Characterizing fibrin glue performance as modulated by heparin, aprotinin, and factor XIII.

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Abstract:

We describe the performance of fibrin glue (FG) as modulated by heparin, aprotinin, or factor XIII levels. In vitro tests and a rat kidney excision model demonstrated that the hemostatic efficacy of fibrin was not modulated by aprotinin. Overlapping rat skin sections demonstrated that adhesion strength (AS) was proportional to the area of overlap as well as to fibrinogen levels. AS was not modulated by exogenous heparin or aprotinin and was independent of the endogenous factor XIII in fibrinogen. SDS-PAGE developed by Coomassie or Western blots with anti-gamma chain antibody confirmed that normal skin sections contain adequate trans-glutaminase to maximally cross-link normal, as well as XIII-depleted, fibrin. Fibrin glue (FG) sprayed onto rat skin incision wounds with a dual channel spray applicator acted in 2 phases: initially (day 1), compared to wounds stapled without or treated with only thrombin, FG significantly increased breaking strength. In the second phase of wound healing (after day 3), all groups achieved increased but equivalent breaking strength. FG containing aprotinin (to 3000 U/m; Immuno, Behringwerke, Germany) exhibited initial tissue bonding strength equivalent to fibrin without aprotinin, but histological examination showed delayed fibrinolysis and a concomitant slower regeneration of granulation tissue. Thus, our data indicated that aprotinin was not particularly beneficial to wound healing and that the endogenous factor XIII level in the fibrinogen did not contribute significantly to skin bonding. Rather, the tissue supplied adequate trans-glutaminase activity required to crosslink fibrin to itself and to the tissue.