THE EFFECT OF PLATELETS ON LUNG CAPILLARY LEAK AFTER FEMORAL FRACTURE AND INTRAMEDULLARY FIXATION

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Purpose:
The purpose of this study was to determine the role of platelets in the lung capillary injury elicited by intramedullary fixation of femoral shaft fractures.

Background:
Pulmonary dysfunction, a common complication observed following femoral fracture and intramedullary fixation, may lead to respiratory failure. Although the etiology of the lung damage following femoral fracture and fixation is ill-defined, it has been suggested that the intravasation of neutral, intramedullary fat is a key initiating event in the pulmonary dysfunction (1). Fat deposition in the pulmonary microvasculature may be chemotactic for circulating neutrophils or may serve as a nidus for platelet aggregation and thrombus formation. A recent study suggested that inflammatory neutrophils may not mediate the pulmonary dysfunction noted after experimental femoral fracture and fixation (2). However, the role of platelets in fracture-induced lung injury remains unclear.

The purpose of this study was to evaluate the role of platelets in the pulmonary capillary leak elicited by experimental femoral fracture and fixation.

Methods:
The pulmonary capillary filtration coefficient, a sensitive and specific index of lung capillary leak (3,4), was determined in rats subjected to bilateral femoral fracture and intramedullary fixation. The role of platelets in the lung injury response was determined in a separate group of animals using: 1) inhibition of platelet aggregation using a GIIb/IIIa receptor antagonist (ReoPro); or 2) platelet depletion with anti-thrombocyte serum.

Results:
Femoral fracture and intramedullary fixation resulted in significant thrombocytopenia (Fig. 1) and produced marked lung capillary leak (Fig. 2) compared to control experiments. However, neither inhibition of platelet aggregation nor platelet depletion blocked the lung damage produced by femoral fracture and fixation (Fig. 2).

Discussion:
Pulmonary dysfunction is a well recognized complication in patients subjected to long bone fractures. Although the pathogenesis of the lung injury is not clear, it has been suggested that pulmonary fat emboli initiate an inflammatory response, culminating in lung capillary leak (1). Although neutrophils have been implicated in this type of lung damage, recent reports do not support a major role for inflammatory leukocytes in the development of fracture-elicited pulmonary vascular injury (2).

It has been suggested that platelets contribute to the lung injury seen in patients with long bone fractures since thrombocytopenia is often observed in patients with long bone trauma who develop pulmonary dysfunction (5). Moreover, an abundance of tissue thromboplastin may be released systemically at the time of fracture and/or fixation, activating the complement and extrinsic coagulation systems. Intravascular coagulation byproducts cause platelet aggregation and induce the release of platelet-derived factors which mediate lung injury manifested by an increase in pulmonary capillary permeability. The purpose of this study was to define the role of platelets in the lung injury elicited by femoral fracture and fixation.

Although femoral fracture and intramedullary fixation was associated with significant thrombocytopenia, neither inhibition of platelet aggregation nor platelet depletion prior to fracture blocked the lung injury associated with femoral trauma.

Conclusion:
Platelets do not appear to mediate the pulmonary capillary leak elicited by femoral fracture and intramedullary fixation.

References: