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Case Report

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Healing Myocarditis from a Hypertrophied Heart with Multifocal Fibrosis Mimicking Cardiomyopathy

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Abstract

We report the case of an 18 year old man who unexpectedly died of healing myocarditis. His heart was hypertrophied with multifocal fibrosis which can be a common histological feature of primary and secondary cardiomyopathy as well as the healing phase of myocarditis. However, the pattern of myocardial fibrosis, inflammation with myonecrosis, sparing of the right ventricular myocardium, and cardiomyocytes features in the remaining areas of the heart were considered as the key elements in determining a diagnosis of myocarditis. This case illustrates that meticulous histologic examination and the analysis of the histologic findings in the hypertrophied heart with multifocal fibrosis can be helpful to make a correct diagnosis.

Keywords: Myocardial fibrosis, Forensic pathology, Ventricular arrhythmia, Subepicardial fibrosis, Cardiomyopathy.

Introduction

Sudden cardiac death is one of the most common causes of death in the practice of forensic pathology. It refers to an unexpected and sudden death where sudden cessation of cardiac activity occurred with the hemodynamic collapse due to ventricular tachycardia, or ventricular fibrillation [1]. This ventricular arrhythmia usually occurs in the setting of an underlying myocardial disease, which can lead to uneven or a disorganized depolarization and repolarization [2,3].

Sudden cardiac death in young adults is an important issue for public health and safety in order to prevent premature death. Myocarditis and cardiomyopathy including hypertrophic cardiomyopathy and arrhythmogenic ventricular cardiomyopathy are the most common underlying myocardial diseases resulting in sudden deaths in this group [4]. Since myocarditis is ongoing into the healed or healing myocarditis; it is difficult to distinguish it from primary cardiomyopathy. Although, in most cases of myocarditis, the role for vaccination and infection control in preventing myocarditis is unknown.

However, vaccination may be effective in preventing some of viral myocarditis [5]. As it can be genetic, it is also important to confirm the specific type of primary cardiomyopathy for the deceased's family and recommend further management. Therefore, we presented a case of a sudden death in a young man with a hypertrophied heart and multifocal fibrosis, which was determined to be healing myocarditis that was distinguished from primary cardiomyopathy.

Case Report

The deceased was an 18-year-old male who was found dead unexpectedly in his bed by his father. When the paramedics arrived clear signs of death and postmortem changes such as livor mortis and rigor mortis were observed. The deceased was healthy, without a significant past medical history and was not known to use illicit drugs or consume significant quantities of alcohol. On external examination, lividity was present on the front of the body. There were no injuries or needle marks. On internal examination, the heart appeared enlarged, globular and somewhat flabby, and sectioning revealed multifocal fibrosis. No blood or effusions were identified within the unremarkable pericardium. There was no evidence of significant atherosclerotic luminal stenosis on the coronary arteries. There were no significant findings on any other internal organs.

The microscopic examination showed subacute lymphocytic myocarditis with evidence of early secondary cardiomyopathic changes; moderate to marked interstitial and replacement-type fibrous tissue deposition in a band-like distribution within the mid myocardial and subepicardial third in the left ventricular wall and the interventricular septum. Moderate to marked lymphocytic infiltrates associated with myonecrosis and cardiomyocyte dropout in the interventricular septum as well as scattered and minute lymphocytic infiltrates in the free wall. Only a focal and minimal interstitial fibrous tissue deposition was noted in the right ventricular myocardium. There were no significant pathological findings in the other organs (**Figure 1**).

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Figure 1: Macroscopic view of a horizontal section of the heart with myocardial fibrosis showing a band-like distribution within the mid myocardial and subepicardial third in left ventricular wall and interventricular septum.

A nasopharyngeal swab was performed for viral studies and Respiratory Syncytial Virus B was detected by nucleic acid amplification test. Toxicological testing showed negative result for drugs and alcohol. Genetic testing was performed and was negative for the cardiomyopathy panel.

Discussion

This case describes the key differential histological findings for a diagnosis of healing myocarditis in a hypertrophied heart with multifocal fibrosis, distinguished from cardiomyopathy. The main macroscopic findings at autopsy showed hypertrophied heart (enlargement of right atrial chamber and both ventricular chambers) with multifocal fibrosis which is sufficient enough to explain a sudden cardiac death. Given that the deceased was young and there was no significant past medical history, primary cardiomyopathy can be considered initially. However, microscopically there were interstitial and replacement-type fibrous tissue deposition in the left ventricular wall, only focal minimal increase in interstitial fibrous tissue deposition is noted in the right ventricular wall, and lymphocytic infiltrates with myonecrosis and cardiomyocyte dropout in the interventricular septum. Therefore, these findings were considered as a reasonable basis to determine a diagnosis of healing myocarditis (Figure 2).

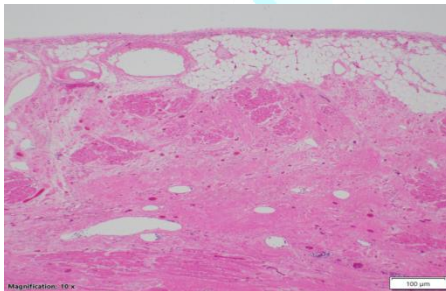


Figure 2: Low power microscopic view (Magnification 10 x 1.25) from left ventricle showing subepicardial fibrosis including moderate to marked interstitial and replacement-type fibrous tissue deposition in a band-like distribution within the mid myocardial and subepicardial third in left ventricular wall.

A hypertrophied heart usually presents macroscopically with a four-chamber dilation and cardiomegaly (beyond normal weight), and microscopically with myocyte hypertrophy and myocardial fibrosis, even though in some subsets of cardiomyopathies, the heart weight can be within normal limits [6,7]. Hypertrophied heart with multifocal fibrosis is a nonspecific pathologic finding, which can be primary or secondary cardiomyopathies. In terms of the diagnosis of secondary

cardiomyopathies, the past history of underlying diseases or drugs, macroscopic and microscopic findings of other organs can be helpful. In this case, there was no significant history, and no pathologic findings were observed in other organs. Etiologically, primary cardiomyopathies can be grouped into three categories which are genetic, mixed, and acquired. Given that the deceased was a young man without any significant history, hypertrophic or arrhythmogenic ventricular cardiomyopathy and myocarditis can also be considered as plausible differential diagnoses. There was no myofiber disarray observed in the multiple sections of the heart which is a pathognomonic finding in hypertrophic cardiomyopathy (Figure 3).

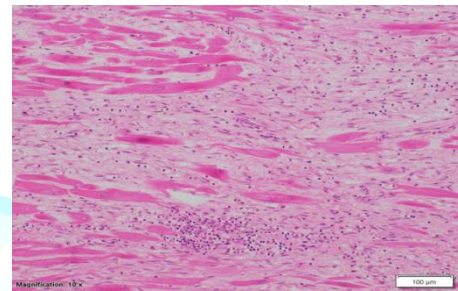


Figure 3: Medium power microscopic view (Magnification 10 x 10) from left ventricle showing myocardial fibrosis and chronic inflammation composed of moderate to marked lymphocytic infiltrates.

Acute myocarditis typically reveals an acute inflammation, which histologic findings should show various components of inflammatory cells depending on each type of myocarditis. However, myocarditis typically evolves through healing or healed stages, which characterizes myocardial fibrosis and myocyte hypertrophy. These findings can mimic arrhythmogenic ventricular cardiomyopathy, which may complicate a diagnosis, or may be impossible to distinguish, especially if there is subepicardial fibrosis in the left ventricle with fat infiltration [8-10]. Our case also revealed myocardial fibrosis in the subepicardial third of the left ventricular myocardium (Figure 4).

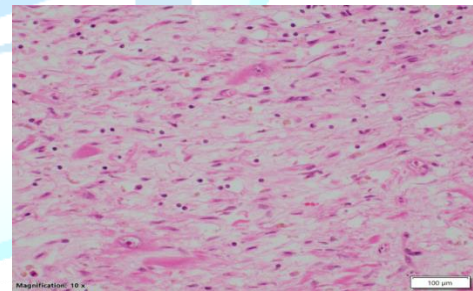


Figure 4: High power microscopic view (Magnification 10 x 20) from left ventricle showing moderate to marked interstitial and replacement-type fibrous tissue deposition and moderate to marked lymphocytic infiltrates associated with myonecrosis and cardiomyocyte dropout in the interventricular septum, scattered and minute lymphocytic infiltrates in the free wall.

However, arrhythmogenic ventricular cardiomyopathy typically reveals fibrofatty replacement with marked myocyte hypertrophy and myocardial degeneration, in random distribution in the right and left ventricular myocardium [5]. In our case, lymphocytic infiltrates with myonecrosis was identified on the muscular septum. Myocardial fibrosis was identified multifocally in the left ventricular wall and within the interventricular septum. It was not a fibrofatty replacement and it showed a band-like distribution within the mid myocardium as well as the subepicardial third of the left ventricle, rather than a random distribution. Only a focal, minimal increase in interstitial fibrous tissue deposition was noted within the right ventricular myocardium.



In addition, the remaining area of the left ventricle, which is spared from fibrosis, revealed just mild myocyte hypertrophy. This band-like pattern of myocardial fibrosis, sparing the right ventricle, inflammatory infiltrates with myonecrosis, and mild myocyte hypertrophy in the remaining myocardium strongly supported healing myocarditis.

Conclusion

We have described a case of a sudden cardiac death in a young man with healing myocarditis. Primary cardiomyopathy and myocarditis can be considered in a young man with a hypertrophied heart with multifocal fibrosis. It is especially hard to distinguish ongoing myocarditis from arrhythmogenic ventricular cardiomyopathy. The pattern of myocardial fibrosis and its distribution, presence of inflammatory cell infiltrates with myonecrosis, and insignificant features of the remaining myocardium can be the key elements to make a diagnosis of healed or healing myocarditis.

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