

## Sepsis associated delirium mimicking postoperative delirium as the initial presenting symptom of urosepsis in a patient who underwent nephrolithotomy

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### Abstract

We report a case of 70 years old male who underwent percutaneous nephrolithotomy for renal calculi. After an uneventful recovery from anaesthesia, the patient developed delirium which manifested as restlessness, agitation, irritability and combative behavior. All other clinical parameters including arterial blood gas, chest X-ray and core temperature were normal and the patient remained haemodynamically stable. But 45 min later the patient developed florid manifestations of septic shock. He was aggressively managed in a protocolized manner as per the Surviving Sepsis Guidelines in the Critical Care Unit and recovered completely. There are no case reports showing postoperative delirium as the only initial presentation of severe sepsis, with other clinical parameters remaining normal. Both urosepsis and sepsis associated delirium have very high mortality. High index of suspicion and a protocolized approach in the management of sepsis can save lives.

**Key words:** Urosepsis; Nephrolithotomy; Postoperative delirium; Sepsis associated delirium; Delirium

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**Core tip:** Postoperative sepsis can initially manifest as delirium only, with other florid manifestations developing later. When delirium is the only initial manifestation, it should be treated with haloperidol, and benzodiazepines

are best avoided. Awareness that delirium can be the only initial presentation of sepsis, having a low threshold for its diagnosis after urological surgery and aggressive early management, can save lives.

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## INTRODUCTION

Percutaneous nephrolithotomy (PCNL) is indicated for large renal stones. Although complications after PCNL have been reported to be as high as 12.5%, the incidence of serious complication like severe sepsis with multiple organ failure, indicated by the Dindo-Clavien classification of surgical complications as IV-b, has been quite low<sup>[1]</sup> (0.1%). The incidence of postoperative delirium (POD) in various studies range from 5%-15%<sup>[2]</sup> but sepsis associated delirium (SAD) has been observed in 9%-71% of patients with severe sepsis<sup>[3]</sup>. Although delirium is a major component of sepsis and "mental dysfunction may even precede the cardinal findings of sepsis"<sup>[4]</sup>, there are no case reports of severe sepsis, initially presenting as SAD mimicking POD with normal hemodynamic parameters and near normal arterial blood gas (ABG). Severe sepsis as the cause of POD could be established only on development of shock and on further investigation. High index of suspicion, especially in urological surgeries, and aggressive management can save lives.

## CASE REPORT

A 70 years old male presented to our emergency department with abdominal pain in the right flank. Supine computed tomography scan of the abdomen and pelvis revealed large obstructing calculi (3 cm × 3 cm) at renal pelvis. Urine analysis showed plenty of pus cells and its culture showed *Escherichia coli* sensitive to ciprofloxacin. He was discharged on a course of oral ciprofloxacin. An elective PCNL was planned for him 3 wk later.

A preoperative kidney, ureter, and bladder X-ray was done on the day prior to surgery (Figure 1).

With the patient under general anesthesia the kidney was accessed through a puncture towards the upper calyx guided by fluoroscope. While no pus was noted during the procedure, the stone of 3 cm × 3.5 cm was noted to have two layers. A white soft outer layer and a hard dark coloured inner layer. The stone was fragmented through a 28 Fr tract by pneumatic lithotripsy and was completely extracted. At the end of the procedure a Double J ureteral stent was placed antegrade and 20 Fr

nephrostomy was placed. The intraoperative period was uneventful with stable hemodynamics during the entire duration of surgery lasting for 115 min. The time interval between the introduction of the nephroscope and the end of surgery was 85 min, during which about 10 L of normal saline was used as the irrigation fluid. At the end of the procedure he received 4 mg of intravenous ondansetron and 75 mg of intramuscular diclofenac sodium. Neuromuscular blockade was reversed with glycopyrrolate and neostigmine. Postoperatively he was fully awake and responding well to verbal commands. He was subsequently kept in the post-anaesthesia recovery area. His pulse rate was 76/min, blood pressure was 140/88 mmHg, and respiration was regular with no apparent discomfort. The respiratory rate remained 14-16/min and oxygen saturation (SpO<sub>2</sub>) remained 100% with supplementary oxygen 4 L/min by face mask. The patient was comfortable for the next 30 min and had no complaints.

Over the next 50 min of his stay in the post-anaesthesia recovery area he was pain free, interacted normally with the doctors and his relatives and was doing well. Then suddenly he started getting agitated and wanted to get up from the bed and go home. He did not complain of any pain at the operated site. In the next few minutes he exhibited restlessness, agitation, irritability and combative behavior. He became disoriented about the time and place, and did not want to acknowledge that he was in a hospital or the fact that he underwent a major surgery a few hours ago. He wanted to pull out the intravenous lines, nephrostomy tube and urinary catheter. Although no formal delirium assessment tools like the confusion assessment method or delirium rating scale<sup>[5]</sup> was administered, his clinical manifestations of disturbance of consciousness (agitation), change in cognition (wanted to get up and go home, did not acknowledge that he was in a hospital), its acute onset (he was normally responsive in the immediate postoperative period) and the fact that it developed in the postoperative period met all the all four criteria (A-D) required to confirm a diagnosis of delirium as per the "Diagnostic and Statistical Manual of Mental Disorders, 4<sup>th</sup> edn, text revision (DSM-IV-TR<sup>®</sup>; American Psychiatric Publishing, Inc., Arlington, VA)"<sup>[6]</sup>. His pulse rate at this time was 91/min, blood pressure 154/86 mmHg and had regular respiration with a rate of 20-21/min with no apparent discomfort. He maintained oxygen saturation (SpO<sub>2</sub>) of 100% with oxygen by face mask at 4 L/min. He was immediately sedated with midazolam 2 mg.

An ABG was done immediately and the report showed pH of 7.31, pCO<sub>2</sub> 29.3 mmHg, pO<sub>2</sub> 107.4 mmHg, lactate 0.9 mmol/L, bicarbonate 17.4 mmol/L and a base deficit of 7.0 mmol/L. The serum electrolytes were within the normal range. The Haemoglobin level was 13.1 g/dL. A chest X-ray done immediately was unremarkable and a 12-lead electrocardiogram was also normal.

However over the next 45 min his consciousness



Figure 1 A kidney, ureter, and bladder X-ray demonstrating the calculi.

level started deteriorating. He became drowsy and unresponsive with a glasgow coma scale (GCS) of E1, V1, and M3: 5/15. His pulse rate was 99/min, blood pressure of 124/76 mmHg, respiration became laboured with a respiratory rate of 24-25/min and the oxygen saturation (SpO<sub>2</sub>) dropped to 76%. Tracheal intubation was done and the patient was ventilated by manually with 100% oxygen. Over the next few minutes the patient developed overt shock with blood pressure of 93/66 mmHg and a pulse rate of 112/min with slightly warm extremities. Central venous access was secured and the central venous pressure (CVP) was observed to be low (2-3 cm of water). Since his blood pressure remained unresponsive to a 1 L of crystalloids transfused over the next 20 min, an infusion of dopamine at 6 µg/kg per minute was started. He was immediately transferred to the critical care unit (CCU).

The differential diagnosis of postoperative delirium includes arterial hypoxemia, preexisting cognitive disorder, hypoventilation with hypercapnia, metabolic derangements, drugs, alcohol withdrawal, electrolyte abnormalities, incomplete muscle relaxant reversal, seizures, acute central nervous event and infection<sup>[2,7]</sup>. All, except drugs and central nervous event, could be excluded from the available history or investigation.

In the CCU, he was mechanically ventilated and the ABG was repeated. The ABG revealed pH of 7.18, pCO<sub>2</sub> 35.5 mmHg, pO<sub>2</sub> 120 mmHg, lactate 8.1 mmol/L, bicarbonate 13.5 mmol/L and a base deficit of 13.9 mmol/L. Cardiac enzymes were within normal limits and an echocardiography showed good ejection fraction (69%). Although old age and many of the perioperatively used drugs including beta-blockers, narcotics, neostigmine can cause postoperative delirium, shock state, high serum lactate level along with lukewarm extremities and low CVP indicated Systemic Inflammatory Response Syndrome. The patient was empirically started on intravenous piperacillin and tazobactam 4.5 g 6 h. The core body temperature measured at nasopharynx was 34.9 °C. The complete blood picture revealed leucocytosis, a raised total count (31000 cells/cumm) with 82% neutrophils and toxic granules on a peripheral smear.

In the CCU, severe shock necessitated intravenous

infusion of crystalloids and inotropes (norepinephrine, epinephrine and dopamine). Inotropes were titrated with an aim to maintain a mean arterial pressure of > 65 mmHg. Over the next 24 h his conscious level gradually improved, core temperature normalised, inotropes were tapered and he was weaned from mechanical ventilation. Tracheal extubation was done 30 h after being shifted to the CCU. Despite negative blood and urine cultures, a diagnosis of severe postoperative urosepsis with septic shock was made and intravenous antibiotics were continued for 7 d.

He was shifted to the ward on the 4<sup>th</sup> postoperative day and discharged from the hospital on the 7<sup>th</sup> day on a 10 d course of oral levofloxacin. He had an uneventful course subsequently and the Double J ureteral stent was removed one month later. Over the next one year he was followed up in the urology clinic and remained asymptomatic.

## DISCUSSION

Delirium is defined as a "transient, usually reversible cause of cerebral dysfunction and manifests clinically with a wide range of neuropsychiatric abnormalities"<sup>[8]</sup>. Postoperative delirium should be differentiated from "emergence from anaesthesia"<sup>[9]</sup>, and by definition "do not have an identifiable aetiology"<sup>[2]</sup>. In our patient, the presentation of restlessness, agitation, irritability and combative behavior appeared about an hour after complete recovery from anaesthesia (50 min after being shifted to the post-anaesthesia recovery area). All the possible causes of distress experienced by the patient were excluded by the available investigations. Due to initial hemodynamic stability, normal core temperature and a serum lactate level of 0.9 mmol/L, sepsis was not suspected as the immediate cause of delirium. Normal respiratory rate, pattern, oxygen saturation, ABG and chest X-ray ruled out the possibility of hypoxemia and an ABG based electrolyte assessment also excluded dyselectrolytemia. Our patient's age and the perioperative use of narcotics, benzodiazepines, cholinesterase inhibitors and drugs with anticholinergic properties initially led us to conclude POD as the cause of the disturbing symptoms<sup>[7]</sup>. However, for treating POD, the drug of choice should have been haloperidol<sup>[7]</sup>. In fact sedatives like benzodiazepines have the potential to aggravate POD<sup>[7]</sup>.

POD is a diagnosis of exclusion<sup>[10]</sup>. The multiple risk factors are illustrated in Table 1<sup>[2,7,9,10]</sup>. Although POD can manifest any time during the perioperative period, with or without any lucid interval, unlike in our patient, it most commonly develops between the 1<sup>st</sup> and 3<sup>rd</sup> postoperative day<sup>[2]</sup>. Modifying or reducing the potentially avoidable perioperative triggers consisting of pain, physical restraints, narcotics or benzodiazepines, anaemia or blood transfusion and urinary catheter can only reduce its risk<sup>[2]</sup>. Haloperidol, a D2 dopamine receptor antagonist, remains the drug of choice for treating POD<sup>[2]</sup>. It is used as an intravenous injection of

**Table 1 Risk factors for postoperative delirium<sup>[2,7,9,10]</sup>**

Age > 65-70 yr
Pre-existing dementia, physical or cognitive impairment
Tobacco or alcohol use
Metabolic derangements (dysselectrolytemia)
Hypoxia, hypercarbia, hypotension
Sepsis
Drug withdrawal
Nature of surgery (cardiac surgery)
Use of certain drugs (narcotics, benzodiazepines, cholinesterase inhibitors and drugs with anticholinergic properties)
Physical restraint
Sleep deprivation
Pain
Anaemia
Urinary catheter

0.5-1 mg every 15 min till the resolution of symptoms. Two to 10 mg titrated over 60-90 min is needed for most patients<sup>[2]</sup>. Careful titration is important as higher doses are associated with over-sedation for prolonged periods<sup>[2]</sup>.

Based on the clinical presentation and in the absence of suggestive investigations, we initially diagnosed the case as POD, but the subsequent manifestations revealed that we were actually dealing with a patient of SAD. SAD is manifested by "acute onset of impaired cognitive function"<sup>[3]</sup> with symptoms ranging from restlessness, irritability and agitation at one end of the spectrum to sluggish mentation, inattention, stupor and coma at the other end<sup>[3]</sup>. Although the incidence of SAD has been reported to be 9%-71%<sup>[3]</sup>, if electro-physiological testing is used to diagnose it, its evidence may be found in almost all cases with severe sepsis<sup>[3]</sup>. SAD manifests early in the course of sepsis, but to diagnose SAD, neurologic dysfunction must be correlated to infection or systemic inflammation<sup>[3]</sup>. SAD continues to remain as an independent predictor of death<sup>[11]</sup>. In SAD mortality increases from 16% to 63% with fall in GCS from 15 to < 8<sup>[3]</sup>.

Septicemia has been infrequently observed after PCNL with an incidence of 0.9%-4.7%<sup>[12]</sup>. Life threatening complication like severe sepsis with multiple organ dysfunction is extremely rare (0.1%)<sup>[11]</sup>. It can occur if infection is introduced during access to the kidney or if the stones are infected<sup>[13]</sup>. It is mandatory to give prophylactic antibiotics and drain a pyonephrotic kidney before doing a PCNL<sup>[13]</sup>. The volume of irrigation fluid used, duration of surgery (> 90 min), pre-existing renal insufficiency and high pressure in the renal collecting system also increases the risk of developing sepsis<sup>[13]</sup>. Although the blood and urine cultures post-operatively were negative in our patient, the clinical features (severe shock, low CVP and hypothermia) and investigations (leucocyte count, ABG) led us to a diagnosis urosepsis. The negative blood and urine cultures could have been due to use of preoperative antibiotics and administration of intravenous piperacillin and tazobactam before the cultures could be sent. In

our case, white soft outer layer of the stone could have consisted of infective debris. However, culture of the stone was missed which probably could have helped in modifying the choice of antibiotics after the culture report. But our patient responded to empirical piperacillin and tazobactam and had shown remarkable clinical improvement by the 3<sup>rd</sup> day. The mortality of urosepsis is high (20%-40%)<sup>[14]</sup>. Identifying severe sepsis early is the biggest challenge and remains the "greatest barrier to implementing the guidelines"<sup>[15,16]</sup>. Early (immediate) initiation of goal directed therapy with particular focus on rapid administration of appropriate antibiotic has been shown to reduce mortality<sup>[17]</sup>.

There is experimental evidence to show that in the initial phase of sepsis, endothelial nitric oxide (NO) synthase derived NO demonstrates "proinflammatory characteristics and contributes to the activation and dysfunction of cerebrovascular endothelial cells"<sup>[18]</sup>. Sepsis is also associated with "mitochondrial dysfunction" and early sepsis can cause cytokine, reactive oxygen species and NO mediated "decrease in mitochondrial ATP generation"<sup>[18]</sup>. This can result in "neural cell apoptosis and an insufficient energy supply to the neurons"<sup>[18]</sup>. The exact mechanism causing delirium is complex and involves the neurological impact arising out of the immune response causing "prolonged inflammation, brain cells activation, over expression of NO, dysfunction of intracellular metabolism and cell death"<sup>[3]</sup>. However, to the best of our knowledge, no case report has ever noted delirium presenting as the only initial manifestation of severe sepsis. None of the causes of neurological symptoms explain why it would only manifest itself without the common clinical signs of severe sepsis like temperature derangement, significant tachycardia or tachypnoea, hyperglycaemia (in absence of diabetes) or any other adverse impact on haemodynamic variables, organ dysfunction or tissue perfusion. Any laboratory investigation like the leucocyte count, plasma C-reactive protein or plasma procalcitonin would delay the diagnosis. Aggressive management based on the clinical signs and symptoms of sepsis without waiting for any laboratory evidence resulted in a positive outcome in our patient.

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## COMMENTS

### Case characteristics

A 70 years old male underwent percutaneous nephrolithotomy under general anaesthesia and developed delirium after complete recovery from anaesthesia.

### Clinical diagnosis

Sepsis associated delirium (SAD).

### Differential diagnosis

He was initially diagnosed as postoperative delirium due to absence of any other manifestation of sepsis.

### Laboratory diagnosis

Subsequent development of severe metabolic acidosis with elevated serum lactate and a complete blood picture revealing leucocytosis with toxic granules on a peripheral smear indicated that the patient's symptoms of delirium was the initial manifestation of impending urosepsis.

### Imaging diagnosis

A normal chest X-ray and echocardiography showing good ejection fraction (69%) excluded respiratory or cardiac cause for the developing symptoms.

### Treatment

The patient managed with organ support and intravenous antibiotics in the critical care unit.

### Related reports

The clinical presentation initially led the authors to a diagnosis of postoperative delirium; however the subsequent manifestations of Systemic Inflammatory Response Syndrome (SIRS) revealed that they were actually dealing with a patient of SAD. The exact mechanism causing delirium is complex and involves the neurological impact arising out of the immune response.

### Term explanation

SAD has an acute onset and is a potentially reversible organic brain dysfunction in patients with SIRS or sepsis. Postoperative delirium is a diagnosis of exclusion and can be attributed to any delirium in the postoperative period which does not have an identifiable cause.

### Experiences and lessons

Urosepsis can initially manifest as delirium without any other common clinical signs of severe sepsis. Haloperidol is the drug of choice in delirium and benzodiazepines are best avoided. Aggressive management of sepsis without waiting for any laboratory evidence can result in better outcomes.

### Peer-review

This is a rare and very interesting case report.

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