J Anesth (2002) 16:242–244

Journal of
Anesthesia



*Clinical reports*

# A case of sepsis that developed during transurethral resection of the prostate

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**Key words** Sepsis · Intraoperative · Complication · TURP

## Introduction

Sepsis is a life-threatening complication of transurethral resection of the prostate (TURP) in the postoperative period [1]. Recent investigations have shown that the incidence of sepsis following TURP is 2.3%–8.0% [2,3]. Although sepsis that develops after TURP is well known, our case showed the initial appearance of septic signs and an atypical hemodynamic presentation during TURP. In this report, we describe a case that showed incipient sepsis during TURP, which developed into septic shock several hours after the operation.

## Case report

A 78-year-old man (height 163 cm, weight 64 kg) was admitted to our hospital with dysuria. He reported having frequent urination and nocturia over the last 2 years. His serum prostate-specific antigen had been slightly elevated a few months before admission. The patient underwent transrectal prostatic biopsy under spinal anesthesia, and a diagnosis of benign prostatic hyperplasia (BPH) was made. A urinary catheter was inserted and kept in place for 8 days. Enoxacin was administered prophylactically. The results of a culture of urine obtained after the biopsy were negative. Ten days later, TURP was scheduled to treat the obstructing BPH. It was noted that the patient had a history of cerebral infarction with no remaining neurological deficit. He was otherwise in good health.

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Received: May 8, 2001 / Accepted: January 16, 2002

On arrival in the operating room, the patient's blood pressure, heart rate, and body temperature were 133/77 mmHg, 69 b.p.m., and 37.0°C, respectively. Spinal anesthesia was induced with 10 mg hyperbaric tetracaine at the L3–4 interspace. A bilateral analgesic level to T8 was obtained. Electrocardiogram (ECG) readings, noninvasive blood pressure, and hemoglobin oxygen saturation ($SpO_2$) were monitored. Intraoperative blood samples were also obtained to measure the serum sodium level, hemoglobin, and hematocrit, and to perform arterial blood gas analysis. Supplemental oxygen was administered by a face mask ($3 l \cdot min^{-1}$), and the patient's $SpO_2$ was stable (98%–100%). Throughout anesthesia induction and the start of surgery, his systolic blood pressure ranged from 100 to 140 mmHg, and his heart rate ranged from 66 to 81 b.p.m.

Sixty minutes after starting the resection, the patient complained of sudden onset of dyspnea. Simultaneously, he became restless. Shivering in the upper body and upper limbs was observed. His heart rate simultaneously increased from 70 to 90 b.p.m. The ECG wave form was unchanged. His serum sodium level, hemoglobin, and hematocrit were $132 mEq \cdot l^{-1}$, $11 g \cdot dl^{-1}$, and 33%, respectively. His systolic blood pressure and heart rate gradually increased with the presence of tachypnea. The resection was terminated 70 min after the start of the operation. The resected mass weighed 25 g. A few prostatic sinuses were opened during the procedure. At the end of the procedure, the patient's systolic blood pressure was 160 mmHg and his heart rate was 120 b.p.m. Arterial blood gas findings showed serum bicarbonate of $17 mEq \cdot l^{-1}$ and pH of 7.34, $PaCO_2$ of 32 mmHg, $PaO_2$ of 120 mmHg, and base excess of $-7.6 mEq \cdot l^{-1}$ (oxygen administered at $6 l \cdot min^{-1}$ using a face mask). At this point, we recognized that the patient was hyperthermal to the touch, and his body temperature was 40.2°C. A diagnosis of sepsis was suspected. We immediately started to cool his body using ice-cold water. His blood pressure returned to baseline level

after two doses of 1 mg midazolam had been administered intravenously (IV). At this time, the ECG showed a sinus tachycardia of 130 b.p.m. Arterial blood gas analysis showed serum bicarbonate of 18 $mEq \cdot l^{-1}$ and pH of 7.34, $PaCO_2$ of 30 mmHg, $PaO_2$ of 165 mmHg, and base excess of $-5.6\,mEq \cdot l^{-1}$ (oxygen administered at $10\,l \cdot min^{-1}$ using a face mask). The patient was initially treated with dibekacin sulfate, 100 mg, soon after a blood sample was taken for culture. A central line was inserted into his right femoral vein. He was then transferred to the intensive care unit (ICU) for subsequent cooling and close observation.

On arrival at the ICU, the patient was conscious and oriented, with a blood pressure of 116/68 mmHg and a heart rate of 100 b.p.m. Central venous pressure was 11.6 $cmH_2O$. His lungs were clear, and $SpO_2$ was 100% while breathing $10\,l \cdot min^{-1}$ $O_2$ by a face mask. However, his respiration was still agonal. The sensory level of the block remained on T10. His body temperature was 40.2°C. The cooling was discontinued 5 h after the operation as his body temperature had returned to 37°C. The patient's condition remained stable, with systolic blood pressure ranging from 110 to 120 mmHg and heart rate ranging from 100 to 120 b.p.m., over a period of about 4 h. Thereafter, his heart rate increased to 130 b.p.m. A short time later, systolic blood pressure decreased to 59–61 mmHg for a period of about 10 min. Ephedrine (10 mg) was administered IV. A fluid bolus of 500 ml colloid was given. Another 10 mg ephedrine was administered IV. Infusion of dopamine was started at $10\,\mu g \cdot kg^{-1} \cdot min^{-1}$. Thirty minutes after the start of dopamine infusion, his blood pressure had returned to approximately 90/60 mmHg. The patient maintained a normal sinus rhythm, ranging from 100 to 120 b.p.m., throughout the hypotensive episode. His hematocrit was 27% before this event. The patient received 2 units of packed red cells. Dopamine ($10\,\mu g \cdot kg^{-1} \cdot min^{-1}$) and norepinephrine ($0.01–0.08\,\mu g \cdot kg^{-1} \cdot min^{-1}$) were required to maintain his systolic blood pressure at above 100 mmHg for the next 17 h. Dopamine and norepinephrine dosages were tapered over the next 25 h.

Laboratory data the next day showed an increase in his white cell count ($26\,500\,mm^{-3}$) with premature neutrophilia, thrombocytopenia ($79\,000\,mm^{-3}$), and increased C-reactive protein ($21.20\,mg \cdot dl^{-1}$). The patient was treated with cefozopran hydrochloride at a dose of $2\,g \cdot day^{-1}$ and isepamycin sulfate at a dose of $400\,mg \cdot day^{-1}$ for 4 days. Gram-negative *Enterobacter cloacae* (*E. cloacae*) was isolated from cultures of blood obtained in the operating room. These clinical symptoms and the laboratory data indicated that the patient had developed incipient sepsis during TURP followed by septic shock.

On the third postoperative day, the patient was hemodynamically stable and was transferred to the ward. He was discharged uneventfully 2 weeks after the operation.

## Discussion

Urosepsis is a rare event, and usually becomes evident in the postoperative period [1–3]. Although sepsis that develops after TURP is well known, this case showed the initial appearance of septic signs and an atypical hemodynamic presentation during TURP, which is relatively uncommon.

A possible explanation for the onset of sepsis in the present case is that a preexisting infection of the prostate was exacerbated by the surgical intervention. It has been reporterd that prostatic bacteria are prevalent in patients undergoing prostatic surgery for obstructive symptoms. However, most patients with positive prostatic cultures show neither bacteriuria nor a positive preoperative urine culture [4]. In our case, although preoperative urine cultures were negative, the isolation of *E. cloacae* from blood culture suggests that there was a considerable amount of the bacteria in the prostatic gland. *E. cloacae* is frequently isolated from patients with clinically infected prostates [5]. The risk of prior prostatic infection is increased by the presence of an indwelling urinary catheter that has been inserted after transrectal biopsy in such patients [1,5]. A duration of more than 70 min for the surgical procedure is a significant postoperative risk factor for infection [2].

There is minor extravasation of the prostatic capsule during TURP, which is of little clinical significance, and bacteremia is usually asymptomatic [6]. Transient bacteremia with positive blood cultures during TURP disappears after a few hours [7]. In this case, it seems that perforation of the prostatic capsule and manipulation of the previously infected prostate caused dissemination of a considerable excess of bacteria into the blood stream, known as intraoperative septic showers, which developed into sepsis several hours postoperatively.

Early manifestations of sepsis are tachypnea, tachycardia, and a change in the mental state, followed by fever and hypotension [8]. However, the initial appearance of septic symptoms is not always clinically obvious, especially in elderly patients. In our case, the early-stage septic reaction may have occurred simultaneously with the signs and symptoms attributed to TURP syndrome [9,10]. Except for fever and tachycardia, the patient's multiple symptoms were interpreted as indicating TURP syndrome. Generally, the body temperature during TURP is not continuously monitored. Touching the patient and monitoring body temperature occasionally are essential in the anesthetic management of a patient with TURP in order to be able to make an early diagnosis of intraoperative septic shower and septic symptoms.

Although intraoperative septic showers are events that most clinicians have witnessed, the diagnosis is difficult.

The atypical hemodynamic presentation in our patient could have been related to rapid volume shifts [8,10]. Intraoperative hypertension may be related to irrigant absorption during TURP. This volume expansion might mask the initial hemodynamic instability (hypotension) attributed to sepsis, and postoperative severe hypotension may occur several hours later.

In summary, this case has suggested that preexisting infection could play crucial role in the development of sepsis during TURP, resulting in postoperative septic shock.

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