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Case report

Delayed Manifestation of Transurethral Syndrome as a Complication of Transurethral Prostatic Resection

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Abstract

Metabolic encephalopathy as a part of 'transurethral syndrome' is an immediate complication following transurethral resection of prostate. It occurs during or few hours after the surgery. (1) However, delayed manifestation of this complication is rare. It is also possible that pretreatment with diuretics can exaggerate this problem by predisposing the patient to electrolyte abnormalities. Here we present a report of such a patient who manifested with neurological complications six days after the prostate surgery.

Case Report

Seventy four year old male was operated for prostate enlargement. He was a known case of hypertension and ischemic heart disease and was on regular treatment with nifedipine, atenolol, indapamide, nitrates, nicorandil and low dose aspirin. Transurethral prostatic resection was done and bladder wash was given using 6 liters of glycine. Post-operative course was uneventful and patient was discharged four days later in a stable condition on pre-existing treatment, antibiotics (cefixime) and doxazocin.

Two days later patient was brought with 7-8 hours history of increasing drowsiness, uneasiness, chest discomfort, hiccups and vomiting. He had tachycardia (heart rate of 104 per minute), blood pressure of 150/90 mmHg, and normal chest signs. He was drowsy, had no focal neurological deficit and had bilateral extensor plantars. His drowsiness increased over the next 4- 5 hours, from being alert to responding sluggishly to verbal commands. He had a generalized tonic convulsion, with up rolling of eyeballs and incontinence. His abdomen gradually distended with only sluggish peristalsis being audible.

His initial investigations revealed serum sodium of 130 meq/lit, potassium of 2.9 meq/lit, chlorides of 92 meq/lit, calcium of 7.8 mg/dl, magnesium of 1.4 mg/dl, and phosphorus of 2.5 mg/dl. His renal and hepatic parameters, hemoglobin, clotting profile and CT scan were normal. There was neutrophilic leucocytosis (WBC count of 12,400 per cubic mm). Electrocardiogram showed generalized T wave inversion.

He was started on replacement therapy with potassium drip. Though calcium replacement could have further dropped the potassium levels, decision was taken to replace the depleted levels of calcium with 10 ml of calcium gluconate intravenously for the first day. Phenytoin was started for preventing further convulsions and antibiotics for preventing infections. He was kept nil by mouth with continuous aspiration of gastric contents.

Repeated electrolyte examinations were performed. Though potassium levels returned to normal, hyponatremia manifested on the second day after admission (129 meq/lit). Correspondingly, abdominal distension decreased and peristalsis reappeared. However,

drowsiness persisted with the patient only opening his eyes voluntarily and not being able to move his limbs. Since intravenous hypertonic saline was not available, sodium replacement was done orally with 8 hourly Ryle tube feeds of 4 gm of table salt in 50 ml distilled water. Sodium levels returned to normal in the next two days to 135 meq/lit. Correspondingly, patient's alertness improved and power in the limbs returned to normal. Oral potassium and table salt supplementation was continued. Subsequent electrolyte examinations were normal except on the sixth day, when his potassium dropped to 2.9 meq/lit. That time, he developed atrial fibrillation with ventricular rate of 140 per minute. Normal sinus rhythm was restored with intravenous adenosine. For the next 12 days in the hospital, patient had normal electrolyte levels. Oral supplementation of electrolytes was tapered and omitted. Further stay in the hospital was complicated by development of deep vein thrombosis, which was appropriately treated. The patient was discharged asymptomatic on the eighteenth day after admission.

Discussion

This case highlights two important points. Though metabolic complications are not uncommon after transurethral prostatic resection, delayed manifestation of this complication is rare. This report suggests that it can occur even after six days of the surgery. Manifestation of this complication depends on the type of the irrigation fluid used, experience of the surgeon (number of venous channels opened), duration of surgery and peripheral venous pressure. Use of glycine as irrigating fluid has vastly reduced the incidence of this complication. However, increased ammonia levels following metabolism of glycine and high levels of glycine per se can give rise to neurological complications.

More importantly, there is a strong possibility that pre-operative treatment with diuretic (indapamide) may have contributed to this complication in this patient. Though, the incidence of hypokalemia with indapamide treatment is very low, mild hypokalemia and possibly hypomagnesemia is possible with long-term treatment.(2) Since there is already a propensity for metabolic changes during prostate surgery, pre-existing electrolyte abnormalities can further complicate the surgery. Hence, the treating physician and surgeon should make a conscious effort to elicit history of diuretic treatment prior to prostate surgery. Hypertension being a common ailment in the age group being operated for prostate enlargement, many of these patients may be on diuretics. We strongly recommend that these patients should have estimation of electrolytes as a part of their pre-operative examination.

References:

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Reviewer's Comments:

It is a well documented fact that hypertensive patients on diuretics are more prone to developing acute manifestations of TUR syndrome due to pre-existing subclinical electrolyte abnormalities. The use of 1.5% Glycine as irrigant solution has reduced the complications related to hypotonicity of irrigating fluids (sterile water), but complications relating to volume load as well as biochemical alterations (hyperammonemia) continue to be matter of concern. The occurrence of clinical and metabolic alterations after a recent TURP has prompted the authors to presume this to be case of delayed TUR syndrome. Tachycardia and neutrophiic leukocytosis are pointers towards sepsis, which by itself could cause metabolic encephalopathy and be responsible for the patient's condition. Recent TURP and diuretic therapy may be only additional factors complicating the clinical picture. However, as the authors state, patients on diuretic therapy pre-operatively should be closely monitored in the perioperative period, clinically and biochemically, to minimize the risk of early or delayed manifestations of a potentially life-threatening clinical entity.