Histological Changes in Parietal Pericardium In Dysfunctional Ischemic Myocardial Diseases

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Abstract

Parietal and visceral pericardium have a limited response to the damage that is initially manifested as an increase in production of pericardial fluid. The leak might be a transudate that mostly contains thin liquid or an exudate that contains large amount of fibrin and variable number of inflammatory cells. Acute pericarditis is often diagnosed 1 to 4 days after the myocardial infarction, and is sometimes accompanied by pericardial exudate.

The aim of the study was to analyze the histological changes in parietal pericardium following the partial pericardiectomy before the surgical intervention in patients with cardiac coronary diseases and with aortic or mitral valve stenosis.

The study was implemented at University Hospital Clinical Centre of Kosovo, using the parietal pericardium biopsy material, obtained in partial pericardiectomy in patients following the bypass surgery and implantation of artificial heart valves. The study included 50 cases of patients diagnosed with coronary artery diseases and aortic or mitral valve stenosis.

Histopathology analysis of tissue samples was performed at Institute of Pathology, Faculty of Medicine, Prishtina. Morphometric analysis was performed using the Leica stereological measurement system. The assessment of dependence of occurrences that are not measurable is shown in contingency table. The reliability of the variance is assessed to be on the level of $p< 0.05$ and higher.

The data obtained show that the initial inflammatory process is present in pericardium in acute coronary disease, the presence of infiltrative inflammatory cells is observed such as granulocytes, lymphocytes, fibrin deposits and increase in hyperactive, hyperplastic, reactive mesothelial cells. Fibrosis is not present. Reparatory changes in parietal pericardium are mild, fresh and hemoliyzed erythrocytes, calcifications, neovascularization and granulation tissue are rarely observed. The mean thickness of pericardium is 2.4 mm, whereas referral values vary up to 2 mm. There is increase in mesothelial cells which are also hyperplastic, and have increased subeptielial and fibrotic reaction.

In patients with coronary artery disease histological changes in parietal pericardium are accompanied with the initial informatory process, the presence of the infiltrative inflammatory cells granulocytes and lymphocytes, fibrin deposits and increase in reactive hyperplastic mesothelial cells. Fibrosis is not present. The mean thickness of pericardium was 2.4 mm. There was increase in mesothelial cells which were also hyperplastic, and had increased subepithelial, organizational and fibrotic reaction. Reparatory changes in parietal pericardium are mild, fresh and hemoliyzed erythrocytes, calcifications, neovascularization and granulation tissue are rarely observed.

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Introduction

Pericardium is a double sack. Externally it consists of parietal pericardium, which is a fibrotic layer of the connective tissue that is rich in elastic and collagen fibers. The fibrosis is supplied by the network of blood and lymphatic vessels that contain macrophages and fibroblasts.

Visceral pericardium is a single serosa layer consisting of flat, irregular mesothelial cells with thin basal membrane, separated from the fibrotic layer with the thin sub-mesothelial space.[1,2] These two layers of pericardium are 1-2 mm thick and form a cavity that contains approximately 15 to 35 ml of pericardial fluid in normal physiological circumstances. Pericardial fluid is formed by ultrafiltration of plasma and mostly contains globular proteins, phospholipids, prostaglandins and surfaktants.[3,4]

Pericardial disease is a common disorder that might be the result of an infectious and non-infectious disease and may manifest as pericarditis with or without leak. Etiology of pericarditis may be viral, bacterial, fungal, uremic, post- acute myocardial infraction, post cardiac neoplastic surgeries, following the radiation of mediastinum, and the consequence of autoimmune systemic diseases.[5,6]

Parietal and visceral pericardium have a limited response to the damage that is initially manifested as increase of pericardial fluid production. The leak might be a transudate that mostly consists of a thin liquid or and exudate that contains large amount of fibrin and variable number of inflammatory cells.[7,8]

Many researchers attempted to link the noninvasive diagnosis of the increased thickening of the pericardium with the pathological findings using computerized tomography (CT), particularly ultrafast and Chinese CT. MRI was mostly used to assess the pericardial thickens. The thickening may be better observed when linked to reasonable pathology findings.[9-12]

Perforation of cardiac wall by the catheter may lead to pericardial reaction similar to the one caused by cardiac surgery. Pressure or penetrating traumas may cause pericardial fibrillary reaction that in some cases may advance to constriction.[13]

Molecular mechanisms of inflammatory and immune damages which are mediated during pericarditis and mechanisms of constrictive pericarditis progression are not entirely understood.[14]

Acute pericarditis is often diagnosed 1-4 after myocardial infarction, and is sometimes accompanied by pericardial exudate. Vascular damages and myocardial necrosis suggest inflammatory damage response. Fibrotic deposits and adhesions are often found in inner and parietal pericardium that covers the infarcted area, but also may include the larger and more spread surface of pericardium.[15-18]

Fibrotic cascade of events is caused by edema of epithelial or endothelial cells that results in initiation of coagulation process. The initial inflammatory response is characterized with release of various pro-inflammatory cytokines, including tumor necrosis factor-α, TNF-α.[19,20]

The loss of architecture of mesothelial cells and mesothelial desquamation is often accompanied by constrictive pericarditis. Mesothelial cells might be subject to phenotypical changes similar to epithelial- mesenchymal transition in order to adopt morphology and fibroblast functioning in healing serosa.[21] Active regulation of pro-inflammatory and anti-inflammatory mediators by mesothelial cells suggests an essential role of the cells in maintaining pericardial homeostasis and also in pericardial fibrosis pathogenesis. Pericardial interstitial cells (PICs) are also involved in producing of extracellular matrix (ECM) and calcification in pericardium.[22]

The aim of the study

The aim of the study was to analyze the histological changes in parietal pericardium following the partial pericardiectomy before the surgical intervention in patients with cardiac coronary diseases and with aortic or mitral valve stenosis. Inflammatory changes, fibrin deposits, and hyperplastic mesothelial cells, pericardial reparative and regenerative changes were found in parietal pericardium. Stereometric analysis of parietal pericardial wall was also performed in the same samples.

Materials and methods

The study was implemented at University Hospital Clinical Centre of Kosovo, using the parietal pericarditis biopsy material, obtained in partial pericardiectomy, in patients following the bypass surgery and implantation of artificial heart valves. The study included 50 cases of patients diagnosed with coronary artery diseases and valvular or mitral stenosis.

The samples of pericardium are fixated in 10% buffered formalin. Histopathology analysis, routine colouring using haematoxylin eosin was

performed at Institute of Pathology, Faculty of Medicine, Prishtina. Morphometric analysis was performed using the stereological measurement system. Leica, after morphometry.

Statistical analysis of the obtained results was performed using the computer program SPSS version 16 For Windows, GrapgPad Instat 3 and Microsoft Excel. Basic analysis of statistical data was performed by determining arithmetic mean value, standard deviation and medians for numerical values provided. The assessment of dependence of occurrences that are not measurable is assessed using the contingency chart. The reliability of the variance is assessed to be 0.05 and higher.

Results

Chart 1 shows the cases in relation to pathology of the pericardial diseases based on the age in 50 examined cases. The youngest age is 22, the oldest age is 80. The arithmetical mean value is 61.4.

Table 1 shows the case studies by gender, 60% of the cases were males and 20% females. There is no statistically important significance in relation to the gender in pericardial diseases (p=0.157).

Table 2 shows the histology examination of pericardium after the partial pericardiectomy in patients with coronary artery diseases (CAD) and other coronary diseases (aortic and mitral stenosis).

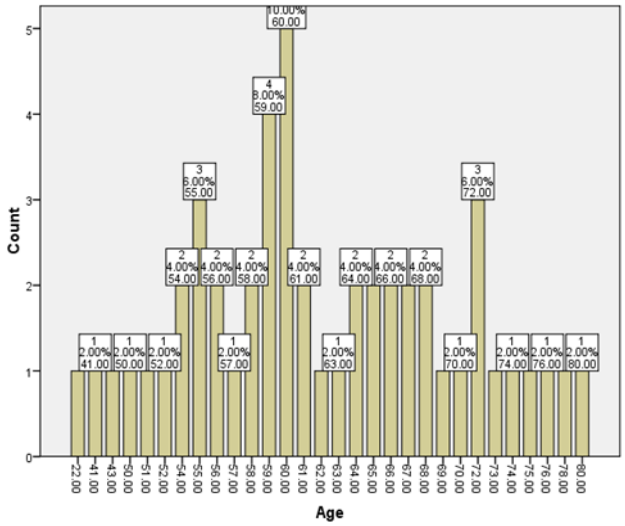

Chart 1. Presents the Frequency of the Cases of Pericardial Diseases by Age.

The data in the table shows that 72% of cases are with coronary artery disease that underwent the bypass surgery, whereas 28% underwent the surgery due to aortic and mitral valve stenosis. The statistical significance is observed in the studied group with coronary artery disease (p=0.0018).

Gender	Cases	%	p value
Male	30	60	0.157
Female	20	40	

Table 1. The Pericardial Disease Cases Studies Based on Gender (No.50).

	Cases	%	p value
Present	36	72	0.0018
Not present	14	28	

Table 2. Presents the Number of Cases Studied with Pericarditis with Coronary Artery Diseases and Other Coronary Diseases.

The presented data show that the inflammatory process of the initial organization stage is present in histology investigation of the pericardium and the presence of infiltrative inflammatory cells such as granulocytes, lymphocytes, fibrin deposits and increase in reactive hyperplastic mesothelial cells is observed. Fibrosis is not present.

	Present		Not present		P value
Histology	Cases	%	Cases	%	
Acute inflammation	21	42.0	29	52.0	P=0.258
Acute fibrillary inflammation	16	32.0	34	68.0	P=0.019
Chronic inflammation	43	86.0	4	14.0	P=0.001
Chronic fibrillary inflammation	19	38.0	31	62.0	P=0.089
Reactive mesothelial cells	35	70.0	15	30.0	P=0.005
Fibrillary with reactive mesothelial cells	27	54.0	23	46.0	P=5.716

Table 3. Shows the Cases Where Histology Investigation of Parietal Pericardium is Performed, Based on Domination of the Type of Inflammatory Infiltrative Cells, Fibrillary Exudate and Hyperplasia of Mesothelial Cells.

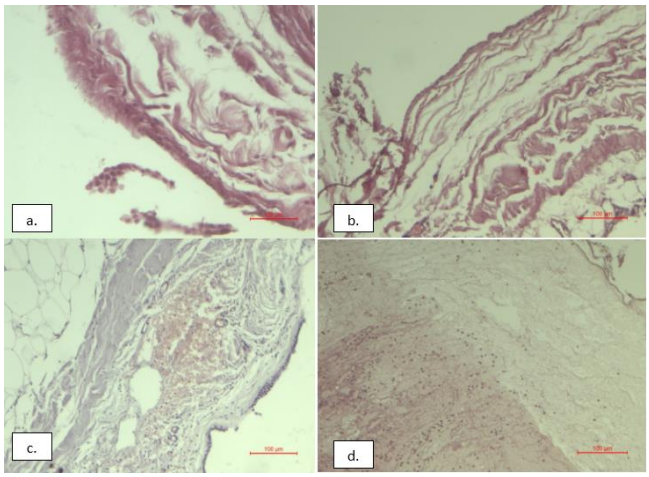

Figure 1. Shows Histology Findings of Pericardium: (a). Hyperplasia of Mesothelial Cells (H&E, 200x), (b). Loose Reactive Subepithelium (H&E, 200x), (c). Subepithelial Extravasal Erythrocytes (H&E, 200x), (d). Fibrillary Deposits and Mixed Inflammatory Infiltrates (H&E, 400x).

The data presented in this table show that reparatory changes in parietal pericardium are mild, fresh and destroyed erythrocytes, calcifications, neo vascularization and granulation tissue are rarely observed.

	Present		Not present		
Histology	Cases	%	Cases	%	P value
Erythrocytes	16	32.0	34	68.0	P=0.010
Calcifications	5	10.0	45	90.0	P=0.001
Neo vascularization	10	20.0	40	80.0	P=0.002
Granulation tissue	2	4.0	48	96.0	P=0.001

Table 4. Shows the Regenerative and Reparative Changes of the Pericardium in Coronary Artery Disease.

Table 5 shows stereomethric histology measurements of parietal pericardium based on histology layers, starting from the thickness of the wall, epithelium, subepithelium, fibrosa and adipose tissue.

Measurements of pericardium in micron	Mean±SEM	p value
Pericardium thickness	2409.0±141.72	p>0.10
Thickness of the pericardium epithelium	16.87±5.37	p<0.001
Thickness of the pericardium sub epithelium	669.26±248.49	p<0.001
Thickness of the pericardium fibrosa	741.09±76.55	p<0.008
Thickness of the pericardium adipose tissue	1127.40±89.78	p=0.0255

Table 5. Stereometric Findings in Parietal Pericardium of the Cases Studied (Mean±SEM).

The data obtained show that the mean thickens of pericardium is 2.4 mm, whereas the referent values are approximately up to 2 mm. There is an increased number of mesothelial cells which are hyperplastic and have increased subepthelial fibrotic reaction.

Chart 2 shows the pericardial thickness with sterometric measurement of the studied cases.

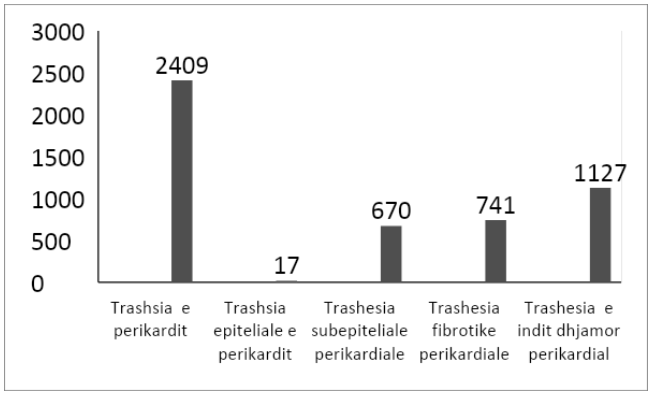

Chart 2. The thickness of the pericardium wall based on histology layers (microns)

Discussion

The fibrin leak is the most common finding regardless of the source of the damage: chemical (uremic, pharmaceutical), physical (open heart surgery, therapeutic ablation,

radiation) or infectious (viral, bacterial, fungal, parasites). As soon as fibrin or fibrinous-hemorrhagic exudate occurs there is usually inflammatory response that would "clean" fibrillary waste. During this stage, the inflammatory cells incite neo vascularization and early deposit of extracellular matrix. If the damaging stimulation ends, the result is usually soft fibrosis. If the damaging stimulation continues pericardium response is extended in relation to exudation, inflammation and reparation process.

If the there is no other damaging stimulus, inflammatory cells within the fibrin exudate incite neo vascularization and spreading of fibroblasts. Extracellular matrix increases and when it matures, the free granulation index becomes organized with more mature fibrous tissue, while neo vascularization and chronic inflammation become less visible.

If the damaging stimulus that caused exudate does not occur again, the healing process finally leads to maturation of granulation tissue into the dense fibrotic layer.[8]

In our study, the histology of examined pericardium after the partial pericardiectomy in patients with coronary artery disease (CAD) and other coronary diseases (aortic and mitral stenosis) it was revealed that 72% of cases are with coronary artery disease that underwent bypass surgery, whereas 28% underwent surgery due to aortic and mitral valve stenosis. The statistical significance is observed in the studied group with coronary artery disease ($p = 0.0018$).

Parietal pericardium is thick because of the dense fibrotic tissue that does not show fibroblasts, inflammatory infiltrates or neo vascularization. Pericardium has normal thickness (< or = 2 mm). The most common causes are previous cardiac surgeries, chest radiation, infract and idiopathic diseases. Microscopically earlier studies show that there was no patient with entirely normal pericardium. Pathological histology findings were mild and focal, including fibrosis, inflammation, calcification, fibrin deposits and non caseous focal granuloams.[23]

From the data presented it is observed that the initial stage of inflammatory process is present in histological investigation of pericardium and the presence of inflammatory infiltrates such as granulocytes, lymphocytes and fibrin deposits is observed and increase of hyperplastic reactive mesothelial cells. Fibrosis is not present.

Moncada et al., used CT to examine pericardium in 15 patients with the symptoms suggesting pericarditis, whose samples show thickened pericardium. Isner *et al*., studied 7 patients with constriction or limiting physiology and verified that 4 had pericarditis on CT (4 mm). Isner *et al*., studied 34 patients with suspected pericardial disease; 9 had pericardial thickening on CT that was verified with tissue analysis. Small number of studies shown a correlation between the normal thickness of the pericardium in radiography images and the thickness in pathology examinations. Oren et al., reported 12 patients with hemodynamics results showing constrictive pericarditis, 5 had pericardial thickness in CT and tissue analysis, 7 had normal thickness of pericardium in both exams. Except for CT and MRI, echocardiography was used to assess the pericardial thickness. However, echocardiography results depend more from operator's technique.[9-12,24]

Data obtained show that the mean value of pericardial thickness is 2.4 mm, whereas referent values are up to 2 mm. There is increase in mesothelial cells which are also hyperplastic and have increased subepthielial and fibrotic reaction.

It is clear that the data suggests the benefit of these replacement techniques for assessment of the thickening of the pericardium. The findings confirmed the concept that constrictive pericarditis may occur in patients with normal thickness of pericardium. In fact, in this study, the pericardial thickness was normal in 18% of patients with evidence of surgery in constrictive pericarditis. Histology view was irregular in all patients with constriction and normal thickness of pericardium, however the changes were mild or focal.[10,12,24]

The blood in the pericardial space in damaged pericardium may lead to fiber pericardial adhesions. The isolated injury of the pericardium serosa in rabbits without bleeding is accompanied only by single fibrillary reaction; if the blood is injected after the damage of the serosa, it may result in pericardial adhesions. Fibrin deposits only on pericardial surface seem not to cause permanent reaction.

Parietal pericardium may contain to inner visceral pericardium at the place of acute

transmural infarct in mice. If the adhesion around the heart occurs before the rupture of the left ventricular wall (due to infarct), bleeding into the space around the heart may result in false aneurism.[25-28]

The data obtained from this study show that reparatory changes in parietal pericardium are mild, fresh and hemoliyzed erythrocytes, calcifications, neovascularization and granulation tissue are rarely observed.

Today cardiac surgery might be the most common cause of pericardial heart diseases. "Rubbed off" pericardial mesothelial cells incite the fibrinous reaction. When the damaged pericardium is healed, the fibrin disappears and two layers of pericardium remain adhered by fiber adhesions. Despite these adhesions, parietal pericardium is rarely thick due to surgical intervention. Certainly the blood and its products enter into pericardial space in every cardiac surgery and the organization of products of the blood may be responsible for fibrotic pericarditis.[25]

Dressler (DS) syndrome usually occurs approximately two weeks after the myocardial infraction. It is unusual presentation of the early reperfusion therapy with thrombolytic therapy and primary percutaneous intervention and with widespread use of heparin. It seems that DS is a repetitive immune-inflammatory syndrome that occurs following the release of auto-myocardial antigens due to necrosis of myocardial tissue. Creation of immunology complexes is believed to cause an oversensitivity reaction due to molecular mimicking and interactions. In reality, the presence of increasing number of antymiocardial antibodies following the injury of the myocardium has been suggested before and supports the possible autoimmune pathogenesis.[29,30,31]

More visible pathological features of the constrictive pericarditis are inflammation and fibrotic thickening of the thin and elastic pericardial layer. Pericardium usually has the inflammatory areas of serosa and fibro – calcification.[13]

The transition accompanied by the "activity" of mesothelial cells and a special enzymatic profile of the cells with the functions focused towards oxidative stress and inflammatory responses has been recently studied. The activated mesothelial cells transport chemokines and adhesion molecules to help in recruitment and migration of leukocytes through mesothelium. They are also known for intermediating the inflammatory process and for producing the ECM components.[2,32,33]

PIC (pericardial interstitial cells) have an immune phenotype comparable to mesenchymal stem cells. PIC cultivated from the human fibrotic samples may differentiate into myofibroblasts and osteoblasts that are essential for fibrosis development and production of extrasceletal calcification. TGF-β and bone morphogenetic protein 2 (BMP-2) is accompanied by the trans-differentiation process. TGF-β increases the expression of PIC mRNA of collagens I and III by lowering the level of matrix metalloprotease-2 and -9 mRNA that are significant for elastin degradation, thus regulating fibrotic process and modulating the genetic expression related to fibrosis.[22]

The main cellular intermediator of fibrosis is myofibroblast which when activated serves as primary cell of collagen production. Myofibroblasts are generated by the range of sources, including mesenchymal resident cells, epithelial and endothelial cells in the so called process of epithelial/ endothelial- mesenchymal transition (EMT/EndMT), and by fibroblast circulation cells named fibrocytes that originate from bone marrow stem cells.

Conclusion

In our study the examined histological cases of pericardium following the partial pericardiectomy in patients with coronary artery diseases (CAD) and other coronary diseases (aortic and mitral stenosis), it is observed that the initial stage of inflammatory process is present in the histology of the pericardium, the presence of infiltrative inflammatory cells is observed such as granulocytes, lymphocytes, fibrin deposits and increase in hyperactive, hyperplastic, reactive mesothelial cells. Fibrosis was not present. The mean value of the thickness was 2.4mm. There was an increase of mesothelial cells which were hyperplastic and with increased subepithelial and fibrotic reaction. Reparatory changes in parietal pericardium are mild, fresh and hemoliyzed erythrocytes, calcifications, neovascularization and granulation tissue were rarely observed.

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