Intervertebral Disc Cell Death in the Porcine and Human Injured Cervical Spine After Trauma: A Histological and Ultrastructural Study

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FROM ABSTRACT

Study Design: Histologic and ultrastructural study of disc cell death after traumatic injury to the human cervical spine.

Objective: To determine the changes in disc cell morphology, viability, and manner of cell death after trauma in human discs.

Summary of Background Data: It is known that compressive or traumatic injuries to cartilage and intervertebral discs can result in cell death by necrosis or apoptosis.

Methods: The anterior portion of intervertebral discs and endplates of 30 patients with traumatic injuries to the cervical spine were studied histologically and ultrastructurally.

Results: Electron and light microscopy showed up to 75% of human disc cells die within the first 24 hours of trauma, mainly by necrosis. Glycogen was commonly found in disc cells after trauma.

Conclusion: Traumatic injuries of the human cervical spine lead to rapid changes in disc cell morphology and cell death, particularly via necrosis.

THESE AUTHORS ALSO NOTE:

“The intervertebral discs are vital to the functioning of the spine in terms of its movement, load bearing, and protection of the spinal cord.”

“The discs in turn are dependent on the cells within them to produce and maintain a fully functioning extra cellular matrix.”

“Trauma has been demonstrated in articular cartilage to lead to apoptosis (so called programmed cell death).”

In the human scoliotic disc where there may be abnormal loading there is increased death of disc cells.

In apoptosis, the death of the cell begins within its nucleus.
In necrosis, the death of the cell begins in its membrane, leading to swelling and eventual rupture of the cell, then on to an inflammatory reaction.

“Patients who have undergone traumatic injuries to the cervical spine with subsequent surgical stabilization provide an ideal population in whom to study the effect of trauma on the cells of the human intervertebral disc. In this study, we have examined the response of disc cells in patients with different types of trauma between 0 and 3 days after injury.”

“Up to 75% of human disc cells die within the first 24 hours of trauma, mainly by necrosis.” [Important]

In this study, samples of human cervical intervertebral discs were obtained from 30 patients, aged 17 to 77 years (mean 41.5), undergoing routine stabilization 0 to 3 days after trauma. “The ultrastructural appearance of the cells within the human discs suggested that healthy cells were a rarity, with the majority of cells appearing necrotic, particularly in the outer regions (60% in the AF and 51% in the NP).”

DISCUSSION

The most likely injury to disc cells is due to ischemia with resultant reduction in nutrient supply and anoxia, in addition to a possible build up of metabolic waste products.

Necrosis is the main cause of cell death after periods of ischemia.

These authors indicate that compressive and flexion-extension loads are spread equally in the nondegenerated disc but cause greater cell death in degenerated discs. [Important]

The increased amount of glycogen found in the injured discs indicates more anaerobic metabolism of the cell, meaning altered metabolism in these disc cells.

This study shows that injured disc cells will respond to injury by “going down apoptopic and particularly necrotic pathways very rapidly, within 24 hours of injury.”

KEY POINTS FROM AUTHORS:

1) “Traumatic injury to the cervical spine causes fracture of the vertebrae and lesion of the intervertebral disc followed by rapid and extensive disc cell death.”

2) Necrosis is the main cause of disc cell death after trauma in humans.
KEY POINTS FROM DAN MURPHY

1) “Traumatic injuries of the human cervical spine lead to rapid changes in disc cell morphology and cell death, particularly via necrosis.”

2) “Up to 75% of human disc cells die within the first 24 hours of trauma, mainly by necrosis.” [Important]

3) Glycogen was commonly found in disc cells after trauma.

4) “Trauma has been demonstrated in articular cartilage to lead to apoptosis (so called programmed cell death).”

5) In the human scoliotic disc where there may be abnormal loading there is increased death of disc cells.

6) In apoptosis, the death of the cell begins within its nucleus.

7) In necrosis, the death of the cell begins in its membrane, leading to swelling and eventual rupture of the cell, leading to an inflammatory reaction.

8) The most likely injury to disc cells is due to ischemia with resultant reduction in nutrient supply and anoxia, in addition to a possible build up of metabolic waste products.

9) Necrosis is the main cause of cell death after periods of ischemia.

10) These authors indicate that compressive and flexion-extension loads are spread equally in the nondegenerated disc but cause greater cell death in degenerated discs. [Important]

11) The increased amount of glycogen found in the injured discs indicates more anaerobic metabolism of the cell, meaning altered metabolism in these disc cells.

12) This study shows that injured disc cells will respond to injury by “going down apoptotic and particularly necrotic pathways very rapidly, within 24 hours of injury.”

13) “Traumatic injury to the cervical spine causes fracture of the vertebrae and lesion of the intervertebral disc followed by rapid and extensive disc cell death.”

14) Necrosis is the main cause of disc cell death after trauma in humans.

COMMENTS FROM DAN MURPHY
This is very important in whiplash cases. Whiplash is proven to cause disc injury and this study documents that disc injury causes“ rapid and extensive disc cell death.”