Open Journal of
Clinical & Medical
Case Reports

Volume 7 (2021)
Issue 05

ISSN: 2379-1039

A case of acute onset heroin induced paraplegia

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Abstract

Here we present a case of a destitute 61-year-old male who developed sudden onset of acute flaccid paraplegia of his lower extremities, impaired sensation of pain and temperature, acute urinary retention and loss of voluntary sphincter tone following an injection of heroin. He has a past medical history significant for coronary artery disease, hypertension, type 2 diabetes mellitus, hyperlipidemia, peripheral artery disease, chronic kidney disease and substance abuse disorder. We diagnosed him with heroin induced infarction with primary hypercoagulable state.

Keywords

Acute paraplegia; complete cord ischemia; hypercoagulable state; factor 8; heroin

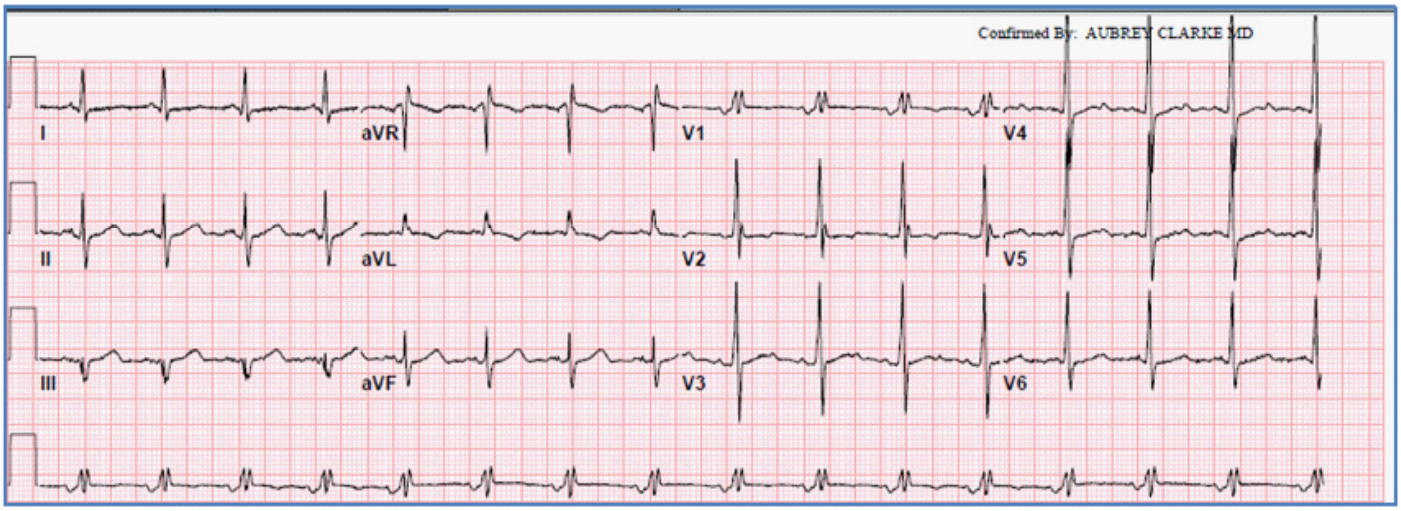
Introduction

Spinal cord ischemia occurs less frequently (0.3-1%) than cerebral infarction but results in significant morbidity, mortality, and reduced quality of life in survivors [1]. Common risk factors for spinal cord strokes are younger age, male gender, hypertension, and diabetes mellitus. The most common etiologies are large vessel atherosclerosis (33%) or aortic type A dissection (16%) [2]. Embolic stroke from aortic atheroma or valvular vegetation are less common. The next most common causes are iatrogenic causes (45% as reported in one case series) usually thoracic aorta aneurysm repairs with radicular arteries reperfusion defects [2]. The most common presentation of a spinal cord infarction is the anterior spinal cord syndrome. Complete cord ischemia is quite rare and usually occurs in trauma, severe tumor compression of the cord, epidural abscess, transverse myelitis, aortic dissection, cocaine induced vasospasm and heroin-induced hypotension. We present a patient with complete cord ischemia, sudden onset without recovery in an elderly male. The possible mechanisms and etiologies for this presentation are discussed below.

Case Presentation

A 61-year-old African American gentleman with a past medical history of uncontrolled diabetes mellitus, uncontrolled hypertension, hyperlipidemia, coronary artery disease, peripheral arterial disease on cilostazol, chronic kidney disease stage 4, tobacco smoker n opioid dependence (heroin), presented to the ED with a first-time episode of sudden onset weakness and numbness on bilateral lower limbs simultaneously associated with bowel incontinence, retrosternal chest pain of 7/10 intensity, after arriving to ED. He used approximately 20 grams of heroin three hours prior to the onset of symptoms. There is no h/o trauma to the back, fever, rash, sudden abdominal pain, vasculitis, neurosarcoidosis, tuberculosis, cancers, multiple sclerosis, aortic surgeries, atrial fibrillation, DVT, mitral valve disorders, ASD. On examination vitals showed low BP 109/55, power 0/5 in bilateral hip, knee, and ankle joint, deep tendon reflexes (knee, ankle, plantar) of 0, bilateral loss of pain, crude touch and temperature sensation below T7 distribution, bilateral loss of vibration and proprioception below knee level, bowel incontinence and bladder retention. Aspirin 325 mg was given stat, followed by dual antiplatelet therapy (Clopidogrel 75 mg and Aspirin 81 mg), Lipitor 80 mg OD, Heparin 5000 SC q 12 hrs. Empiric steroid treatment was initiated (Dexamethasone 10 mg IV stat with Solumedrol 1g IV for 5 days) with no improvement in symptoms. MRI C and T spine showed T2 hyperintense signal at T2 through T6-T7 (Image 2). This signal extended to the lower level of T1 in a couple of days. Stroke workup showed elevated factor VIII of 293% (Table 4). Other investigations were normal (normal lumbar tap except for diabetic changes of IgG. CT Thorax and Abdomen showed no aortic dissection but diffuse atherosclerotic changes. He was diagnosed with paraplegia secondary to complete cord ischemia at the T6 level from primary hypercoagulable state with Type 2 MI (3 serial EKG showed RSR' pattern in V1, one troponin reading of 0.016; pro BNP 778) and acute kidney injury (creatinine of 3.3 to 5.2 mg/dl). Of note, patient developed high BP (BP in 170s and 180s) following steroid administration and required labetalol 600 mg and Nifedipine 120 mg daily. Patient continued to remain paraplegic (power improved to 3/5). A suprapubic cystotomy with Foley insitu was done prior to discharge to relieve bladder retention. Patient was reviewed via telephone call after day 20 of incident. There was no improvement from discharge. Given poor social support his prognosis is poor.

Neuroimaging (08/26/2020)



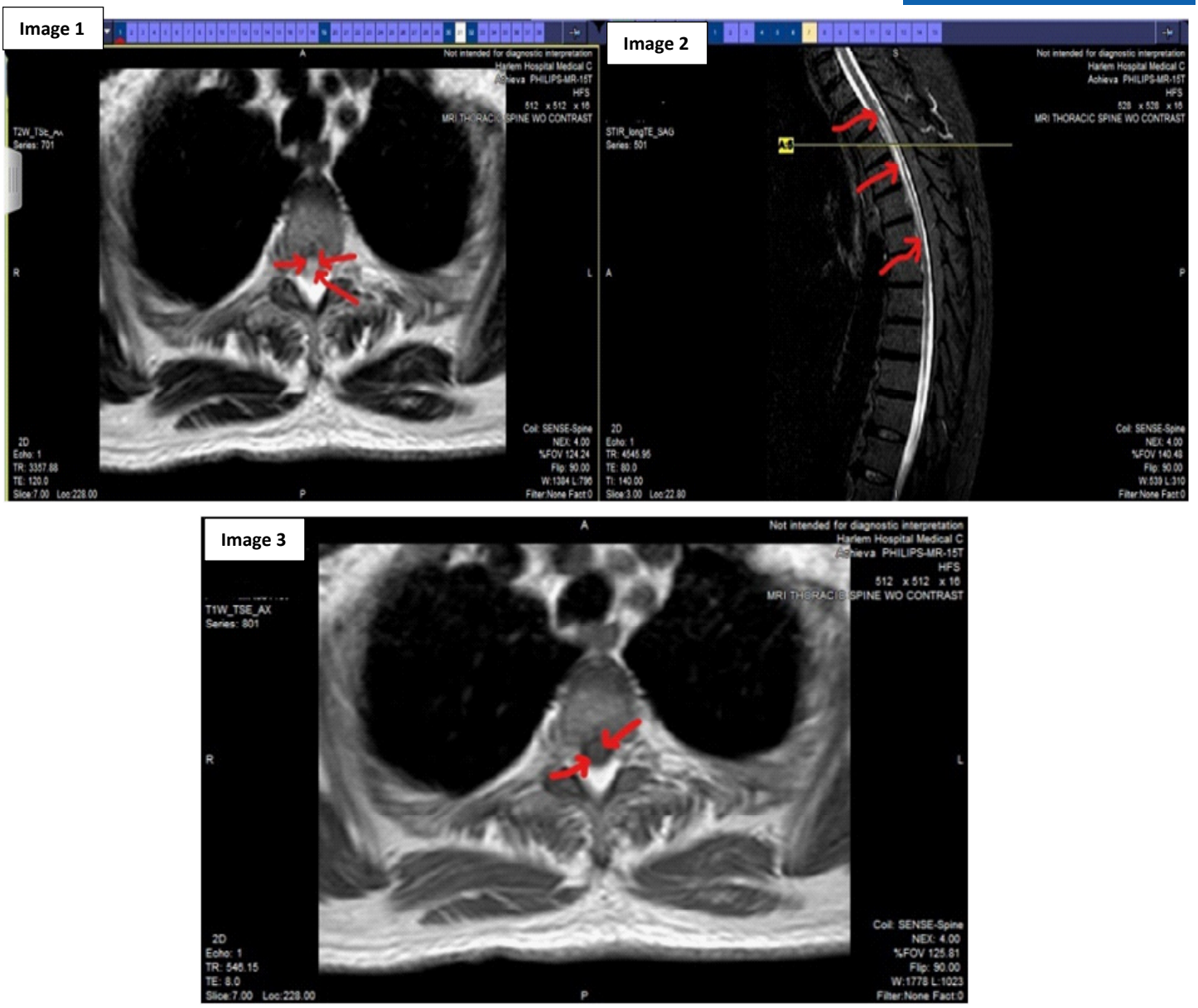


Image 1 & 3 (Cross Section): Anterior cord lesion and dorsal column lesion left>right.
Image 2 (Sagittal): Hyperintense lesions with cytotoxic edema in the T1 segment; thickened at T3-4 level, ends at T6 level

Discussion

In order to understand a spinal cord infarction, it is necessary to have a good understanding of the spinal cord circulation. The spinal cord receives its blood supply (Figures 1 and 2) from three major arteries: A single anterior spinal artery (ASA) and two posterior spinal arteries (PSA) [3].

The anterior spinal artery originates from the vertebral arteries at the level of the medulla at the vertebrobasilar junction (Figure 1). The anterior spinal artery travels down the middle of the spinal cord through the anterior sulcus (Figure 1). As it travels through the cervical portion of the spinal cord it gets fed by several feeders from various arteries like deep cervical artery branch of subclavian artery; costocervical trunk, thyrocervical trunk, ascending cervical arteries and 2-3 anterior radiculomedullary arteries (branch of superior intercostal artery (Figure 2). The thoracic and lumbar ASA gets feeders from the anterior rami of the segmental arteries (radiculomedullary arteries) (Figures 1 and 2). The segmental arteries are the

right and left branches of the descending aorta. There are usually 9 pairs of segmental arteries and one pair of subcostal arteries that feed the thoracic spinal cord. Artery of Adamkiewicz (0.5-1 mm diameter) which is the largest radiculomedullary artery, arises on the left, and supplies the T8-L2 areas of the spinal cord (in 75% of cases) [4]. The ASA continues caudally to the conus to form a rich anastomotic plexus with the PSAs (the 'rami cruciantes' and pial plexus/vasocorona) (Figure 2) [4].

The 2 PSAs also originate at the level of the foramen magnum by branches of the ipsilateral vertebral or posterior inferior cerebellar arteries (PICAs) [3,4]. Occasionally, they arise from the pre-atlantal vertebral arteries. The upper cervical part of the cord is supplied by the PICAs. The thoracic and lumbar PSAs are supplied by radicular arteries, branches of the segmental arteries. The lower lumbar portions of the PSA form a pial plexus with the ASA and supply the conus medullaris. The system of posterior spinal arteries is a discontinuous network which is often misrepresented in several textbooks as continuous system of two posterior spinal arteries on the right and left sides of the spinal cord [4].

The ASAs (diameters 0.2 to 0.8 mm) and the PSAs (diameters <0.5 mm) are small in diameter and hence susceptible to major infarctions from vasospasms or occlusions to these arteries [4]. The upper thoracic portion of ASA has a smaller diameter when it passes through the thoracic spinal cord as compared to its cervical and lumbar sections. Thus, it is the most vulnerable to having its blood supply cut off. This is also the watershed area of the spinal cord, especially at the border, with opposing flow from adjacent ascending and descending radicular branches that supply the ASA. Hence infarctions in the thoracic portion of the spinal cord are quite common [3,4].

The ASA supplies the anterior two-thirds of the spinal cord tissue (including the anterior horns, and the spinothalamic and corticospinal tracts) by central and pial branches. Blockage of the ASA results in loss of bilateral motor power, pain, temperature, and crude touch [5]. The PSA supplies the dorsal third of the spinal cord including the dorsal grey horns, spinal ganglia, and the nerve roots. Blockage of the PSA will cause UNILATERAL and IPSILATERAL effects (as paired) with loss of vibration and proprioception along with fine touch ipsilaterally [5].

FIG 1

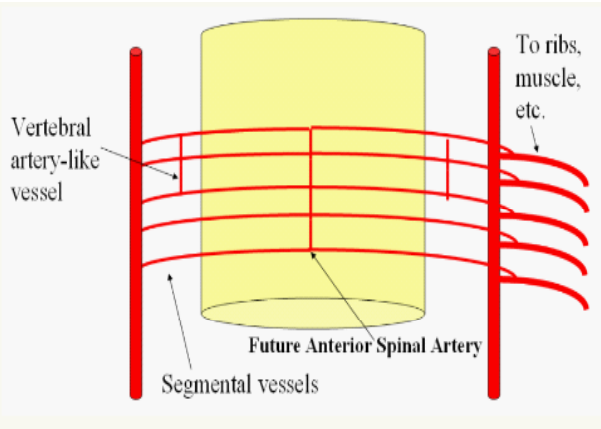
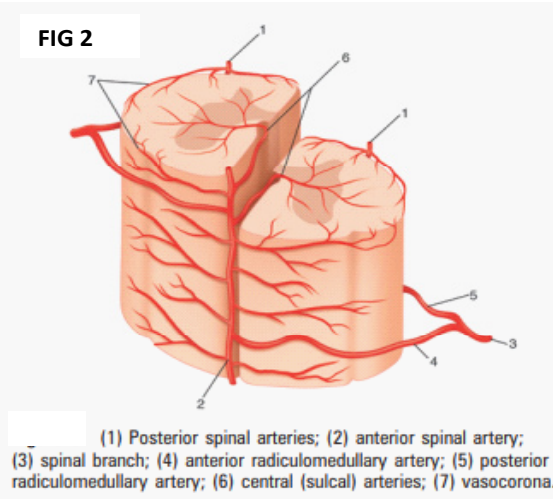


FIG 2



(1) Posterior spinal arteries; (2) anterior spinal artery; (3) spinal branch; (4) anterior radiculomedullary artery; (5) posterior radiculomedullary artery; (6) central (sulcal) arteries; (7) vasocorona

Our patient presented with complete cord ischemia involving the anterior cord and the posterior cord with type 2 MI. The MRI C and T Spine revealed hyperintensity deficits in T3-T7 (the watershed areas (Image 2)) [2]. An aberrant single artery blood supply to the cord could be the cause for complete cord ischemia. This aberrancy could be a single direct branch off the segmental artery that feeds the ASA and PSAs directly (Figure 1). A thrombus in this direct branch would cut off blood supply to both the ASA and PSAs instantaneously causing a complete cord ischemia. The second explanation could be from a single PSA in the midline (responsible for BILATERAL dorsal column lesions like the ASA). (Bilateral dorsal column lesions are uncommon as there are paired PSAs). Angiogram studies would be the best test to show this anomaly, but it wasn't done as our patient had CKD Stg 4 (with a creatinine of 5) Heroin-mediated injury is high on the list of differentials, either through direct toxicity to the vessels and organs or through immune response (from adulterants like talcum, starch, curry powder, Vim, Ajax, caffeine («Chinese heroin»), strychnine, mannitol, quinine, or lactose) leading to a thrombotic/vasculitic phenomenon [6].

In our patient, multifactorial causes could have contributed to the multiorgan involvement - complete cord ischemia, mildly elevated troponin (0.016 ng/ml and proBNP 778 pg/ml) and acute kidney injury (creatinine from 3.3 mg/dl to 5.2 mg/dl). These likely causes are the underlying hypercoagulable state from elevated factor 8, uncontrolled hypertension, uncontrolled diabetes (170s to 250s) and hyperlipidemia (TG – 284 mg/dl) [7,8]. The spinal arteries, having a small diameter, were easily clogged as they were susceptible to the Virchow's triad of poor circulation hypercoagulability (elevated factor 8) and vascular damage from hypertensive/diabetic vasculopathies and atherosclerosis. Hypercoagulable state in our patient was additionally worsened by elevated vWF in CKD 4 (Von Willebrand factor), buprenorphine 8 mg intake and metoprolol intake. Buprenorphine injections are known to cause emboli. 9 vWF levels which are elevated in CKD patients cause increased platelet adhesion and aggregation.10 Metoprolol increases F2 iso-prostane concentrations which increases platelet adhesions [11].

Conclusion

In a patient with acute paraplegia suspect drug induced infarctions in your differential. These could be from vasoactive drug induced spasms or immune response. Hypercoagulable states can appear as a late manifestation. Complete cord ischemia is highly unusual but can occur in those with multiple comorbidities, especially if an aberrant blood feeder to the spinal arteries is present.

Learning point: Heroin intravenous drug use can cause acute onset transverse myelitis. Primary hypercoagulable state can cause multiorgan failure.

Table 1

CSF Parameters	Value	Ref range
Color	Colorless	Color less
Appearance	Clear	Clear
Appearance Spun		negative
WBC manual CSF	<=5%	1
WBC Ct	<=5 cu/mm	1
RBC manual CSF	<=0%	0
RBC Ct	<=0 cu/mm	0
Glucose	117 mg/dl	(40-70 mg/dl)
Protein	68 mg/dl	(15-45 mg/dl)
LDH	68 U/L	-
CSF culture	No growth	-
IgG CSF	9.4 mg/dl	<=3.4 mg/dl
CSF Albumin	56.8 mg/dl	14-25.0 mg/dl
IgG/Alb serum	0.31	<=4.00
Oligoclonal banding	absent	absent

Table 2

IgG subset	Value	Reference Range
IgG Subset 1	583 mg/dl	248-810 mg/dl
IgG Subset 2	152 mg/dl	130-555 mg/dl
IgG Subset 3	57 mg/dl	15-102 mg/dl
IgG Subset 4	22 mg/dl	2-96 mg/dl
Total IgG	897 mg/dl	603-1613 mg/dl
IgG index	0.5	<=0.7
IgG	946	610-1660 mg/dl
IgG synthesis	5.3 mg/day	<= 8.0 mg/day
S Albumin	3064	3500-5200 mg/dl
IgG/Alb serum	0.31	<=4.00
IgG/Alb CSF	0.17	<=0.25

Table 3

EKG	NSR right ventricular conduction delay (RSR' pattern in V1 serial EKGs)
Creatinine	3.3 → 5.2 → 6.3 mg/dl
Troponin	0.016 (N <=0.016)
Pro BNP	778 pg/ml (N <= 125 pg/ml)
Echocardiogram	Normal systolic function, mild LVH, diastolic relaxation abnormal, normal atrial size, normal valvular abnormal
Glucose studies	170s - 250s HbA1c 4.7
Lipid Panel	TG – 284 mg/dl Chol - 179 mg/dl HDL – 31 mg/dl LDL – 91 mg/dl

Table 4

Autoimmune/ Hypercoagulable Parameters	Value	Ref range
Factor VIII	293%	60-125%
Factor V	115%	50-150%
Antithrombin III	134 %	(85-135%)
Protein C	114%	74-150%
Protein S	80%	67-141%
Homocysteine	11.3	<=15 mmol/L
Phosphatidylserine Serum Ab		
IgA	<20 U/mL	<20 U/mL
IgG	<10 U/mL	<10 U/mL
IgM	<25 U/mL	<25 U/mL
Serum NMO antibodies	Negative	
Beta2 Glycoprotein 1 Ab (B2G)	Negative	Negative
RF	10	0-13 IU/ml
ANA	Negative	< 1:80
ESR	38 mm	0-15 mm/hr
CRP	3.51	0.00-0.40mg/dl

Table 5	
MRI T Spine	Long segment of T2/STIR extending from T2 caudally (through T6-T7); thickness T3-T4
MRI C Spine	T2 hyperintense signal at the T1 mid vertebral body level (increased from previous scan with extension of lesion from inferior end plate of T2 to T1)
MRI Brain	Acute lacunar infarction (right thalamus) Chronic lacunar infarcts (deep white matter; frontoparietal lobes centrum semi-ovale left > right); lesions on the left rosary like distribution s/o carotid arterial stenosis Calcification of bilateral globus pallidus Atherosclerotic calcification of cavernous segment of bilateral ICA Chronic microvascular disease in the frontoparietal white matter n pons
CT head without contrast	Left centrum semi-ovale – remote infarct
MRI Lumbar Spine	Nil significant (mild foraminal narrowing L4-L5; L5-S1)
CT Angio Carotid arteries/ Circle of Willis	Bilateral ICA narrowing – mild to moderate atherosclerotic stenosis Eccentric mural filling defect – left internal jugular vein (non-occlusive thrombus) Patent circle of Willis
CT Angio chest, abdomen, pelvis	Atherosclerotic calcification in abdominal aorta and distal branches. Left adrenal adenoma 3x2 cm. Auto infarcted spleen. Cholelithiasis. No evidence of traumatic aortic injury.

Table 6 on Inpatient treatment		
Steroids	Dexamethasone 10 mg IV Stat Solumedrol 1g IV X 5 days	
Antihypertensives	Nifedipine ER 90 mg once daily - increased to 120 mg once daily Labetalol 300 mg twice daily Metoprolol and Amlodipine were discontinued	BP - 150s (few reading in 170s and 180s)
Stroke medicines	Aspirin 81 mg Clopidogrel 75 mg Atorvastatin 80mg	
Diabetes	Sliding scale with regular Insulin Aspart (Novolog) 8 units thrice daily Levemir (detemir) 27 units twice daily	
Addiction	Methadone 20 mg once daily	Discontinued on day of discharge
Diabetic neuropathy	Gabapentin 300 mg thrice daily	
Surgical	Suprapubic cystotomy	Day 9 of inpatient stay
Rehabilitation	Inpatient rehabilitation	Referred to subacute rehabilitation

References

1. Romi F, H Naess. Spinal Cord Infarction in Clinical Neurology: A Review of Characteristics and Long-Term Prognosis in Comparison to Cerebral Infarction. Eur Neurol. 2016; 76: 95-98.

2. Caton MT, Huff JS. Spinal Cord Ischemia. [Updated 2020 Jul 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021.

3. Gofur EM, Singh P. Anatomy, Back, Vertebral Canal Blood Supply. [Updated 2020 Jul 27]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021.

4. Santillan A, Nacarino V, Greenberg E. Vascular anatomy of the spinal cord. J NeuroIntervent Surg. 2011.

5. VK. Kunam, V Velayudhan, ZA. Chaudhry, M Bobinski, WRK. Smoker, DL. Reede. Incomplete Cord Syndromes: Clinical and Imaging Review. RadioGraphics. 2018; 38: 1201–1222.

6. LR Caplan, DB Hier, G Banks. Current concepts of cerebrovascular disease — Stroke: Stroke and drug abuse. Stroke. 1982; 13(6): 869-872.

7. Sharma S, Hashmi MF, Bhattacharya PT. Hypotension. [Updated 2021 Feb 11]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021.

8. N Vongveeranonchai, M Zawahreh, D Strbian, S Sundararajan. Evaluation of a Patient With Spinal Cord Infarction After a Hypotensive Episode. Stroke. 2014; 45: e203-e205.

9. T Lim, KM Lee, SH Sim. Injection abuse of buprenorphine and midazolam tablets: Brain MRI findings and follow up of embolic stroke.

10. N Jain, SS Hedayati, R Sarode,S Banerjee, RF Reilly. Antiplatelet Therapy in the Management of Cardiovascular Disease in Patients with CKD. What Is the Evidence? Clin J Am Soc Nephrol. 2013; 8(4): 665–674.

11. K Ayers, LM. Byrne, A DeMatteo, NJ. Brown. Differential Effects of Nebivolol and Metoprolol on Insulin Sensitivity and Plasminogen Activator Inhibitor in the Metabolic Syndrome. Hypertension. 2012; 59(4): 893-898.

Manuscript Information: Received: January 11, 2021; Accepted: May 14, 2021; Published: May 17, 2021

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Citation: Savarimuthu MK, Abdellatief A, Simon K, Salama F. A case of acute onset heroin induced paraplegia. Open J Clin Med Case Rep. 2021; 1749.