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Endovascular coil embolization of segmental arteries induces vascular remodeling and consequently reduces paraplegia after subsequent thoracoabdominal aortic aneurysm repair – an experimental model

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Thoracoabdominal aneurysm repair (TAAA) remains a challenging surgical procedure looking at postoperative paraplegia rates – whether to be performed in open surgical technique, endovascularly or in hybrid procedures is a decision to be made for each patient individually. The possibility of endovascular repair would enable treatment of patients for whom open surgery currently poses an unacceptably high risk. Hybrid procedures combine the two approaches and are able to consolidate the benefits of both when applied with consideration of patient history and aneurysm morphology. Splitting the intervention in two stages appears to be a promising alternative. However, a high percentage of patients with aneurysm or other extensive aortic anomalies are not suitable for two demanding operations.

The hypothesis behind this work was that spinal cord vasculature is not based upon one single conduit but rather on a complex interplay between various collaterals, which are able to remodel and adapt to a period of ischemia to ensure spinal cord integrity given that a certain amount of time is provided for the collateral network to recover. Current understanding of spinal cord injury, and particularly of the spinal cord circulation and its response to deprivation of segmental artery input, has been pieced together from both clinical observations and laboratory evidence. Similarly, the likely success of the two-stage strategy for preventing paraplegia was suggested by clinical observation, but its validity was confirmed and its implications extended in laboratory experiments.

In this experimental model, the aim was to find a preliminary procedure before singlestage TAAA repair that offers diverse protective measurements: minimally invasive approach for low intraoperative risk, no risk of paraplegia with few SAs sacrificed, sufficient ischemic stimulation of spinal cord vasculature and triggering of vascular remodelling. Since operation time was short and no perioperative complication occurred, coil embolization can be considered a safe procedure. Hemodynamic evaluation intra- and postoperatively underline that statement. All pigs fully recovered after coiling with no signs of neurological impairment shortly after extubation. The benefit of an ischemic stimulus could be observed in pigs with 2 or more SAs coiled and was reflected in the 0% paraplegia rate in group 2 (4 to 5 SAs coiled) after TAAA repair performed one week after coiling embolization. TAAA repair was simulated in a hybrid method by open surgical ligation of lower thoracic and lumbar vessels and subsequent endovascular stenting of the thoracic part of the aorta. One segmental artery, however, precedently occluded did not seem to be a sufficient stimulus, as the corresponding pig was paraparetic. Paraplegia rate without coiling (control group) was 60% and thereby significantly higher than in group 2. Histopathological injury after sacrifice was also significantly lower in group 2 than in control and was most impressive in the coiled region. Additionally, high neurological injury scores and high numbers of injured levels within the spinal cord could be defined as predictive factors for paraplegia. All pigs with 2 or more SAs coiled were not within the range calculated for those parameters. Unfortunately, it was not possible to evaluate the histopathological impact of coil embolization on a molecular level - serum levels of vascular endothelial growth factor did not seem to be the appropriate setting in which changes can be affirmed.

Nonetheless, coil embolization prior to extent TAAA repair, significantly reduced paraplegia and might be suitable for both patients undergoing elective endovascular or open surgical TAAA repair. Especially patients with aneurysms of large extent and high comorbidity could profit from a low-risk-preliminary procedure that would reduce their risk of postoperative paraplegia compared to conventional single- or two-stage procedures. Before being applied to humans, it would be ideal to find a way to monitor or evaluate the efficacy of ischemic preconditioning through coiling. Markedly, further investigations upon cellular mechanisms enhancing spinal cord blood flow and inducing vascular remodeling are needed. Despite the aforementioned limitations a clinical trial in a patient cohort at high risk for paraplegia might be appropriate.