



Valvular Heart Disease

MITRAL VALVE CHORDAL RUPTURE IS ASSOCIATED WITH ALTERED ELASTIC FIBRIL ARCHITECTURE AND DIMINISHED ELASTIN DENSITY IN FIBRO ELASTIC DEFICIENCY: IMPLICATIONS IN LEAFLET STABILITY

Poster Contributions

Poster Sessions, Expo North

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Session Title: Evolving Concepts for the Optimal Management of Mitral Regurgitation from E-Clip to Robotics

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Background: Chordae rupture triggering severe mitral regurgitation is a life-threatening event requiring emergent surgery. Elastic fibrils within the chordae tendineae maintain the integrity and distensibility of the mitral leaflets. In Degenerative Mitral Valve Disease (DMVD), chordal rupture is more prevalent in Fibro Elastic Deficiency (FED) when compared to Barlow's Disease (BD). We tested the hypothesis that reduced elastin content and altered elastic fibril architecture may be more commonly seen in FED with chordal rupture when compared to BD without chordal rupture.

Methods: Twenty human posterior mitral valve leaflets were studied, 9 FED with rupture and 11-BD without rupture. Elastin protein density and architecture (elastic fibril fragmentation and disorganization), and valve morphology, were quantified using histochemistry and immunohistochemistry.

Results: Elastin protein density was reduced and fragmentation/disorganization increased in FED (Table and Figure).

Conclusion: Mitral valve chordal rupture is associated with diminished elastin protein density and altered elastic architecture in fibro elastic deficiency in DMVD. Future development of pharmacological or molecular technology preserving elastin protein content and architecture may have the potential to reduce chordae rupture, reducing the need for emergent surgery in DMVD.

Elastic Fibril Composition	FED with Chordal Rupture (n=9)	BD without Chordal Rupture (n=11)	p-Value
Elastin protein density	0.13 ± 0.01	0.31 ± 0.03	0.0001
Elastic fibril fragmentation	2.86 ± 0.04	1.98 ± 0.21	0.002
Elastic fibril disorganization	2.71 ± 0.07	1.95 ± 0.2	0.003

