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Does FXIII Deficiency Impair Wound Healing after Myocardial Infarction?

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Inadequate healing of myocardial infarction may contribute to local expansion of the infarct, frequently leading to chamber dilation, heart failure, or myocardial rupture. Experimental evidence in mouse models suggests that Factor XIII might play a key role in wound healing and low persistent values lead to increased incidence of cardiac rupture following myocardial infarction. For example in heterozygous FXIII deficient mice (characterized by 50% plasma levels of FXIII) 100% die from cardiac rupture following MI (Figure 1 A-C) and FXIII replacement therapy reverses these findings [2]. Here we would like to share our initial clinical experiences with strikingly similar observations in patients with this grave disease.

REFERENCES


Figure 1.
A: Short axis high resolution, high field cardiac MRI of a FXIII−/− mouse 2 days after coronary ligation. Arrows: intrathoracic hematoma adjacent to experimental anterolateral infarction.
B: Autopsy confirms a blood clot (asterisk) originating from myocardial rupture at the border zone (arrow) of the myocardial infarct.
C: Histology of 1A shows rupture channel (arrows), filled with blood.
D: In patients with ruptured MI, FXIII levels were significantly reduced (*p<0.01).
E: Color Doppler echo of patient with new ventricular septum defect 7 days after myocardial infarction (arrow).
F: MRI after VSD repair with patch (arrows).
G–I: Explantation site of saphenous veins for CABG surgery displays delayed healing.
J: 73 days after initial surgery, 3 revisions and 2 weeks after i.v. FXIII augmentation, the wound is closed.
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