Hyponatremia And Neurological Manifestations of TURP Syndrome

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Citation

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Abstract

The absorption of large volumes of bladder irrigation fluid during transurethral resection of prostate can produce a complication known as transurethral resection of prostate syndrome (TURP). We compared two patients who have transurethral resection syndrome. We report about a 75-year-old man with postoperative Na+ level of 111mEq/lt and mild CNS symptoms, as well as a 67-year-old man with postoperative Na+ level of 115mEq/lt and severe CNS symptoms. We think that propably hyponatremia may not be the only cause of CNS signs of TURP.

MAY HYPONATREMIA BE THE ONLY CAUSE FOR NEUROLOGICAL MANIFESTATIONS OF TUR SYNDROME?

TUR syndrome is a term applied to a constellation of symptoms and signs caused primarily excessive absorption of the irrigating fluid [1]. Irrigant absorption may occur in up to 46% of resections [2] with 5-10% of patients absorbing 1 litre or more [3, 4] . TUR syndrome is observed in 2-10% of all prostate resections [5,6,7]. Neurologic manifestations, such as restlessness, agitation, confusion, altered sensorium, seizure, and coma, result from water intoxication and dilutional hyponatremia, which collectively produced cerebral edema. The cardiovascular effects reflect volume overload and hyponatremia. If serum sodium levels rapidly decrease to less than 120 mEq/L, negative inotropic effects are manifest as hypotension and ECG changes of widened QRS complexes, ventricular ectopy[1], ST-segment depression, or T-wave inversions [8].

We report two patients with TUR syndrome. Suprisingly, not only the classic neurologic symptoms but also the other sypmtoms of TUR don't show parallelism with the hyponatremia levels. Despite the extreme hyponatremia, clinical manifestations were mild.

CASES 1 AND 2

Transurethral prostat resection was performed in both patients under total intravenous anaesthesia. Standart monitoring (5-lead electrocardiograph, noninvasive blood pressure, pulse oximeter, end-tidal carbon dioxide using Datex-Ohmeda S/5 monitor) was applied on arrival in the operating theatre. At induction of anaesthesia, the patients received a bolus of remifentanil 0.5 µg/kg i.v. followed by a bolus of propofol 1.5 mg/kg i.v. Both patients received vecuronium 0.1 mg/kg to produce neuromuscular block. Maintance was with remifentanil (0.1-0.5 µg/kg/min) and propofol (3-6 mg/kg/h) i.v. infusions and %66 nitrous oxide in oxygen. The bladders were irrigated with 1.5% glycine solution.

A 75-year-old man with prostatic hypertrophy underwent transurethral resection of the prostate. He had no history of angina, stroke, or myocardial infaction. Laboratory studies revealed Na^+ 140 mmol/L, K^+ 3.55 mmol/L, creatinine kinase 53 U/L, creatinine kinase MB 10 U/L. ECG was normal.

During the surgery his blood pressure decreased from 138/79 to 73/50 mm Hg. Hemodynamic variations were first treated by changing the anesthetic (remifentanil-propofol) infusion rate. Then he was given Ephedrine (5 mg) and a fluid bolus (500 ml). The ECG revealed 3 mm of anterolateral ST-segment elevation in lead V5 for 15 min. Surgery was stopped and the bladder irrigation fluid was changed to normal saline solution. The procedure lasted 83 min and 35.5 litres of irrigation solution were used. The prostatic tissue resected weighted 35 g. In the recovery room, despite a sodium of 111 mmol/L, the patient was alert and oriented. Ten hours after the procedure the patient had nausea. He developed bradycardia, hypotermia and hypotension. The patient reported “dark and blurry” vision. The patient was

treated with hypertonic saline. 24 hours after the procedure, ECG was normal, visual changes resolved, and serum Na^+ was 133 mmol/L. The patient was discharged 3 days later with normal creatinine kinase and creatinine kinase MB levels.

A 67-year-old man had an elective transurethral resection of the prostate. He had an history of hypertension that was under control. His preoperative serum sodium was 141 mmol/L. Laboratory studies revealed K^+ 4.72 mmol/L, creatinine kinase 60 U/L, creatinine kinase MB 12 U/L. ECG was normal. During the surgery his blood pressure decreased from 161/83 to 97/58 mm Hg. The prostatic tissue resected weighted 29g. The procedure lasted 60 min and 21 litres of irrigation solution were used.

While in the recovery room the patient became agitated and confused. He had nausea and vomiting and developed hypotermia, bradycardia, hypotension. His serum sodium concentration was down to 115 mmol/L. ECG was normal. The patient was treated with a loop diuretic and hypertonic saline. 24 hours after the procedure, ECG was normal and serum Na^+ was 133 mmol/L. 36 hours after procedure the patient's condition improved and he was discharged 5 days later with normal creatinine kinase and creatinine kinase MB levels.

DISCUSSION

At sodium levels below 115 mEq/L, electrocardiographic changes are made manifest by QRS widening and ST-segment elevation [1].

In our first case, sodium level was 111 mEq/L so the ECG revealed 3 mm of anterolateral transient ST-segment elevation in lead V5.

The central nervous system (CNS) symptoms, which include irritability, apprehension, confusion, and headache, provide early warnings signs of rapidly developing hyponatremia. They become apparent at sodium levels below 120 mEq/L and further progression of hyponatremia (sodium less than 102 mEq/L) and decreased serum osmolality lead to the development of seizures and coma [1]. But classic CNS signs of TURP are not caused by the hyponatremia per se but due to the accompanying acute serum hypo-osmolality allowing water movement into the cells causing cerebral edema [1, 9].

Hyperammonaemia may occur in patients who have absorbed large quantities of glycine solution. The increase in blood ammonia is correlated with a decrease in serum sodium[10].

The above cases shows that hyponatremia may not be the only cause for neurologic manifestations of TUR syndrome. Despite the extreme hyponatremia, in our first case, neurological manifestations were mild when compared with the second one. Perhaps cerebral edema or hyperammonaemia didn't develop in first one.

So we think that propably hyponatremia may not be the only cause of CNS signs of TURP.

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