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COMPLETE BURST FRACTURES OF THE THORACOLUMBAR JUNCTION WITH SPINAL CORD INJURY: SURGICAL MANAGEMENT AND CLINICAL OUTCOME IN A CASE SERIES OF 27 PATIENTS

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Background: Thoracolumbar burst fractures are caused by high-energy trauma such as suicidal jumping or falling from great heights. They may lead to a narrowing of the spinal canal with spinal cord compression and cause severe neurological impairment. While most patients suffer from multiple injuries, the optimal treatment remains a matter of debate.
Methods: We retrospectively analyzed all patients treated for complete burst fractures of the thoracolumbar junction suffering from a spinal cord injury from 2011 to 2021. We only included patients suffering from preoperative neurological deficits (ASIA Score A-D).
Results: 27 patients were treated for complete burst fractures (type A4 AO spine classification, 3 additionally suffered from a type C or B3 fracture) of the thoracic (6/27), or lumbar (18/27) spine. In 3/27, we detected multiple burst fractures. In more than half of the cases, the cause of injury was attempted suicide by jumping (14/27). Most patients presented with an ASIA C (37%) or A (33%) spinal cord injury, 89% of the patients underwent a staged procedure: dorsal fixation first and a ventral vertebral body replacement after a median of 7 days. Most patients recovered from at least one grade on the ASIA scale until discharge (ASIA D in 41% cases). In 6 cases, we did not perform a dorsal decompression, but opted for an urgent ventral decompression. We did not detect a significant difference in outcome in patients with and without dorsal decompression. Interestingly, 6/27 patients suffered from a concomitant sacrum fracture and required sacral decompression.
Conclusion: Patients suffering from complete burst fractures of the thoracolumbar junction may present with severe neurological deficits and benefit from an early surgical treatment. Whether a posterior decompression is necessary remains a question of debate. In our cohort, we did not detect a significant difference in clinical outcome depending on the surgical approach.

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A NEUROKININ-1 RECEPTOR ANTAGONIST MODULATES THE BLOOD-SPINAL CORD BARRIER AND IMPROVES FUNCTIONAL RECOVERY AFTER THORACIC SCI

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Objective: Traumatic spinal cord injury (SCI) remains a devastating event with no neuroprotective treatment currently available. Disruption of the blood-spinal cord barrier (BSCB) with subsequent edema formation and neuroinflammation after has been linked to the release of the neuropeptide Substance-P (SP). In this study, we aimed to antagonize the binding of SP to its neurokinin-1 (NK1) receptor with N-acetyl-L-tryptophan (NAT) in a rodent SCI model.
Methods: Female Wistar rats (N=66) were subjected to either a thoracic clip-contusion/compression SCI at the T10 level or sham surgery (laminectomy). Additionally, an osmotic micropump was implanted, and infusion of NAT or Vehicle into the intrathecal space over the spinal cord lesion was initiated. Basso, Beattie, Bresnahan (BBB) score, Gridwalk test, and CatWalk gait analysis were performed to assess functional recovery. After infusion of NAT/Vehicle for 1, 3, or 7 days, animals were sacrificed, immunohistological analyses were conducted, and results were statistically compared ($p < 0.05$ was considered significant).
Results: The inhibition of SP via NAT in the injured spinal cord showed no significant effects on macrophages' infiltration and their polarization towards the M1- or the M2-subtype 7 days after SCI. However, the invasion of T-lymphocytes was significantly reduced with the NAT treatment. Furthermore, a reduced leakage of Fibrinogen and an attenuated expression of ß-Catenin was found, indicating BSCB-stabilizing capabilities of NAT. Correspondingly, a trend towards reduced spinal cord edema could be observed. Furthermore, after 7 days of NAT-infusion, a significantly increased BBB score and significantly improved recovery in the CatWalk gait analysis were observed.
Conclusion: The intrathecal administration of NAT led to increased integrity of the BSCB in the acute phase after thoracic SCI, potentially attenuating aspects of neuroinflammation, reducing edema formation, and improving functional recovery. Thus, NAT might have neuroprotective properties and should be further assessed in the context of SCI.

5.5 Neurocritical Care for TBI and SCI

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DIRECT ORAL ANTICOAGULANTS VS. LOW-MOLECULAR-WEIGHT HEPARIN FOR PULMONARY EMBOLISM IN PATIENTS WITH GLIOBLASTOMA

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Background: Glioblastoma (GBM) is a cancer type with high thrombogenic potential and GBM patients are therefore at a particularly high risk for thrombotic events. To date only limited data on anticoagulation management after pulmonary embolism (PE) in GBM is available and the sporadic use of DOACs remains off-label.
Methods: A retrospective cohort analysis of patients with GBM and postoperative, thoracic CT-scan confirmed, PE was performed. Clinical course, follow-up at 6 and 12 months and the overall survival (OS) were evaluated using medical charts and neuroradiological data.
Results: Out of 584 GBM patients, 8% suffered from postoperative PE. Out of theses, 30% received direct oral anticoagulants (DOACs) and 70% low-molecular-weight heparin (LMWH) for therapeutic anticoagulation. There was no significant difference in major intracranial hemorrhage (ICH), re-thrombosis or re-embolism between the two cohorts. Although statistically non-significant, a tendency to reduced mRS at 6- and 12 months was observed in the LMWH cohort. Furthermore, patients receiving DOACs had a statistical benefit in OS.
Conclusion: In our analysis DOACs showed a satisfactory safety profile in terms of major ICH, re-thrombosis and re-embolism compared to LMWH in GBM patients with postoperative PE. Prospective, randomized trials are urgent to evaluate DOACs for therapeutic anticoagulation in GBM patients with PE.

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ESTIMATION OF INTRACRANIAL PRESSURE USING FUNDOSCOPY

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Introduction: Monitoring of intracranial pressure (ICP) remains invasive with external ventricular drain being the golden standard. Although several different methods such as optic nerve sheath diameter and pupillometry have shown some promise, non-invasive ICP monitoring is still not validated and implemented, especially in the acute setting. Correlation between intraocular hemodynamics, intraocular pressure and ICP is well-known, but not fully understood.
The purpose of this study is to evaluate if non-invasive fundoscopy of the retinal vessels can estimate absolute and/or changes in intracranial pressure in adult patients admitted to the neurosurgical intensive care unit (NICU).
Methods: Design: Observational cohort study, single-blinded (analyst)
Study setup: Fundoscopy without the use of mydriatic drugs was performed using CE branded capturing device, where the arteriole(A) and venule(V) diameter was measured, and A/V-ratio calculated. Ratios were correlated to the patient's invasive ICP monitoring device. Intraocular pressure was also acquired. A mixed-effect linear regression model with random intercepts was used to assess association between mean ICP and median A/V-ratio.