



## Case Report

## Use of midodrine and fludrocortisone in neurogenic shock: A case report

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## ABSTRACT

Our case report includes a 42 year old male who had C6–C7 fracture dislocation with complete spinal cord injury following a motor vehicle accident. The patient underwent combined anterior and posterior fixation of the cervical spine. However, the patient had hypotension as a part of neurogenic shock and required vasopressor to maintain mean arterial pressure. The patient was treated in Intensive Care Unit with noradrenaline infusion to maintain mean arterial pressure but it was really challenging to stop the vasopressor. The patient was started on oral Midodrine and Fludrocortisone; vasopressor dose was tapered and finally stopped.

## 1. Introduction

Neurogenic shock following a complete spinal cord injury is difficult to treat and its prognosis remains grave in most of the times. The incidence of traumatic spinal cord injury with neurogenic shock is high in the young population with majority being the male subgroup [1]. The duration of neurogenic shock usually varies from two to six weeks. However, it may last for several months [2]. Prolonged shock can be stressful to the patient and it can delay the early rehabilitation process. We report a 42 year old male who had neurogenic shock following a traumatic spinal cord injury. The shock persisted for four weeks requiring vasopressor which was successfully tapered with the introduction of midodrine (alpha agonist) and fludrocortisone (mineralocorticoid).

## 1.1. Case presentation

A 42 year old male patient with no known co-morbidities presented to our emergency room with paraplegia following a motor vehicle collision. At the time of presentation, his Glasgow Coma Scale was E<sub>4</sub>V<sub>5</sub>M<sub>6</sub> and he was hemodynamically stable. His motor power in bilateral lower limb was 0/5 and there was sensory loss up to T6 level. Rest of the neurological examination including anal sphincter tone revealed no abnormalities. There was no urinary and fecal incontinence or retention. There was no significant family and drug history. Initial laboratory investigations were within normal limits. Computed Tomography scan showed C6–C7 fracture dislocation with complete spinal cord injury. According to the American Spinal Injury Association (ASIA)

impairment scale, the neurological severity of the spinal cord injury was class A. Combined Anterior and Posterior fixation of cervical spine was done by the spine orthopaedics team and postoperatively the patient was shifted to ICU for further management.

On the first day of admission, his mean arterial pressure above 80 mm Hg was well maintained with no vasopressor requirement. He was extubated after successful breathing trial. However, fall in BP was noted in the 2nd day of admission with blood pressure of 72/48 mm Hg with MAP of 56 mm Hg and bradycardia with heart rate of 45 beats per minute. Therefore, the patient was started on fluid resuscitation and noradrenaline infusion. Other possible causes of hypotension including hypovolemia, sepsis, and cardiac were ruled out by relevant clinical examinations and investigations including electroencephalogram and echocardiography. Thus, we attributed spinal shock to be the cause of hypotension. All standard ICU care and monitoring including physiotherapy, rehabilitation, and nutrition were continued. But, the patient continued to have persistent hypotension (with MAP below the target value) requiring vasopressor and it was very difficult to taper and stop the vasopressors till 21st day of ICU admission. Considering this scenario, tablet Midodrine 5 mg was added three times a day and the dose was gradually increased to 15 mg thrice daily. In addition, tablet Fludrocortisone 0.1 mg was started once daily. After the addition of these two drugs, vasopressor dose was slowly tapered and stopped on the following one week. The patient was shifted to general ward for further rehabilitation after 30th day of total ICU admission with residual motor and sensory deficits. Both midodrine and fludrocortisone were gradually tapered and stopped.

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## 2. Discussion

Decreased sympathetic activity as a result of spinal cord injury with intact parasympathetic tone from vagal nerve causes hypotension and bradycardia. Time course of development of neurogenic shock after spinal cord injury is not clearly demarcated [3]. In our particular case, the patient developed neurogenic shock from second day of admission in ICU.

The primary aim of treatment of neurogenic shock is to maintain perfusion to the body and compromised spinal cord thereby reducing secondary cord damage. Early identification of neurogenic shock followed by appropriate treatment and maintenance of perfusion is one of the few interventions linked with better neurological outcome. Neurogenic shock in our patient was treated with fluid resuscitation and noradrenaline. However, noradrenaline support could not be tapered and stopped as expected. With the addition of midodrine and fludrocortisone, neurogenic shock was dramatically improved. Midodrine is a peripheral alpha adrenergic agonist which undergoes enzymatic hydrolysis to form its active metabolite desglymidodrine. It exerts its sympathomimetic effect via activation of alpha adrenergic receptors in the blood vessels which causes resultant increase in venous return and blood pressure. It has been found to be effective in patients with orthostatic hypotension with minimal side effects [4,5]. Fludrocortisone is a potent mineralocorticoid drug with minimal glucocorticoid activity. It acts on distal tubule of nephron where it reabsorbs sodium with resultant increase in intravascular volume and blood pressure [6]. However, caution should be taken while prescribing fludrocortisone in patients with hypertension, congestive heart failure and coronary artery disease. In addition, electrolyte and acid base balance should also be kept in mind due to its effect on hydrogen and potassium ion [7]. Few cases have been reported in the literature where neurogenic shock has been successfully treated with midodrine and fludrocortisone [5,7,8]. A case report by Taikwan Kim and Cheol su Jwa showed that the patient showed recovery from neurogenic sock followed by administration of midodrine [9]. Another case report by T E Grooms showed that a 28 year old woman with C5 quadriplegia was successfully treated for hypotension with fludrocortisones in addition to other drug therapies [10]. However, more promising trials supporting the evidence for efficacy of these drugs are yet lacking.

## 3. Conclusion

This case suggests that the use of midodrine and fludrocortisone is effective in treating the refractory neurogenic shock associated with spinal cord injury thereby enabling the patient to participate in early rehabilitation program. However; we suggest further studies should be done for evaluating the evidence of these drugs in the treatment of neurogenic shock.

## Sources of funding

This study has not received any funding.

## Ethical approval

This study was conducted in accordance with ethical standard.

## Consent

Informed written consent was taken from patient for the publication of this case report.

## Author contribution

- I. KC Kripa took relevant history, clinical examination, collected relevant investigations of the patient and wrote the report. And she was directly involved in patient's care during his stay in ICU.
- II. Sushil Khanal worked for literature review and revision of the case report into its final version. He was directly involved in patient's care during his stay in ICU.

## Registration of research studies

1. Name of the registry: Not applicable
2. Unique Identifying number or registration ID:
3. Hyperlink to your specific registration (must be publicly accessible and will be checked):

## Guarantor

KC Kripa.

## Declaration of competing interest

No any conflicts of interest.

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