Case Report

Transient Vision Loss in Transurethral Resection of Prostate Syndrome

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Abstract

We had an unusual experience of patient developing transient loss of vision following transurethral resection of prostate. Various pathophysiological changes contributing the vision loss during transurethral resection of the prostate has been discussed. The patient recovered his vision fully after therapeutic interventions. This paper discusses the possible etiologies for the transient vision loss and treatment of same.

Key words: Glycine, hyperammonemia, hyperglycinemia, hyponatremia, transurethral resection of the prostate syndrome, visual disturbances

Introduction

Transurethral resection of the prostate syndrome is a constellation of symptoms and signs caused mainly by absorption of irrigating fluid in patients undergoing endoscopic surgery of prostate gland. This syndrome includes cardiovascular, neurological, gastrointestinal manifestations such as disorientation, restlessness, confusion, agitation, drowsiness, convulsions, vision loss, nausea, vomiting, hemolysis, hypothermia, dyspnoea, pulmonary edema, and cardiac arrest.[1,2] The primary concern associated is the intravascular absorption of the large volume of nonelectrolyte irrigating fluid through the exposed venous sinuses of surgical capsule.[2,3] The metabolic disturbances such as dilutional hyponatremia, hyperglycinemia, hyperammonemia, and water toxicity play a role in the pathophysiological sequence of events.[4]

Case Report

A 61-year-old man with the diagnosis of benign prostate hypertrophy was planned for transurethral resection of prostate under spinal anesthesia with no significant history suggesting cardiovascular and neurological illness and was diagnosed as having hypertension for 2 weeks and put on treatment tablet amlodipine 2.5 mg. General physical, systemic examination and airway assessment could not reveal any abnormalities. Vital parameters were found to be under normal limits. Since the patient was hypertensive preoperative assessment revealed no changes in electrocardiogram (ECG) and echocardiography. Investigations were done to know any end organ damages such as fundoscopy, showing no retinopathy changes, and renal parameters with normal values. Preoperative hemoglobin was 12.7 g% and serum electrolytes were sodium = 141 mEq, potassium = 3.8 mEq, and chloride = 101 mEq. With this preanesthetic evaluation, the patient was accepted under American Society of Anesthesiology II in view of hypertension. Spinal anesthesia is given with 0.5% hyperbaric bupivacaine 2.8 ml and injection fentanyl 0.4 ml and analgesia covering up to dermatomal level T10. Transurethral resection of the prostate (TURP) started using 1.5% glycine as irrigating fluid with the plan to remove 90 cc of prostatic tissue in 60 min. After 1 h of starting the procedure, the patient desaturated to 80–88%. The patient was oxygenated with bains circuit with 8 l/min of 100% of oxygen with the improvement to 90–92%, oxygen increased to 10 L/min, and saturation picked up to 96–98%. Intraoperatively, 2–3 venous sinuses were opened as judged by surgeon and haemorrhage noted, to treat that

Access this article online

Quick Response Code:

Website: www.karnatakaanaesthj.org

DOI: 10.4103/2394-6954.190773

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How to cite this article: Kempachary, Raghavendra TR, Vinutha DK, Vasanth CG, Prasannan K. Transient vision loss in transurethral resection of prostate syndrome. Karnataka Anaesth J 2016;2:28-30.

injection tranexamic acid 500 mg was put in infusion of 500 ml of dextrose normal saline. The patient was conscious, comfortable, and the procedure continued. Auscultation of respiratory system showed basal crepitations with continuation of cough intermittently. Injection furosemide 20 mg was given and procedure continued. Overall, after 90 min from starting the surgery, the patient started feeling discomfort, fatigability, and feeling of suffocation. To our surprise, patient started complaining of blurring of vision not associated with pain. Immediately, surgeon was asked to stop the procedure. On table examination showed patient to be conscious, pulse - 82/min, blood pressure - 130/90 mmHg, Spo2-98%, crepitations all over the lung field with occasional rhonchi, and pupils were reacting bilaterally. After sometime, the patient complained of complete loss of vision including the perception of light.

Procedure completely terminated following immediately injection frusemide 20 mg repeated and O2 administration at 10 L/min. On table blood sample was sent to a laboratory for hemoglobin, serum electrolytes, and blood ammonia immediately. Serum glycine level was not done as facilities were unavailable. Nebulization done with duolin respule 2.5 mg and *in vitro* fertilization, normal saline continued ophthalmological opinion sought which revealed normal fundus and preservation of all reflexes.

Thirty minutes after resuscitative measures perception of light appeared. On auscultation chest was clear with occasional rhonchi and absence of cough. The patient was shifted to surgical Intensive Care Unit for further management avoiding injection diclofenac sodium. Laboratory investigations showed hemoglobin = 10.2 g%, sodium = 110, potassium = 4.2, and chloride = 78. Blood ammonia levels were found to be 30 mmol/L. Then, the patient was advised to continue ringer lactate and dextrose normal saline for maintenance and injection sodium chloride 3% at rate of 25 ml/h slowly.

After 1 h, he started developing a vision, which was blurred, then after 2 h complete reversal of loss of vision appeared. The total duration of surgery was about 1 h 45 min.

Discussion

For the etiology of the visual loss, various theories are proposed, but it has been attributed to the rapid absorption of a large amount of irrigating fluid into the systemic circulation causing metabolic disturbances requiring intensive care. Pathophysiological mechanism consists of pharmacological effects of irrigant solutes, the volume effect of irrigant water, dilutional hyponatremia, pulmonary, and cerebral edema.[5] Currently, the most commonly used irrigant solution is glycine 1.5%.

Fluid absorption occurs rapidly through the open venous sinuses during prostatectomy resulting in hypervolemia and hyponatremia. A drop of 20–30 mEq/L or a value of 120 mEq/L of serum sodium indicates severe reaction. Symptoms include visual disturbances such as blurred vision, pupillary dilatation, and transient cerebral edema. Serum sodium level alterations affect central nervous system functions causing cerebral edema involving occipital cortex resulting in vision loss.[2] Hypervolemia is followed by hyponatremia, hypocalcemia,[6] low serum osmolality, and hypothermia.[7] Cerebral edema and changes in osmolality due to water intoxication will disturb visual pathway[1,2] and cause transient cortical blindness, In addition to that perception of light, blink reflex, rapid eyelid closure will be lost, This could be ruled out as the patient had all intact visual reflexes, accommodation reflexes, and normal fundus.

Hyperammonemia is known to cause central nervous system depression by hyperammonemic encephalopathy.[8] Ammonia as a product eliminated from glycine metabolism by oxidative deamination in liver. Blood ammonia concentration >100 mmol/L are associated with neurological symptoms. Studies shows poor correlation between hyperammonemia and TURP syndrome.[9] Our patient had blood ammonia levels 30 mmol/L which falls in the normal range (10–35 mmol/L).

Hyperglycinemia has been attributed as a cause of visual disturbances.[9-11] Glycine is nonessential aminoacid commonly used (1.5%). The plasma concentration in human is 0.3 mmol/L where 5–8 mmol/L noted in visual disturbances and still higher levels produces transient blindness. Glycine is an inhibitory neurotransmitter in the retina and the central nervous system[12] acting via N-methyl-D-aspartate receptor activity. In glycine toxicity, visual symptoms range from blurred vision to complete blindness. However, intraocular pressure, fundoscopy, pupillary reflexes, brain computed tomography scan will usually be normal with the exception of pupils which may or may not be dilated. Transient prickling sensation,[13] burning sensation in face and neck, facial warmth, becoming restless, the presence of headache, and chest pain are reported sometimes. Bradycardia and arterial hypotension, shortness of breath, uneasiness, and pulmonary edema are other features to occur. Hypoxia has been reported and also the ECG showing bradycardia, prolongation of PQ time, widening of QRS complexes.[14] In our patient serum, glycine could not be done as facilities were unavailable.

Conclusion

With unknown levels of glycine, ammonia levels being under normal limits and the presence of hyponatremia, hypoosmolality contributed to the visual disturbances. The clinical signs and symptoms were in more favour of hyperglycinemia and would be the probable cause of transient blindness in the patient. As soon as TURP symptoms started, immediate intervention should be done meanwhile surgeon needs to be intimated about fluid absorption whether it exceeds 1 h or 1 L.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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