REVIEW ARTICLE

Transurethral Resection of the Prostate (TURP) Syndrome: A Review of the Pathophysiology and Management

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Whenever irrigation fluid enters the intravascular space, dangerous complications can arise. This is best described as the transurethral resection of the prostate (TURP) syndrome. The syndrome has also been reported after endometrial ablation (1–5) and ureteroscopic procedures with irrigating solutions (6–8). TURP syndrome may occur as quickly as 15 minutes after resection starts (9–11), or up to 24 hours postoperatively (12). Of approximately 400,000 TURP procedures each year (13), 10% to 15% incur TURP syndrome (14,15) and the mortality is 0.2% to 0.8% (16,17). TURP syndrome affects many systems and manifests itself mainly through acute changes in intravascular volume and plasma solute concentrations (Figure 1). Despite this seemingly consistent etiology, TURP syndrome lacks a stereotypical presentation; therefore, its diagnosis is difficult (Table 1) (14,18–23). Further, recent work suggests that the conventional perioperative management of both TURP and the TURP syndrome may have to be revised.

The TURP Procedure

Surgical features of TURP vary (Table 2) (17,18,24–31). The irrigation used ranges from distilled water to a variety of nonhemolytic glycine, sorbitol, and mannitol solutions (17,32,33). No consistent correlation has been found between the volume of fluid absorbed, the duration of TURP, and the weight of removed tissue (11,14,29,30). The irrigation fluid either gains direct intravascular access (through the prostatic venous plexus), or is more slowly absorbed from the retroperitoneal and perivesical spaces (25,26,29) (Figure 2).

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TURP Syndrome: Intravascular Volume Shifts

Intravascular Volume Expansion

Both hypertension and hypotension (14,34–36) occur during TURP syndrome. The rapid volume expansion [one patient gained 3.3 kg in 20 minutes (11)] from absorbed irrigant during TURP can explain hypertension with reflex bradycardia (19,37,38). Absorption rates can reach 200 mL/min (29). Patients with poor left ventricular function may develop pulmonary edema from acute circulatory volume overload (39). A report of five patients with severe TURP syndrome (two deaths, two seizures, and one ventricular arrhythmia) found "no significant variations" in serum osmolalities before and after TURP (35), which suggests that intravascular volume changes independent of osmolality may play an important role in the morbidity and mortality associated with TURP syndrome. Several factors contribute to the volume gained, prominent among which are the intravesicular pressure (governed by the height of the irrigation bag above the prostatic sinuses) and the number of prostatic sinuses opened (37,39). Antidiuretic hormone produced by the stress of surgery (40), increased renin, and aldosterone secretion (41) may also contribute to volume expansion by promoting water retention.

Intravascular Volume Loss

Perioperative hypotension during TURP is sometimes preceded by hypertension (37,38,42). Profound hyponatremia by itself does not explain the hypotension (43). However, hyponatremia with hypertension may lead to net water flux along osmotic and hydrostatic pressure gradients out of the intravascular space and into the lungs, which triggers pulmonary edema and hypovolemic shock (37,42). This concept is consistent with the findings of Hahn (38), who reported that 12 patients absorbed more than 1 L of isotonic irrigant intravascularly during TURP. After the first 20 minutes of the procedure, the patients were hypervolemic and hypertensive and had increased central venous

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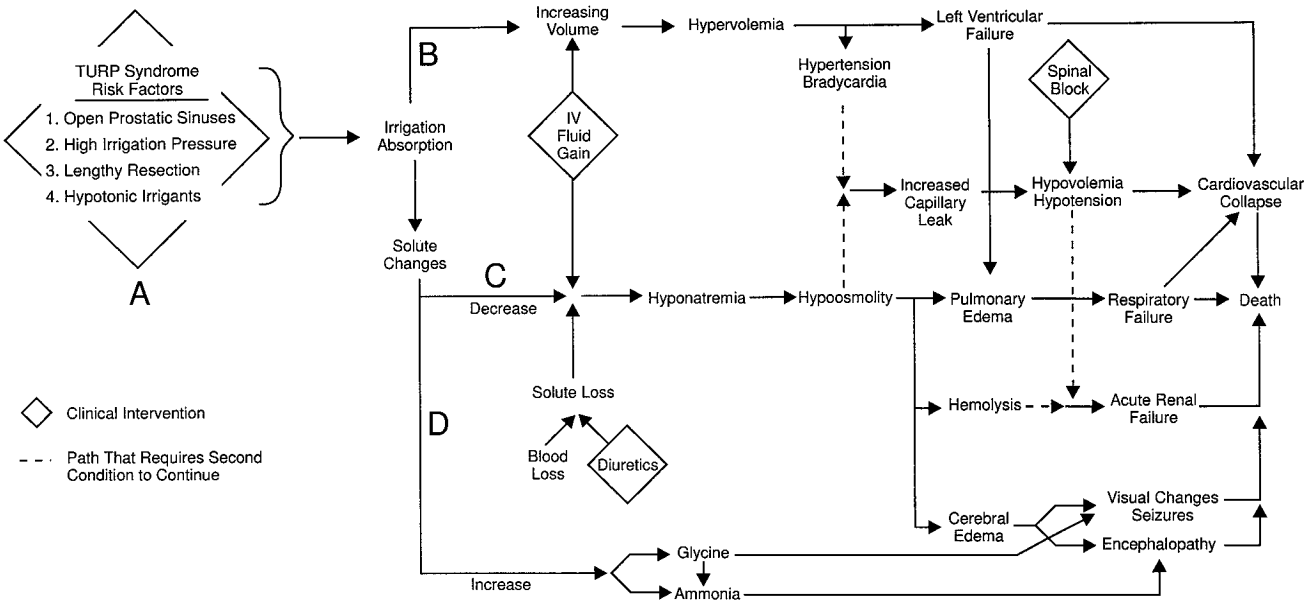

Figure 1. The variety of mechanisms and pathways that lead to transurethral resection of the prostate (TURP) syndrome. The triggering event is the entry of irrigation solution into the intravascular compartment (A), which increases intravascular volume (B) with its sequelae and decreases (C) and/or increases (D) solute concentration. The figure shows the complex interactions that need to be considered when the TURP syndrome unfolds. IV = intravenous.

Table 1. Signs and Symptoms Attributed to Transurethral Resection of the Prostate Syndrome by Major Physiologic System and Increasing Severity

Cardiopulmonary	Hematologic and renal	Central nervous system
Hypertension	Hyperglycinemia	Nausea/vomiting
Bradycardia	Hyperammonemia	Confusion/restlessness
Dysrhythmia	Hyponatremia	Blindness
Respiratory distress	Hypoosmolality	Twitches/seizures
Cyanosis	Hemolysis/anemia	Lethargy/paralysis
Hypotension	Acute renal failure	Dilated/nonreactive pupils
Shock	Death	Coma
Death		Death

pressure (CVP). After 30 to 35 minutes, when the rate of irrigant absorption slowed, flow from the plasma to the interstitium increased to an average of 75 mL/min and CVP decreased. Three patients then became suddenly hypotensive (systolic blood pressure ≤80 mm Hg), two of whom became hypotensive again after the procedure. Three other patients suddenly became hypotensive within the first postoperative hour. Such fluctuating intravascular fluid volume may explain the intraoperative hypervolemia and hypertension followed by postoperative hypovolemia and severe hypotension.

Sympathetic blockade induced by regional anesthesia may compound TURP syndrome. Intraoperative endotoxemia can occur in up to 45% of patients with negative preoperative urine cultures despite routine antibiotic prophylaxis (44).

Absorption of distilled water during TURP can cause acute hypoosmolality with massive hemolysis (45). Bleeding and red blood cell destruction are additional sources of volume and oxygen-carrying capacity losses. The hemoglobinemia that follows such hemolysis, coupled with hypotension, can cause acute renal failure and death (18,37,46).

Osmotically Active Solutes

Glycine, sorbitol, and mannitol are electrically nonconducting, but osmotically active, solutes that are added to irrigation fluids to decrease the risk of massive intravascular hemolysis. Their use in irrigation solutions has reduced the occurrence of significant hemolysis and death by more than 50% (46).

Although distilled water may still be used by some clinicians (17,18,24,36), the irrigation solutions most often used now range in calculated osmolality from 178 mOsm/kg water for 3% sorbitol to 200 mOsm/kg for 1.5% glycine solutions or to isotonic sorbitol or

Table 2. Surgical Features of Transurethral Resection of the Prostate by Patient Age

	Average	Maximum
Patient age (yr) (17,18,24)[a]	63–73	>90
Resection time (h) (17,18,23,24)	<1.2	3.5
Resected mass (g) (17,18,24,25)	22–24	110
Absorbed volume (L) (26,27)	1	8.8
Absorption rate (mL/min) (27–29)	10–30	200
Blood loss (L) (26,30,31)	0.176–0.534	3

[a] Numbers in parentheses are reference numbers.

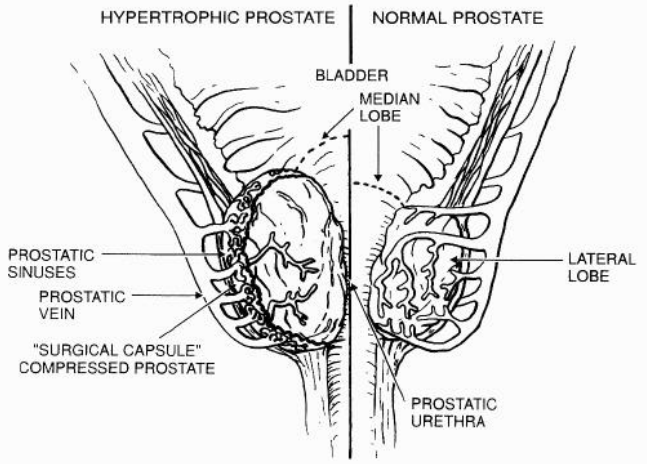

Figure 2. Anatomy of hypertrophic prostrate. The hypertrophic gland represents glandular and leiomyomatous hyperplasia of the submucosal glands and the smooth muscle of the prostatic urethra, which pushes the normal prostatic tissue to create a "surgical capsule." (Reproduced with permission from Wong KC, Liu W-S: Anesthesia for urologic surgery. In: Stoelting RK, Barash PG, Gallagher TJ, eds. Advances in anesthesia. Vol 3. Chicago: Year Book Medical, 1986:379).

glycine solutions. Osmolality calculations for irrigation solutions assume that there is no interaction between solute particles. Since these interactions do occur, calculated osmolality values are slightly greater (10–20 mOsm/kg) than the solution's measured osmolality.

Treatment of Intravascular Fluid Volume Shift. Massive absorption is more likely if intravesicular pressure increases above 30 mm Hg. Limiting the height of the irrigation bag to 40 cm above the prostate (47) or using continuous irrigating resectoscopes or suprapubic trocar drainage (48) can minimize absorption. If intravesical pressure is kept below 15 cm H_2O, absorption virtually ceases (10).

Resection time under one hour and leaving a rim of tissue on the capsule until near the end of the procedure, where it can either be left (if signs of TURP syndrome are evident) or removed all at once, may reduce the time that a large number of prostatic sinuses are open and thus capable of absorbing fluid (14,37) (Figure 2).

Table 3. Factors That, Alone or in Combination, Can Affect the Central Nervous System During Transurethral Resection of the Prostrate

	Substrate	
Drugs	Solute	Hypoxia
Benzodiazepine	Hyperammonemia	Congestive heart failure
Local anesthetic	Hyperglycinemia	High spinal anesthesia
Narcotic	Hypoglycinemia	Myocardial infarction
	Hyponatremia	Pulmonary edema
	Hypoosmolality	Sepsis
		Stroke

The most widely used indicator of volume gain is serum sodium dilution (14,31) or breath-alcohol level when ethanol is used as a tracer in the irrigation solution (1,30,49). Other methods follow volumetric fluid balance (14,38,49), CVP trend (44), plasma electrolyte concentrations (e.g., magnesium and calcium) (50), irrigation solutes [glycine (31), sorbitol (33)], transthoracic impedance change (51), and the patient's weight gain (52).

No method guarantees that TURP syndrome will be avoided (36). Before reflexively treating TURP syndrome with hypertonic saline, hypervolemia with near-normal osmolality must be excluded (39,53). Symptomatic cardiovascular or pulmonary compromise requires aggressive intervention. After adequate pulmonary gas exchange and hemostasis are established, administration of blood, the positive inotropic agents calcium and magnesium (50), or diuretics or augmentation of intravascular volume may be needed.

TURP Syndrome: Plasma Solute Effects

Solute changes may alter neurologic function independent of volume-related effects (Table 3). Acute hyponatremia caused by the rapid absorption of a large volume of sodium-free irrigation fluid—one form of acute water intoxication—can trigger the central nervous system (CNS) complications but other factors may play a role (11,37,54). Spinal anesthesia associated with hypotension can contribute to nausea and vomiting (20). CNS symptoms may also be caused by the derangements of sodium, osmolality, ammonia, glycine, and also benzodiazepines and narcotics.

Hyponatremia

Profound hyponatremia has been implicated as the cause of visual aberrations, encephalopathy, pulmonary edema, cardiovascular collapse, seizure, and

death (54). The incidence of serum sodium concentration less than 125 mmol/L after TURP may reach 15% (15) with a mortality of 40% when hyponatremia is symptomatic (headache, nausea, vomiting) (53,55). Dilutional hyponatremia may be aggravated by electrolyte losses into accumulations of infused but extravasated nonelectrolyte fluid (43).

Hyponatremia is common, and serum sodium concentration decreases of 6 to 54 mmol/L have an incidence ranging from 7% to 26% (34,39). Decreases from a normal preoperative level to 113 and 104 mmol/L after just 15 minutes of resection with isotonic irrigants have been reported (9,11).

Even markedly hyponatremic patients may show no signs of water intoxication (31,39). Neither a decrease in serum sodium concentration from 34 to 54 mmol/L without TURP syndrome nor a significant change in serum osmolality has been reported (39). When 3% mannitol irrigation was used on one TURP patient, serum sodium concentration decreased from 133 to 99 mmol/L (56). Osmolality was measured postoperatively at 290 mOsm/kg but was calculated at 239 mOsm/kg. This difference was attributed to the osmotic effect of mannitol not accounted for by the calculation. Although severe hyponatremia has been associated with hemolysis and renal failure (36), cardiovascular and electrocardiogram changes (11,35,50), respiratory compromise (11,57), seizure (35,58,59), coma (11,58), and death (11,34,35,57,58), other hyponatremic patients did well. Hyponatremia may not be the sole or even the primary cause of the neurological manifestations of TURP syndrome.

Hypoosmolality

The crucial physiological derangement of CNS function is not hyponatremia *per se,* but acute hypoosmolality. This is to be expected because the blood-brain barrier, with an effective pore size of 8 Å, is essentially impermeable to sodium but freely permeable to water (60). In rabbits, signs of water intoxication induced by administration of vasopressin and 2.5% glucose solution were reversed by administration of osmotically active agents such as urea and mannitol without correcting the serum sodium concentration (61).

One method of determining the independent neurophysiological effects of serum sodium concentration and osmolality has been to measure field potentials from prepared brain slices. Field potentials can be triggered or arise spontaneously and represent the voltage generated by the synchronous discharge of many neurons aligned in parallel.

Hypotonic saline (Na^+ of 123 mmol/L and a tonicity of 245 mOsm/kg) as a brain slice superfusate increased the amplitude of field potentials in rat hippocampal slices. Correcting the osmolality with mannitol, while maintaining the low sodium concentration, returned field responses to near control levels. The increased field response, therefore, results from a decreased osmolality, and not a decreased sodium concentration, which is consistent with predictions based on the Nernst equation (61).

The Nernst equation predicts that the decrease in extracellular sodium concentration that accompanies the hypoosmolality seen with TURP only minimally alters neuronal excitability. Replacing a Na^+ value of 140 mmol/L with 100 mmol/L in the Nernst equation increases the calculated transmembrane resting potential of −60 mV by 9 mV. Thus, theoretically, serum sodium concentration should not substantially contribute to neuronal excitability independent of serum osmolality, even when these changes are of the magnitude typically associated with severe TURP syndrome.

The brain reacts to a sustained hypoosmotic stress within seconds to minutes, with intracellular decreases in Na^+, K^+, Cl^-, and in so-called "idiogenic osmoles," which act to decrease intracellular osmolality and prevent swelling (61). However, with acute osmotic change (within hours or even minutes), such compensatory mechanisms may not work fast enough (61). Cerebral edema caused by acute hypoosmolality can increase intracranial pressure, which results in bradycardia and hypertension by the Cushing reflex (62). Furthermore, cerebral edema is not caused by decreased serum colloid oncotic pressure, but by decreased osmolality (63).

Only a few studies correlate a patient's fate after TURP with both serum sodium concentration and osmolality (14,39,46). In a series of 72 patients undergoing TURP, serum sodium concentration decreased by 10 to 54 mmol/L in 19 (26%), while osmolality changed in only two (3%) (39). The two patients who had both hyponatremia (serum sodium concentration decreases of 27 and 30 mmol/L) and hypoosmolality (serum osmolality of 260 and 256 mmol/L) developed pulmonary edema and encephalopathy. The five patients in this series with the largest decreases in serum sodium concentration (by 34 to 54 mmol/L) had no changes in serum osmolality and no signs of TURP syndrome.

Treatment of Hyponatremia and Hypoosmolality. Precautions can be taken to avoid severe hyponatremia and hypoosmolality. Diuretics have been implicated in the rapid onset of hyponatremia (64). When used routinely or to treat hypervolemia after TURP, they may worsen hyponatremia and hypoosmolality and, thus, lead to TURP syndrome. Furosemide and bumetanide act within minutes on the ascending loop of Henle where they inhibit chloride uptake, which causes urinary sodium loss and promote salt-wasting after TURP (41,50). Mannitol also causes sodium losses during the first 12 hours after TURP but does

not lower serum level during the first three to five postoperative hours (65).

A patient's serum sodium concentration and osmolality may continue to decrease for some time after the procedure because much irrigant is slowly absorbed from the perivesicular and retroperitoneal spaces (26). The TURP syndrome can start 4 to 24 hours later (12,14,27,54) with coma, blindness, grand mal seizures, and hemiplegia (14,54). Problems may be forestalled by using loop and other salt-wasting diuretics with a concomitant infusion of saline—even in the presence of near normal serum sodium concentration—during the first 12 postoperative hours (41,65).

Pretreatment with hypertonic saline may decrease the degree of dilutional hyponatremia (66). This approach may decrease the incidence of TURP syndrome caused by hypoosmolality, but likely will exacerbate the incidence and severity of the syndrome's hypervolemia manifestations. Because the serum sodium concentration need not reflect serum osmolality (35,39,56), serum sodium concentration should be reported together with osmolality when the irrigant solution contains osmotically active solutes (such as glycine, mannitol, or sorbitol). If osmolality is near normal, no intervention to correct sodium is recommended for asymptomatic patients, even in the face of reduced serum sodium concentration.

The most feared complication of correcting hyponatremia is central pontine myelinolysis (CPM). Because demyelination can occur in extrapontine areas, the disease is also referred to as "osmotic demyelination syndrome" (67). Although CPM is most often seen in women [probably due to sex differences in cellular ion pump capacity (55)], it has been reported after rapid as well as slow correction of serum sodium concentration in TURP patients (59,68).

When treatment is instituted too slowly for symptomatic hyponatremia ($\leq$0.7 mmol $\cdot$ L^{-1} $\cdot$ h^{-1}) (57,68), it has been associated with a higher morbidity and mortality than has rapid correction ($\geq$1.0 mmol $\cdot$ L^{-1} $\cdot$ h^{-1}) (64). Many reports (58,59, 69–71) suggesting that a 1.5- to 2.0-mmol $\cdot$ L^{-1} $\cdot$ h^{-1} correction rate is safe have failed to consider changes in osmolality. Several investigators have suggested that osmotic stress is probably greater when correcting chronic compared with acute hyponatremia (72,73).

Extracellular changes in blood osmolality equilibrate within minutes across the blood-brain barrier and brain cell membranes (61). Hypoosmotic stress affects intracellular ion and amino acid concentrations (72,73). There is up-regulation of the processes for the export of organic osmolytes from the cell and down-regulation of the synthesis of amino acids in response to hypoosmotic stress (61). Once induced by hypoosmolality, it is not known how fast the elimination of these amino acids can be reversed by the correction of the hypoosmolality. If the reversal is slow, the rapid correction of acute asymptomatic hyponatremia or hypoosmolality may be clinically indistinguishable from correction of chronic hyponatremia and hypoosmolality.

The presence of symptoms has been described as the single most important factor determining morbidity and mortality from hyponatremia (55). The safest treatment of hyponatremia and hypoosmolality may be symptomatic (74). Instituting therapy in the absence of symptoms risks too rapid a correction because the correction rate is difficult to control (59). Therefore, osmolality should be monitored and corrected aggressively only until symptoms substantially resolve; then correction should be continued slowly (Na^+ correction $\leftarrow$ 1.5 mmol $\cdot$ L^{-1} $\cdot$ h^{-1}).

Hyperammonemia

The portal bed and kidneys can metabolize glycine that gains intravascular access. The primary pathway used by the liver and kidneys is oxidative deamination (75), which leads to the formation of two potentially toxic metabolites: glyoxylic acid and ammonia (76). The brain also contains a glycine cleavage enzyme system that splits glycine into carbon dioxide, a one-carbon fragment, and ammonia (77).

It seems clear that an increase of serum ammonia during TURP is the result of glycine absorption because patients undergoing retropubic resections without glycine do not develop hyperammonemia (78). The alteration of CNS function caused by hyperammonemia may be a factor in perioperative management, however, the role of hyperammonemia in TURP syndrome remains unclear.

In the treatment of hyperammonemia, the methods for limiting the increase of plasma ammonia concentration when glycine irrigants are used include L-arginine, which acts in the liver by preventing hepatic release of ammonia and accelerating ammonia conversion to urea. The time necessary to deplete endogenous arginine stores may be as little as 12 hours, which approximates preoperative fast time (79). Prophylactic administration of intravenous L-arginine markedly moderated the increase in blood ammonia concentration in fasting patients receiving intravenous glycine. Infusion of L-arginine with or at the conclusion of glycine administration prevented further increases in blood ammonia concentration and accelerated its return to normal (79). Doses between 4 g (20 mmol) infused over three minutes and 38 g (180 mmol) infused over 120 minutes have been recommended (79). No toxicity was noted with either of these regimens. The purchase cost of L-arginine is approximately $85 per 300-mL bottle of a preservative-free 10% L-arginine solution with a calculated osmolality of 950 mOsm/kg water (R-GENE 10®; Kabivitrum, Franklin, OH).

Hyperglycinemia

Glycine is a major inhibitory neurotransmitter like γ-aminobutyric acid (GABA) in the spinal cord and midbrain and may have a significant role in higher cortical neurotransmission (80). Its role in post-TURP encephalopathy and seizures is suggested by nonketotic hyperglycinemia or glycine encephalopathy. This disease is a heritable affliction characterized by a defect in the glycine cleavage enzyme system, disturbed electrophysiologic function, intractable seizures, lethargy, spasticity, mental retardation, and death within the first few months of life (81). These patients have a plasma glycine level up to 10 times greater than that of normal infants (mean range, 266–2027 μmol/L; normal infant level, 209 μmol/L) (77,81).

Glycine may lead to encephalopathy and seizure via *N*-methyl-D-aspartate (NMDA), an excitatory neurotransmitter. NMDA receptor activity is markedly potentiated by glycine and, along with its role as an inhibitory transmitter, may facilitate excitatory transmission in the brain through an allosteric activation of the NMDA receptor (82).

A large concentration of glycine may be harmless in plasma but can be fatal in the brain (77). Signs of glycine toxicity include nausea, vomiting, headache, malaise, and weakness. They manifest at an infusion rate of 3.5 mg · kg^{-1} · min^{-1} (83), which, in a 70-kg man, represents an intravascular absorption of 1.5% glycine solution at the rate of 54 mL/min. Serum glycine after TURP has been reported at a level greater than 14,300 μmol/L (14,31,78). This concentration is 17 times greater than that in children dying from glycine encephalopathy and over 65 times that in adults (normal adult level, 219 μmol/L) (77).

Visual disturbances in TURP syndrome vary in severity from blurred vision (23) to complete blindness (22,54,84,85). Several investigators have remarked on the unusual calmness of their patients facing what would seem to be an extremely frightening complication (22,85). Some patients present with sluggish or fixed and dilated pupils and total loss of light/dark discrimination (21,22,27,84,85). Atropine (84) or hyponatremia and cerebral edema from overhydration may contribute to these visual disturbances (27,54). Patients with cortical blindness, on the other hand, lose all visual sensation (light perception and the blink reflex) but retain the pupillary responses to light and accommodation (86). Although it is difficult to separate the effects of serum sodium concentration from those of other retinal transmitters, sodium appears to play only a minor role in the visual disturbances (87).

Glycine is now gaining acceptance as the most likely cause of visual aberrations during the TURP syndrome (23). A wide range of serum glycine levels have been documented in patients with visual change. These have led to speculation of a serum glycine concentration threshold for symptomatic visual impairment (>4000 μmol/L) (23) and blindness (>13,734 μmol/L) (22). Glycine is probably a major inhibitory neurotransmitter in the retina (80). It is found in the inner plexiform layer, amacrine, and bipolar cells of the human retina and causes hyperpolarization of ganglion cells (87). The sensitivity of oscillatory potentials of the electroretinogram and visual evoked potentials to glycine in the absence of large osmolality changes has been demonstrated (23). Therefore, glycine appears to affect the retinal physiological condition independent of cerebral edema caused by hypoosmolality.

Glycine may also exert toxic effects on the kidney (88). A study in rats found histologic evidence of glycine toxicity in their kidneys six hours after either intravenous or intraperitoneal administration of large doses of 1.5% glycine solution. No toxicity was found after injecting similar volumes of retroperitoneal water or lactated Ringer's solution. This study did not investigate whether the kidney would eventually recover from the apparent toxic insult. Hyperoxaluria from metabolism into oxalate and glycolate has also been proposed as a route whereby glycine could cause renal failure in susceptible patients (89).

In considering the treatment of hyperglycinemia, glycine may be involved with TURP encephalopathy and seizure through its positive action on the NMDA receptor-channel system, as it is in glycine encephalopathy (90,91). Seizures after TURP associated with hyponatremia and hypoosmolality are likely to be resistant to benzodiazepine and anticonvulsant therapy; in fact, such treatment may provoke apnea (74). Theoretically, a NMDA-receptor antagonist (91) or glycine antagonist (90) are better choices.

Magnesium exerts a negative control on the NMDA receptor (82,91). A serum magnesium level lowered by dilution may increase susceptibility to seizures. Magnesium may be dramatically lowered after TURP in patients who have been treated with a loop diuretic (50). Therefore, a trial of magnesium therapy for seizures in patients in whom a glycine irrigant was used during TURP deserves consideration, especially if measured osmolality is near normal.

Vision returns to normal within 24 hours as glycine approaches normal (14,22,85). This is predictable because the half-life of glycine is approximately 85 minutes (31). Reassurance that unimpaired vision is expected to return may be the best treatment.

TURP Syndrome: Other Considerations

Benzodiazepines are known to act at the GABA receptor, and thereby may mediate some compromise of vision through the activation of the retinal GABA receptor. Diazepam increases the latency of visual

evoked potentials and decreases their amplitude in both rats (92) and rabbits (93). Narcotics can contribute to sedation and nausea. When these drugs are used their effects must be considered in the differential diagnosis of the TURP syndrome.

Conclusion

Our understanding of the pathophysiology leading to the TURP syndrome has improved in recent years. Complex changes in intravascular volume, solute, and neurophysiologic function mark the TURP syndrome. The prevention, diagnosis, and treatment of TURP syndrome is challenging, because aberrations of solute and volume can occur simultaneously and may suggest opposing diagnoses and treatments (Figure 1).

Although monitoring serum sodium concentration during TURP is common practice and is effective for assessing intravascular absorption, there may be a benefit from monitoring serum osmolality as well. Hypoosmolality appears to be the principle culprit contributing to neurological and hypovolemic changes. Supportive care remains the mainstay of management for renal, pulmonary, and cardiovascular complications of TURP syndrome. Several therapies warrant consideration in formulating a management plan for hyperammonemia, hyperglycinemia, hyponatremia, hypoosmolality, encephalopathy, and seizures after TURP.

It is also likely that these pathophysiological processes and therapeutic interventions can be applied to a nearly identical syndrome occurring in women undergoing endometrial ablation with saline, glycine, or sorbital solutions (2–5). When 32% dextran 70 irrigation (Hyskon; Medisan, Parsippany, NJ) is used, however, the major concerns pertain to dextran reaction, which may occur with absorbed volumes of 500 mL or more (2). Hypoosmolality is not a concern since Hyskon® has a calculated osmolality of 3005 mOsm/kg.

It is clear from the decreasing incidence of TURP syndrome over the past 40 years that progress has been made in its prevention and treatment. Despite this progress, many questions remain. Definitions of what constitutes chronic and acute hyponatremia and osmolality elude us. The etiology of the encephalopathy associated with TURP syndrome remains a matter of speculation. The roles of hyponatremia and hypoosmolality and how to avoid CPM when correcting them are yet to be described. The introduction of new therapies for the medical and surgical management of prostatic hypertrophy (94–97) may minimize risks of TURP syndrome in the future.

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