Continuing Medical Education

The TURP syndrome

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This article discusses the presentation, aetiology, treatment and prevention of central nervous system disturbances after transurethral resection of the prostate. Nausea and vomiting, visual symptoms, and altered states of consciousness have been reported as complications due to intravascular absorption of irrigating fluid. Hypotonicity after absorption of the irrigating fluid causes cerebral oedema. Hyperglycinaemia may cause visual disturbances and hyperammonaemia may cause delayed coma.

Cet article discute de la présentation, l'aétiologie, le traitement et la prévention des problèmes du système nerveux central après RTUP. Les nausées, les vomissements, les symptômes visuels, l'altération de la conscience ont été rapportés comme complications dû à une absorption intra-vasculaire du liquide d'irrigation. L'hypotonicité après absorption du liquide d'irrigation provoque l'œdème cérébral. L'hyperglycinémie peut causer des perturbations de la vision et l'hyperammoniémie peut provoquer un coma retardé.

Key words

COMPLICATIONS: TURP syndrome;
SURGERY: urological;
IONS: sodium.

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Central nervous system (CNS) complications after intravascular absorption of irrigating fluid during transurethral resection of the prostate (TURP) range in severity from minor to life-threatening and depend on the rate and amount of absorption. Glycine, 1.5 per cent, is the most commonly used irrigating fluid. Because glycine is a permeant solute, intracellular oedema may occur upon absorption. Toxic effects may occur from high concentrations of ammonia, which is a by-product of the metabolism of glycine, and from high concentrations of glycine itself. Acute severe hypotonicity leading to cerebral oedema is the most serious complication and requires therapy with hypertonic saline. The rate at which the hyponatraemia should be corrected is controversial. Hyperglycinaemia may cause visual disturbances and

TABLE I TURP syndrome – presentation

<i>CVS Components</i>	<i>CNS Components</i>
Increase in central venous pressure	Apprehension
Bradycardia	Disorientation
Hypertension	Nausea
Angina	Visual disturbances
Electrocardiographic changes	Stupor
CV collapse	Coma
	Seizures

hyperammonaemia may cause delayed coma but both require only supportive therapy. Nausea and vomiting may be side-effects of hyponatraemia or may result from elevated concentrations of nonessential amino acids which are metabolites of glycine. The TURP syndrome is an iatrogenic surgical complication but the responsibility for its diagnosis and treatment often falls upon the anaesthetist.

Transurethral resection of the prostate is frequently complicated by absorption of the irrigating fluid which is used to distend the bladder during surgery.¹⁻³ Because of this absorption, there are continuous and complicated shifts of water and electrolytes in the body.⁴

Presentation

The TURP syndrome comprises CNS and cardiovascular system (CVS) alterations (Table I) resulting from intravascular absorption of irrigating fluid.^{1,5}

Nausea and vomiting, visual disturbances, and altered states of consciousness have been reported to occur after TURP.¹ The aetiology of these CNS disturbances has been attributed to hyponatraemia, hyperglycinaemia, and/or hyperammonaemia. Hyponatraemia may occur when any of the irrigating fluids is used, but hyperglycinaemia and hyperammonaemia may occur only when glycine is used as the irrigating fluid. This review will consider the presentation, aetiology, treatment, and prevention of these CNS disturbances.

Altered states of consciousness

Acutely altered states of consciousness from mild confusion to stupor and coma have been reported after TURP.^{1,2,6} Hoekstra noted that one of 30 patients had an altered sensorium after TURP using glycine 1.5 per cent as the irrigating fluid.⁷ Henderson reviewed the charts of 2000 patients who had undergone TURP with glycine 1.5 per cent and identified 78 cases of hyponatraemia. Of these, an unidentified number had agitation or vomiting or confusion, and 14 deteriorated into a comatose or semi-comatose state.⁶

The onset of the comatose state is quite variable, ranging from 15 min after surgery has started to ten hours

after surgery has been completed. The neurological examination is consistent with a metabolic encephalopathy. In the more severe cases the patient may have focal or generalized seizures. Mild papilloedema and decerebrate movements may be present. The coma lasts from 8 to 120 hr. Upon discharge from hospital, the 14 cases of coma after TURP reported by Henderson were neurologically unchanged from the premorbid state.

Visual disturbances

Visual disturbances have been reported as a complication of TURP but only when glycine has been used as the irrigating fluid and when hyponatraemia has occurred.⁸ The reports range from "dimming of vision", "light perception only," to "no light perception" for several hours.⁹ Patients are usually alert when altered vision is present but they also complain of nausea. The onset of symptoms may occur from 30 min after the start of resection to six hours following its completion. Vision returns to normal within 2 to 12 hr. Fundoscopic examination is normal with no papilloedema and the pupillary response to light is maintained in those patients who are minimally affected. However, patients whose visual acuity is decreased to light often only have dilated and unresponsive pupils. The intraocular pressure remains normal.¹⁰ Electroretinograms are abnormal in patients with visual impairment,^{9,11} while visual evoked potentials are not consistently affected.¹¹

Aetiology

Irrigating fluid

PROPERTIES

The properties of the ideal irrigating fluid are shown (Table II) because intravascular absorption of the irrigating fluid is the cause of the TURP syndrome.¹² The fluid must be nonelectrolytic to disperse the electrical current. Water allows clear visibility by the surgeon but it is hypotonic and causes haemolysis when absorbed.² Glycine, sorbitol, mannitol, and urea are solutes which have been added to water to bring its osmolality closer to that of

TABLE II The ideal irrigating fluid

Isotonic
Nonhaemolytic
Non-toxic when absorbed
Nonelectrolytic
Not metabolized
Allows clear visibility
Does not influence osmolality
Rapidly excreted
Osmotic diuretic

TABLE III Factors influencing absorption of irrigating fluid

Hydrostatic pressure of irrigation fluid
Number and size of venous sinuses opened
Peripheral venous pressure
Duration of surgery
Experience of surgeon

plasma. Ideally these solutes and their metabolites should be nontoxic. Mannitol is not metabolized but undergoes urinary elimination; sorbitol is metabolized to glucose; and glycine and its metabolites may not be as nontoxic as was initially thought.

ABSORPTION

During TURP both intravascular and extravascular absorption of irrigating fluid may occur. Intravascular absorption occurs through open prostatic vessels when the irrigating pressure exceeds venous pressure. Perivesical absorption occurs with perforations of the prostatic capsule. A radioisotope technique found that only 29 per cent of the absorbed fluid was absorbed intravascularly.¹³ Factors which influence the extent of absorption are listed in Table III.³ Irrigating fluid absorption may occur throughout the TURP although the risk appears to be greatest 30 min after surgery commences.¹⁴ Increased blood loss during TURP coincides with intravascular but not extravascular absorption.¹⁴

Effects of absorption

SODIUM

The absorption of large amounts of electrolyte-free irrigating fluid intravascularly leads to dilutional hyponatraemia.¹ With extravascular absorption, hyponatraemia is less pronounced and could be delayed to the morning after the TURP.¹⁵ A decrease in serum sodium concentration to less than $120 \text{ mmol} \cdot \text{L}^{-1}$ indicates a severe TURP reaction.^{1,2} A decrease in serum (Na) of 20 to $30 \text{ mmol} \cdot \text{L}^{-1}$ implies absorption of 3 to 4 L.⁴ Hahn concluded that continuous and complicated shifts of water and electrolytes occur in the body during TURP.⁴ The changes in serum (Na) were related to glycine absorption during the course of TURP. The serum (Na) at the end of TURP only roughly correlated with the volume of irrigating fluid which was intravascularly absorbed. The absorption time is also an important factor; only rapid massive absorption can produce the very low sodium concentrations typical of severe TURP reactions. Diffusion of water into the intracellular space and renal elimination of excess water are factors which reduce the degree of hyponatraemia.⁴

Certain signs and symptoms have been observed as

TABLE IV Symptoms and acute changes in Serum (Na)

Serum (Na)	Electrocardiogram	CNS
$120 \text{ mmol} \cdot \text{L}^{-1}$	Possibly widening QRS	Restlessness Confusion
$115 \text{ mmol} \cdot \text{L}^{-1}$	Widened QRS Elevated ST segment	Nausea Semicoma
$100 \text{ mmol} \cdot \text{L}^{-1}$	Ventricular tachycardia or fibrillation	Seizures Coma

serum Na concentrations decrease (Table IV).^{1,16} Symptoms of hyponatraemia are related to the speed of its development. Acute changes produce more symptoms than chronic hyponatraemia.¹⁷ After 24 to 48 hr of sustained hypotonicity, the brain cells adapt by extruding solute, which reduces cell swelling.¹⁸

OSMOLALITY

Osmolality is an expression of the total number of particles in a given weight of solvent. Osmolality refers to the number of osmoles per kilogram of solvent and osmolarity refers to the number of osmoles per litre of solution. At the solute concentrations of the body fluids, the two measurements correspond so closely that no correction is required.¹⁹ Glucose, urea and sodium and its accompanying anions contribute almost 100 per cent of the measured osmolality of plasma under normal circumstances. The formula used to calculate plasma osmolality is $2 \times (\text{Na}) + (\text{glucose}) + (\text{urea})$ where the concentrations of these compounds are measured in $\text{mmol} \cdot \text{L}^{-1}$. The actual measurement of osmolality is based on freezing point depression and the difference between measured and calculated osmolality is called the osmolal gap.¹⁹

Glycine, 1.5 per cent, has an osmolality of $188 \text{ mosm} \cdot \text{kg}^{-1}$ which is lower than the normal plasma osmolality of $285 \text{ mosm} \cdot \text{kg}^{-1}$.²⁰ When this solution is absorbed intravascularly, hyponatraemia may occur but the serum osmolality may remain close to normal. Desmond studied 72 patients undergoing TURP with glycine 1.2 per cent.²¹ Nineteen of these 72 patients developed decreases in serum (Na) but only two patients had decreases in serum osmolality. In a patient with TURP syndrome, the osmolality gap will be greater than $10 \text{ mosm} \cdot \text{kg}^{-1}$, which indicates the presence of solute other than that included in the calculated osmolality.¹⁹

TONICITY

Tonicity describes the osmotically effective solute in a solution. Effective solutes are relatively restricted to one particular body fluid compartment and are able to exert an osmotic force for water movement from other compartments. Tonicity cannot be measured but can be estimated as follows: $2 \times (\text{sodium}) + (\text{glucose})$.²² Mannitol and

sorbitol are osmotically effective solutes and would be included in this calculation if they were absorbed intravascularly. Urea passes freely across all membranes and is not an effective solute.¹⁹ Glycine is a small amino acid which moves intracellularly upon absorption.¹²

In summary, sorbitol and mannitol contribute to tonicity and will increase the extracellular fluid volume when absorbed. However, urea and glycine do not contribute to tonicity and will therefore expand both the intracellular and extracellular spaces. The changes in body fluid tonicity, rather than changes in osmolality, lead to alterations in cell volume. Acute hypotonicity is associated with cerebral oedema.²² The signs of metabolic encephalopathy associated with the TURP syndrome have been attributed to cerebral oedema¹³ but other authors have also implicated the toxicity of glycine and its break-down products.¹

COLLOID OSMOTIC PRESSURE

With an irrigant absorption of between 0.5 and 2.8 L, plasma protein concentrations decrease to 70 to 80 per cent of preoperative levels.²³ However, this effect alone does not promote cerebral oedema. An acute decrease in oncotic pressure does not promote an increase in cerebral water content in the noninjured brain.²⁴

POTASSIUM

There is no change in serum (K) during TURP. However, there may be a small increase of $0.5 \text{ mmol} \cdot \text{L}^{-1}$ in those patients experiencing intravascular absorption of glycine.¹⁵ The cause is not known, but it has been speculated that high concentrations of glycine may interfere with transmembrane electrolyte exchange.¹² Some haemolysis has been reported with the use of glycine, 1.5 per cent,²⁰ and this may contribute to the increase in serum (K).

Glycine

HIGH CONCENTRATIONS OF GLYCINE

There may be a higher incidence of CNS disturbances with glycine than with other irrigating solutions. Two groups of patients undergoing TURP were compared when glycine 1.5 per cent and sorbitol three per cent were used.²⁵ Three of the 21 patients in the glycine group exhibited signs of encephalopathy but the 12 patients in the sorbitol group did not.

The 1.5 per cent solution of glycine contains 1000 times the normal concentration of glycine in plasma.²⁶ The serum glycine concentration after TURP was elevated to ten times normal levels in one study even though sodium concentrations remained normal.²⁷ Two of 21 patients in this series had some nausea associated with the elevated glycine concentrations but the others showed no adverse effects. Visual impairment may occur when

FIGURE Metabolism of glycine. (Modified from Hahn.²⁹)

serum glycine concentrations are 30 times normal (at $4000 \mu\text{mol} \cdot \text{L}^{-1}$).¹¹ Glycine is an inhibitory neurotransmitter in the mammalian central nervous system²⁸ and may act on receptors in the retina to cause blindness. The mechanism of visual loss may be a function both of CNS toxicity from glycine and hypotonicity resulting in cerebral oedema.⁸

METABOLISM

Glycine is a simple amino acid which is involved with numerous reactions in the metabolic chain. The Figure shows a simplified version of the metabolism of glycine. Ammonia is a by-product of this reaction and other nonessential amino acids are metabolites. The symptoms of nausea and vomiting have been associated with a rapid increase in the concentrations of several amino acids, including serine, alanine, and glutamate.²⁹ Urinary excretion is not important for elimination of glycine from the body.¹²

AMMONIA

High ammonia concentrations have been implicated in the pathogenesis of coma after TURP. The cerebral depression due to hyperammonaemia may enhance or act independently of dilutional hyponatraemia. Deterioration in cerebral function has been documented when blood ammonia concentration exceeds $150 \mu\text{mol} \cdot \text{L}^{-1}$.³⁰ Hyperammonaemia causes an excess of cerebral inhibitory neurotransmitters and a deficit of excitatory neurotransmitters which lead to coma.⁷

A marked elevation of glycine concentration may be associated with normal ammonia concentrations.⁷ Conversely, some patients with high ammonia levels after TURP have only mild glycine elevations, possibly reflecting a more rapid breakdown of absorbed glycine.⁷ Ammonia is normally converted to urea in the liver through the urea cycle. Patients with a nutritional deficiency of arginine³ and those with pre-existing liver disease³¹ are more likely to develop high ammonia concentrations.

Treatment

The mildly symptomatic patient

If a patient exhibits nausea, vomiting, agitation, confu-

TABLE V Principles of management of comatose patients

- 1 Assure oxygenation
- 2 Maintain circulation
- 3 Lower intracranial pressure
- 4 Stop seizures
- 5 Treat infection
- 6 Restore acid-base balance and electrolyte balance
- 7 Adjust body temperature

sion and/or visual disturbances, while the CVS is stable, then monitoring and observation should continue until the symptoms resolve. Blood should be sent for electrolyte estimation. A serum sodium concentration of $120 \text{ mmol} \cdot \text{L}^{-1}$ is the borderline between mild and severe reactions.¹ A loop diuretic may be given if there is evidence of fluid overload, but most patients in this category will diurese spontaneously and may require no further treatment.^{1,2}

If there are visual disturbances, an ophthalmologist should be consulted to eliminate other causes of impaired vision. There is no specific treatment for the visual disturbances of the TURP syndrome. The patient should be reassured that vision will return by the next day.⁸

The unconscious patient

The approach to the unconscious patient is well outlined in the monograph by Plum and Posner.³² Certain general principles of management should be addressed (Table V). The differential diagnosis of states causing sustained unresponsiveness is described in Table VI. If the patient has metabolic encephalopathy, the anaesthetist must rule out other potentially treatable conditions such as diabetic coma, hypoxia, or drug interactions before the TURP syndrome is diagnosed. The immediate tests to evaluate metabolic coma are listed in Table VII, and in the case of the TURP patient, may include serum glycine and ammonia concentrations.

The presence of an acute hypotonic state is life-threatening if the serum (Na) is less than $120 \text{ mmol} \cdot \text{L}^{-1}$ and the patient is severely symptomatic.²² The therapy of symptomatic hypotonicity is directed at increasing extracellular fluid tonicity in order to shift water from the intracellular to the extracellular fluid, thereby ameliorating cerebral oedema.^{1,22} This may be accomplished with the use of hypertonic saline in the patient with normal renal function^{1,6,20,33} or with the use of haemodialysis if the patient has chronic renal failure.³⁴ There is no specific treatment for hyperglycinaemia or hyperammonaemia but the concentrations of these substances have been reported to return to normal by the next day.^{25,27}

Additional drugs in the treatment of the TURP syndrome have been suggested. Sodium bicarbonate, 8.4 per cent, may be used if hypertonic saline is not available.³⁵

TABLE VI Differential diagnosis of sustained unresponsiveness

- 1 Supratentorial mass lesions compressing or displacing the diencephalon or brainstem
- 2 Subtentorial masses or destruction
- 3 Metabolic coma
- 4 Psychiatric unresponsiveness

Administration of calcium may be beneficial in the presence of an acute reduction in serum calcium concentration.³⁵

Hypertonic saline

Even though there is agreement that acute symptomatic hypotonicity should be treated, the details of therapy are controversial. Henderson recommended the administration of 100 ml, five per cent saline over one to two hours, repeating the measurement of serum sodium concentration and then giving another 100 ml if necessary.⁶ Beal suggested the use of 100 to 200 ml of three per cent hypertonic saline but the rate of administration was not reported.²⁰ Some authors recommend the simultaneous administration of furosemide and hypertonic saline to reduce fluid overload.^{1,20,33} Hypertonic saline is only infused until the serum (Na) has been increased to $120 \text{ mmol} \cdot \text{L}^{-1}$. Complete correction of the hypotonicity is then achieved gradually by fluid restriction over the next few days.²² An alternative approach is to calculate the sodium requirements based on total body water and then to administer this amount as hypertonic saline.²² However, there is such great variability in response that undercorrection or overcorrection may occur.³⁶

Concern has been expressed that the administration of hypertonic saline in the treatment of the TURP syndrome may cause hyperosmolality.³⁴ However, there is little evidence that hyperosmolality *per se* is harmful, and the major concern depends on whether its development has been accompanied by major fluid shifts.³⁷ If the patient is

TABLE VII Evaluation of metabolic coma

- A Venous Blood
 - 1 Glucose
 - 2 Electrolytes (Na^+ , K^+ , Cl^- , CO_2 , Ca^{++} , PO_4^{--})
 - 3 Urea or creatinine
 - 4 Osmolality
 - 5 Glycine
 - 6 Ammonia
- B Arterial blood
 - 1 HCO_3^-
 - 2 pH
 - 3 PO_2
 - 4 PCO_2
- C Electrocardiogram

symptomatic from hypotonicity, the appropriate therapy is to normalize the tonicity, even though osmolality would further increase.²²

Neurological complications

Neurological complications have been reported after very rapid correction of hyponatraemia, but there is still a lack of agreement regarding the safe rate of correction. If hyponatraemia is corrected too quickly, the rapid intracellular volume contraction could lead to the abrupt decrease of cerebral volume and, consequently, to intracranial haemorrhage.²² Another concern is that cerebral demyelinating lesions have been associated with excessively rapid correction of chronic hyponatraemia.³⁸ Gradual neurological deterioration has developed one to several days after complete or partial correction of chronic hyponatraemia, leading to pseudobulbar palsy and quadripareisis. However, the pathophysiology of chronic hyponatraemia is different from that of acute hyponatraemia. Demyelinating lesions have not yet been reported after correction of acute hyponatraemia in the TURP patient. A recent animal study showed that rapid correction of hyponatraemia, without overcorrection, can cause neurological disease.³⁶ The same study concluded that myelinolysis was greater in the group with protracted hyponatraemia than the group with hyponatraemia of short duration.³⁶

Prevention

The CNS complications after TURP are preventable with the efforts of both the surgeon and the anaesthetist. The resection time should be limited to one hour.² The hydrostatic pressure of the irrigating fluid should not exceed 6.9 kPa (70 cm H₂O).¹ The early detection of cerebral symptoms is more feasible if a spinal anaesthetic is administered than if general anaesthesia is used.¹ The anaesthetist should institute the same monitoring regime with the spinal anaesthetic as would be used with a general anaesthetic. Rapid measurement of serum (Na) should be available within the institution. Hypotonic intravenous fluids should not be administered. Hypotension from the spinal anaesthetic is better treated with vasopressors rather than with large boluses of intravenous crystalloid. Lastly, open prostatectomy should be considered a valid alternative to transurethral prostatectomy. The evidence indicates increased rates of reoperation and reduced five-year survival after transurethral prostatectomy.³⁹

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Self-Assessment Questions**A. SELECT THE SINGLE CORRECT ANSWER**

1 Which of the following is not a metabolite of glycine?

- A mannitol
- B ammonia
- C serine
- D pyruvate

2 The serum (Na) which is the borderline between mild and severe TURP reactions is:

- A $130 \text{ mmol} \cdot \text{L}^{-1}$
- B $120 \text{ mmol} \cdot \text{L}^{-1}$
- C $115 \text{ mmol} \cdot \text{L}^{-1}$
- D $110 \text{ mmol} \cdot \text{L}^{-1}$

3 When a patient develops the TURP syndrome, the osmolal gap is:

- A increased more than $10 \text{ mosm} \cdot \text{kg}^{-1}$
- B unchanged
- C decreased
- D increased less than $10 \text{ mosm} \cdot \text{kg}^{-1}$

4 Visual impairment after TURP is associated with glycine concentrations which are:

- A normal
- B decreased
- C increased ten times normal
- D increased thirty times normal

5 The ideal irrigating fluid has the following properties except:

- A nonelectrolytic
- B allows clear visibility
- C rapidly excreted
- D haemolytic

B. FOR EACH QUESTION, ONE OR MORE OF THE COMPLETIONS IS CORRECTLY ANSWERED AS FOLLOWS:

- A If only 1, 2 and 3 are correct
- B If only 1 and 3 are correct
- C If only 2 and 4 are correct
- D If all are correct

6 Visual disturbances after TURP are associated with:

- A normal fundoscopic examination
- B normal serum glycine concentrations
- C normal intraocular pressure
- D permanent alterations in vision

7 Recommendations for prevention of the TURP syndrome include:

- A spinal anaesthetic
- B hydrostatic pressure of irrigation fluid to be 6.9 kPa ($70 \text{ cm H}_2\text{O}$) or less
- C open prostatectomy
- D hypotonic intravenous fluid

8 Treatment of severe metabolic encephalopathy after TURP should be directed at:

- A decreasing plasma glycine concentrations
- B increasing extracellular fluid tonicity
- C decreasing plasma ammonia concentrations
- D ruling out potentially treatable conditions

9 Which of the following solutes contributes to plasma osmolality but not to tonicity:

- A urea
- B mannitol
- C glycine
- D sorbitol

ANSWERS:
1 A 2 B 3 A 4 D 5 B 6 B 7 C 8 C 9 B