

CLINICAL INFORMATION

Perioperative stroke following transurethral resection of prostate: high index of suspicion and stabilization of physiological parameters can save lives

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Abstract We report a case of a 72 year old hypertensive male who developed severe hypertension followed by neurological deterioration in the immediate postoperative period after transurethral resection of prostate. While arterial blood gas and laboratory tests excluded transurethral resection of prostate syndrome or any other metabolic cause, reduction of blood pressure failed to ameliorate the symptoms. A cranial CT done 4 hours after the onset of neurological symptoms revealed bilateral gangliocapsular and right thalamic infarcts. Oral aspirin was advised to prevent early recurrent stroke. Supportive treatment and mechanical ventilation ensured physiological stability and the patient recovered completely over the next few days without any residual neurological deficit.

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PALAVRAS-CHAVE

Ressecção
transuretral de
próstata;
Perioperatório;
Acidente vascular
cerebral;
Hipertensão;

**Acidente vascular cerebral no perioperatório após ressecção transuretral de próstata:
alto índice de suspeita e estabilização de parâmetros fisiológicos podem salvar vidas**

Resumo Relatamos o caso de um paciente hipertenso, 72 anos, que desenvolveu hipertensão grave seguida de deterioração neurológica no pós-operatório imediato após ressecção transuretral de próstata. Embora os testes laboratoriais e a gasometria tenham excluído a síndrome de ressecção transuretral de próstata ou qualquer outra causa metabólica, a diminuição da pressão sanguínea não conseguiu melhorar os sintomas. Uma tomografia computadorizada craniiana, realizada 4 horas após o aparecimento de sintomas neurológicos, revelou infartos gangliocapsular bilateral e talâmico à direita. AAS oral foi aconselhado para prevenir

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Manifestações
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Inconsciência

um acidente vascular cerebral recorrente precoce. O tratamento de apoio e a ventilação mecânica garantiram a estabilidade fisiológica e o paciente obteve recuperação completa durante os próximos dias, sem qualquer déficit neurológico residual.
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Introduction

Perioperative stroke has been reported to have an incidence of 0.1% in 523,059 patients undergoing non-cardiac, non-neurological and non-vascular surgical procedures.¹ None of these cases have been reported following urology surgeries. Although TURP is most commonly performed urological procedures, to the best of our knowledge, postoperative stroke has not been reported after TURP. No incidence of stroke was reported in any patient in the perioperative period in 7707 patients across 3 German centres between 1989 and 2005.² Although Raj et al. reported two cases of Transient Ischaemic Attack (TIA) or stroke amongst 305 patients undergoing TURP, this study consisted of 111 patients who had medical indication like history of stroke or TIA and coexisting atrial fibrillation, for receiving anticoagulants.³ However, the authors had not specified whether the two cases were specifically TIA or stroke.

Case report

A 72 year male weighing 62 kg with history of hypertension for the past 12 years was scheduled for a Transurethral Resection of Prostate (TURP) for his symptoms of bladder outlet obstruction. His hypertension was controlled on oral amlodipine 5 mg once daily and all preoperative routine investigations were unremarkable except for the Electrocardiograph (ECG) which showed a left bundle branch block pattern. Transthoracic echocardiograph revealed left ventricular hypertrophy with an ejection fraction of 58%. He had no history of any central nervous system symptoms and was not on any antiplatelet medication.

The patient received alprazolam 0.5 mg orally on the night before surgery and also on the morning 2 h prior to surgery. Amlodipine was continued on the morning of surgery. In the operation theatre his initial baseline readings were: pulse rate of 74 min, blood pressure of 179/92 mmHg and room air saturation (SpO_2) of 98%. 10 min after 1 mg of intravenous midazolam his vitals remained stable with a pulse rate of 74 min, blood pressure of 160/86 mmHg and room air saturation (SpO_2) of 97%. TURP was performed under subarachnoid block with 2 mL (10 mg) 0.5% bupivacaine heavy with 25 mcg of fentanyl. The total resection time was 50 min and 17L of glycine was used. During the surgery the heart rate remained between 69 and 83 beats per minute and the blood pressure remained between 142/77 mmHg and 177/83 mmHg.

The patient was subsequently monitored in the post-anesthesia recovery area for 60 min and then shifted to the High Dependency Unit (HDU). The patient remained

comfortable and hemodynamically stable for the initial 150 min with return of sensory-motor power of the lower limbs. Then his blood pressure rose from 157/77 mmHg (pulse rate of 67 min) to 210/94 mmHg (pulse rate of 62 min) over 1 h. He remained fully conscious and alert, was comfortable and did not complain of any pain, distress or headache. He was immediately given 5 mg of amlodipine orally and was observed over the next few hours. Two hours later he slowly started getting confused and disoriented with gradually waning consciousness. His blood pressure was 220/110 mmHg (pulse rate of 68 min), Glasgow Coma Scale Score was 8/15 (E1, V2, M5) and he was neglecting his left side. The patient was sedated with intravenous midazolam 2 mg, intubated and mechanically ventilated. An infusion of nitroglycerin was started at $2 \mu\text{g}.\text{kg}^{-1}.\text{min}^{-1}$ and titrated with an aim to reduce the mean arterial pressure by 25% in the next hour and maintain it around 160/100–110 subsequently.

An Arterial Blood Gas (ABG) sampling was done and all routine investigations, including serum electrolytes were sent to the laboratory. A cranial Computed Tomography (CT) was advised.

A provisional diagnosis of TURP Syndrome was made with the differential diagnosis as hypertensive encephalopathy or stroke. At this point he ABG on room air revealed a pH of 7.410, pCO_2 of 39.6 mmHg, pO_2 of 124.7 mmHg, Hb of 13.4 g.dL⁻¹, Na^+ 140 mmol.L⁻¹, K^+ 5 mmol.L⁻¹, and Cl^- 104 mmol.L⁻¹, Lactate of 1.4 mmol.L⁻¹, HCO_3^- of 22 mmol.L⁻¹ and a base excess of 1.4 mmol.L⁻¹. The laboratory results showed a haemoglobin level of 13.1 g.dL⁻¹, K^+ of 3.7 mmol.L⁻¹, Na^+ of 149 mmol.L⁻¹, a Total Leucocyte Count (TLC) 7900/cumm (N66, L31, E1, M2) and a random blood sugar of 100 mg.dL⁻¹. A normal pH, sodium level, bicarbonate level and anion gap (14 mmol.L⁻¹) ruled out TURP syndrome as the cause of the deterioration of consciousness levels. No other metabolic cause could be appreciated based on the investigations done. A normal lactate level and total leucocyte count also excluded sepsis as the possible cause of neurological symptoms. A cranial CT done 4 h after the onset of neurological symptoms revealed acute bilateral gangliocapsular and right thalamic infarcts. Rest of the brain parenchyma showed only age related atrophic changes.

Appreciating stroke as the cause of neurological deterioration, nitroglycerin infusion was slowly tapered and stopped. The blood pressure during the next 24 h ranged from 167/84 mmHg to 116/78 mmHg and the heart rate ranged from 98 to 73 beats/min. The patient was given 325 mg of aspirin by a nasogastric tube and intravenous pantoprazole 40 mg was given for gastric ulcer prophylaxis. Over the next 2 days his GCS gradually improved with supportive care and mechanical ventilation. His hypertension was

gradually controlled on amlodipine 5 mg and losartan 25 mg once daily by nasogastric tube. He continued to receive 150 mg of aspirin daily. He was weaned from mechanical ventilation and tracheal extubation was achieved on the third postoperative day. He remained drowsy but arousable on stimulation with a Richmond Agitation Sedation Scale (RASS) score of -1 for the next 2 days. However there was no residual sensory-motor deficit.

His awareness gradually improved over the next 2 days and he was shifted to the ward on the 5th postoperative day. He was finally discharged on the 7th postoperative day on oral amlodipine 5 mg, losartan 25 mg and aspirin 75 mg once daily. A subsequent colour-Doppler sonography and CT-angiography did not reveal any stenosis or plaque. He has been subsequently followed up at our hospital and has had an uneventful course over the next 3 months.

Discussion

The risk factors for perioperative stroke include advanced age, hypertension, myocardial infarction within the last 6 months and previous history of renal disease, stroke or TIA. Continuation of the beta-blockers and statins can have a protective effect. Recent evidence also shows that continuation of aspirin in non-cardiac surgery has not been associated with any increased risk of bleeding.¹ Our patient had only two of the known risk factors, advanced age and hypertension. The modifiable risk factor (hypertension) in our patient was managed by preoperative control of blood pressure with oral amlodipine.

Our patient initially presented with a blood pressure of 179/92 mmHg in the operating room. Although preoperative hypertension is the commonest medical cause of postponement of surgery,⁴ various studies have shown that hypertension <180/110 mmHg is not an independent risk factor for perioperative cardiovascular complications.⁵ Mere anxiolysis with 1 mg of intravenous midazolam brought the blood pressure down to 160/86 mmHg within 10 min and we proceeded with the surgery. Our aim was to avoid decrease in the blood pressure to <50% of the preoperative levels and avoid any 10 min period where the blood pressure was >33% for 10 min. Both have been shown to be independent risk factors for adverse perioperative events.⁶ The perioperative aim in hypertensive patients is to maintain an arterial pressure at "70–100% of baseline" and avoid tachycardia.⁷

Neurological symptoms in the immediate postoperative period with hypertension led us to initially suspect fluid overload and TURP syndrome. TURP syndrome manifests within 15 min of starting of resection to 24 h postoperatively with cardiovascular, haematological and renal symptoms along with central nervous symptoms.⁸ Cardiovascular manifestations can consist of hypertension, bradycardia, dysrhythmias, cyanosis and shock.⁸ Haematological and renal symptoms consist of hyponatraemia, hypoosmolality, acidosis or alkalosis, haemolysis, anaemia and acute renal failure.⁸ Central nervous system features can manifest as nausea, vomiting, confusion, restlessness, seizures, paralysis or coma.⁸ However a normal ABG with normal sodium level excluded TURP syndrome as the cause of neurological symptoms. The calculated

osmolality $2 \times (\text{Na} + \text{K}) + (\text{glucose}/18)$ was also normal at 295.6 mOsm.kg⁻¹.

Neurological manifestation is a major component of sepsis and "mental dysfunction may even precede the cardinal findings of sepsis".⁹ However a normal lactate level in ABG, normal total leucocyte count and a higher blood pressure excluded sepsis as the possible cause of neurological symptoms. Hypertensive encephalopathy may manifest with headache, alteration in vision, nausea and vomiting and altered level of consciousness. Reduction of blood pressure by a nitroglycerin infusion failed to reduce the symptoms. An ophthalmoscope examination could have aided the diagnosis, but was not done as the cranial CT took priority over other investigations.

Rapid neurologist deterioration necessitated initial stabilization and cranial CT could only be done 4 h after the onset of neurological symptoms. Current recommendations suggest non-contrast Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) of the brain to determine ischaemic or haemorrhagic nature of stroke immediately on the suspicion.¹ Despite diagnosing ischaemic stroke recombinant Tissue Plasminogen Activator (rtPA) could not be considered in view of major urological surgery few hours ago.¹ The relative exclusion criteria for recombinant tissue plasminogen activator include "recent gastrointestinal or urinary tract haemorrhage (within previous 21 days)" and "major surgery or serious trauma within previous 14 days".¹⁰ However, the final decision on intravenous rtPA in such cases would be based on the "risk/benefit" ratio and "severity and location of the stroke".¹ Although not a substitute to intravenous rtPA, our patient received the 325 mg of aspirin as the safer option according to the current guidelines and evidence (Class I; Level of Evidence A)¹ in an effort to prevent early recurrent stroke.¹⁰

As the patient was asymptomatic, we initially tried to treat hypertension in the post operative period by oral amlodipine. However on failure to achieve adequate control, an infusion of nitroglycerin was started. The suggested goal of treatment is to reduce the mean arterial pressure by "no more than 25 percent"¹¹ within the 1st hour, and subsequently to "160/100–110 mmHg within the next 2–6 h".¹¹ This guideline was followed in managing our patient also. Currently there is no evidence to suggest that for a patient who presents with severe hypertension, failure to aggressively reduce blood pressure "is associated with any increased short-term risk".¹¹ The exception to the above recommendation applies to patients with ischaemic stroke who do not receive fibrinolysis. In such patients current recommendations suggest that anti-hypertensive therapy should be withheld "unless the systolic blood pressure is >220 mmHg or the diastolic blood pressure is >120 mmHg".¹⁰ As per these guidelines, nitroglycerin infusion was tapered and stopped after the cranial CT revealed an ischaemic stroke to be the cause of neurological deterioration. As observed in our patient, the blood pressure often spontaneously declines "during the first 24 h after onset of stroke".¹

The American Society of Anesthesiologists has defined "perioperative stroke" as "brain infarction of ischaemic or haemorrhagic aetiology that occurs during surgery or within 30 days after surgery".¹ It can have "catastrophic outcome" in non-cardiac, non-neurosurgical patients with

"adjusted 8 fold increase in mortality".¹ As compared to an overall mortality of 12.6% in patients with stroke, perioperative stroke after general surgery has been reported to be as high as 26%, possibly due to delay in diagnosis and a "synergistic interaction between the inflammatory changes" after stroke and normally occurring after surgery.¹² While early mortality in perioperative stroke results from delayed diagnosis, intracranial hypertension and cerebral oedema, delayed mortality maybe caused by "aspiration, pneumonia, metabolic derangement, sepsis, or myocardial infarction".¹² The "need for intubation has prognostic implications" with "up to 50% mortality within 30 days after stroke".¹⁰ Our patient was fortunate enough to overcome the crisis and have an uneventful recovery.

Learning points

High index of suspicion and early radiological investigation can diagnose and save lives in patients developing perioperative stroke.

Perioperative stroke can mimic as TURP syndrome after urological procedures.

Maintenance of physiologic stability is imperative in care of patients with stroke.

Although 50% mortality is seen patients with stroke needing intubation, it maybe required to protect the airway along with need-based ventilatory support.

Thrombolytic therapy with recombinant Tissue Plasminogen Activator (rtPA) is not an absolute contraindication in the perioperative period and can be offered to patients on a case to case basis.

Aspirin should be considered in all indicated cases of perioperative stroke.

In patients with stroke, anti-hypertensive therapy should be withheld unless the systolic blood pressure is >220 mmHg or the diastolic blood pressure is >120 mmHg.

Conflicts of interest

The authors declare no conflicts of interest.

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