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Full Length Research Paper

Volumetric overload shocks in the patho-etiology of the transurethral resection prostatectomy syndrome and acute dilution hyponatraemia: The clinical evidence based on 23 case series

Khaled A. Ghanem[1] and *Ahmed N. Ghanem[2]

Mansoura University Hospital, Mansoura 35511, Egypt.

*Corresponding author email: khaledghanem899@hotmail.com, an_ganem@hotmail.com

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ABSTRACT

Introduction and Objective: To report 23 case series demonstrating that volumetric overload shocks (VOS) cause the transurethral resection prostatectomy syndrome and dilution hyponatraemia (HN) Patients and methods: Representatives of 23 case series are reported showing the insult of volumetric overload type and quantity causing increase in plasma and interstitial fluid volumes with dilution of serum contents. Results: VOS 1 is induced by 3.5-5 litres of sodium free fluid while VOS2 is induced by >10 litres of sodium based fluids. VOS1 induces acute HN while VOS2 has no such clear marker. This causes increase in the volume of plasma and interstitial fluid with dilution of its contents. After the initial vascular shock VOS manifests with multiple vital organ dysfunction or failure. In VOS1 HN encephalopathy coma predominates while in VOS2 the adult respiratory distress syndrome predominates. Conclusion: The clinical evidence based on 23 case series that volumetric overload shocks is the patho-aetiology of the transurethral resection prostatectomy syndrome and acute dilution HN is reported. After presentation with shock and multiple vital organ dysfunction/ failure VOS1 manifests next day with encephalopathy HN coma. The evidence on volumetric overload type and quantity and its effect on plasma and interstitial fluid volume as well as dilution of serum content concentration are presented. While VOS1 is characterised with acute dilution HN, VOS 2 as no clear such marker and is presented as the adult respiratory distress syndrome. Treating VOS like any known shock with volume expansion is lethal while hypertonic sodium therapy is lifesaving.

Keywords: Shock; Hyponatraemia (HN), transurethral prostatectomy syndrome (TURS), adult respiratory distress syndrome (ARDS)

INTRODUCTION

The transurethral prostatectomy syndrome (TURS) is a severe vascular hypotension reaction that complicates endoscopic surgery as a result of massive irrigating fluid absorption causing severe acute dilution hyponatraemia (HN) of <120 mmol/l. (Ghanem and Ward, 1990).

Volumetric Overload Shock (VOS) is a condition caused by massive fluid infusions and is of two types; Type one (VOS1) and Type two (VOS2). VOS1 is

ABBREVIATIONS

VO: Volumetric overload
VOS: Volumetric overload shocks
VOS1: Volumetric overload shock, Type 1
VOS2: Volumetric overload shock, Type2
TURP: The transurethral resection of the prostate
TURS: The transurethral resection of the prostate syndrome
ARDS: The adult respiratory distress syndrome
MVOD: The multiple vital organ dysfunction/ failure syndrome
HN: Hyponatraemia
HST: Hypertonic sodium therapy of 5% NaCl or 8.4% Sodium Bicarbonate
CT: Conservative treatment with volume expansion
BP: Blood pressure
CVP: Central venous pressure
ICU: I*ntensive Care Unit*
PV: Plasma volume
ISF: Interstitial fluid volume
mmol/l: Mille mole/litre
mmhg Mille meter Mercury
U/L: Unit per litre
NaCl: Sodium chloride

induced by sodium-free fluid gain such as 1.5% Glycine used as irrigating fluid during endoscopic surgery such as the transurethral resection of the prostate (TURP) (Ghanem and Ward, 1990). It has been reported with other fluids such as Glucose, Mannitol and Sorbitol. It is known as TURS or HN shock (Harrison III et al., 1956) as HN is a marked serological marker for the condition. (Arieff, 1986) VOS2 is induced by massive infusion of sodium-based fluids such as normal saline, Ringer, Hartmann, plasma and plasma substitutes and/or blood transfusions that may complicate the therapy of VOS1. VOS2 also complicates fluid therapy in critically ill patients suffering from other known shocks such as trauma, hypovolaemic, haemorrhagic and septicaemia shocks and presents with the multiple vital organs dysfunction (MVOD) or failure syndrome. The adult respiratory distress syndrome (ARDS) (Ashbaugh et al., 1967) is another name under which VOS2 is reported. Both VOS1 and VOS2 are complications of fluid therapy. VOS1 has been induced in animals under clean experimental conditions in the absence of haemorrhage and sepsis (Danowski et al., 1946).

TURS was first reported by Creevy (1947) as acute water intoxication when distilled water was used as irrigating fluid for TURP. (Creevy, 1947) Water intoxication caused intravascular red cell haemolysis and acute renal failure. Shift to osmotic solutions was made and 1.5% Glycine gained popularity. Harrison et al. (1956) reported TURS as acute dilutional HN shock after massive gain of Glycine irrigant. However, TURS is not limited to TURP. It may affect any endoscopic surgery and has been reported in women undergoing Transcervical Endometrial Resection. (Arieff and Ayus, 1993; Istre et al., 1994). It may also affect women undergoing any surgery following excessive 5% Glucose infusions. (Arieff, 1986) TURS manifests as shock during surgery and by next morning it manifests as HN encephalopathy coma. (Henderson and Middleton, 1980) TURS may be mistaken for other recognized shocks such as septicaemic (Bertrand et al., 1981), hemorrhagic (Bird et al., 1982; Friedman et a., 1969; Ekengreen and Hahn, 1993) and cardiogenic (Evans et al., 1992; Charlton, 1980) shock. VOS 2 may complicate all types of shocks during fluid therapy and the transition is seamless and hard to detect. It may be called the irreversible shock. The only way to detect VOS 2 is the sudden acute increase in body weight or accurate fluid balance during resuscitation. The serum solutes changes of VOS1 particularly HN have been reported by all authors. (Desmond, 1970; Beirne et al., 1965; Berg et al., 1962).

TURS may be presented as HN encephalopathy coma (Arieff, 1986; Arieff and Ayus, 1993; Istre et al., 1994; Henderson and Middleton, 1980), cardiogeic shock or cardiac arrest (Desmond, 1970), respiratory failure or arrest (Jacobson, 1965) and acute renal failure among other vital organs involved. Visual loss has also been reported. (Kay et al., 1985) Post-mortem examination has been documented. (Lessels et al., 1982). TURS has been attributed to Glycine and ammonia toxicity (Hoekstra et al., 1983) but it has also been reported with Mannitol (Hoekstra et al., 1983) and Glucose. (Arieff, 1986).

Professor Hahn et al reported 480 articles of which >340 articles are on TURS [PubMed search December 2016] investigating the fluid and electrolytes dynamics (Hahn, 1990), effect of over-hydration on cardiac muscle (Hahn et al., 1996) and other tissues (Hahn et al., 1996), effect on renal function (Hahn et al., 1996) and compared Glycine to Mannitol (Hahn et al., 1998). Professor Hahn favoured the toxicity of Glycine as the patho-etiological cause of TURS. Ghanem and Ward introduced the concept of volumetric overload in the patho-etiology of TURS in 1990. Ghanem and Ward (1990) confirmed the effectiveness of hypertonic 5%NaCl or 8.4% Sodium Bicarbonate both as anecdotal evidence (Ghanem et al., 1987) in a prospective study, Ghanem and Ward (1990) also investigated the underlying faulty physiological law of Starling for the capillary interstitial fluid transfer. (Ghanem, 2001; Ghanem and Ghanem, 2016).

In our previous reports on VOS (Ghanem and Ghanem, 2016; Nisha et al., (In the press) the clinical evidence was lacking. Here we rectify this issue.

PATIENTS AND METHODS

We report 23 case series divided into 3 groups of patients. Group 1 of 3 patients were treated as one of the known shocks by conservative treatment (CT) of volume expansion and all died. Group 2 of 10 patients were

treated as volumetric overload shock with hypertonic sodium therapy (HST) of 5% NaCl or 8.4% Sodium Bicarbonate and all survived. Group 3 were 10 symptomatic patients encountered during a prospective study on 100 patients (Ghanem and Ward, 1990) and were randomised between CT and HST; 5 patients in each group named group 3.1 and 3.2, respectively.

Accurate data recording on each patient included age, body weight and volumetric fluid balance during the operation, pre- and postoperative times. Serum solute concentration changes were also recorded at pre and post operative times.

Case Report 1: a representative of Group 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 on the 5th post operative day he became progressively oedematous wit 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 Report 2: another example of Group 1

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 mmhg, 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. 1500 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 Reports: representative of Group 2

Case 3

Six hours after endoscopic bladder tumour resection on 67 year old fit man, he became comatosed and hypotensive (BP 70/50 mmhg). He was thought to be in hypovolaemic shock and was transfused wit 5 units of blood and 3 litres of colloids and crystalloids. His blood pressure remained below 90 mmhg and CVP at -5 cm saline. He devolved generalised convulsive fit. 12 hour later a neurological assessment confirmed coma with fixed dilated pupils and quadriplegia. He was thought to have suffered cerebro-vascular 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% NeCl, 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 rose temporarily from 129/89 to 160h/100 mmhg. He later became hyhpotensive and developed brochospasm 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 dysrythmia re-occurred. He remained socked, hypothermic, comatosed an annuric. His BP was 80/50

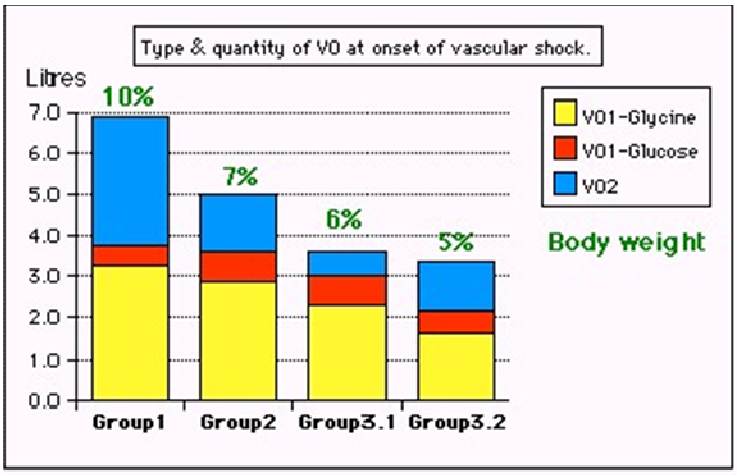

Figure 1. shows the type (Sodium free fluid VO1 and sodium based fluid VO2) and mean quantity of fluid in litres and as percent of body weight at the time of occurrence of volumetric overload shock (VOS).

mmhg CVP ranging between -9 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 217mosm/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.

Case 5

At the end of TURP on a fit 79 years old man who became hypotensive and shocked. He had some bleeding from the prostatic veins. Fluid absorption was suspected and 5 litres of the irrigating 1.5% Glycine was calculated missing from the efflux. His serum sodium concentration dropped from 138 to 101 mmol/l . He underwent an intravenous infusion of 700 ml 1.8% sodium chloride and 200 ml of 8.4 Sodium Bicarbonate within 2 hours. This raised his serum sodium to normal. He was fully awake within 4 hours during which he passed 5.2 litres of urine. He made full recovery and was discharged home on the 6th postoperative day.

Group 3 cases were 10 symptomatic cases encountered during a prospective study on TURP patients whose treatments were randomised between CT and HST, reported previously. (Ghanem and Ward, 1990).

RESULTS

The reported cases demonstrate that the volumetric overload (VO) is responsible for the induction of dilution hyponatraemia (HN) and TURS. A summary of VO causing both types of VOS1 and VOS2 is sown in Figure 1. Table 1 shows the summary and comparing data on the 3 groups of patients. Figure 2 shows the drop in serum sodium concentration (HN) and osmolality; characterising VOS1. The effect of VOS on the intravascular fluid plasma volume (PV) and the interstitial fluid volume (ISF) is shown in figure 3 and 4, respectively. The dilution changes affecting the serum solutes content affecting the 10 symptomatic TURS cases is shown in Figure 5. Figure 6 shows the serum osmolality Gaps; the initial caused by Glycine and the terminal is caused by the contents of plasmolysed body cells leaking into plasma; occurring only in patients who died.

It is quite clear that VOS1 is induced by sodium free fluids of 1.5% glycine and 5% glucose characterised with acute dilution HN causing dilution of both PV and ISF volumes as shown in Figures 1-4. VOS2 causes dilution of both proteins and haemoglobine but not as marked as HN. Also the clinical picture of VOS1 and VOS2 is characterised by shock and the multiple vital organs dysfunction/ failure as demonstrated by the reported cases. This is also clear on the post-mortem examinations on patients who died as all Vital organs

Table 1. Shows the mean summary of data, therapy and outcome comparing the 3 groups of patients.

	A	B	C	D	E	F	G	H
1		**Gr1**	**Gr2**	**Gr3**	**Gr3.1**	**Gr3.2**	**Normal**	**Units**
2	Number of patients	3	10	10	5	5	mean	
3	Age	71	70	75	72	78	72	Years
4	Body weight (BW)	69	70	68	71	65	69	
5	Post operative serum solute concentration						Preoperative	
6	Osmolality	271	234	276	282	271	292	Mosm/1
7	Na+	110	108	120	119	121	139	Mmol/1
8	Ca++	1.69	1.79	1.85	1.84	1.86	2.22	"
9	K+ (P<.05)	5.6	4.8	5.0	4.9	5.0	4.46	"
10	C_02 (P=.002)	23.0	23.0	25.5	240	26.4	27.30	"
11	Glucose	13.2	17.3	16.4	15.9	16.9	6.20	"
12	Urea (P=.0726)	26.5	9.0	6.6	6.8	6.4	6.7	"
13	Bilirubin (P<.05)	19	16	8	6	9	7	"
14	AST	124	32	20	18	21	20	"
15	Protein	43	52	48	44	52	62	g/l
16	Albumin	23	30	30	28	32	39	"
17	Hb (P=.0018)	119.3	127.9	114.5	105.2	123.8	123.8	"
18	WCC (P<.005)	18.9	16.2	7.5	7.8	7.2	8.0	per HPF
19	Glyoine			10499			293	µmol/1
20	Therapy	CT	HST	Random	HST	CT©		
21	Outcome	Death	Full Rec.		Full Rec.	Morb©		

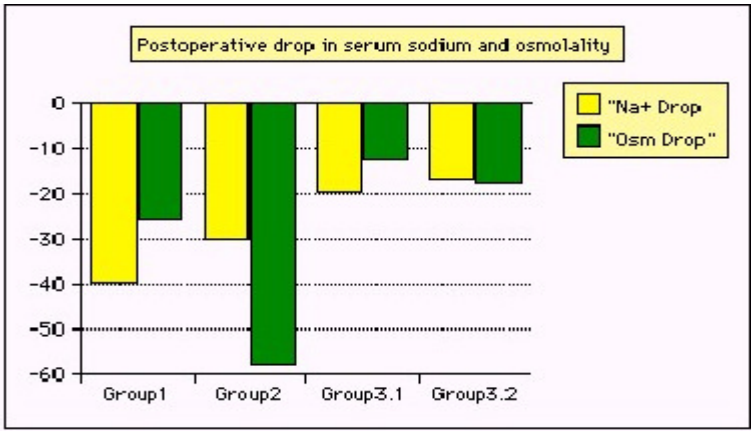

Figure 2. Shows the mean postoperative drop in serum sodium and osmolality in the 3 patients groups.

were congested and oedematous as demonstrated above. Next postoperative day clinical picture is HN encephalopathy coma in VOS1 and the adult respiratory distress syndrome in VOS2.

The volume inducing VOS1 is 3.5-5 litres. The volume inducing VOS2 is two to three times as much as shown in Figure 1 in litres and as % body weight. Mistaking VOS for a recognized sock and treating it with further volume expansion is lethal. While using HTS of 5% NaCl or 8.4% Sodium Bicarbonate is lifesaving. The HTS therapy induces massive diuresis and full recovery. This is proved by both case series reported here as well as the prospective randomised trial on the 100 TURP patients (Ghanem and Ward, 1990) among whom the 10 symptomatic cases of Group 3 were randomised between therapies.

DISCUSSION

The presented evidence demonstrates that sodium free fluid of 1.5% Glycine and 5% Glucose induces VOS1 causing dilution of all serum solutes concentration of which HN is the most marked. It presents with shock during surgery and by next day it becomes HN encephalopathy coma. But, all vital organs are affected with dysfunction or failure due to congestion and oedema. Thus the clinical picture of VOS1 is coma,

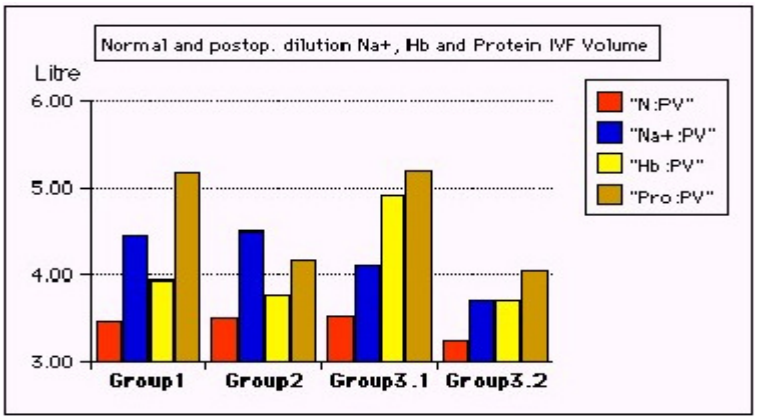

Figure 3. Shows the mean intravascular plasma volume (PV) in normal condition and as based on dilution of sodium (Na+:PV), haemoglobin (Hb:PV) and protein (Pro:PV).

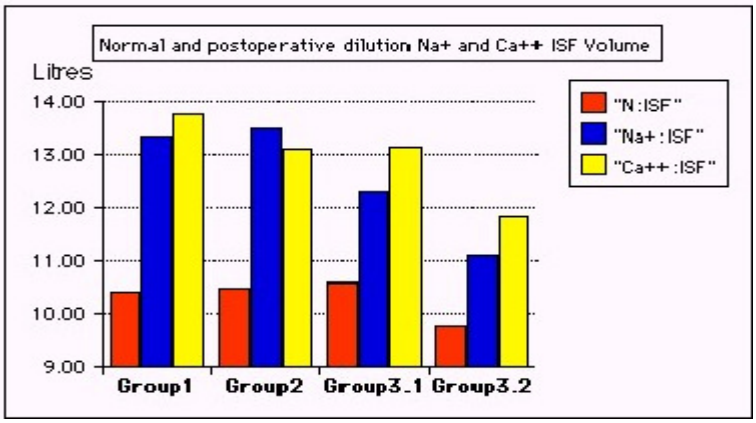

Figure 4. Shows the mean normal interstitial fluid space (N:ISF) and compared to that based on sodium (Na+:ISF) and calcium dilution (Ca++: ISF)

convulsion, disrythmia, annuria, respiratory distress, liver dysfunction, haematological disorder and paralytic ileus plus oedema or anasarca. The volume of gained fluids inducing VOS1 and VOS2 is shown in figure 1. So, these patients though in shock are not hypovolaemic but rather hypervolaemic as evidenced by the dilution of PV and ISF (Figures 3 and 4). Its effect on serum sodium and osmolality is shown in figure 2. Its effect on all serum solute concentration contents is shown in figure 5.

Harrison et al. (1956) reported TURS as acute dilutional hyponatraemic shock after massive gain of Glycine irrigant. However, TURS is not limited to TURP. It may affect any endoscopic surgery and has been reported in women undergoing Transcervical Endometrial Resection. (Arieff and Ayus, 1993; Istre et al., 1994). It may also affect women undergoing any prolonged surgery following excessive 5% Glucose infusions. (Arieff, 1986) TURS manifests as shock during surgery and by next morning it manifests as HN encephalopathy coma. (Henderson and Middleton, 1980) TURS may be mistaken for other recognized shocks such as septicaemic (Bertrand et al., 1981), hemorrhagic (Bird et al., 1982; Friedman et a., 1969; Ekengreen and Hahn, 1993) and cardiogenic (Evans et al., 1992; Charlton, 1980) shock. VOS2 may complicate all types of shocks during fluid therapy and the transition is seamless and hard to detect. It may be called the irreversible shock. The only way to detect VOS2 is the sudden acute increase in body weight by >10% or accurate fluid balance during resuscitation. The serum solutes changes of VOS1 (Figure 5) particularly HN have been reported by all authors. (Desmond, 1970; Beirne et al., 1965; Berg et al., 1962) TURS may present as HN encephalopathy coma (Arieff, 1986; Arieff and Ayus, 1993; Istre et al., 1994; Henderson and Middleton, 1980), cardiogeic shock or cardiac arrest (Desmond, 1970, respiratory failure or arrest (Jacobson, 1965) and acute renal failure among other vital organs involved. Visual loss has also been

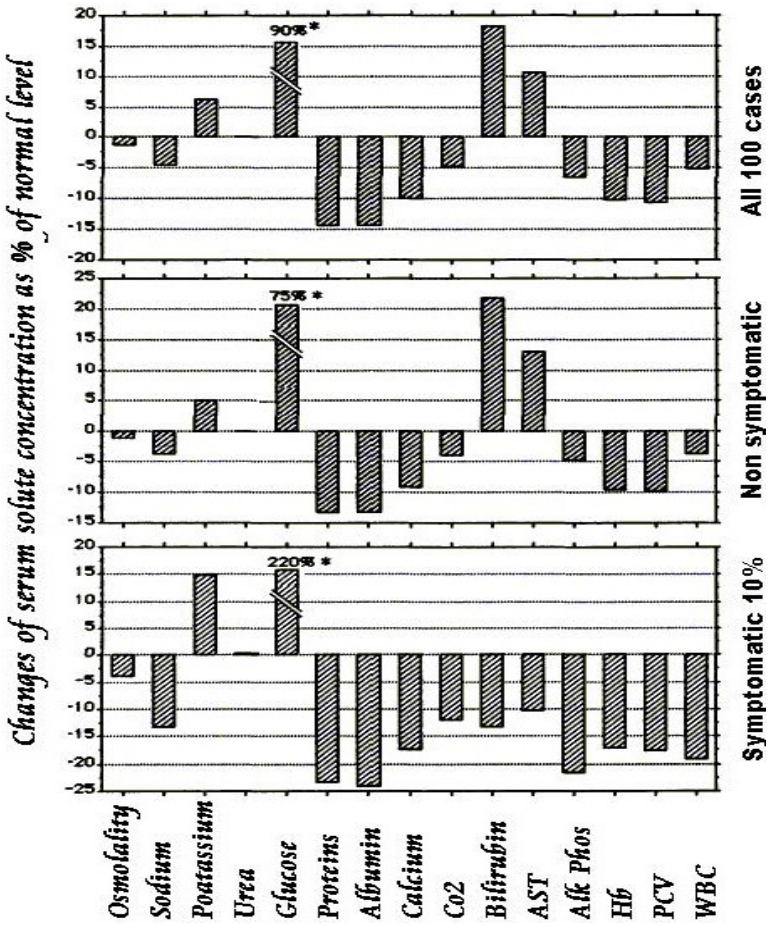

Figure 5. Shows the mean changes in serum solutes concentration as % of normal in 100 patients prospectively studied of whom the symptomatic group is the Group 3 of 10 patients mentioned in the text.

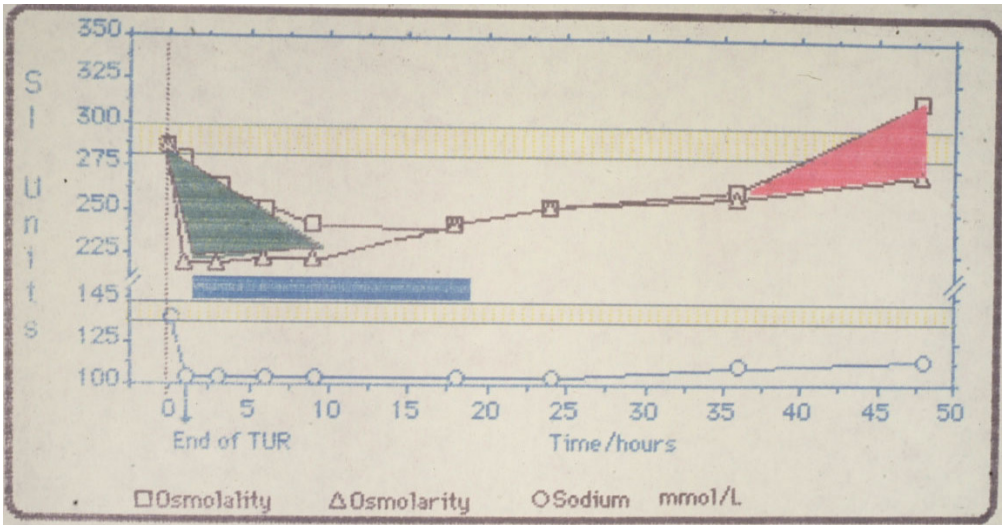

Figure 6. Shows the changes in serum sodium and osmolality demonstrating the serum osmolality Gaps. This is based on a line graph of measured and calculated osmolality. The initial osmolality Gap is negative represented by the green triangle while the terminal osmolality Gap is positive represented by the red triangle.

reported. (Kay et al., 1985) Postmortem examination has been documented. (Lessels et al., 1982) TURS has been attributed to Glycine and ammonia toxicity (Hoekstra et al., 1983) but it has also been reported with Mannitol (Hoekstra et al., 1983) and Glucose. (Arieff, 1986).

Professor Hahn et al reported 480 articles of which >340 articles are on TURS [PubMed search December 2016] investigating the fluid and electrolytes dynamics (Hahn, 1990), effect of overhydration on cardiac muscle (Hahn et al., 1996) and other tissues (Hahn et al., 1996), effect on renal function (Hahn et al., 1996) and compared Glycine to Mannitol (Hahn et al., 1998). Professor Hahn favoured the toxicity of Glycine as the patho-etiological cause of TURS. Ghanem and Ward introduced the concept of volumetric overload in the patho-etiology of TURS in 1990. (Ghanem and Ward, 1990) confirmed the effectiveness of hypertonic 5%NaCl or 8.4% Sodium Bicarbonate both as anecdotal evidence (Ghanem et al., 1987) and in a prospective study (Ghanem and Ward, 1990) and also investigated the underlying faulty physiological law of Starling for the capillary interstitial fluid transfer. Ghanem, 2001; Ghanem and Ghanem, 2016) VOS in the patho-aetiology of TURS has recently been reported. (Ghanem and Ghanem, 2016; Nisha et al., In the press) These reports on VOS lacked the clinical evidence that is rectified here.

CONCLUSION

The clinical evidence based on 23 case series that volumetric overload shocks is the patho-etiology of the transurethral resection prostatectomy syndrome and acute dilution hyponatraemia is reported here. After presentation with shock and multiple vital organ dysfunction/ failure VOS1 manifests next day with encephalopathy HN coma. The evidence on VO type and quantity and its effect on PV and ISF as well as dilution of serum content concentration is presented. While VOS1 is characterised with acute dilution HN, VOS 2 as no clear such marker and is presented as the adult respiratory distress syndrome. Treating VOS like any known shock with volume expansion is lethal while HTS therapy is life saving.

Conflict of interest

No conflict of interest was declared

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