Transurethral prostatectomy complicated by intraperitoneal extravasation of irrigating fluid

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A patient underwent transurethral resection of the prostate (TURP), which was complicated at the outset of the procedure by an inadvertent puncture wound of the dome of the bladder and the peritoneum. Shortly after resection was initiated, the patient developed shoulder pain and a tensely distended abdomen, although at this time the serum sodium concentration remained normal. Over the next several hours the patient developed significant hyponatremia. The prolonged and gradual time course of this development suggests that fluid and electrolyte shifts occurred via diffusion across the peritoneal membrane. This case illustrates a rare but potentially dangerous complication of TURP that requires recognition prior to initiation of appropriate therapy.

The "TUR syndrome" is a well described complication of transurethral resection of the prostate (TURP).^{1,2} The symptomatology is believed to be due to intravascular absorption of bladder irrigation fluid, which may then result in acute water intoxication. Dilutional hyponatremia and hyposmolality are thought to be the underlying mechanisms for the symptom complex which can include agitation, confusion, nausea, vomiting, coma, and possibly death. Absorption can occur not only through open prostatic venous sinuses, but via other routes, such as surgically created defects in the prostatic capsule or perforations

of the bladder.^{3,4} Such occurrences can allow significant volumes of irrigation solution to be sequestered in the perivesical and retroperitoneal spaces.^{1,5,6} We describe a case in which a patient developed the gradual onset of severe hyponatremia over a period of several hours after a TURP complicated by bladder perforation and intraperitoneal extravasation of irrigation fluid. This demonstrates a rare variant of the "TUR syndrome," in which the time course for pathophysiological changes seems to be considerably prolonged. Appropriate diagnosis and intervention by the anaesthetist is important to prevent unnecessary morbidity and mortality in this situation.

Case report

A 63-year-old 70 kg man underwent TURP. His history included insulin dependent diabetes mellitus treated with daily split doses of lente insulin. His spot blood glucose values were less than 11.1 mmol·L⁻¹ for several days preoperatively. Other medications included indomethacin and amitriptyline. He received no preoperative intravenous fluid therapy. Other preoperative laboratory values included: sodium 141 mmol·L⁻¹, potassium 3.9 mmol·L⁻¹, blood urea nitrogen 5.6 mmol·L⁻¹, creatinine 239 µmol·L⁻¹, and haemoglobin 94 g·L⁻¹. His pre-anaesthetic arterial blood pressure was 130/70 mmHg, with a pulse rate of 100 beats/min. Preoperative medications included meperidine 50 mg and promethazine 25 mg IM, and lente insulin 25 units SC (half of usual morning dose).

Intravenous therapy with five per cent dextrose in lactated Ringer's solution was initiated. Spinal anaesthesia with tetracaine 12 mg in hyperbaric dextrose solution was successfully performed with a 22 g needle at the L₂-L₃ interspace. Anaesthesia up to the T₁₀ dermatome was obtained without significant change in blood pressure or pulse rate. After 15 minutes of resection the patient complained of bilateral shoulder and neck pain. At this time it was noted that the arterial blood pressure had decreased to 90/60 mmHg, with a pulse rate of 92. The anaesthetist examined the patient and discovered that he had a tensely distended, non-tender abdomen. No

Key words

SURGERY: transurethral resection of prostate; COMPLICATIONS: bladder perforation; hyponatremia.

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change in ventilatory pattern was noted. The surgeon was notified and the operation immediately halted. Four litres of three per cent sorbitol irrigation had been used during the procedure, but only about one and a half litres were recovered. A presumptive diagnosis of bladder and peritoneal perforation was made. Laboratory results at this time showed: sodium $134 \text{ mmol} \cdot \text{L}^{-1}$, potassium $3.6 \text{ mmol} \cdot \text{L}^{-1}$, and serum glucose $15 \text{ mmol} \cdot \text{L}^{-1}$. A Foley catheter was inserted, and the patient was taken to the Recovery Room for further evaluation. He was awake and alert, with continued shoulder pain.

The patient was observed closely for several hours in the Recovery Room while the urology team evaluated his condition. Two and a half hours after the first intraoperative laboratory values, the tests were repeated. At this time sodium was $123 \text{ mmol} \cdot \text{L}^{-1}$, glucose $28.3 \text{ mmol} \cdot \text{L}^{-1}$, and potassium $3.7 \text{ mmol} \cdot \text{L}^{-1}$. Because of the patient's hyperglycaemia, we calculated the value of serum sodium that would maintain the same osmolality with a normal blood sugar (i.e., adding $1.6 \text{ mmol} \cdot \text{L}^{-1}$ to the sodium value for each $5.5 \text{ mmol} \cdot \text{L}^{-1}$ of glucose above the first $5.5 \text{ mmol} \cdot \text{L}^{-1}$). The adjusted sodium value was $129 \text{ mmol} \cdot \text{L}^{-1}$. The patient's urine output after three hours in the Recovery Room totalled 15 ml, and furosemide 10 mg was administered intravenously. The systolic arterial blood pressure ranged between 120 and 95 mmHg, with pulse rates ranging between 80 to 100.

Two and a half hours later laboratory tests were again repeated. Sodium was $113 \text{ mmol} \cdot \text{L}^{-1}$ ($119 \text{ mmol} \cdot \text{L}^{-1}$ when adjusted for hyperglycaemia), glucose $29.6 \text{ mmol} \cdot \text{L}^{-1}$, potassium $4.0 \text{ mmol} \cdot \text{L}^{-1}$, and haemoglobin $117 \text{ g} \cdot \text{L}^{-1}$. No evidence of haemolysis was found. The patient was awake, but restless and nauseated. His urine output was still minimal despite repeated doses of furosemide. A cystogram showed bladder, peritoneum, and colon perforation. At this time he was returned to the Operating Room for exploratory laparotomy.

After arterial and central venous pressure monitoring was established (initial CVP 3 mmHg), rapid sequence induction was performed without difficulty. The initial intraoperative sodium was $115 \text{ mmol} \cdot \text{L}^{-1}$ ($121 \text{ mmol} \cdot \text{L}^{-1}$ when adjusted for hyperglycaemia). Following incision, nearly three litres of clear fluid were removed. Chemical analysis for electrolyte and glucose concentration was not performed. Inspection of the dome of the bladder showed a small perforation, and it was hypothesized that a catheter guide inserted at the outset of the TURP had perforated the bladder dome and entered the colon. The defect was repaired, a loop colostomy was performed, and a suprapubic cystostomy tube placed. Three per cent saline solution 320 ml was given over the next two hours, as well as furosemide 20 mg IV. Immediately after infu-

sion of the three per cent saline solution, the sodium was $117 \text{ mmol} \cdot \text{L}^{-1}$ ($123 \text{ mmol} \cdot \text{L}^{-1}$ when adjusted for hyperglycaemia). Central venous pressure rose to 5 mmHg. Intraoperative urine output, after the suprapubic cystostomy tube placement, was 400 ml.

Postoperatively, the patient was brought to the Intensive Care Unit. Continued therapy consisted of vigorous hydration with normal saline and Plasmanate, and continuous infusion of regular insulin at $10 \text{ units} \cdot \text{hr}^{-1}$ until adequate control of the hyperglycaemia was obtained. Antibiotics were started to prevent peritonitis. One hour after the operation, sodium was $120 \text{ mmol} \cdot \text{L}^{-1}$ ($126 \text{ mmol} \cdot \text{L}^{-1}$ when adjusted for hyperglycaemia), and glucose was still $29.4 \text{ mmol} \cdot \text{L}^{-1}$. The patient improved gradually throughout the night, and by the following morning his sodium was $133 \text{ mmol} \cdot \text{L}^{-1}$ ($135 \text{ mmol} \cdot \text{L}^{-1}$ adjusted for hyperglycaemia) and his glucose was $14.3 \text{ mmol} \cdot \text{L}^{-1}$. The remainder of the postoperative course was uneventful, with normal values for serum sodium and glucose, and the patient attained full recovery.

Discussion

Perforation of the dome of the bladder during TURP can be associated with intraperitoneal extravasation of irrigation fluid. We found that this can cause metabolic derangement with a time course significantly different from that usually seen in the TURP water intoxication syndrome.

There are no reports in the literature on the incidence of bladder perforation with intraperitoneal extravasation of fluid, but it has been reported as a rare complication.³ A study of 2015 TURPs found 23 cases of extravasation (1.1 per cent), without noting the relative incidence of intraperitoneal versus extraperitoneal extravasation.⁴ In a more recent study of 511 successive TURPs a postoperative cystogram was routinely done. A total of 66 perforations of the bladder neck were found (13 per cent), and of these 45 would have gone undetected except for the cystogram (nine per cent).⁷ No perforations of the bladder dome were found. These studies present no evidence that bladder neck perforation (a more common occurrence) can be associated with the clinical situation described above for bladder dome perforation.

In this case, perforation of the bladder dome resulted in the placement of nearly 3 L of three per cent sorbitol solution into the patient's peritoneal cavity. The clinical presentation included shoulder pain (probably due to diaphragmatic irritation), a moderate decrease in blood pressure, and a tense, distended, non-tender abdomen. Although initial measurement of serum sodium showed a minimal decline (from 141 to $134 \text{ mmol} \cdot \text{L}^{-1}$), subsequent measurements showed a continuous gradual decrease over a period of six hours, reaching a minimum value of

113 mmol·L⁻¹. Treatment with furosemide resulted in minimal urine output through the Foley catheter, possibly due to urine extravasation from the bladder into the peritoneum. The patient suffered from nausea, restlessness, and shoulder pain, but remained haemodynamically stable and did not lapse into confusion or coma. His hyperglycaemia was probably stress-related, as he was a brittle, insulin-dependent diabetic. After a six-hour period of evaluation and unsuccessful conservative therapy, the patient's abdomen was surgically explored. Removal of the peritoneal fluid, placement of a suprapubic cystostomy catheter, and hypertonic saline administration led to reversal of the hyponatremia.

This case illustrates several important points about the diagnosis and treatment of this complication of TURP. Some of these points were made by Desmond⁸ in a review of this topic. Regional anaesthesia facilitates the early detection of mental status change or diaphragmatic irritation. Also, careful measurement should be made of the volume of irrigation solution entering and exiting the patient's bladder, and the abdomen should be intermittently examined for signs of distention. The operating urologist may note an abnormal irrigating pattern when this situation occurs. Such findings should trigger immediate investigation.

There are differences of opinion concerning the need for, and the timing of, surgical intervention in such cases. Mulkey and Witherington⁹ presented eight cases of vesical rupture that were successfully managed with only an indwelling urethral catheter and antimicrobial therapy. Others^{2,10,11} have presented similar findings. Kenyon² suggests that in the case of extensive perivesical extravasation or intraperitoneal involvement, a urethral catheter should be introduced as soon as the diagnosis is made and the patient should receive supportive treatment for four to six hours. After this time, surgical intervention may be carried out more safely. The urologic surgeons at our institution elected to attempt conservative treatment of the bladder perforation. However, the subsequent discovery of a colon perforation by the cystogram made exploratory laparotomy mandatory.

The finding of a slow and gradual development of hyponatremia was unexpected. Because the pathophysiology leading to such hyponatremia is probably not unique to this case, we now believe that the diagnosis of bladder perforation with intraperitoneal extravasation of irrigation solution calls for therapy including immediate removal of the fluid. Such action will probably prevent the gradual onset of hyponatremia. In contrast, treatment of the developing hyponatremia with furosemide (especially prior to placement of a suprapubic cystostomy catheter) may not be helpful because extravasation of urine into the

peritoneum can occur. The net effect on hyponatremia due to vigorous renal sodium and water excretion followed by peritoneal reabsorption of the resulting fluid is unpredictable.

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Résumé

Un patient a subi une résection transurétrale de sa prostate (TURP) s'étant compliquée par une perforation accidentielle du sommet de la vessie et du péritoine. Peu après le début de la résection le patient a développé une douleur à l'épaule ainsi qu'un abdomen tendu. A ce stade le sodium sérique était normal. Au cours des heures suivantes le patient a développé une hyponatrémie significative. Le temps d'installation prolongé et graduel de cette hyponatrémie suggère que le transfert de liquide et électrolytes est survenu par diffusion à travers le péritoine. Ce cas illustre une complication rare et potentiellement dangereuse de la résection transurétrale de la prostate et dont la détection doit se faire avant le début du traitement adéquat.