

Bilateral blindness complicating the transurethral resection of the prostate (TURP) surgery

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Introduction and objective: The ophthalmological complications of the transurethral of the prostate (TURP) surgery is reviewed and analyzed. Bilateral blindness may be induced by the TURP syndrome without being implicated for it.

Patients and methods: Analytical Literature Review On The Eye Manifestation Is Reported. A reported case of permanent bilateral blindness is analyzed as an example of the illusive masks the TURP syndrome uses.

Results: Reported data indicate that VO was responsible for the dilution of serum solute contents and clinical picture of hyponatraemia (HN) of the TURP syndrome. Volumetric overload was the only highly significant factor in relation to the severity of the TURP syndrome. The syndrome presented with myocardial, cerebral, respiratory, renal and hepatic failures masks or ARDS. We provide evidence that it is also responsible for the

permanent bilateral blindness in the reported case. Hypertonic sodium therapy has saved the lives of 20 patients.

Conclusion: Volumetric overload shocks are the patho-etiology of TURP, HN and ARDS. This has uncovered most masks behind which the TURP syndrome hides. The eye manifestations including permanent bilateral blindness may occur without incriminating the TURP syndrome. Hypertonic sodium therapy is curable bringing patients back from dead and may restore his vision.

Key Words: *Hyponatraemia; The TURP syndrome; The multiple vital organ dysfunction/ failure syndrome; ARDS; Shock, Blindness; Capillary-interstitial transfer; Haemodynamic; Fluid therapy; Volumetric overload; hypertonic sodium therapy.*

Abbreviations: *VOS: Volumetric overload shocks; TURP: The transurethral resection of the prostate; ARDS: The adult respiratory distress syndrome; MVOD/F: The multiple vital organ dysfunction/failure; HN: Hyponatraemia; HST: Hypertonic sodium therapy; CVP: Central venous pressure; PBB: Permanent Bilateral Blindness; SSC: Serum sodium concentration*

INTRODUCTION

Lately, the TURP syndrome presented with “Permanent Bilateral Blindness” (PBB) without even being incriminated. A highly accessed case report of “nonarteritic ischemic optic neuropathy” complicating the TURP surgery was reported [1]. On reading the title, I immediately suspected the TURP syndrome and was sure as I finished the report. I was disappointed but not surprised as the TURP syndrome was not considered the diagnosis. The reason was clear from prepublication history particularly the impressive third reviewer’s comments that brought the TURP syndrome into authors’ attention for the first time during editorial consideration! This is indeed a devastating condition to endure after the TURP surgery that I find most provocative and diabolical.

The authors considered the TURP syndrome retrospectively on the 3rd reviewer’s suggestion [1] but excluded it after looking up a recent review on the complications of the TURP surgery [2]. In their discussion [1], the authors based their opinion of excluding the TURP syndrome on one paragraph covering the “definition and disappearance of the TURP syndrome from urology” [2].

In my view, the recently published review article is excellent and comprehensive but unfortunately fails to enlighten about the diagnosis. One might argue that a physician armed with that review cannot recognize the TURP syndrome when he encounters one! In my experience the clinicians involved across specialties cannot agree on the diagnosis of this syndrome when faced with it. To further make it difficult we cannot exclude it retrospectively based on current investigations which I will explain later.

In my opinion, it is the TURP syndrome that is the real culprit for “PBB” in this case. The fact that it may totally lack hyponatraemia (HN) that defines and characterizes it, took 25 years to investigate and unravel. The “nonarteritic ischaemia” is unlikely to be caused by local arterial

insufficiency, but was secondary to optic disc oedema, which is part of an obscure systemic pathological mechanism with which the TURP syndrome and HN insult every cell and vital organ in the body.

The Case Report in which the TURP syndrome was not incriminated for PBB is freely accessed [1]. Reading this report may help readers identify how and where the culprit is hiding, while examining the evidence exposing and incriminating it.

CASE SCENARIO

The following case scenario is fair representation of most cases and contains most data needed to answer questions. The scenario re-plays the events in slow motion utilizing cool methodical analysis rather than that encountered in the hectic murky clinical setting.

The extreme HN with acute serum sodium concentration (SSC) nadir of <100 mmol/l is invariably lethal during or immediately after surgery. The apparent cause of death is vascular collapse [3-7], cardiac or respiratory arrest behind which the TURP syndrome is offending but invisible. A nadir of <120 mmol/l is the HN marker that defines and characterizes the TURP syndrome, only if measured and detected at the immediate postoperatively period after the bolus of sodium-free fluid VO1 or irrigant absorbed causing HN enters the patient vascular system diluting all his serum contents.

However, HN is in fact dynamic and may be erased in few minutes or hours! An “acute dilution HN” nadir of 120 mmol/l may either be self-corrected in hours, by intracellular shift of water, excretion of water, or in minutes by the intravenous infusion of sodium-based fluids given by the treating team for hypotension shock, assuming the kidney remains functional, there will only be mild mental confusion next day.

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The same HN nadir of 120 mmol/l may be completely erased in minutes while the condition deteriorates despite the apparent correction of SSC. The acute dilution HN induced this clinical condition in the first place with sodium-free fluids VO1 bolus which is presenting as hypotension shock calling for further "volume expansion" using saline fluids! If blood sample was taken after resuscitating the patient with couple of bags of Hartmann's, Ringer's fluid, plasma and/or blood, HN may never be seen while the patient goes into coma.

He may progress into vegetative state by the time he is seen by a physician or neurologist! The TURP syndrome is again lurking behind, invisible, now wearing the mask of encephalopathy coma and/or cerebrovascular accident of relevance to the reported case: eye manifestations of the TURP syndrome are not new [3].

It has been documented that the TURP syndrome may cause sudden "Temporary Bilateral Blindness" preceded by diplopic blurred vision. Such eye manifestations have been attributed to glycine neuro-toxicity impeding nerve conduction. If you opt for "Toxic-Septic" hypothesis for the TURP syndrome, as the greatest researcher of the TURP syndrome of all times does, please remember before you incriminate the "glycine amino acid", that it is NOT mandatory for inducing the TURP syndrome at all as it occurs with glucose, mannitol and sorbitol. In fact if you think harder, one may find that neither glycine, nor TURP surgery, nor the syndrome name, nor definition and nor serum HN are mandatory either! Glycine may be just like alcohol an excellent marker for irrigating fluid absorption during TURP and may have similar toxicity at higher doses.

The visual symptoms are only reported when the patient is conscious during a procedure performed under regional anesthesia. For obvious reasons the eye symptoms can neither be reported by patients under general anesthesia nor when he goes into coma due to cerebral edema.

Eye examination at such time reveals the bilateral swollen optic discs. At this time visual loss is usually "temporary and recoverable", after couple days it is "permanent", but can blindness be detected if the patient is comatose? Can you guess any way you may prevent or treat this? Such therapy may have nothing to do with ophthalmologists at all!

It is most interesting that the eyes not only show optic disc "oedema ischaemia hypoxia", but also its "vitreous body fluid" shows another unique finding of the TURP syndrome: severe HN matching that of the blood. Such HN of "vitreous body fluid" was reported by a group of pathologists on PM examination of patients killed by the TURP syndrome [4]. For mentioned reasons the serum sodium concentration (SSC) was normal and the syndrome was not considered a clinical diagnosis.

The authors of this unique letter [4] suggested that estimation of sodium concentration of the "vitreous body fluid" at PM examination is diagnostic of the TURP syndrome even in the absence of serum HN. Even after the apparent correction of SSC level, vitreous body maintains HN for couple of days longer than serum HN nadir.

This remains true even when erasure or apparent correction of SSC is induced by sodium-based fluid infusions given with the good intension of "resuscitation" but in fact cause deterioration, internal drowning and death.

Thus, I would agree with the authors and congratulate them on their case being the first I read as "PBB" to complicate the TURP surgery and for which the TURP syndrome was not incriminated [1]. I accept their reported findings including predisposing factors of hypotension shock though the apparently low hemoglobin and hematocrit may indicate dilution rather than anemia of blood loss. The reported intra-ocular signs may urge them to reconsider the TURP syndrome as a culprit in the light of this discussion.

There are identical cases of blindness in which serum sodium may genuinely be normal, the authors referenced some, and we shall be seeing more in future.

It shares most of what is mentioned above particularly cell edema of HN manifesting as the multiple vital organ dysfunction/failure (MOVD/F) and internal drowning with interstitial tissue of mainly trunk edema seen so

commonly on ICU to complicate excessive sodium-based fluid infusions in cases of the acute respiratory distress syndrome (ARDS). Patients who improve must lose it before discharge and those who die go with it.

However, the cell edema differs on two issues: HN is completely out of the picture and with which goes the definition and name of the TURP syndrome.

The cell "oedema ischemia hypoxia" mechanism remains, but its explanation exists elsewhere at the capillary wall as a result of failure of the optimum dynamic capillary-interstitial fluid circulation [5]. Here normal tissue/cell irrigation that is vital for viability of cell is replaced by fluid drowning that kills viability of cells. This has been recognized as volumetric overload shocks [6,7].

DISCUSSION

Last but not least, I wish to affirm that both the TURP syndrome and acute dilution HN have a successful curative therapy of hypertonic sodium therapy (HST) of 5% NaCl or 8.4% NaCO₃ commonly available at resuscitation trolleys and equally effective. 29.2% NaCl is most effective but not recommended as it causes severe phlebitis and thrombosis. Such therapy guarantees complete recovery from shock, ARF and coma, when and if "accurately timely and promptly given" that prevents the patient deteriorating into the hopeless vegetative state physicians have to face as chronic HN when brain damage is irreversible. It recovers the state of bilateral blindness. Yet HST of 5%NaCl still worth a try as one can only be pleasantly surprised by seeing a Lazarus or two- the man who came back from the world of dead.

CONCLUSION

The therapy should be given promptly, rapidly without dithering or waiting for radiology to confirm the diagnosis. If the immediate postoperative biochemical and hematological evidence do not convince you nothing else will. I would recommend that after taking the blood sample for analysis to start therapy immediately without even waiting for the results.

Just make sure you do not infuse any other fluids or blood. Please also refrain from replacing urine output when the patient passes 3-5 liters in response to HST therapy as he recover from coma and ask for a drink-both you and your patient deserve one.

During the final year of my medical study, a Professor of Ophthalmology boasted in a lecture contrasting the surgeon and ophthalmologist. He said: "When a surgeon makes a serious blunder he may kill his patient and his error may be buried with the patient! However, if ophthalmologist makes an error, the patient is blinded but stays alive to haunt him until one of them dies".

I was not sure to be so faultless and hated being haunted thus ophthalmology was excluded early as career choice. When I choose urology as specialty later, however, I never thought our trade procedure, TURP surgery, might blind a patient.

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