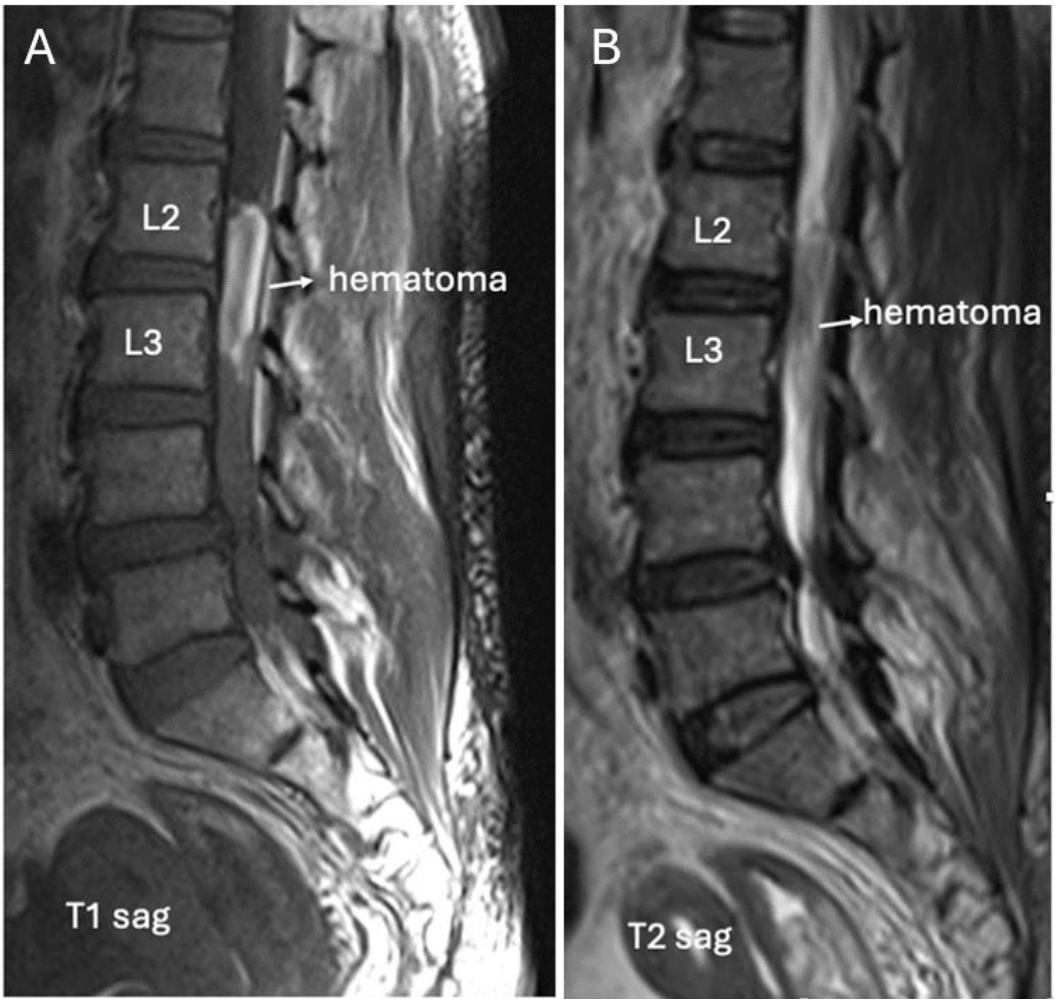


Figure 4. MRI lumbar spine showing intradural hematoma at L2–L3 with spinal cord compression.

3. Narrative Review of the Literature and Discussion

3.1 Obstructive Urosepsis and Source Control

Obstructive urosepsis represents a time-critical clinical emergency in which delayed decompression of the urinary tract is strongly associated with increased morbidity and mortality [1–3]. The coexistence of urinary tract obstruction and bacterial infection promotes rapid bacterial proliferation, elevated intrarenal pressure, impaired renal drainage, and systemic dissemination of pathogens, ultimately contributing to septic shock and multiorgan dysfunction. In addition, obstruction impairs antibiotic penetration into the infected collecting system, further complicating management and increasing the risk of persistent infection.

Early source control through urinary decompression remains one of the most important determinants of survival in obstructive urosepsis. Current evidence consistently demonstrates that delayed decompression is associated with prolonged hospitalization, increased intensive care admission, and higher mortality rates [1–7]. Both percutaneous nephrostomy and retrograde ureteral stenting are considered effective decompressive strategies, with selection depending on local expertise, anatomical considerations, and patient stability.

In septic patients, timely initiation of effective antimicrobial therapy is equally important. Kumar et al. demonstrated that each hour of delay in appropriate antimicrobial administration in septic shock is associated with increased mortality [7]. Similarly, early

implementation of sepsis bundles and prompt hemodynamic resuscitation have been shown to significantly improve patient outcomes [1, 5, 6]. These findings emphasize the importance of rapid recognition, multidisciplinary coordination, and early intervention in patients with obstructive urosepsis.

3.2 Antimicrobial Resistance and ESBL Infections

The increasing prevalence of antimicrobial resistance has substantially complicated the management of severe urinary tract infections worldwide. Extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae can hydrolyze most penicillins and cephalosporins, thereby limiting therapeutic options and increasing the likelihood of inadequate empirical treatment [8–12]. Infections caused by ESBL-producing organisms are associated with delayed effective therapy, prolonged hospitalization, increased healthcare costs, and higher mortality rates, particularly among critically ill patients [8–10]. The emergence of ESBL-producing Klebsiella pneumoniae is especially concerning because of its association with multidrug resistance and nosocomial transmission.

Current Infectious Diseases Society of America (IDSA) guidance recommends carbapenems as first-line therapy for severe ESBL-associated infections, especially in septic or hemodynamically unstable patients [13]. Although alternative non-carbapenem regimens have been explored, carbapenems continue to demonstrate superior outcomes in severe invasive infections caused by ESBL-producing organisms [14]. In the present case, initial empirical therapy was insufficient because of antimicrobial resistance, requiring escalation of treatment after microbiological identification of ESBL-producing Klebsiella pneumoniae. This clinical course illustrates the importance of early recognition of resistant pathogens and timely adjustment of antimicrobial therapy in critically ill patients.

3.3 Spinal Intradural Hematoma Following Neuraxial Anesthesia

Spinal intradural hematoma is a rare but potentially catastrophic complication of neuraxial anesthesia [15–18]. Although spinal epidural hematoma is more commonly reported, intradural hematoma represents an important differential diagnosis in patients presenting with acute neurological deterioration after spinal procedures. Reported incidences of neuraxial hematoma range from approximately 1:150,000 to 1:220,000 procedures, although the true incidence may be underestimated because of underreporting and diagnostic variability [15–17].

The exact pathophysiology remains incompletely understood. Proposed mechanisms include injury to radicular vessels during needle insertion, bleeding into a traumatically created cleavage plane within the dural border cell layer, or hemorrhage originating from fragile venous structures within the spinal canal. Progressive accumulation of blood may result in compression of the conus medullaris and/or cauda equina, causing ischemia and potentially irreversible neurological injury. Several risk factors for neuraxial hematoma have been identified, including anticoagulant therapy, coagulopathy, thrombocytopenia, traumatic needle placement, multiple puncture attempts, advanced age, and spinal anatomical abnormalities [15,16]. Critically ill septic patients may represent a particularly vulnerable population because coagulation status may fluctuate rapidly during illness.

Clinically, spinal intradural hematoma often presents with acute back pain followed by progressive neurological deficits, including motor weakness, sensory disturbances, radicular pain, and bladder or bowel dysfunction. Because neurological deterioration may progress rapidly, early recognition remains essential. MRI is considered the diagnostic modality of choice because it allows rapid visualization of hematoma location, extent of neural compression, and associated spinal pathology.

Neurological outcome is strongly dependent on the timing of surgical decompression. Multiple studies have demonstrated significantly improved neurological recovery when decompression occurs within 12–24 hours after symptom onset, whereas delayed intervention is associated with persistent deficits and poorer functional outcomes [22, 23].

Despite timely decompression in the present case, neurological recovery remained incomplete, underscoring both the severity of this complication and the narrow therapeutic window available for intervention.

3.4 Sepsis-Associated Thrombocytopenia and Procedural Risk

Sepsis-associated thrombocytopenia is frequently encountered in critically ill patients and reflects a complex interaction between inflammation, coagulation activation, endothelial dysfunction, platelet consumption, immune-mediated destruction, and impaired platelet production [1, 6]. Importantly, platelet counts may decline rapidly over a short period of time, even in patients demonstrating apparent hemodynamic improvement. In the present case, the patient underwent spinal anesthesia during a phase of transient clinical stabilization. Clinical decision-making was based on laboratory values from the preceding day, which demonstrated a platelet count of 150 $\times 10^9$/L. However, retrospective review later revealed a platelet count of 63 $\times 10^9$/L on the day of the procedure.

Although precise platelet thresholds for neuraxial anesthesia remain debated, thrombocytopenia is widely recognized as an important risk factor for neuraxial hemorrhagic complications [16,19–21]. Recent systematic reviews and expert consensus statements suggest that procedural risk increases substantially at lower platelet levels, particularly in patients with rapidly evolving hematological abnormalities or additional coagulation disturbances [19–21]. This case highlights an important clinical principle: apparent hemodynamic stabilization following sepsis does not necessarily indicate normalization of coagulation-related procedural risk. Reliance on previously obtained laboratory values may therefore result in underestimation of bleeding risk. Reassessment of coagulation parameters immediately prior to neuraxial procedures is essential, particularly in critically ill patients recovering from septic shock or recent hemodynamic instability.

3.5 Overall Clinical Implications

This case illustrates the complex interaction between obstructive urosepsis, antimicrobial resistance, sepsis-associated thrombocytopenia, and a rare but severe complication of neuraxial anesthesia. While timely source control and appropriate antimicrobial escalation resulted in hemodynamic stabilization, underlying hematological instability persisted and likely contributed to the development of spinal intradural hematoma. From a broader clinical perspective, this case emphasizes the importance of dynamic risk assessment in critically ill patients. Physiological and laboratory parameters may evolve rapidly during the course of sepsis, requiring continuous reassessment and multidisciplinary coordination among anesthesiology, urology, intensive care, microbiology, radiology, and neurosurgery teams.

In resource-limited settings, delayed access to MRI and neurosurgical expertise may further compromise neurological outcomes. Heightened clinical vigilance, rapid recognition of neurological symptoms, and immediate intervention therefore remain essential. Ultimately, this case demonstrates that critically ill septic patients undergoing neuraxial procedures require careful reassessment of evolving coagulation abnormalities, even during apparent clinical stabilization.

4. Conclusion

This case highlights the complexity of managing septic shock secondary to obstructive uropathy complicated by antimicrobial resistance and a rare neuraxial hemorrhagic complication. Continuous reassessment of coagulation parameters prior to neuraxial procedures is crucial in critically ill septic patients. Apparent hemodynamic stabilization does not necessarily reflect normalization of coagulation-related procedural risk. New neurological deficits following neuraxial anesthesia should be regarded as a medical emergency requiring immediate imaging and urgent neurosurgical evaluation.

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Research Ethics Committee Approval: Written informed consent was obtained from the patient for publication of this case report and accompanying images. We declare that the study was conducted in accordance with the ethical principles established by the Declaration of Helsinki.

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Conflicts of Interest: All other authors declare no conflicts of interest.

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