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Volumetric overload shocks in the patho-etiology of the transurethral resection prostatectomy syndrome and acute dilution hyponatraemia

Nisha Pindoria[1], Salma A Ghanem[2], Khalid A Ghanem[3] and Ahmed N Ghanem[4*]

[1]North Middlesex University Hospital, UK

[2]Barts and the Royal London NHS Trust- Royal London Hospital, UK

[3]Mansoura University Hospital, Egypt

[4]Retired Consultant Urologist, Egypt

Abstract

The transurethral prostatectomy syndrome (TURS) is defined as 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. The vascular shock is usually mistaken for one of the recognized shocks and Volumetric Overload Shock type 1 (VOS1) is overlooked making Volumetric Overload Shock Type 2 (VOS2) unrecognizable. VOS1 is induced by the infusion of 3.5-5 liters of sodium-free fluids and is known as TURS or HN shock. VOS2 is induced by 12-14 liters of sodium-based fluids and is known as the adult respiratory distress syndrome. The most effective treatment for VOS1 and VOS2 is hypertonic sodium of 5% NaCl or 8.4% Sodium Bicarbonate. The literature is reviewed and the underlying patho-etiology is discussed. As Starling's law for the capillary-interstitial fluid transfer proved wrong an alternative mechanism was found by studying the hydrodynamics of the porous orifice (G) tube. Incorporating the G tube in a chamber (C), representing the interstitial space surrounding a capillary, demonstrated a rapid dynamic magnetic field-like fluid circulation between the C and G tube lumen. The G-C phenomenon is autonomous having both filtration and absorption forces making a true replacement for Starling's law.

Abbreviations: VOS: Volumetric overload shocks, VOS1: Volumetric overload shock, Type 1, VOS2: Volumetric overload shock, Type2, TURP: The transurethral prostatectomy, TURS: The transurethral prostatectomy syndrome, ARDS: The adult respiratory distress syndrome, MVOD: The multiple vital organ dysfunction/ failure syndrome, HN: Hyponatraemia, HS: Hypertonic sodium, G Tube: The Porous orifice tube

Definitions

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 [1].

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 induced by sodium-free fluid gain such as 1.5% Glycine used as irrigating fluid during endoscopic surgery such as the transurethral resection prostatectomy (TURP) [1]. It has been reported with other fluids such as Glucose, Mannitol and Sorbitol. It is known as TURS or HN shock [2] as HN is a marked serological marker for the condition [3]. 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 septicaemic shocks and presents with the multiple vital organs dysfunction (MVOD) or failure syndrome. The adult respiratory distress syndrome (ARDS) is another name under which VOS2 is reported. Both VOS1 and VOS2 are complications of fluid therapy [4].

Introduction

Why should TURS be recognized as VOS? As shown here VOS1 is the real patho-etiology of TURS which has HN as a clear serological marker. This makes it easier to recognize VOS2 which unlike VOS1 has no clear serological marker. It also helps to establish the correct and lifesaving therapy of hypertonic 5% NaCl or 8.4% Sodium Bicarbonate. It has also helped in realizing that the physiological law of Starling, which underlies the principles of fluid therapy in clinical practice, is in fact incorrect. From the literature review it will be realized that TURS presents as vascular hypotension shock to the anaesthetists and surgeons during the surgery that should not be mistaken for one of the recognized shocks. By next morning it presents as HN coma to physicians. VOS1 has been induced in animals under clean experimental conditions in the absence of hemorrhage and sepsis [5].

Correspondence to: Ahmed Ghanem, Retired Consultant Urologist, Egypt, Email: an_ghanem@yahoo.com

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Literature review

TURS was first reported by Creevy in 1947 as acute water intoxication when distilled water was used as irrigating fluid for TURP [6]. Water intoxication caused intravascular red cell haemolysis and acute renal failure. Shift to osmotic solutions was made and 1.5% Glycine gained popularity. Harrison 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 [7,8]. It may also affect women undergoing any surgery following excessive 5% Glucose infusions [3]. TURS manifests as shock during surgery and by next morning it manifests as HN encephalopathy coma [9]. TURS may be mistaken for other recognized shocks such as septicaemic [10], hemorrhagic [11-13] and cardiogenic [14,15] 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 change particularly HN have been reported by all authors [16-18].

TURS may present as HN encephalopathy coma [3,7-9], cardiogeic shock or cardiac arrest [16], respiratory failure or arrest[19] and acute renal failure among other vital organs involved. Visual loss has also been reported [20]. Postmortem examination has been documented [21]. TURS has been attributed to Glycine and ammonia toxicity [22] but it has also been reported with Mannitol [22] and Glucose [23].

Professor Hahn *et al.* reported 480 articles of which >340 articles are on TURS investigating the fluid and electrolytes dynamics [24], effect of overhydration on cardiac muscle [25] and other tissues [26], effect on renal function [27] and compared Glycine to Mannitol [28]. Professor Hahn favoured the toxicity of Glycine as the patho-etiological cause of TURS. Ghanem *et al.* introduced the concept of volumetric overload in the patho-etiology of TURS in 1990 [1]. Ghanem confirmed the effectiveness of hypertonic 5%NaCl or 8.4% Sodium Bicarbonate both as anecdotal evidence [29] and in a prospective study [1] and also investigated the underlying faulty physiological law of Starling for the capillary interstitial fluid transfer [30,31].

Aetiology

VOS1 is induced by the infusion of 3.5-5 liters of Glycine irrigating fluid through the peri-prostatic veins during TURP [1]. Intravenous infusion of 5% Glucose augments this effect. It is important to realize the significance of time; 3.5 liters of fluids is a normal daily intake harmless if gained over 24 hours but is certainly pathological when gained over one hour. In VOS1 one liter of fluid causes a drop in serum sodium concentration of 7 mmol/l. VOS2 is induced by the gain of 12-14 liters of sodium-based fluids [3]. The problem here is that every hypotension is considered synonymous with hypovolaemia and is treated with massive volume expansion. In the past VOS1 was wrongly attributed to one of the known shocks and treated with sodium-based fluids inducing VOS2.

Patho-physiology

The occurrence of massive interstitial tissue oedema with congestion of vital organs, pleural and peritoneal effusions, in the presence of severe hypotension shock, casted doubt on Starling's law!

Shock is a disturbance at the capillary cellular level impairing the capillary-interstitial fluid transfer; delivery of oxygen and removal of waste products. This process and oedema formation is governed by Starling's law [32]. In this law the arterial pressure is considered the force causing capillary filtration! If this is true, how come that arterial hypertension though common never causes oedema? Starling based his hypothesis on Poiseuille work on strait uniform brass tubes. Latter evidence however demonstrated that the capillary is a porous narrow orifice (G) tube as it has a pre-capillary sphincter [33] and pores that allow the passage of plasma proteins [34]. Because the capillary pores allow the passage of plasma molecules, hence no oncotic pressure force, a call for reconsideration of Starling's hypothesis was made [35]. At this time an alternative to Starling's law was not found yet.

The hydrodynamics of the G tube demonstrated that the proximal (arterial) pressure induces a negative side pressure gradient on the wall of the G tube causing inflow suction (absorption) most prominent over the proximal half (Figure 1) and turns into positive pressure over the distal half causing fluid outflow (filtration) [30,31] (Figure 2). Incorporating the G tube in a chamber (C) (Figure 3,4), representing the interstitial space surrounding a capillary, demonstrated a rapid dynamic magnetic field-like fluid circulation between the C and G tube lumen (Figure 5,6). Incorporating the G tube and C in a circulatory model driven by electric pump inducing proximal pressure similar to arterial pressure in human circulatory system (Figure 7); caused suction from C into the lumen of G tube. The pressure in C is negative (Figures 3,4). The pressure in the interstitial fluid space is also negative of -7 cm H_2O [36]. Distal (venous) pressure is responsible for augmenting filtration. This proves that the circulatory system is not an all positive pressure, the arterial pressure causes suction not filtration

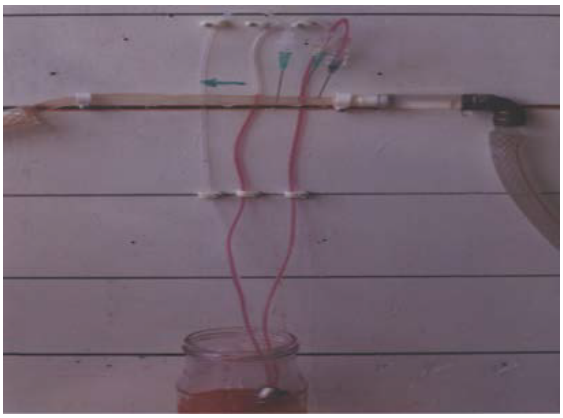

Figure 1. Demonstrates a rubber tube with a narrow inlet and water passing through causes suction over the proximal part of the tube by sucking red water into manometer tubes from a jar 35 cm below the G-Tube.

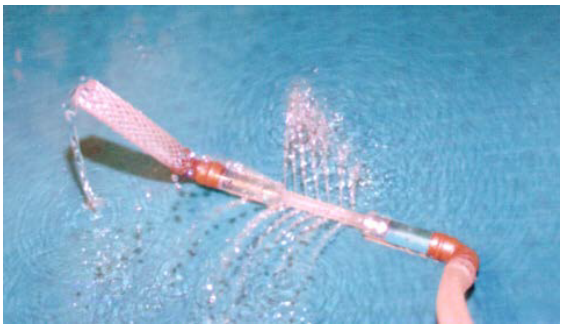

Figure 2. The G-Tube demonstrating the pressure gradient on its wall causing fluid to come out maximal near the exit. The negative pressure on the proximal part was shown in Figure 1. Close inspection shows a magnetic field like fluid circulation of water film on upper part of the blue surface background.

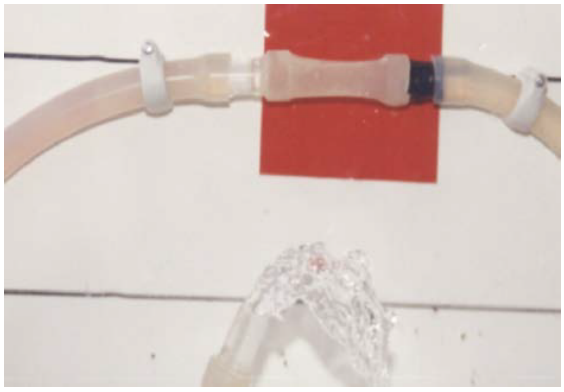

Figure 3. The G-Tube enclosed in a rubber chamber (C) which is sucked in as water passes through the G tube indicating net negative pressure in C.

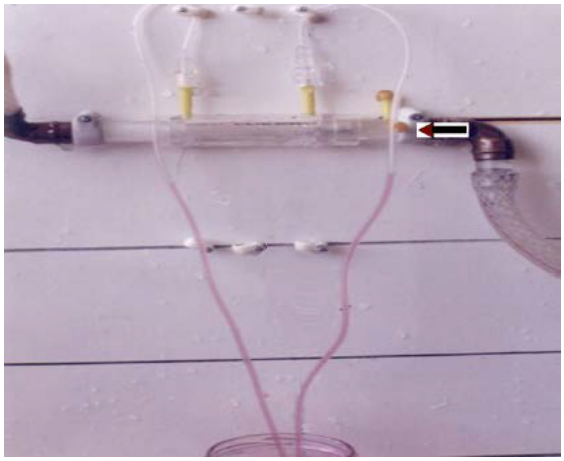

Figure 4. The G-C model measuring the negative pressure in C with manometers sucking water from a jar 30 cm below.

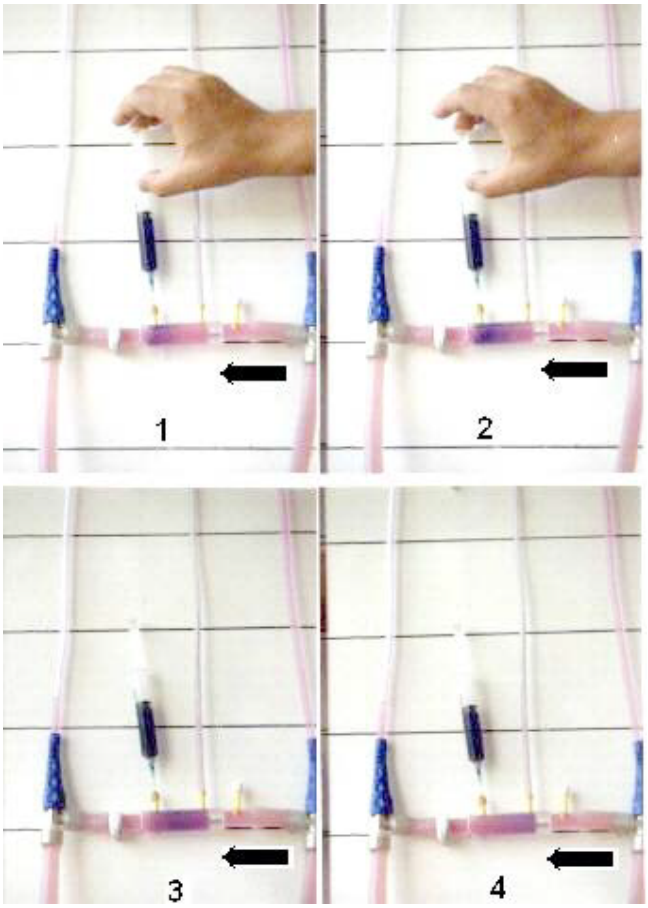

Figure 5 (sequence of 1-4). Link injected into the distal part of C moves, in an opposite direction to the flow inside the G-Tube, towards the proximal part where it gets absorbed and cleared fast.

at the capillary interstitial fluid transfer, and hence Starling's law is wrong [31]. The G-C circulatory phenomenon is autonomous having both filtration and absorption forces makes it a true replacement for Starling's law.

Clinical picture

VOS1 has the following clinical picture before it transfers into VOS2 with a full blown picture of MVOD or failure or ARDS characterizing both conditions. It is noted that VOS1 presents during surgery as hypotension shock and next day as HN coma. When the TURP is done under general anesthetic cardiovascular signs and cyanosis appear first and when done under spinal or epidural anaesthetic cerebro-nervous signs appear first.

Cerebro-nervous system

Numbness and tingling sensation, sudden bilateral blindness and clouding of consciousness have been reported under spinal or epidural anesthesia. Convulsions may occur. The patient proceeds into high scale coma and does not recover from the general anesthetic.

Cardiovascular system

Hypotension and bradycardia are early features also in patients having general anesthetic. Hypertension is rarely detected. Other types of disrythmia with cardiac arrest and sudden death may occur. Cardiac

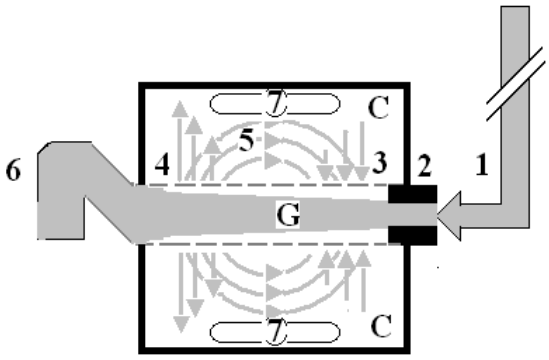

Figure 6. Diagram of the porous orifice (G) tube enclosed in chamber (C) based on several photographs demonstrating the magnetic field-like G-C circulation phenomenon. The proximal inflow (arterial) pressure (1) pushes fluid through the orifice (2) creating fluid jet in the lumen of the G tube. The fluid jet creates negative side pressure gradient causing suction maximal over the proximal half of the G tube near the inlet (3) that sucks fluid into lumen. The side pressure gradient turns positive pushing fluid out of lumen over the distal half maximally near the outlet (4). Thus the fluid around G tube inside C moves in magnetic field-like fluid circulation (5) taking an opposite direction to lumen flow of the G tube. The inflow (arterial) pressure (1) and orifice (2) induce the negative side pressure energy creating the dynamic G-C circulation phenomenon that is rapid, autonomous and efficient in moving fluid out from the G tube lumen at (4), irrigating C at (5), then sucking it back again at (3), maintaining net negative energy pressure (7) inside C. The distal outflow (venous) pressure (6) enhances outflow at (4) and its elevation may turn the negative energy pressure (7) inside C into positive, increasing volume and pressure inside C chamber.

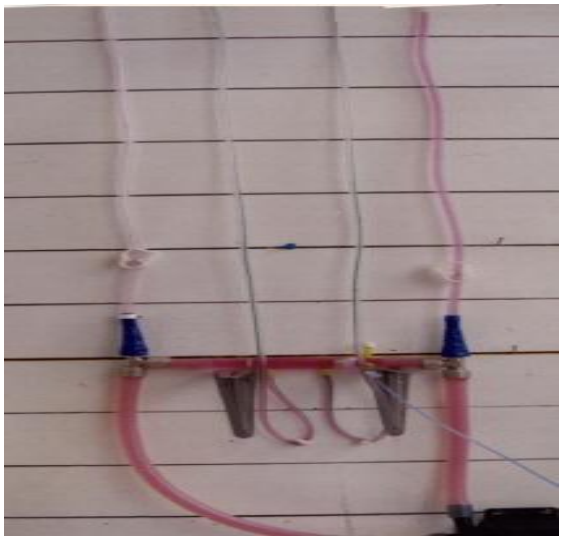

Figure 7. A circulatory system incorporating the G-C model with manometers measuring pressure at various points of the system. It is quite remarkable in mimicking circulatory system and the capillary-interstitial fluid circulation in health and disease.

enzymes are elevated. Cardiovascular shock prevails.

Respiratory system

Cyanosis

The lungs are involved with shock lung or ARDS.

Renal

The kidneys develop annuria which is unresponsive to diuretics. Acute renal failure occurs. Serum urea and creatinine rise.

Hepatic system

The liver function tests are elevated.

General

Trunk oedema or anasarca develops.

Therapy

As soon as the diagnosis of VOS is made further isotonic fluid infusions is contraindicated. VOS1 is treated with hypertonic sodium solutions in the form of 5% Sodium chloride and if not available 8.4% Sodium Bicarbonate is equally effective. This is given in fractionated doses of 200 ml over 10 minutes through a central venous line. The dose may be repeated up to 5 times. The effect of this therapy is magical [1,29]. The cardiovascular shock is corrected with elevation of arterial pressure. The patient recovers from coma. The kidneys respond with massive dieresis that should not be replaced. The treatment has proved equally effective in VOS2 that complicates VOS1 particularly when given early before the vital organs congestion developed into areas of necrosis and infarctions, and before MVOD becomes failure. Supportive measures on intensive care units are most helpful. If the kidneys do not respond by diuresis try haemodialysis- setting the net fluid balance to negative. Both VOS1 and VOS2 were induced in animals and treated successfully with hypertonic 5% NaCl [5].

Conclusion

TURS presents with VOS1 and volumetric overload represents its real patho-etiology. Previous authors have advanced Glycine toxicity as aetiology and the shock was mistaken for one of the recognized shocks. The massive interstitial tissue oedema affects all tissues and vital organs but signs of one organ may predominate. Recognizing TURS as VOS1 with its HN as marker helps to recognize VOS2. The correct and life saving therapy for VOS1 and VOS2 is hypertonic 5% NaCl or 8.4% Sodium Bicarbonate as prescribed. It is important to correct the faulty underlying Starling's law and advance the new mechanism for the capillary interstitial fluid transfer based on the hydrodynamics of the porous orifice (G) tube. The autonomous rapid dynamic magnetic field-like fluid circulation between the C and G tube lumen is self sufficient to explain the capillary-interstitial fluid exchange and replace Starling's law. I am certain that when the concepts presented here become fully appreciated, recognized and comprehended, it will save thousands of lives every year all over the World.

Conflict of interest

None declared.

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