



Volumetric Overload Shocks (VOS) Causing the Transurethral Resection of the Prostate (TURP) Syndrome: Case Reports

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Abstract

Introduction and Objective: The TURP syndrome complicates 1.5% glycine absorption during the TURP procedure. It presents as vascular shock previously reported as hyponatraemic shock. However, it is commonly mistaken for haemorrhagic or septic shock and is treated with volume expansion causing lethal outcome. The first two case reports reported here represent this practice. The remaining two cases report the concept of VOS causing TURP syndrome and the life saving treatment of Hypertonic Sodium Therapy (HST).

Patient and Methods: Four case reports are presented. Two represent the old school of thinking using volume expansion for treatment of TURP syndrome with lethal outcome. Two cases represent the modern school advancing the concept of VOS for the patho-etiology of TURP syndrome and using HST for treatment.

Results: The first two cases were mistaken for a recognized shock and treated with volume expansion; both died. The remaining two cases were identified as VOS1 and treated with HST whose lives were saved.

Conclusion: The concept of VOS identified in the patho-etiology of the TURP syndrome as VOS1 characterized with acute hyponatraemia allows using HST as life saving therapy. VOS2 is that induced by saline-based fluids and has no such clear marker.

Introduction

The TURP syndrome is well known in urology [1,2]. It complicates 1.5% glycine absorption during the TURP procedure. It presents as vascular shock previously reported as hyponatraemic shock [2]. However, it is commonly mistaken for haemorrhagic or septic shock and is treated with volume expansion causing lethal outcome [3]. The first two case reports reported here represent this practice.

Recently, however, the role of VOS in the patho-etiology of the TURP syndrome has been recognized [4,5]. It clearly demonstrates that further volume expansion is absolutely contraindicated in the treatment of the TURP syndrome, for which Hypertonic Sodium Therapy (HST) is the correct life-saving treatment. The remaining two cases reported here represent this practice.

Case Reports

Case 1

At the end of a 2 hours TURP procedure on a fit 78 years old man suffered severe hypotension shock and cardiac arrest on the operating table. He was resuscitated with 4 units of blood, one litre of Haemaccel, one litre of Hartmann and 200 ml of sodium Bicarbonate after which his serum sodium concentration was 124 mmol/L. He remained shocked, in coma, respiratory distressed requiring Dopamine infusion and assisted ventilation. He was thought to remain hypovolaemic and volume expansion policy aiming at elevating his Central Venous Pressure (CVP) continued; further infusion of 5 units of blood and 10 litres of colloids and crystalloids were given in 24 hours and failed to elevate his pressures. Although fluid restriction and peritoneal dialysis were started

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on the 5th post operative day he became progressively oedematous with bilateral plural effusions. Progressive cerebral, renal, cardiac, respiratory and gastro-intestinal failures led to his death on the 21th post operative day. He had sterile cultures of urine and blood. Post mortem examination was not done.

Case 2

Three hours after TURP with resection of 127 grams of tissue on a previously fit 74 year old man under spinal anaesthetic, he became unconscious, shocked and suffered respiratory arrest. His blood pressure dropped to 79/40 mm hg, pulse to 36 beats per minute and CVP to -1 cm saline. His serum sodium concentration dropped to 103 mmol/L. He was given 6 units of blood and 3 litres of colloids and crystalloids after which his serum sodium was raised to 123 mmol/L. He was intubated, ventilated and received supportive measures on ICU. He underwent further infusions of 21 units of blood, 3 litres of colloids and 4 litres of crystalloids but his pressures remain persistently low. Fourteen hours later severe catheter bleeding required open packing of the prostatic cavity. Gastric and wound capillary bleeding occurred despite normal coagulation screen and repeated platelet infusions. It became clear he was fluid overloaded. Although fluid restriction and peritoneal dialysis were started on the 2nd day progressive cerebral, cardiovascular, respiratory, renal and hepato-biliary failure occurred and culminated in his death on the 6th postoperative day. His serum sodium and osmolality prior to death were 130 and 321 respectively. Increased cardiac enzymes activity suggested myocardial infarction [Creatinine kinase 16 (<8 U/L), Hydroxybuterate dehydrogenase 557 (<120 U/L) and Aspartate Transferase (<40 U/L).

Post-mortem examination showed enlarged congested and oedematous lung, liver, heart and kidneys. All tissues were laden with water, 1,500 ml of blood stained fluid was found in the plural spaces and 3 litres in the peritoneal cavity. The myocardium was oedematous but there was neither infarction nor coronary artery disease.

Case 3

Six hours after endoscopic bladder tumour resection on 67 year old fit man, he became comatose and hypotensive (BP 70/50 mm hg). He was thought to be in hypovolaemic shock and was transfused with 5 units of blood and 3 litres of colloids and crystalloids. His blood pressure remained below 90 mm hg and CVP at -5 cm saline. He developed generalized convulsive fit, 12 hour later a neurological assessment confirmed coma with fixed dilated pupils and quadriplegia. He was thought to have suffered cerebrovascular accident. At this time bladder perforation was diagnosed and his serum sodium dropped to 110 mmol/L. He underwent a rapid infusion of 500 ml 5% NaCl, followed by laparotomy and over sewing of bladder perforation. Three litres of fluid was drained from his peritoneal cavity. Postoperatively he passed 4.5 litres of urine and recovered fully from coma and quadriplegia. He was discharged home on the 14th postoperative day.

Case 4

During TURP on a fit 74 year old man, his BP raise temporarily from 129/89 mm hg to 160/100 mm hg. He later became hypotensive and developed bronchospasm and pulmonary crepitations. Frusimide, atropine and aminophylline were given. The patient had undergone an infusion of 2 units of blood, one litre of Haemaccel and one litre

of Hartmann. On recovery from the anaesthetics, he suffered a generalised convulsive fit and went into coma. Pulmonary oedema, bronchospasm and cardiac dysrhythmia re-occurred. He remained shocked, hypothermic, comatose and anuric. His BP was 80/50 mm hg CVP ranging between -9 cm and -4 cm saline; giving an impression of hypovolaemic shock. However, a volume of 5.5 litres of the irrigating fluid 1.5% Glycine was calculated missing from the returned fluid; remaining inside the patient's body. His immediate postoperative serum sodium concentration was 101 mmol/L and serum osmolality was 270 mOsm/L. The osmolality further dropped to 217 mOsm/L after 4 hours. Volumetric overload shock was realised and a fluid restriction policy was adopted in spite of the low BP and CVP. He was given a rapid infusion of 1.8% sodium chloride and 400 ml of 8.4% sodium bicarbonate, given in 200 ml increments and each was followed by estimation of serum electrolytes and osmolality. Over the next 24 hours he lost 5.1 litres of urine and 1.7 litres of gastric aspirate leading to his full recovery. He was discharged home on the 6th postoperative day.

Discussion

Here we report 4 cases of the TURP syndrome of which two died and two survived. The two who died are representative of most previous reports on the TURP syndrome. Their conditions were mistaken for one of the recognised shocks such as haemorrhagic or septic shock and were treated with further volume expansion. Such practice should be made obsolete.

The remaining two case reports are representative of the modern school of practice. In this school volume expansion is absolutely contraindicated. The role of VOS in the patho-etiology of the TURP syndrome has been reported [4,5].

It advances Hypertonic Sodium Therapy (HST) of 5% sodium chloride or 8.4% sodium bicarbonate as the treatment of choice which is life saving.

The advantage of recognising VOS is not only saving the lives of the TURP syndrome identified as VOS1 patients but also paves the way for recognising the condition induced by sodium-based fluids identified as VOS2. The latter has no clear serum marker such as hyponatraemia seen in VOS1 and is more difficult to identify. VOS2 presents with the Adult Respiratory Distress Syndrome (ARDS).

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