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Original Article

Histological changes of myocardium in dilated cardiomyopathy

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ABSTRACT

Introduction: For patients with acute dilated cardiomyopathy, definition of prognosis and of clinical features predictive of outcome is particularly important due to the availability of cardiac transplantation and other innovative treatment strategies. Exact prevalence of DCM in India is not known. The disease is reported to be more prevalent and aggressive in Blacks and in females, while a few other reports had shown a preponderance of males.
Methods: The present study was undertaken on 60 heart samples, out of which 10 heart samples were enlarged and dilated compared to the control samples. The heart muscle samples were dissected and stained for microscopic evaluation.
Result: The heart tissue affected with dilated cardiomyopathy showed variation in myocyte size, transmural scars, fibrofatty change, fibrosis and deposition of collagen fibres, as compared to the normal heart tissue,
Discussion: Dilated cardiomyopathy leads to heart failure which can cause death. So, it is important to know the changes occurring in a heart due to dilated cardiomyopathy for the prognosis and further treatment for the disease. The finding ranges from minimal change to many other variation in the myocytes which is discussed further.

1. Introduction

Cardiomyopathyrepresents a diverse group of heart muscle disorders, which are further subdivided on the basis of their anatomic and hemodynamic findings. More than 80% of cardiomyopathies are classified as dilated or congestive.[1] These disorders increase both myocardial mass and volume, such that, despite moderate myocyte enlargement, or hypertrophy, the heart appears thin walled and distended[1] Dilated cardiomyopathy (DCM) is a disease of the cardiac muscle, characterized by dilatation and impaired contractility of the left or both ventricles, with progressive development of congestive heart failure (CHF) and occurrence of serious arrythmiologic events.[2] Diminished contractile function is the critical hemodynamic feature of dilated cardiomyopathy, an abnormality that triggers complex neurohumoral responses, which increase circulatory volume so as to maintain cardiac output. Although such events are initially compensatory, these responses ultimately become maladaptive and contribute to clinical deterioration and onset of heart failure. Only 50% of patients with dilated cardiomyopathy survive 15 years after diagnosis[2]; premature death occurs from unmitigated pump failure and from co-morbidities such as thromboembolic events and arrhythmias. Despite current strategies to aggressively manage dilated cardiomyopathy, the disorder remains a common cause of heart failure and a prevalent diagnosis in individuals referred for cardiac transplantation.[3]

A great variety of factors (toxic, infectious, metabolic, immunologic etc.) have been etiologically implicated in DCM. This correlation is of great importance, as myocardial damage may be reversible in some of these cases. However, DCM is often characterized, as "idiopathic", when no etiologic factor is revealed.[4]

The main objective of our study is to evaluate histological changes of heart muscles in patients with dilated cardiomyopathy in North-Indian population.

2. Material and method

The Control and Dilated cardiomyopathy heart samples were collected from the Department of Anatomy, Institute of Medical Sciences, Banaras Hindu University, Varanasi (U.P). A morphologic examination of myocardial tissue was performed in these 60 heart

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samples. cardiac tissue samples from each heart for histological study were stained with Haematoxylin and Eosin staining and Masson's Trichrome.

Dissection of the heart

Firstly the heart samples were properly washed with water and then the left ventricular chambers were opened by giving a longitudinal incision starting from left auricle up to the apex of heart. Clots were removed and the chambers were washed under running tap-water.

To open the left atrium, incision was made from pulmonary vein opening up to the left auricle. For right atrium, incision was given from superior vena cava opening up to the right auricle and for right ventricle; incision was further extended beyond the right auricle up to the apex of heart. Again the heart chambers were properly washed under running tap-water to remove any clots. The heart samples were preserved in 10% formalin. Thereafter a small tissue sample from the left ventricular wall was taken and subjected them for Haematoxylin and Eosin staining and Masson's Trichrome staining.[5]

The histological examination was carried out at a magnification of 10X, 40X and 100X ***(Nikon, Japan)***, to observe the the cardiomyocytes and other tissues of heart muscle.

3. Result

On histology examination branching cardiomyocytes were seen in cardiac muscle, where in the nucleus was located centrally. Few cardiac muscles were bi-nucleated. Intercalated discs were seen. Individual cardiac muscles were covered with the connective tissue endomysium (Fig. 1A).

The histological features of dilated cardiomyopathy: the findings ranged from minimal variation in myocyte size to typical features of myofiber loss, interstitial fibrosis (Fig. 2A and B), and marked variation in myofiber size. In hearts, the findings were diffuse findings of myocytes, including variation in size, nuclear variation (Fig. 1B) and interstitial fibrosis (Fig. 2A). Interstitial and replacement fibrosis were also common (Figs. 2–4). Occasionally, there was fibrofatty change. Transmural scars were also observed in dilated cardiomyopathy.

Quantitation of collagen showed four times the normal collagen concentration, with a decrease in mature cross-linked collagen. Few of the myocytes were hypertrophied, as well as atrophied (Fig. 4). The volume density of myofibrils is reduced, and mitochondrial density is normal, but the mitochondria were more numerous and small (Figs. 1–4).

Histologic examination of myocardial biopsy samples and necropsy material has revealed marked disorganization of myocardial fibers (Fig. 4) (involving at least 5% of the tissue surface) in about 95% of patients with DCM (This cellular disarray involves >25% of myocardium in 50% and >50% of the tissue in 25% of the patients and was characterized by oblique and perpendicular arrangement of adjacent muscle fibers. Myofiber disorganization in DCM was not necessarily confined to the most hypertrophied LV segments. Indeed, there was little correlation between wall thickness and extent of cellular disorganization. Thus, cellular disorganization involves an average of 40% of the ventricular septum and 33% of the LV free wall despite differences in the magnitude of hypertrophy in these regions.

Masson's Trichrome staining of heart tissue (DCM) demonstrated the fibrosis and deposition of collagen fibres which was absent in Controls (Fig. 3).

4. Discussion

Defined by ventricular dilation and diminished contractile function, dilated cardiomyopathy is a prevalent world-wide disorder that is estimated to affect 36.5 per 100,000 individuals.[6] DCM results in heart failure, serious arrhythmias and thromboembolic events which ultimately proves fatal for the affected individual. The pathologic manifestations of dilated cardiomyopathy are often nonspecific. Although cardiac mass is increased, there is often only modest ventricular wall hypertrophy while atrial and ventricular chambers can be mildly or markedly distended. Microscopic examination may reveal no abnormalities or evidence of abnormal histopathology in the myocardium such as myocyte hypertrophy, myocyte degeneration, and increased interstitial fibrosis. Unlike hypertrophic cardiomyopathy, substantial distortions of cell architecture or myocyte disarray are not features of dilated cardiomyopathy.[7]

Signs and symptoms in the early stages of DCM are vague and the affected person shows symptoms of easy fatigue, dyspnoea or palpitations. Further deterioration in contractile function and progression toward heart failure or onset of atrial and ventricular arrhythmias worsens symptoms. Diagnosis of dilated cardiomyopathy is based on the finding of increased cardiac systolic and diastolic dimensions with diminished contractile function. When underlying causes such as coronary artery disease, chronic alcohol abuse, thyroid disease, or viral infection are excluded as etiologies, a diagnosis is often made of idiopathic dilated cardiomyopathy.[7]

The histologic features of dilated cardiomyopathy: the findings range from minimal variation in myocyte size to typical features of myofiber loss, interstitial fibrosis and marked variation in myofiber size. In hearts, the findings were diffuse findings of myocytes, including variation in size, nuclear variation and interstitial fibrosis. Interstitial and replacement fibrosis are also common. Occasionally, there can be fibrofatty change.

5. Conclusion

DCM is the most common cardiomyopathy, occurring due to multiple factors, including long-standing hypertension, ischaemic heart disease, infection and sarcoidosis.[8] For a doctor, it is very

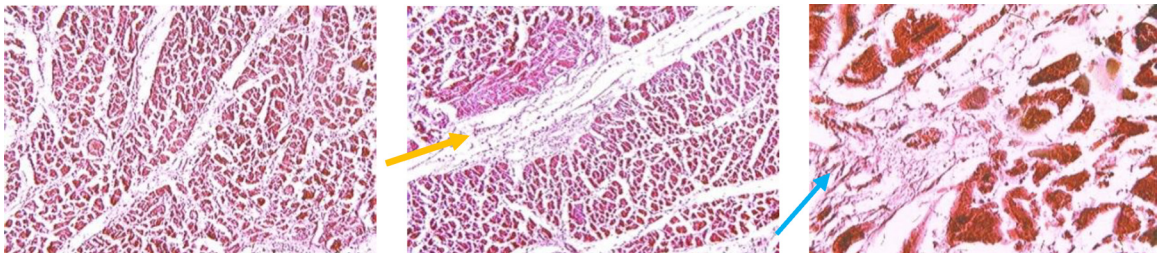

Fig. 1. (A). H & E staining 100X of heart tissue (Control) shows normal architecture. (B). H & E staining 100X of heart tissue (DCM) shows evidence of fibrosis (orange arrow). (C). H & E staining 400X of heart tissue (DCM) shows the deposition of collagen fibres (blue arrow).

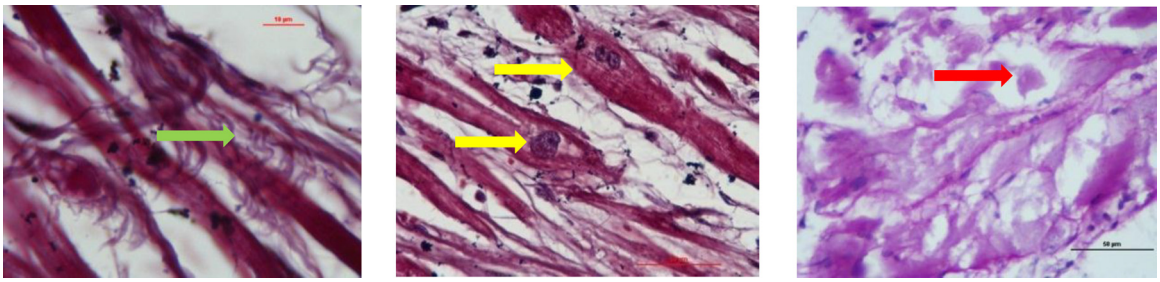

Fig. 2. (A). Photomicrospic heart section from cardiac explants in a patient with end-stage cardiomyopathy. There is focal interstitial fibrosis (green arrow). (B). variation in nuclear size (yellow arrow). The change is nonspecific and can be seen in heart failure from any cause. (C). the intracellular accumulation of amorphous material (basophilic degeneration) (red arrow). The change is nonspecific and can be seen in heart failure from any cause.

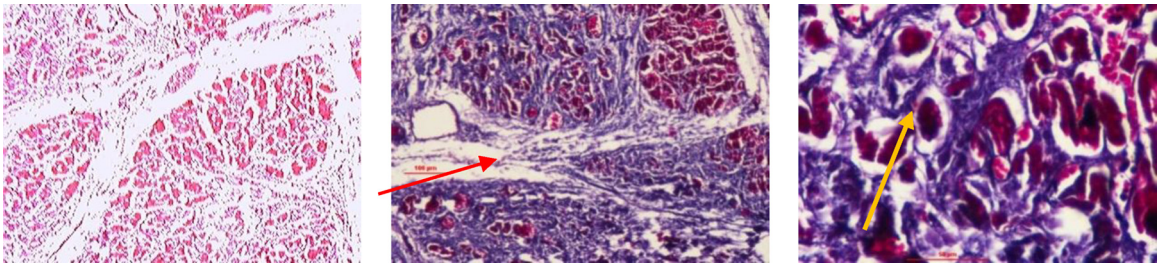

Fig. 3. (A). Masson's Trichrome staining 100X of heart tissue (Control) shows normal architecture. (B). Masson's Trichrome staining 100X of heart tissue (DCM) shows evidence of fibrosis (Red arrow). (C). Masson's Trichrome staining 200X of heart tissue (DCM) shows deposition of collagen fibres (Orange arrow).

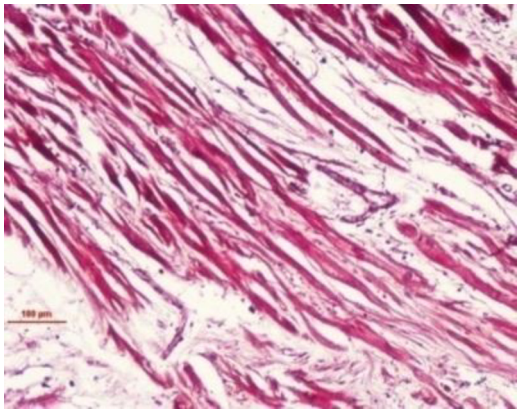

Fig. 4. The histology of dilated cardiomyopathy heart shows hypertrophy and degeneration of myocytes (dark red) without disarray. Increases in interstitial fibrosis (pale pink) are evident.

important to differentiate between familial DCM, idiopathic DCM and the other aetiologies, since management differs for each. Mainstay of the treatment is early intervention and accurate diagnosis.

Histological features can be used to identify patients at particularly high risk for death. It also helps know the progress of the disease and the treatment mode for dilated cardiomyopathy.

Once this is fully understood, new and improved therapeutic strategies will be developed to combat these diseases which are responsible for death associated with dilated cardiomyopathy. Focusing research efforts on the cascade of events involved in development of a cardiomyopathic disorder or its final common pathway should ultimately lead to a longer and healthier life for all.

Conflict of Interest

There is no conflict of interest to be declared by authors.

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