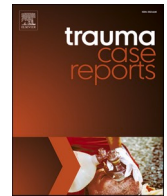




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# Trauma Case Reports

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## Cervical cord injury complicated by acute mesenteric ischemia

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### ABSTRACT

Acute mesenteric ischemia is a rare, life-threatening complication of cervical cord injury. This case report emphasized the importance of prompt diagnosis and treatment of this complication.

A 60-year old Japanese man with no co-morbidities was diagnosed as a C2 cervical cord injury and traumatic brain injury after a fall-down trauma. He was admitted to the intensive care unit after cord decompression surgery. Nine days later, he presented tachycardia and hypotension. Ultrasound showed dilated intestines and ascites. Intestinal ischemia was suspected based on the contrast-enhanced computed tomography scan. We performed prompt surgical resection of a broad part of the small intestines. The ischemic region of the intestines was segmental and the cause was attributed to a non-occlusive mechanism.

Acute mesenteric ischemia can be fatal and a delay in diagnosis leads to poor outcomes. We focus on predisposing factors in patients with cervical cord injuries and how to prevent this serious diagnosis.

### Background

There are some fatal complications in the acute phase of cervical cord injury. Of those, neurogenic shock, respiratory failure or thromboembolism is relatively common [1]. However, acute mesenteric ischemia may not be well-recognized as an acute phase complication. A misdiagnosis or delayed diagnosis may lead to a fatality.

### Case presentation

A 60-year old Japanese man without co-morbidities was transferred to the hospital after he slipped and fell down from a 3-meter height at a construction site. On arrival, his blood pressure was 84/50 mmHg, pulse 48/min, respiratory rate of 22/min, and O<sub>2</sub> saturation of 99% on a reservoir mask. The Glasgow coma scale was E3V4M6. A whole computed tomography (CT) scan revealed cranial epidural and subdural hematomas and a C2 spinal process fracture (Fig. 1). Examination showed complete quadriplegia and abdominal breathing. Subsequent magnetic resonance imaging (MRI) revealed a C2 cervical cord injury and a narrowing spinal canal due to a vertebral disc hernia and an epidural hematoma (Figs. 2, 3). Spinal cord decompression surgery was performed and he was admitted the intensive care unit (ICU) after surgery. On ICU admission, blood pressure was 163/79 mmHg and pulse was 56/min on 0.1 µg/kg/min of norepinephrine. Norepinephrine was discontinued on day 3 of the ICU stay. He remained quadriplegic after surgery. Although his mental status had been clear, his consciousness was abruptly deteriorated to E1VTM1 on day 5 which was not explained

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## C2 spinal process fracture (day 1)



Fig. 1. C2 spinal process fracture (day 1).

## MRI T1W1 (day 1) white arrow indicates epidural hematoma

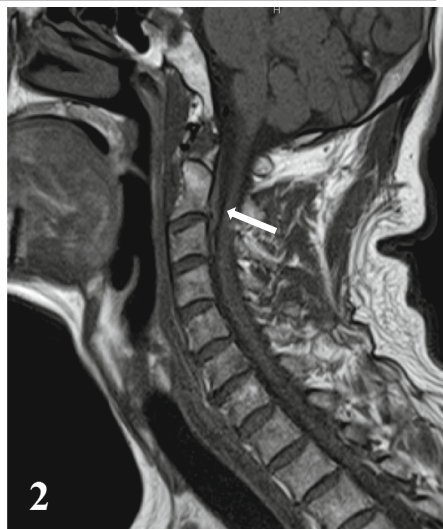


Fig. 2. MRI T1W1 (day 1) white arrow indicates epidural hematoma.

by CT and MRI images. Lumbar puncture and electronic encephalography (EEG) was unremarkable, although the EEG was not continuous. Levetiracetam was soon initiated for suspected status epilepticus but mental status remained unchanged.

After discontinuation of vasopressors on day 3, he presented transient hypertension from day 5 to 9. Systolic blood pressure varied significantly from 90 to 170 mmHg without any evident triggers. Furthermore, high fever above 40 degrees Celsius persisted from day 3 to 10 which was refractory to cooling and antipyretic drugs. He presented with tachycardia at 110/min on day 10 and became hypotensive, requiring fluid resuscitation, although his blood pressure had been maintained without vasopressors and there had been no signs indicating low-flow states such as decreased urine output or hyperlactatemia until that day. On physical examination, the abdomen was distended and ultrasound showed dilated intestines. Subsequent intravenous contrast-enhanced CT scan revealed a poorly enhanced region of the small intestines and mesenteric venous gas (Fig. 4). Emergent open abdominal surgery was performed

MRI STIR T2W1 (day 1) white  
arrow indicates cervical cord injury



Fig. 3. MRI STIR T2W1 (day 1) white arrow indicates cervical cord injury.

poorly enhanced small intestines  
and mesenteric venous gas (day 10)

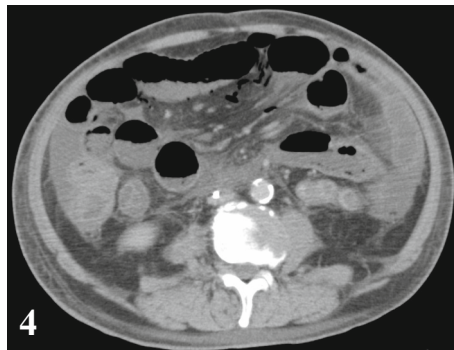


Fig. 4. poorly enhanced small intestines and mesenteric venous gas (day 10).

by surgeons and a broad part of small intestines - from jejunum at 60 cm from Treitz ligament to ileum at 20 cm to terminal - was resected. The distal ischemic lesion was segmental and no thrombus was found in the superior mesenteric artery or vein in CT scan, which indicated that the cause of ischemia was non-occlusive. After abdominal surgery, he received a tracheostomy on day 20 for persistent unconsciousness and was discharged from the ICU on day 27. He was transferred to another hospital for rehabilitation 10 days later.

### Discussion

Acute mesenteric ischemia is caused by occlusive or non-occlusive mechanisms. An occlusive mechanism is further defined as a mesenteric arterial embolism, mesenteric arterial thrombosis, or mesenteric venous thrombosis [2].

Traumatic patients can have bowel injuries which are not initially apparent. In this case, enteral nutrition was initiated on day 2 and normal defecation was observed on days 4 and 5, and bowel distention nor an increase in gastric residual volume were not observed until day 10, suggesting that the trauma did not cause bowel injuries.

The mesenteric circulation is a high-resistance vascular bed in which impaired regional perfusion owing to vasospasm can develop. The resulting ischemia is referred to as nonocclusive mesenteric ischemia (NOMI) [2]. NOMI is most often associated with cardiac insufficiency or low-flow states [2]. There were no signs of low-flow states until day 10, and the patient's cardiac function was normal on ICU admission. Furthermore, this patient had no previous history of atrial fibrillation and arrhythmias were not observed during ICU stay. Nor the risk factors of atherosclerosis were found in his past medical history such as hypertension, diabetes mellitus, or smoking. The operative findings were consistent with NOMI – no thrombus was discovered and segmental ischemic lesions were evident. Initially, he appeared to have no risk factors for NOMI. However, upon consideration, there were indeed predisposing factors including autonomic dysreflexia (AD) and a sustained high fever.

In AD, a noxious stimulus triggers an unregulated sympathetic cascade, leading to focal vasoconstriction [3]. Classically, if the neurological level of injury is at or above T6, this vasoconstriction can involve the splanchnic vessels and lead to progressive hypertension [4], predisposing patients to NOMI. Experimentally, it has been shown that mesenteric blood flow is markedly diminished in rats 3 days after a T3-spinal cord injury [5]. In our case, from day 5 transient high blood pressure was observed without any triggers or causes, which may imply patient's hyper-sympathetic activity.

Insensible water loss from respiration increases with a high fever [6], which can make patients prone to dehydration. The cause of our patient's high fever remained unclear; repeated blood culture was negative. The traumatic brain injury or the unidentified cause of the coma may have been responsible for the high fever. Moreover, impaired thermoregulation which can occur after a spinal cord injury may have exacerbated our patient's high fever [1].

In the acute phase of cervical cord injury, vasopressors may be required due to neurogenic vasodilatation. Vasopressors can be a precipitating factor for NOMI, inducing mesenteric vasoconstriction. Therefore, those patients should be managed with minimized vasopressors to maintain patients' individual perfusion pressure and adequate preload.

Research has shown that patients with spinal cord injuries have increased risks of ischemic bowel syndrome [7]. The potential risk of acute mesenteric ischemia in this case may have been underestimated. In summary, signs of hyper-sympathetic activity such as hypertension should not be ignored. These signs may imply visceral vasoconstriction, leading to poor perfusion of visceral organs. In addition, care should be taken to ensure that those patients do not become dehydrated. To avoid delayed diagnosis of NOMI, physicians should not miss signs of low-flow states such as reduced urine output or cold extremities, and signs of tissue hypoxia such as hyperlactatemia.

## Conclusion

We presented a case of cervical cord injury complicated by acute mesenteric ischemia.

Autonomic dysreflexia and dehydration due to sustained high fever may be predisposing risk factors.

## Abbreviations

CT	computed tomography
MRI	magnetic resonance imaging
ICU	intensive care unit
EEG	electronic encephalography
NOMI	non-occlusive mesenteric ischemia
AD	autonomic dysreflexia

## Ethics approval and consent to participate

Not applicable.

## Consent for publication

Informed consent was obtained in advance from the patient's family for publication of this case report and any accompanying images.

## Availability of data and material

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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### Declaration of competing interest

The authors declare that they have no competing interests.

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Not applicable.

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