Disturbance of fracture healing by blunt thoracic trauma

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Introduction: Life-threatening complications from patients suffering from multiple injuries are not caused solely by the sum of the individual injuries but from the local and systemic inflammatory response to trauma. According to epidemiological data of 2,069 patients with compound injuries from the Trauma Registry of the German Society of Trauma Surgery (1), injuries of the extremities was the most common type of injury (68.9%), followed by chest trauma (44.5%) and severe head injury (39.2%). Most importantly, blunt chest trauma has been found to have a crucial role in the prognosis of severely injured patients (9). These observations have increased efforts researching pathophysiology and healing of organs following multiple injuries such as bone healing. Severe head trauma has been found to stimulate bone healing with accelerated osteogenesis and formation of hypertrophic bone callus (8). Although several factors such as hormones were discovered to potentially influence bone metabolism, the mechanism of the interaction remains unclear (2). Recent research has found that the lung plays a central role in the inflammatory response to compound injuries (3,4). However, there are no studies proving the connection between chest trauma and bone healing. Therefore, a hypothesis was made arguing that chest trauma alters the process of fracture healing and an experimental study was created to investigate this potential effect.

Materials and Methods: Male Wistar rats (weight 250 to 300 g) were randomly assigned into two groups of 48 animals each: Group A containing rats with an isolated fracture of the lower leg, and group B containing rats with additional blunt thoracic trauma. The fracture of the right lower leg created in all animals was produced by a guillotine model and was then stabilized by a 0.7 mm K-wire after closed reduction. A custom-made blast wave generator induced blunt thoracic trauma only to rats in group B. The blast wave was generated by a pressure reservoir with a polyester diaphragm. When the pressure exceeded the resistance of the diaphragm a single blast wave hit the thorax of the rat. Local blood flow was measured at the fracture site before and after trauma using laser Doppler flowmetry. Seven rats were available for sham procedure. Rats were killed after 1, 3, 7, 14 days (n=8 rats per subgroup), and 28 days after operation (n=16 rats per subgroup). Blood flow measurements were repeated before sacrificing. After preparation of the tibia, a maximum of n=8 specimens of each subgroup were stained with Giemsa and fracture healing was analyzed using light microscopy. The remaining n=8 specimens of day 28 were scanned using micro-computertomography to examine standardized callus volume and density and were then tested biomechanically using three-point bending.

Results: The chest trauma (group B) significantly decreased maximum failure load by 29% compared to group A (Fig. 1). According to that, callus volume was reduced by 15% after thoracic trauma which could be attributed to the periosteal callus. Microscopic analysis revealed a significant delay in chondral callus formation from the effects of blunt thoracic trauma at later stages of healing process. Local blood flow from the soft tissue envelope at the lower leg was also altered due to chest trauma (Fig. 2). Blood flow regeneration after chest trauma and fracture did not exceed hypercompensation between day 7 and day 28 observed after isolated fracture. Bone density showed no significant differences between the groups.

Discussion: We have shown in this experimental study for the first time that a blunt thoracic trauma is capable to influence fracture healing. Chest trauma led to a delay in chondral callus maturation and caused subsequent minor maturation of bony callus volume observed on the 28th day of healing. In addition, thoracic trauma reduced local blood flow at the lower leg between day 7 and the end of the experiment, but the reason of the soft tissue vascularization disturbance remains unclear. There are no reports of possible long-term effects of a chest trauma due to local blood flow. However, it is known that disturbances of fracture healing can easily occur if the fracture is not supplied with sufficient blood. Other factors may affect bone healing after chest trauma. High concentrations of cytokines released by a systemic inflammatory reaction (6,7) and prostaglandins (5,6) are known to induce bone resorption. This experiment raises questions referring to the delay in fracture healing after chest trauma and requires further investigation.


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