Histopathological Findings of Myocardial Tissue in Congestive Heart Failure, Myocardial Infarction and Cardiac Arrest

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INTRODUCTION

- Coronary arteries are responsible for perfusing the myocardium with oxygen rich blood.
- Based on bodily requirements, these vital arteries can quickly adjust oxygen delivery to the myocardium to meet demand. Coronary arteries can become narrowed by pathologies such as atherosclerosis leading to ischemic heart disease.
- The most common form of IHD is coronary artery disease.
- With over 17.9 million lives lost annually, CAD remains the leading cause of death.
- CAD progressively develops overtime due to the formation of atherosclerotic plaques.
- These plaques can eventually rupture and result in complete occlusion of the coronary artery leading to a myocardial infarction, a severe and life-threatening condition.
    - Treatment includes immediate medical intervention including percutaneous coronary interventions.

Methods

- Using cadaveric specimens, hematoxylin and eosin staining was performed on myocardial tissue.
- Was subsequently analyzed via microscopy
- Specimens were collected from myocardial tissue that had varying pathologies including the following:
    - Cardiac arrest
    - Congestive heart failure
    - Myocardial infarction.
- Tissue was collected from various regions of the myocardium including the following:
    - Apex
    - Right atrium

RESULTS

- Microscopic analysis revealed that extensive changes occur to the myocardium follow ischemic and non-ischemic insult [Figures 1-4].

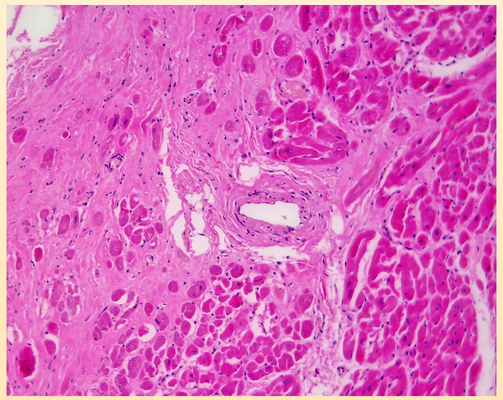

Figure 1: Myocardial apex tissue demonstrating myocardial fibrosis from a possible myocardial infarction.

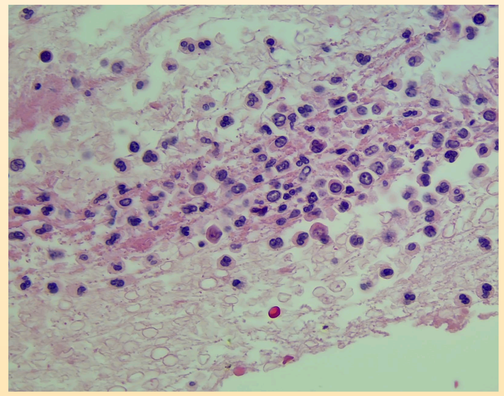

Figure 2: Myocardial tissue highlighting inflammatory cells from a previous myocardial infarction.

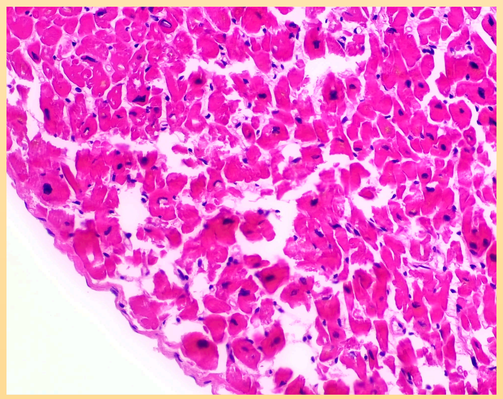

Figure 3: Myocardial apex tissue revealing patchy hypertrophy of myocytes secondary to long-standing congestive heart failure.

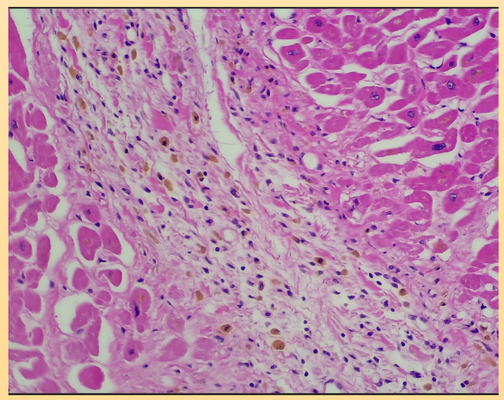

Figure 4: Myocardial tissue revealing patchy granulation tissue surrounding myocytes secondary to long-standing congestive heart failure along with cardiac arrest.

CONCLUSION

- The histopathological findings of these pathologies reveals several key features including
    - Inflammatory cell migration
    - Fibroblast activation resulting in subsequent fibrosis
    - Hypertrophy of myocytes
    - Deposition of granulation tissue
- The histopathological changes that occur during a myocardial infarction can be broken down into immediate and late changes
    - Acute changes include the following:
        - Wavy myocytes – 30 minutes to 4 hours
        - Early coagulation necrosis and hemorrhaging – 4 – 12 hours
    - Subacute changes include:
        - Neutrophilic infiltrate – 1 to 3 days
        - Macrophage migration – 3 to 7 days
- Other pathologies including heart failure have been shown to result in myocardial hypertrophy
- The hypertrophy arises as a result of increased physiologic demands on the heart
- The hypertrophy can be broken down into type subtypes
    - Concentric hypertrophy
    - Eccentric hypertrophy
- Overall, this analysis demonstrates the histopathological findings that are common following insults to the myocardium.

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