Blue Vision (Cyanopsia) Associated With TURP Syndrome: A Case Report

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There have been many complications associated with transurethral resection of the prostate (TURP), known as TURP syndrome. Of the various irrigation fluids used for TURP, glycine irrigant has been historically popular given its relatively low cost. It is also a nonconductive solution and only slightly hypoosmolar, reducing the risk of burn injury or significant hemolysis. However, there have been many case reports of central nervous system toxicity such as transient blindness and encephalopathy related to glycine toxicity. Here, we report blue vision (cyanopsia), which has never been reported as a symptom of TURP syndrome. (A&A Practice. XXX;XXX:00–00.)

A rare but potentially life-threatening complication of transurethral resection of the prostate (TURP) is TURP syndrome. A large volume of solution is used for distention and irrigation during TURP. Historically, fluids utilized were hypoosmolar solutions such as distilled water, glucose, glycine, mannitol, and sorbitol. It is hypothesized that during prostate resection, exposure of the venous sinuses and damage to the prostatic capsule lead to absorption of large volumes of irrigant, resulting in pulmonary edema, congestive heart failure, hyponatremia, seizures, or coma. We report here what we believe is the first report of blue vision (cyanopsia) associated with glycine toxicity and severe hyponatremia after TURP.

Verbal and written consent was obtained from the patient for the publication of this case report.

CASE DESCRIPTION

A 76-year-old man with benign prostatic hypertrophy status post transurethral microwave therapy presented for TURP for increased urinary frequency and urgency. His other medical history included steroid-dependent asthma, adrenal insufficiency with chronic steroid use, diabetes mellitus, cervical myelopathy associated with ataxia, and lumbar degenerative disk disease.

On the day of surgery, before administration of spinal anesthesia, he was administered intravenous hydrocortisone 100 mg, midazolam 1.5 mg, and fentanyl 50 µg. He was positioned in the sitting position. Hyperbaric bupivacaine 11.25 mg and fentanyl 20 µg were injected into the subarachnoid space at the level of L3–4. A few minutes after placing the patient recumbent, anesthesia was documented to the T5 level. He was then positioned in the lithotomy position. Approximately 10 minutes before the procedure ended, I noted a slightly blurry vision. When the procedure finished, I was unable to visually recognize individuals talking to me and could barely see the individuals transporting me to the recovery room. My hearing was not impaired and I could readily converse with the staff. The voices appeared to be coming from a foggy light blue background, and individuals or objects such as ceiling lights could not be recognized except their outline as silhouettes. The visual appearance continued en route to the recovery room.

On arrival in the postanesthesia care unit (PACU), I had difficulty recognizing individuals and the room appeared like a blue fog. I had no generalized pain or headache. I was able to see the attending nurse, however, individuals, and objects at the end of the bed, as well as the overhead panel lights appeared silhouetted and partially hidden by a blue fog.

After about 30–50 minutes, I was able to see the intravenous bags, and after 1–2 hours, I was able to see the clock at the end of the room but not the precise time. Interestingly, the colors were also changing from a darker blue to a lighter blue and then later to a white-gray fog. As my vision improved, the silhouettes subsided.

A venous blood sample obtained in PACU revealed severe hyponatremia with a serum sodium value of 112 mEq/L. Hematocrit and other electrolytes were within the normal range. Preoperative serum sodium was 138 mEq/L. He was started on a 3% hypertonic saline infusion at 125 mL/h. Hourly serial venous blood samples were estimated blood loss was minimal and clear urine was noted at the end when a 3-way Foley catheter was inserted.

Over the last 10 minutes of the procedure, he began to complain of blurry vision. He did not have respiratory distress, hemodynamic instability, or nausea and vomiting. He recorded his observations in a letter summarized below:

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- On arrival in the postanesthesia care unit (PACU), I had difficulty recognizing individuals and the room appeared like a blue fog. I had no generalized pain or headache. I was able to see the attending nurse, however, individuals, and objects at the end of the bed, as well as the overhead panel lights appeared silhouetted and partially hidden by a blue fog.
- After about 30–50 minutes, I was able to see the intravenous bags, and after 1–2 hours, I was able to see the clock at the end of the room but not the precise time. Interestingly, the colors were also changing from a darker blue to a lighter blue and then later to a white-gray fog. As my vision improved, the silhouettes subsided.
- 3–4 hours after admission to the PACU, my vision slowly improved and I was able to read a nursing chart of size 16–18 font and a few words but not complete sentences of the newspaper. The beds and individuals on the other side of the recovery room were now recognizable as if they slowly emerged from a fog. Seven hours after arrival to the PACU, I was able to read a few sentences of the newspaper.

A venous blood sample obtained in PACU revealed severe hyponatremia with a serum sodium value of 112 mEq/L. Hematocrit and other electrolytes were within the normal range. Preoperative serum sodium was 138 mEq/L. He was started on a 3% hypertonic saline infusion at 125 mL/h. Hourly serial venous blood samples were
subsequently 120, 123, 125, and 128 mEq/L. The hypertonic saline infusion was discontinued after 4 hours with a total of 545 mL being administered.

His symptoms slowly improved during the administration of hypertonic saline. Serum sodium values were stable at 140 mEq/L. He had complete resolution of all visual disturbances. He was discharged on postoperative day 2 and ophthalmological examination revealed no retinal abnormalities.

**DISCUSSION**

Glycine 1.5% solution is commonly used for TURP because it is inexpensive, nonconductive, and only slightly hypoosmolar, reducing the risk of burn injury or significant hemolysis. Glycine is a nonessential amino acid and it readily crosses the blood-brain barrier. It is an inhibitory neurotransmitter in the central nervous system and retina. Toxicity may lead to visual aberrations such as transient blindness. Hyperammonemia may also result as a byproduct of glycine metabolism. Some authors argue that glycine should be abandoned completely given its toxicity, and alternative nonconductive irrigation should be used for TURP.2

Interestingly, the visual disturbance experienced by our patient describes a phenomenon known as cyanopsia, a medical term for seeing everything tinted with blue. There have been case reports of patients experiencing cyanopsia after cataract removal and intraocular lens implantation or with the use of phosphodiesterase 5 (PDE-5) inhibitors. However, cyanopsia associated with TURP syndrome has not been reported.

PDE-5 inhibitors are used for the treatment of erectile dysfunction and pulmonary hypertension. Most PDE-5 inhibitors also have inhibiting effects on PDE-6, an enzyme actively present in retinal photoreceptors.3 The result is an increase in cyclic guanosine monophosphate concentration, which causes depolarization of the rod cells.4 Rod cells are most sensitive to light of wavelengths near 498 nm, which appears bluish-green.4 Vision under dim lighting conditions, known as mesopic vision, enhances both rod and cone cells, which may induce the bluish hue experienced by some patients taking PDE-5 inhibitors.5

High glycine levels are suspected of causing abnormal electroretinograms (ERG) and visual evoked potentials (VEP).6 Glycine has been shown to inhibit ganglion cells in the retina, as well as suppress the waveforms of ERG and VEP in rabbits and dogs. One study that looked at preoperative and postoperative ERG and VEP in patients undergoing TURP with glycine irrigant found that, among the patients who complained of visual impairment, serum glycine levels >4000 µmol/L were associated with complete dropout of both oscillatory potentials in the ERG and 30-Hz flicker after both ERG and VEP recordings from the occipital scalp.4 There was also a significant correlation with elevated serum glycine levels and severe hyponatremia. Other findings showed that the ocular fundus appeared normal and pupillary response to light remained unchanged. Vision also returned to normal within 2–12 hours after the procedure.

It has been suggested that severe hyponatremia may lead to cerebral edema of the occipital cortex, leading to visual aberrations.7 In the previous study, no patients had low serum osmolality despite hyponatremia. Ammonia levels did not significantly correlate with serum glycine or sodium levels, making hyperammonemia also less likely the cause for visual disturbances. The visual disturbances experienced by our patient do not fit the description of cortical blindness, either. Visual sensation including perception of light, corneal reflex, and lid-closure reflex would be absent in cortical blindness if edema of the occipital cortex was the cause.7 Our patient had blurry vision and cyanopsia but appeared to have intact reflexes.

A study looking at a dose–response relationship between glycine administration and visual disturbances in humans found marked variability in the development of visual symptoms. In the most pronounced case, deterioration of visual acuity occurred after 4.4 g of glycine administration, equivalent to only 300 mL of glycine 1.5% solution.8 The visual disturbances noted in this study were always transient and during the early stages of glycine administration. The tendency for visual symptoms to resolve spontaneously despite further administration of glycine suggested that absorption of glycine must be rapid.8

It is challenging to explain how TURP syndrome with glycine irrigant is associated with cyanopsia. Animal and human research has shown that glycine receptors are found on small-field amacrine cells, bipolar cell axons, and ganglion cell dendrites in the retina.9 What effects high glycine levels or low sodium levels, or a combination of both may have on photoreceptors is not discernible. The Purkinje effect is the tendency for the peak luminance sensitivity of the human eye to shift toward the blue end of the color spectrum during mesopic vision.10 As intensity dims, color-sensitive cones are taken over by light-sensitive rods. Before color disappears completely, the rod cells become most sensitive to bluish-green light.10 Glycine’s inhibitory effects along with the environmental factor of lighting may disrupt the balance of light absorption between the 2 cells, causing this phenomenon.

To our knowledge, cyanopsia has not been reported as a symptom of TURP syndrome. We speculate that this was due to a unique characteristic of this patient such as a genetic anomaly involving his retinal photoreceptors. ■

**DISCLOSURES**

**Name:** William C. Fox, MD.

**Contribution:** This author helped collect the data, review the literature, and prepare the manuscript.

**Name:** Richard E. Moon, MD, CM, MSc, FRCP(C), FACP, FCCP.

**Contribution:** This author helped review the data and literature, and edit the manuscript.

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**REFERENCES**


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