Clinical Study

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Mechanisms of Ventricular Fibrillation in Sudden Cardiac Death

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ABSTRACT

A concept has been formulated regarding the mechanisms of the pathogenesis of ventricular fibrillation in sudden cardiac death. A predisposing factor is hereditary deficiency of cell adhesion proteins in the intercalated discs of cardiomyocytes. Impairment of coronary circulation leading to myocardial ischemia/reperfusion causes hypercontraction of cardiomyocytes due to uncontrolled calcium ion influx into the sarcoplasm. Due to the insufficiency of intercalated discs, uncoupling (dissociation) of cardiomyocytes occurs, disrupting the transmission of the electrical impulse through the intercalated disc. This is accompanied by electrical instability of the myocardium and leads to ventricular fibrillation.

Keywords: Cardiac death, Cardiomyocytes, Sarcoplasm, Coronary circulation

Abbreviations: VF: Ventricular Fibrillation; ACM: Arrhythmogenic Cardiomyopathy

1. Clinical Study

Ventricular Fibrillation (VF) is the leading mechanism of cardiac arrest in sudden cardiac death. The factors provoking VF are myocardial ischemia/reperfusion due to impaired coronary circulation caused by coronary atherosclerosis. As well as electrical instability of the myocardium resulting from disrupted conduction of the electrical impulse through the intercalated discs of cardiomyocytes[1]. Intercalated discs are the point of uncoupling (dissociation) of cardiomyocytes under conditions of myocardial ischemia/reperfusion, which is morphologically clearly documented by fragmentation of muscle fibers (Figure 1).

Dissociation of cardiomyocytes is caused by myocardial ischemia/reperfusion, leading to an uncontrolled influx of calcium ions into the sarcoplasm. This results in hypercontraction of cardiomyocytes, which is morphologically manifested as contracture damage (Figure 2).

The dissociation of cardiomyocytes is facilitated by the disruption of intercalated discs due to reduced levels of cell adhesion proteins (molecules) within them[2]. Intercalated discs are a complex system that includes desmosomes, adherens junctions and gap junctions[3]. Deficiency of cell adhesion proteins in intercalated discs is most often caused by a hereditary (genetic) factor. A striking example of such a pathology is

Arrhythmogenic Cardiomyopathy (ACM), in which a deficiency of desmosomal proteins in intercalated discs is observed due to mutations in the genes encoding them (Table 1).

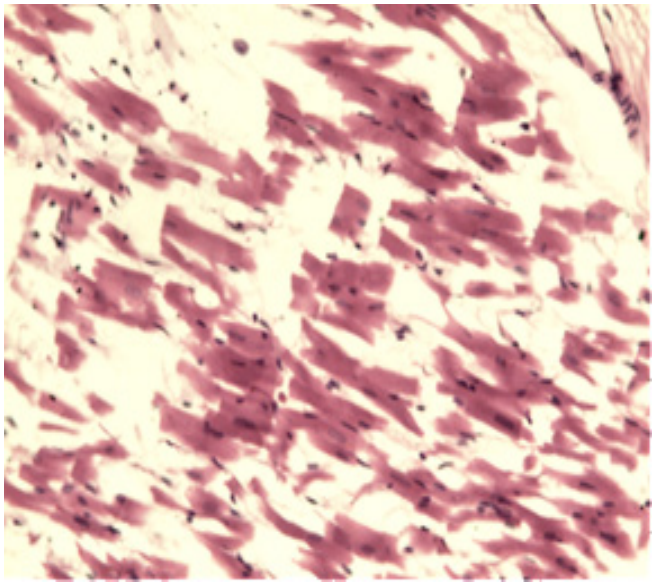


Figure 1: Dissociation of cardiomyocytes in VF. Hematoxylin and eosin stain, x 100.

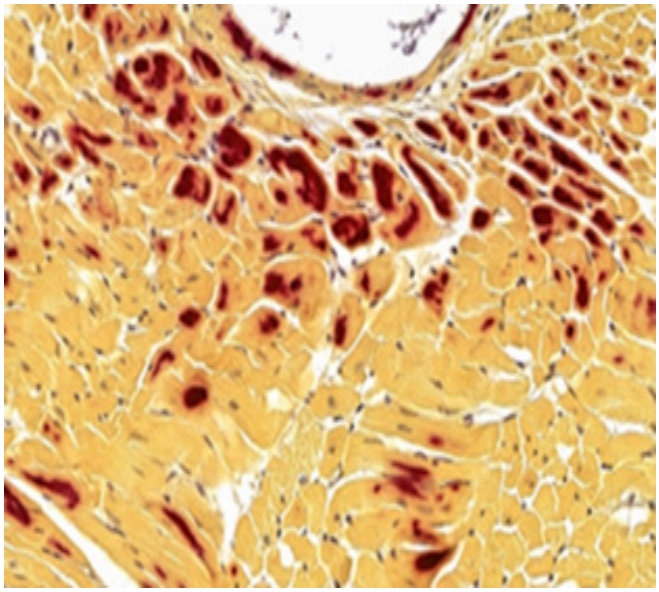


Figure 2: Contracture damage of cardiomyocytes. Lie stain, x 100.

Table 1: Gene mutations in ACM[4].

Gene	Encoded Protein	Subcellular Localization	Chromosomal Locus
JUP	Junction plakoglobin	Desmosome	17q21.2
DSP	Desmoplakin	Desmosome	6p24.3
PKP2	Plakophilin-2	Desmosome	12p11.21
DSG2	Desmoglein-2	Desmosome	18q12.1
DSC2	Desmocollin-2	Desmosome	18q12.1

Immunofluorescence showed the disappearance of cell adhesion proteins - plakoglobin and connexin-43 - from the intercalated discs of patients with arrhythmogenic cardiomyopathy (Figure 3)[5].

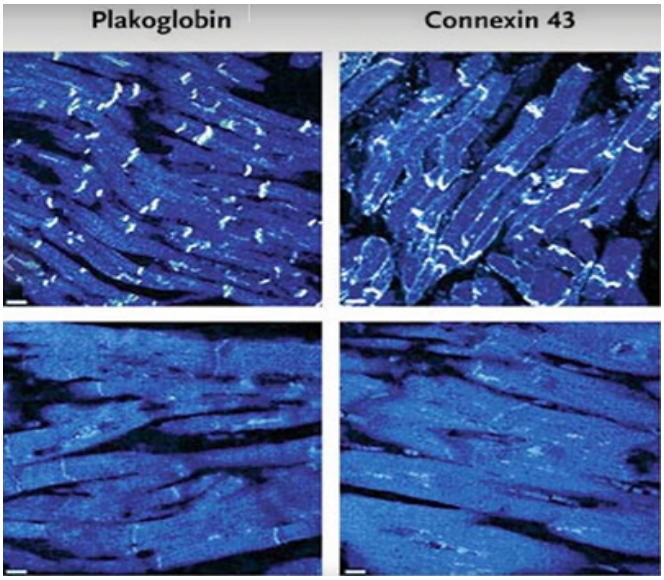


Figure 3: Disappearance of plakoglobin and connexin-43 from intercalated discs in ACM. Immunofluorescence[5].

Along with mutations of desmin protein genes in ACM, destruction of intercalated discs with widening of their gap and destruction of adjacent myofilaments has been revealed (Figure 4)[6].

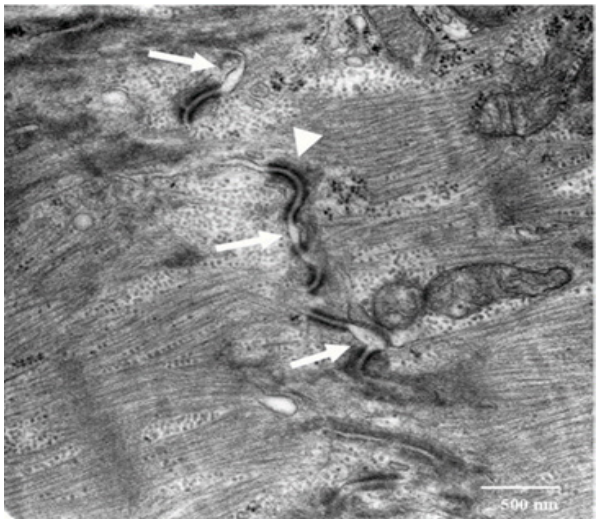
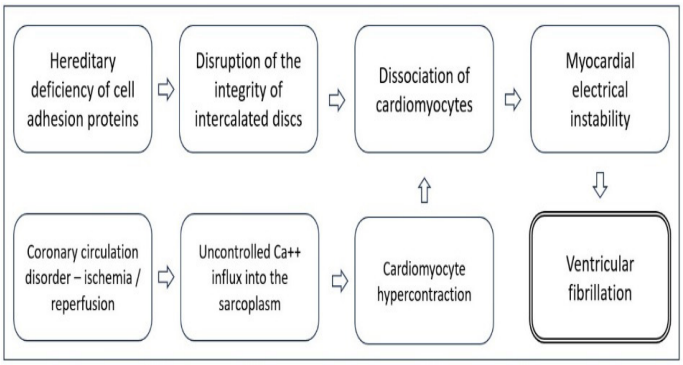


Figure 4: Ultrastructural damage to intercalated discs in ACM. Focal widening of the adherens junction gap (arrows), areas of myofilament destruction[6].

Thus, insufficiency of intercalated discs caused by a genetically determined deficiency of cell adhesion proteins predisposes to the development of VF. During myocardial ischemia/reperfusion, uncoupling of intercellular contacts occurs in the region of intercalated discs, which leads to disruption of electrical impulse transmission, resulting in electrical instability of the myocardium and VF. The last one is clearly documented morphologically as dissociation of cardiomyocytes. Individuals with hereditary deficiency of cell adhesion proteins constitute a risk group for sudden cardiac death. A fatal outcome may never occur throughout the patient's life if they do not experience coronary circulation disorders that cause ischemia/reperfusion. Reperfusion under the prolonged, irreversible myocardial ischemia plays a significant trigger role in the development of VF, as the restoration of blood flow to ischemic cardiomyocytes with damaged sarcolemma leads to an uncontrolled influx of calcium

ions (Ca++) into the sarcoplasm of the cardiomyocytes, causing their hypercontraction. Against the background of defective intercalated discs, this results in uncoupling (dissociation) of cardiomyocytes, culminating in VF.

This chat can be represented in the form of the following scheme



Chat 1: Mechanisms of Ventricular Fibrillation.

2. Conclusion

Hereditary deficiency of cell adhesion proteins in the intercalated discs of cardiomyocytes is a risk factor for ventricular fibrillation of the heart in sudden cardiac death. Impaired circulation in the coronary arteries of the heart with the development of ischemia/reperfusion is accompanied by an uncontrolled influx of calcium ions into cardiomyocytes, their hypercontraction and in individuals at risk - by uncoupling (dissociation) of cardiomyocytes, resulting in ventricular fibrillation and sudden cardiac death.

3. References

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