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Extended Abstract
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The correct replacement for the wrong Starling's law is the hydrodynamic of the porous orifice (G) tube: Volumetric overload shocks in the patho-etiology of the transurethral resection prostatectomy syndrome and acute dilution hyponatraemia

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Findings: The TURP syndrome presents with shock usually mistaken for recognized shocks and treated with volume expansion with lethal outcome. The effective treatment is hypertonic sodium therapy (HST). Starling's law which dictates faulty rules on fluid therapy proved wrong. Replacement was found by studying the hydrodynamics of the porous orifice (G) tube.

Meaning: The TURP syndrome presents with volumetric overload shock (VOS) best treated with HST, Starling's law is wrong and the correct replacement is the hydrodynamic of the G tube.

Hypothesis: 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.

Objective: To report VOS and its successful treatment of hyper-tonic sodium therapy that is life saving. To report that Starling's law is wrong and the correct replacement is the hydrodynamic of the porous orifice (G) tube.

Methods: We conducted the following studies:

1. Prospective study on 100 consecutive TURP patients among whom 10 developed the TURP syndrome with acute dilution hyponatraemia (HN) and vascular shock.

2. A case series of 23 TURP syndrome cases.

3. A physics study on the hydrodynamic of the G tube.

Key points

Question: Does TURP syndrome present with shock, how best it should be treated and what is its relevance to Starling's law?

Results: The TURP syndrome 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. In adults VOS1 is induced by the infusion of 3.5-5 litres of sodium-free fluids and is known as TURP syndrome or HN shock. VOS2 is induced by 12-14 litres of sodium-based fluids and is known as the adult respiratory distress syndrome. The most effective treatment for VOS1 and VOS2 is hypertonic sodium therapy (HST) of 5%NaCl or 8.4% Sodium Bicarbonate. The literature on TURS is reviewed and the underlying patho-etiology is discussed. Starling's law proved wrong and the correct replacement is the hydrodynamic of the G tube.

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

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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.

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 enzymes are elevated. Cardiovascular shock prevails.

Respiratory system

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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: Volumetric overload causes shock of two types, VOS1 and VOS2. VOS 1 is characterized with acute dilution HN and is known as the TURP syndrome. Mistaking VOS1 for a recognized shock and treating it with vascular expansion is lethal while HST is life-saving. Starling's law which dictates the rules on fluid therapy proved wrong and the correct replacement is the hydrodynamic of the G tube.

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