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CASE REPORT

Oral ephedrine is useful to wean patients off long term parenteral vasopressors after cervical spinal cord injury

Muhammad Faisal Khan, Khalid Maudood Siddiqui, Muhammad Ali Asghar, Hameed Ullah

ABSTRACT

Traumatic spinal cord injury (SCI) in young adults not only increases the risk of mortality but more commonly it complicates with life-long disability. Cervical SCI patients are particularly susceptible and sensitive to phases of cardiovascular instability and respiratory failure directly consequential from their injuries. Furthermore, long term vasopressor requirement is not uncommon though weaning from parenteral vasopressor is a challenge.

We document a case of the use of oral ephedrine, which we used to wean our patient from parenteral vasopressors. Oral ephedrine can be an appropriate option to get rid of long term use of infusion of vasopressor.

Key words: Cervical spinal cord injury (SCI); Vasopressor; Oral ephedrine; Critical care

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Department of Anesthesiology, Aga Khan University Hospital, Stadium Road, P.O. Box 3500, Karachi 74800, (Pakistan)

Correspondence: Dr Khalid Maudood Siddiqui, Aga Khan University Hospital, Stadium Road, P.O. Box 3500, Karachi 74800, (Pakistan); Mobile: 92 3332117475; E-mail: khalid.siddiqui@aku.edu

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INTRODUCTION

Spinal cord injury is a devastating event that may affect every aspect of an individual's life.[1] Cervical spinal cord transection causes major derangement of the sympathetic control of blood pressure, heart rate and body temperature.[2] Patients with a cervical cord injury may develop neurogenic shock, characterized by bradycardia and hypotension in early period. These changes are related to increased vagal tone, decreased sympathetic input, and possibly changes in the heart itself.[4] Pharmacologic and electrical interventions (i.e, pacemakers) may be necessary in these patients if fluid resuscitation alone cannot maintain adequate tissue perfusion. Failure to adequately treat neurogenic shock may result in further ischemic injury of an already compromised nervous system (secondary injury).[4] Aggressive treatment with fluids and vasopressors, and appropriate invasive monitoring, including arterial and central venous access, is paramount.[5]

We report a case of a patient with cervical cord injury complicated with severe vascular dysfunction and vasopressor dependency demanding prolonged dopamine infusion. Oral ephedrine was started and after few days the patient was successfully weaned off from vasopressor infusion.

CASE REPORT

A 41 year old male was admitted in ER after a gunshot injury at the back of neck. On arrival, he was awake with GCS of 15/15, maintaining saturation at 97% on room air but unable to move his lower limb with power of 0/5, while upper limb power was 3/5 with brisk reflexes. Blood pressure and heart rate were normal. On auscultation, there was decreased air entry on left side.

On secondary survey, there was an entry wound on the back of neck but no exit wound identified. CT

scan demonstrated comminuted fracture of C6, C7 and T1 with multiple bony chips in spinal canal at cervicothoracic junction with fracture of body of sternum along with left upper lobe contusion. Chest tube was inserted owing to left hemopneumothorax. In the emergency room, all of a sudden, patient developed signs of hemodynamic instability. He was resuscitated with crystalloid fluid bolus and infusion of approximately 6 L over 4 h, but later required dopamine infusion in the dose of 10-15 μg/kg/min. Subsequently, patient had to be intubated due to worsening respiratory failure and persistent hemodynamic instability. ABG's revealed pH 7.24, PCO_2 45, PO_2 55, HCO_3 16 on 10 L of oxygen through face mask. Patient was shifted to intensive care unit (ICU) for further management.

In the ICU, controlled ventilation was started with 0.5 FiO_2. Invasive arterial line was inserted. On day 1, despite adequate fluid resuscitation and dopamine infusion his mean arterial pressure (MAP) remained below 60 mmHg, so norepinephrine was added. Initially he was managed for spinal shock. During the ICU course he constantly required intravenous vasopressors due to blood pressure swings, loss of sinus arrhythmia, and decreased sweating. After excluding other causes of hypotension, diagnosis of autonomic dysfunction was made on clinical grounds. On 7th ICU day, he was off norepinephrine but dopamine infusion had to be continued. Tracheostomy was done to remove the endotracheal tube. Patient ICU stay was prolonged due to continued support of dopamine, so on 13th ICU day, oral ephedrine 30 mg every 6 h was started to wean off dopamine, after excluding other causes of hypotension (Table 1). The dose of ephedrine was increased up to 60 mg every 6 h on the next day with simultaneous tapering off of dopamine. On 16th ICU day, the oral ephedrine had totally replaced dopamine infusion. Oral ephedrine 60 mg Q 6 hourly was continued for the next two weeks. Meanwhile, he was given trial of track mask ventilation which was successful. He was observed for hemodynamic stability with no further hypotension so he was weaned off ventilator. On 19th ICU day, he was transferred in a stable normotensive condition to an in-patient rehabilitation unit. He remained in the rehab unit for next two weeks and then got discharged from hospital.

DISCUSSION

Cardiovascular complications in the acute stage following traumatic spinal cord injury (SCI) require prompt medical attention to avoid neurological compromise, morbidity, and death.[6] Hypotension (both supine and orthostatic), autonomic dysreflexia, and cardiac arrhythmias (including persistent bradycardia) are attributed to the loss of supraspinal control of the sympathetic nervous system that commonly occurs in patients with severe spinal cord lesions at T-6 or higher.[6] Providing adequate fluid resuscitation is paramount in patients presenting with acute spinal cord injury.[7] Vasopressors and inotropes may be indicated in the presence of decreased systemic vascular resistance, despite adequate volume expansion. In patients with acute spinal cord injury, the vasopressor of choice depends on the patient's hemodynamic profile, but often it is the one that has both α- and β-adrenergic activity.[7]

In our patient dopamine was uninterruptedly required to maintain patient's blood pressure that led to his prolonged ICU stay. Oral ephedrine was successfully added to taper off dopamine. Ephedrine, a nonselective α- and β-agonist, which stimulates the

Table 1: Blood Trend and Drug Administered

Day	Mean Blood Pressure (mmHg)	Heart Rate Mean	Drug
1	92/57	50	Nor* /Dop**
3	90/69	68	Nor* /Dop**/Flu***
5	112/89	60	Dop**/Flu***
7	100/54	55	Dop**/Flu***
9	99/55	61	Dop**/Flu***
13	100/64	55	Dop**/Flu***/Eph****
15	110/65	61	Dop**/Flu***/Eph****
16	120/74	68	Flu*** /Eph****
17	115/64	68	Flu***/Eph****

* Norepinephrine ** Dopamine *** Fludricortisone **** Ephedrine

release of norepinephrine, produces vasoconstriction and perhaps some vasodilation secondary to its stimulation effects on cardiac beta adrenergic receptors. A net increase in heart rate and blood pressure result from ephedrine administration.

It is suggested to include a short term trial of oral ephedrine in intravenous vasopressors dependent patients secondary to autonomic dysfunction in neurogenic shock.

CONCLUSION

Spinal cord injury is a lethal entity, especially cervical part of spinal cord trauma increases the risk of disability and mortality manifold. Vascular dysfunction is a unique challenge that warrants swift management. Oral ephedrine can be an appropriate alternative in specific group of patients who require intravenous vasopressors for long periods of time and difficult to wean off the parenteral infusions.

Authors Contribution:
MFK: Concept and manuscript writing
KMS: Literature search and editing
MAA: Literature Search
HU: Review

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