

Does Neutrophil Elastase Degradation of Plasminogen Contribute to Intrapleural Fibrinolytic Failure?

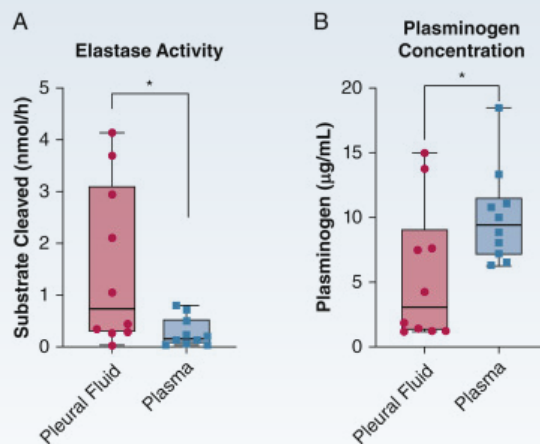
STUDY DESIGN


Evaluation of infected pleural fluid and circulating plasma from hospitalized adults (n = 10) before intervention (intrapleural fibrinolytics vs surgery) and on days 1, 2, and 3 after intervention


Testing included:

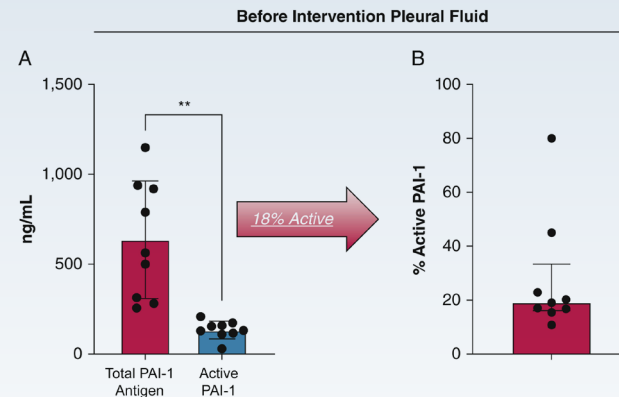
- Activity assays
- Enzyme-linked immunosorbent assays
- Western blot analysis
- Turbidimetric measurements of fibrinolysis

RESULTS



Elastase: Pleural fluid 4x  than plasma

PLASMINOGEN ANTIGEN:
Pleural fluid 3x  than plasma



Plasminogen activator inhibitor 1 (PAI-1), native tissue plasminogen activator inhibitor, had high levels before intervention, but 82% of that PAI-1 was not active

- All activity was lost by day 2 after intervention

Our results indicate that inflammatory plasminogen deficiency, not high PAI-1 activity, is a significant contributor to intrapleural fibrinolytic failure.