Author's response to reviews

**Title:** Delayed posttraumatic spinal cord infarction in an adult following minor head and neck trauma: a case report

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**Author's response to reviews:** see over
Dear Editor,

We thank the reviewers and the Associate Editor for their valuable comments. Based on their suggestions and questions we made the following modifications to the manuscript.

**Associate editor:**

**Question:** Interesting case, well written. I would agree with the reviewer No 2. that the authors should consider a change of the manuscript title to: Delayed posttraumatic spinal cord infarction in an adult following minor head and neck trauma: a case report

**Answer:** We changed the title accordingly.

**Question:** I would also recommend the authors to describe terminal phase of the patient’s life in detail: they report that ...Twelve hours after hospitalization the patient became unresponsive and despite full resuscitation efforts he expired....
- was his unresponsiveness sudden or gradual? if gradual, describe its timeline
- were there any EWS (early warning signs) of deterioration? - hypotension, anuria, tachy/bradycardia? If he was found unresponsive, describe his neurological status in detail - GCS, seizures, etc?
- was this coma primarily associated with cardiovascular failure - arrest, VT, VF, bradycardia?
- was there spontaneous breathing?

**Answer:** We thank the editor for this comment since he pointed out the crucial message of our report. The key point is that the patient’s overall clinical status was not concerning enough for the patient being placed on continuous monitoring. This is the reason why he was waiting for an intermediate unit bed in the emergency department with neurological exams every 2 hours. The patient was hemodynamically stable and had adequate oxygen saturation, complaining only of whole body aching and waxing-waning paresthesias in the extremities. Because of difficulty voiding, he had a urinary catheter placed. This, retrospectively, was the only objective sign of worsening. Yet, 2 hours after the urinary catheter placement he was still stable. It was during the next 2-
hour check (4 hours after the catheter placement) that he was found dead. For the aforementioned reasons there are no known details on the terminal phase of the patient’s life. This unanticipated dramatic event prompted us to adopt a new hospital policy. Based on this policy every minor head or neck trauma patient with symptoms of cervical spondylotic myelopathy is put immediately on continuous vital sign monitoring and gets transferred to the surgical/neurosurgical intensive care unit for the maintenance of adequate arterial blood pressure.

We made appropriate changes into the Case presentation section to clarify this point.

Reviewer #1:

**Question:** This is a case report that details an extremely unusual clinical presentation of mild head trauma. The authors have described the clinical setting adequately. They have hypothesized the mechanism of the spinal cord necrosis but could have elaborated the pathophysiology with respect to the vascular territory involved e.g.: anterior versus posterior spinal artery territory or pial versus sulcal artery involvement. They could have used the autopsy findings (gross and microscopy) to advance the hypothesis.

**Answer:** Elaborating the pathophysiology with respect to the vascular territory involved is a very interesting topic. Pathological studies of infarcted human spinal cords indicate that infarctions occur most frequently in the metabolically most active lumbosacral and cervical segments (Duggal et al 2002). This finding is contrary to the historical concept of ischemic vulnerability of the “watershed zone” centered at the mid-thoracic levels, however it is comprehensible in perspective to the angiosome concept of blood supply to the spinal cord. Based on the angiosome concept, the arterial supply to the spinal cord follows a segmental, rather than longitudinal, distribution (Hong et al. 2008). So, during arterial hypotension, the most vulnerable spinal cord segments are those that are the most active, metabolically. The fact that our patient suffered infarction to the cervical intumescence is in good agreement with the aforementioned findings.

With respect to pial versus sulcal artery involvement, Ishizawa (2005) in a histopathological study on hemodynamic spinal cord infarction, showed that the infarction involved the gray matter supplied by the central artery, while the periphery of the spinal cord supplied by the pial arteries, remained intact. In our patient, the pathological finding was altered by the pre-existing cervical spondylotic myelopathy and delineation between pial versus sulcal vascular territories were not possible.

We added a paragraph to the Discussion section to elaborate the question of vascular territories.
**Question:** Figure 2 showing the autopsy specimen should be labelled indicating the orientation. In addition an axial section at the region of the abnormality will be interesting to the readers.

**Answer:** The labeling on Figure 2 has been added, indicating the orientation as well as the infarcted and normal parts of the spinal cord.

**Reviewer #2:**

**Question:** Interesting case, well explained. Manuscript well written and the authors have addressed all the questions from previous reviewers. My only question would be about the title of the case and whether "... following minor head and neck trauma" or something similar would be better than "following minor head injury" since there was some mechanism involving the neck during the trauma as well and not to mislead readers as something that would occur after a simple hit on the head with minor head injury...

**Answer:** We changed the title to: “Delayed posttraumatic spinal cord infarction in an adult following minor head and neck trauma: a case report”.

**Formatting changes**

1. We removed the subsections from the Case presentation section
2. We highlighted all changes in our revised manuscript

Sincerely,

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