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**MINISTRY OF HEALTH OF THE REPUBLIC OF MOLDOVA  
PUBLIC INSTITUTION  
STATE UNIVERSITY OF MEDICINE AND PHARMACY  
«NICOLAE TESTEMITANU»**

**Viorica OCHIȘOR. Valeriu REVENCO**

# **CHRONIC PERICARDIAL DISEASES**

**Guidelines**

**Chișinău  
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CARDIOLOGY  
DEPARTMENT OF INTERNAL MEDICINE

Viorica OCHIȘOR. Valeriu REVENCO

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Intended for students, medical residents.

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## LIST OF ABBREVIATIONS

CK-MB	–	creatininkinaze MB fraction
CT	–	computed tomography
DNA	–	desoxyribonucleic acid
ECG	–	Electrocardiography
EchoCG	–	Echocardiography
ESR	–	erythrocyte sedimentation rate
IAS	–	interatrial septum
IVS	–	interventricular septum
LA	–	left atrium
LDH	–	lactate dehydrogenase
LV	–	left ventricle
MRI	–	magnetic resonance imaging
MV	–	mitral valve
NSAIDs	–	non-steroidal antiinflammatory drugs
PA	–	pulmonary artery
PCR	–	C reactive protein
RA	–	right atrium
RNA	–	ribonucleic acid
RV	–	right ventricle



## DEFINITION AND CLASSIFICATION OF PERICARDITIS

Pericardial diseases encompass an extremely wide range of conditions which involve the pericardium, isolatedly appeared or associated to other heart or systemic pathologies.

Pericarditis represents the inflammation of the pericardium and can be acute, recurrent or chronic.

### **Clinical forms of pericardial diseases**

1. Acute pericarditis
  - Fibrinous pericarditis
  - Exudative pericarditis
2. Cardiac tamponade
3. Chronic pericarditis
  - Exudative pericarditis
  - Effusive-constrictive pericarditis
  - Constrictive pericarditis
4. Other pericardial diseases
  - Chylopericardium
  - Pericardial cyst
  - Congenital pericardial absence

## CHRONIC PERICARDITIS

Chronic pericarditis (duration > 3 months) includes the following forms:

1. Exudative (inflammatory, hydropericardium in heart failure)
2. Effusive-constrictive
3. Constrictive

### **Chronic exudative pericarditis**

#### ***Definition***

Pericarditis is considered *chronic*, if the pericardial effusion persists more than three months regardless of its etiology.

Pericardial effusion is frequently asymptomatic or oligosymptomatic and can be diagnosed by chance during a routine clinical, radiological or echocardiographic examination. In other cases the etiology is diagnosed previously, during the course of acute pericarditis.

If chronic pericarditis is associated with hydrothorax and/or ascites, then congestive heart failure, hepatic cirrhosis, nephrotic syndrome and other polyserositis are to be considered.

Pericardial effusion may be located or circumferential. In chronic pericarditis the symptomatology is exhibited in dependance of the amount of collected pericardial effusion, level of cardiac compression and presence of pericardial inflammation.

If pericarditis is of symptomatic evolution, the patient may show general fatigue, precordial chest pain, palpitations.

**Diagnosis** will be established on base of anamnestic data, clinical symptoms and paraclinical data. Echocardiographic data are of major importance.

The evaluation of chronic pericarditis etiology is required, possibly by performing of pericardiocentesis, pericardioscopy and/or pericardial biopsy. Diagnostic algorithm of chronic pericarditis is similar to that of acute pericarditis.

The determination of chronic pericarditis causes (for example myxedema, tuberculosis, toxoplasmosis, autoimmune and systemic diseases etc.) assures an efficient treatment.

### **Treatment**

*Therapeutic strategy* includes symptomatic and etiologic medication similar to the one in acute pericarditis.

#### 1. Nonsteroidal anti-inflammatory drugs

*Ibuprofen* is preferred considering its rare side effects and favourable influence on the coronary flow. Doses depend upon pain severity and response to treatment. 300-800 mg are administered every 6-8 hours for several days or weeks, until the effusion disappears.

Indometacine is not indicated in the elderly due to its effect of coronary flow reduction.

2. *Acetylsalicylic acid* can be administered - 300 - 600 mg every 4- 6 hours (both are administered with gastric protection).

3. *Colchicine* 0,5 mg bid is administered in monotherapy or in association with NSAIDs in acute episodes, as well as in the prevention of recurrences.

4. Systemic corticosteroid therapy – is indicated in connective tissue diseases, autoreactive or uremic pericarditis.

*Prednison* 60mg/day for 2 days with a gradual decrease of doses during a week. Nevertheless, corticosteroid therapy may favourize recurrences. In autoreactive forms intrapericardial instillation of corticosteroids in the form of non-absorbable crystalloids is very efficient.

5. Anticoagulants when needed *Heparine* (under strict observation).

6. *Pericardiocentesis* is indicated in therapeutic purpose for: cardiac tamponade, suspicion of purulent neoplastic pericarditis or in the case of large or symptomatic effusions, despite the received medical treatment. Pericardiocentesis guided by fluoroscopy is performed in the cardiac catheterisation laboratory with ECG monitoring. The drainage of fluid will be done cautiously in steps with the removal of less than 1 liter, in order to avoid acute dilation of the right ventricle.

7. *Surgical drainage* is preferred in traumatic haemopericardium and purulent pericarditis.

8. *Pericardiectomy* is indicated for etiologic diagnosis and in patients with tamponade, in presence of pericardial effusion maintenance or creation of constriction phenomenon.

In the case of symptomatic recurrences *balloon pericardiotomy* or *pleuro-pericardial fenestration* are to be considered.

In persistant /recurrent chronic effusions *pericardiectomy* may be performed if intrapericardial therapy is not efficient.

**Effusive – constrictive pericarditis** referres to the association of pericardial effusion with heart constriction at the level of the visceral pericardial layer.

### ***Etiology***

The most frequent causes of effusive-constrictive pericarditis are idiopathic, tuberculous, neoplastic, postradiation, connective tissue diseases, bacterial infections.

Regarding the initial haemodynamic aspect, in diastole, prior to pericardiocentesis performance, right atrial pressure waveform shows a prominent „x” descent and absence of „y” descent. This aspect is similar to the waveforms in cardiac tamponade. After pericardial fluid drainage, an additional „y” descent to the existing „x” descent appears on the right atrial pressure waveform. In the right and left ventricular waveform appears an initial descent in diastole, followed by a diastolic plateau. Central venous pressure is evidently increased.

**Clinical features** may have an acute onset with fever, pericardial friction rub and chest pain, and after several weeks-months constrictive pericarditis like symptoms will occur: dyspnea, turgescient jugulars, hepatomegaly, periferal edema. In contrast with constrictive pericarditis, the presence of pulsus paradoxus is determined more frequently, and more rarely pericardial knock and Kussmaul's sign.

### **Paraclinic investigations**

*Electrocardiography* shows changes of ST segment and T-waves. Low QRS voltage is determined.

*Chest X-ray* marks an enlargement of the cardiac shadow and sometimes pericardial calcifications.

*Echocardiography* may determine pericardial effusion, pericardial thickening and RA colapse.

At *Computed tomography* the presence of pericardial effusion and pericardial thickening may be noted.

The diagnosis will be established through the determination of venous pressure prior to pericardial puncture and after the evacuation of fluid. Maintenance of elevated venous pressure after pericardiocentesis is explained by the association of constriction. In effusive-constrictive pericarditis there are tamponade-like pressures at the level of the RA, RV and LV before the evacuation of pericardial fluid. After the evacuation of pericardial fluid the pericardium looks constricted.

In order to appreciate the etiologic diagnosis of effusive-constrictive pericarditis the analysis of pericardial fluid and pericardial biopsy is required. The efficiency of pericardiocentesis is of short-term due to the presence of constriction.

### **Treatment**

It consists in a total pericardectomy of the parietal and visceral layers in case of constriction. Additionally, symptomatic and etiologic therapy is administered.

### **Constrictive pericarditis**

Constrictive pericarditis represents a severe complication of a rare frequency, which implies fibrosis formation, calcifications and adhesions between parietal and visceral layers of the pericardium, inducing deficient ventricular filling and ventricular failure.

Pericardial fibrosis may develop rapidly, due to an acute etiologic factor, possibly subacute, in several months or it may develop gradually

over years, causing diastolic heart failure with elevated venous pressure, hepatomegaly, edema and ascites.

### *Etiology*

Constrictive pericarditis may develop after any pericarditis. The most frequent etiologies are:

- idiopathic
- postradiation
- tuberculous
- postpericardiotomy
- infectious (bacterial, viral infections)
- neoplastic
- autoimmune (connective tissue diseases)
- uremic
- post-traumatic
- sarcoidosis
- irritation of mediastinum.

### *Morphopahtology and physiopathology*

It has an onset with acute or silent evolution, followed by fibrinous accumulated material associated frequently with pericardial fluid. As a result of fibrinous effusion resorbition, fibrinous material is formed, and proliferation of scar connection tissue with zonal obliteration of the pericardial cavity. Initially the formed adhesions are delicate, easily detachable, but later, they become solid and difficult to separate. Therefore, constrictive pericarditis implies marked fibrinous thinckening of the pericardium, which later becomes rigid and sometimes calcified. Fibrosis process occurs in the visceral and parietal pericardial layers, with symetric extension to the level of all cavities, followed by a partial or total obliteration of the pericardial cavity.

These pericardial changes usually affect diastolic filling, sometimes systolic contraction and lead to characteristic hemodynamic anomalies. Constrictive pericarditis is a symetric process, but there are cases of strictly located constriction. Located forms of constrictive pericarditis determine particular haemodynamic aspects.

Haemodynamics in constrictive pericarditis is influenced by important restriction of ventricular diastolic filling with the increase and equalization of filling pressures.

In the beginning of diastole the filling of cavities is accelerated, due to increased venous pressures, with an abrupt stop, when the intracardiac volume reaches the distensibility limit of fibrous pericardium. In this way, ventricular filling phenomenon occurs early in the initial third of diastole. In the RV and LV the so-called „dip and plateau” sign occurs. There are two „x” negative waves during ejection at the RA level and jugular veins and a very prominent „y” negative wave, which correspond to protodiastole, with a M or W aspect.

Systolic pressure in the pulmonary artery is moderately increased (<40 mmHg), the ejection fraction is normal (50-70%), but in cases of inflammation function and myocardial fibrosis, it may diminish due to contractive function. LV filling is reduced in inspiration, which determines increase of RV filling and left movement of the interventricular septum. Reverse changes develop in inspiration.

Compression of coronary arteries, myocardial fibrosis and atrophy association leading to myocardial retraction is possible.

**Clinical features** are dyspnea, general fatigue, signs of heart failure, mainly right one, normal heart dimensions, lack of pulmonary changes in a patient not suffering from some other cardiovascular pathology (coronary artery disease, valvulopathy), but with history of acute pericarditis.

As a rule, there is a long delay between the initial pericardial inflammation and the onset of constriction. Effort dyspnea, orthopnea, cough, general fatigue, weight loss reduction, flatulence and abdominal pain, peripheral edema are more frequent. In more advanced stages, systemic congestion, hepatomegaly, pleural effusions, ascites and anasarca, painful hepatomegaly, palmar erythema and subclinical jaundice develop. Ventricular filling impairment results in reduced cardiac output leading to muscular weakness, fatigue, weight loss, cachexy. Hydrosaline retention is determined by cardiac output reduction, elevated systemic venous pressure and inhibition of atrial natriuretic peptide production.

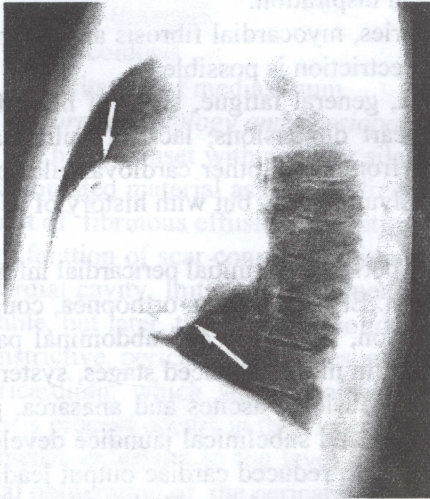
Physical examination detects jugular turgescence (raised venous pressure index), Kussmaul's sign (inspiratory distension of jugular veins caused by elevation of venous pressure in inspiration), low or normal arterial tension, sometimes presence of pulsus paradoxus, systolic retraction of the apical impulse, pericardial knock after the second heart sound (caused by the early stop of ventricular filling) and widened

splitting of the second sound (through fixed volume of the RV and premature closure of the aortic valve in inspiration, through reduction of the LV stroke volume). Atrial fibrillation and tricuspid regurgitary systolic flow murmur may be present.

#### *Electrocardiography*

Electrocardiographic record may be normal or registering low voltage QRS, changes of LA (record of jagged dilated p waves), flattened or diffusely negative T-waves, atrio-ventricular blocks, atrial fibrillation, more rarely pseudoinfarction changes ( Q waves).

*Chest roentgenography* detects a heart of normal or slightly enlarged dimensions, the superior mediastinum may be sometimes enlarged because of the superior vena cava, LA which may be dilated, pericardial calcifications, sometimes pleural effusion (fig. 1).



*Fig. 1.* Pericardial calcifications in constrictive pericarditis

*Echocardiography* determines pericardial thickening and other constriction-suggestive characteristics. *M mode and 2D Echocardiogram 2D examination* denotes pericardial thickening and presence of calcification in the region of LV posterior wall, early closure of MV, abnormal movement of the IVS, mesodiastolic flat movement of the LV free wall, protodiastolic abrupt posterior movement of the aortic wall, pulmonary valve premature opening, normal ventricles dimensions with preserved function and large atria, the

LV diameter which is not increasing after the early rapid filling phase, reduction of the angle formed by LA posterior wall junction/ LV posterior wall (below  $150^\circ$ ), IAS shift to LA during inspiration, dilation of inferior vena cava, hepatic veins without respiratory variations.



At *pulsed Doppler examination* dissociation between intrathoracic and intracardiac pressure and exaggerated ventricular interdependence in diastolic filling are determined:

- transmitral flow: high E wave, low A wave ( $E/A > 2$ ), reduced deceleration time (TDE)  $< 160$  ms; in inspiration E wave is reduced  $\geq 25\%$  vs basal index, isovolumetric relaxation time (IVRT) is prolonged by  $> 20\%$ ;
- transtricuspidal flow: E wave  $> A$ , E wave raises  $> 35\%$  in inspiration;
- flow in the pulmonary veins: D wave and prominent atrial reverse, more pronounced in expiration;
- flow in the hepatic veins: typical aspect in W, diastolic wave reduces in expiration and retrograde flow increases.

At *tissue Doppler examination*:

Aspect of normal compliance is present, but relaxation is altered: E' velocity at the mitral ring level remains normal or elevated;

In *colored M mode examination*, transmitral diastolic flow propagation speed is normal or elevated ( $\geq 100$  cm/s).

### ***Computed tomography and magnetic resonance imaging***

Computed tomography and magnetic resonance imaging at CT global or located pericardial thickening is noted (normal pericardium determined by TC is  $< 2$  mm; by MRI is  $\leq 4$  mm); pericardial calcifications, atrial dilation, stenosis of one or both atrio-ventricular grooves, tubular configuration of one or both ventricles, dilation of the cava veins, presence of ascites, pleural effusions. Pericardial thickening may be local or generalized. Through computed tomography may be determined the presence of myocardial atrophy described by: IVS thinning and of postero-lateral wall ( $< 1$  cm), reduction of wall thickening during the cardiac cycle ( $< 40\%$ ) and diminishing of muscular mass LV/LV telediastolic volume ( $< 1$ ).

Myocardial fibrosis is to be considered if the thickened/calcified pericardium is not separated of the myocardium through subepicardic fat and when the myocardial wall is thinned and shows undulant movement.

MRI examination determines ventricular filling anomalies in constrictive pericarditis comparative to restrictive cardiomyopathy. MRI



examination with gadolinium hypercontrast denotes pericardial inflammation.

#### *Laboratory analysis*

Hypoproteinemia, hypoalbuminemia, hyperglobulinemia, hyperbilirubinemia, altered hepatic functional tests, anemia are depicted in some cases.

#### *Cardiac catheterisation*

It determines pressure elevation in the RA (20mmHg), diastolic pressure in the RV, in pulmonary capillaries and diastolic pressure in the LV with its equalization (difference between telediastolic pressures in the LV and RV <5 mmHg). Moderate elevation of systolic pressures in the LV and PA (35-45 mmHg). Pulmonary hypertension is not characteristic of constrictive pericarditis in the absence of pre-existing pulmonary pathology or other cardiac disease. Fixed cardiac output (which is not effort induced) is distinctive. Reduction of the stroke flow with preserved relaxation cardiac output is due to compensatory tachycardia. Telesystolic and telediastolic volumes are normal or reduced.

#### *LV/RV Angiography*

It allows the determination of ventricular systolic and diastolic functions, shows reduction of ventricular dimensions, atrial dilation, rapid premature ventricular filling during diastole without the following increase („dip and plateau”), ventricular systolic function being normal in the absence of myocardial inflammation and fibrosis.

#### *Coronarography*

It is indicated prior to surgery in all patients to exclude coronary involvement especially in patients over 35 years or with history of mediastinal irradiation regardless of age. It can identify coronary arteries particularities (hypermobile septal coronary arteries and less mobile epicardial ones), increase of distance between the coronary arteries and cardiac silhouette.

*Myocardial biopsy* is informative in exclusion of restrictive cardiomyopathy, endomyocardial fibrosis or eosinophilic myocardial syndrome.

***Differential diagnosis*** of constrictive pericarditis will be done with: right heart failure of various etiologies (pulmonary embolism, RV myocardial infarction, tricuspidal valvulopathy, mitral stenosis, chronic pulmonary obstruction), cardiac tamponade, effusive-constrictive peri-

carditis, superior vena cava obstruction, RA myxoma, hypertrophic cardiomyopathy, nephrotic syndrome.

The most difficult differential diagnosis is the one with restrictive cardiomyopathy, which is similar to constrictive pericarditis clinically and haemodynamically.

Lower LV output, higher LV diastolic pressure towards RV diastolic pressure ( $> 5\text{mmHg}$ ), greater elevation of RV systolic pressure ( $>50\text{ mmHg}$ ) are in favour of restrictive cardiomyopathy.

Constrictive pericarditis **treatment** comprises the following:

- bed rest
- sodium restriction
- diuretics (for reduction of hydrosaline retention)
- Digoxin (in atrial fibrillation with rapid ventricular rate, for its reduction, but not below 80-90/min);

Beta-blockers and calcium channel blockers should be avoided due to sinus tachycardia which is a compensatory mechanism.

• In transitory forms of constrictive pericarditis, the symptomatology and haemodynamic anomalies disappear completely after around three months of medical treatment regardless of etiology. In these cases pericardectomy is not needed.

• Treatment of choice is complete pericardiectomy in the affected regions of the RV and LV, if necessary, with the extension to large vessels and ventricular grooves.

*Pericardectomy indications:*

- reduction of functional capacity associated with presence of fibrosis or extended pericardial calcifications
- haemodynamic consequences with clinical features (elevated jugular venous pressure, signs of hepatic failure, diuretic therapy necessity),
- echocardiographic ones and through cardiac catheterism.

*Pericardectomy contraindications:*

- constriction in an early stage (asymptomatic patients or those with functional class I (NYHA) heart failure
- transitory constriction
- fibrosis and/or extended myocardial atrophy determined by CT or MRI evaluation

- constriction in advanced stage (class IV NYHA- high surgical mortality).

### *Pericardectomy*

Performed in 2 standard approaches which supposes radical resection of the parietal and visceral pericardium if it is affected:

- antero-lateral thoracotomy or
- median sternotomy

Ultrasonic debridement or use of Excimer laser may be alternative or adjuvant to the surgical method.

Major complications of pericardectomy are perioperative heart failure and ventricular wall rupture.

Cardiac mortality and morbidity associated to pericardectomy are determined by presurgical presence of myocardial atrophy and fibrosis, not diagnosed on time.

Early postoperative mortality is caused by low cardiac output (in patients with prolonged extracorporeal circulation, difficult pericardial dissections), septicemia, haemorrhagies, respiratory and renal failure. The highest mortality rate is noted in patients with preoperative congestive heart failure (III-IV NYHA). Long-term prognosis is negatively influenced by history of post-radiation therapy, renal dysfunction, low LV ejection fraction, relatively increased systolic pressure in the pulmonary artery, hyponatremia, advanced age. A more favorable prognosis is seen in the case of pericardectomy performance at an earlier stage.

## **ETIOLOGIC FORMS OF PERICARDITIS**

### **Idiopathic pericarditis**

Idiopathic pericarditis is a form of pericarditis, the etiology of which has not been determined regardless the diagnostic investigations done. The incidence of idiopathic pericarditis is high (>50% according to some studies).

### **Viral pericarditis**

Viral pericarditis represents the most common cause of pericardial lesion due to direct viral attack or the immune response of the body (antiviral or anticardiac). Multiple types of viruses have been identified in the etiology of viral pericarditis. The most frequent ones are Echo-

and Coxsackie viruses. Most patients have a recent history of flu-like syndrome (with fever, cough, myalgia) or respiratory infection.

*Clinical features* are characterized by acute pericarditis, which normally regress in 1-3 weeks, more rarely it lasts for 3-6 weeks. Recurrences develop in 15-30% cases, in various time periods of up to 15 years.

*The definitive diagnosis* of viral pericarditis is not made without the evaluation of pericardial fluid and/or pericardial/epicardial tissue by PCR or *in situ* hybridisation. A fourfold elevation in viral antibodies level is suggestive, but it is not diagnostic for viral infection. Viral isolation in blood, pericardial fluid, stool is rarely possible.

*The treatment* of acute viral pericarditis is especially symptomatic (NSAIDs). In patients with chronic pericardial effusions or symptomatic recurrences with confirmed viral infection, specific treatment with *hyperimmunoglobulin* (cytomegalovirus pericarditis), *interferon* (Coxsackie B pericarditis), *immunoglobulins* (adenovirus and parvovirus B 19 perimyocarditis) is tested. In HIV patients, pericardial involvement is the most frequent cardiac manifestation of HIV. Pericardial effusions may be small and asymptomatic or generalized which extend to the peritonem and pleura. Large effusions develop in advanced stages of the disease. Asymptomatic patients with small, moderate pericardial effusions do not require any treatment, but large effusions and tamponade impose *pericardiocentesis* performance. Treatment with *corticosteroids* is *contraindicated*, except secondary tuberculous pericarditis, where these are add-on to tuberculostatic treatment.

**Bacterial pericarditis** represents a rare form of purulent exudative pericarditis in adults, always fatal if untreated.

*Causes* of bacterial pericarditis are pulmonary infections, septicemia, surgical interventions or thoracic traumatism, infectious endocarditis, esophageal lesions or fistula, sclerotherapy of esophageal varices etc. Pathogenic agents which may cause bacterial pericarditis are extremely variated. Predisposing factors depend upon the pathways of infectious penetration at the pericardial level, with the association of chronic pathologies, immunosuppression, burns, cytostatic treatment, radiation etc. In some cases bacterial pericarditis develops after heart, mediastinal surgery or chest trauma.

## *Diagnostics*

### *Clinical features*

Bacterial pericarditis is manifested by fever and chills, perspiration, dyspnea, precordial pain dependent on breathing and position, pericardial friction rub. It is complicated with cardiac tamponade in 30-40% of cases. It is accompanied by manifestations other septic as pneumonia, pleuritis, endocarditis, mediastinitis or arthritis are often associated.

*Echocardiographic examination* may denote encystation and septation of the pericardial effusion, changes of the cardiac and valvular cavities.

Chest *radiographic examination* may show the level of air and liquid.

*Laboratory analysis* are suggestive for a septic infection. Leukocytosis, elevated ESR, positive blood cultures are determined. Percutaneous pericardiocentesis is required urgently. The obtained pericardial fluid is turbid, with a mass of leukocytes, elevated proteins and LDH, reduced glucose values. The extracted fluid must be analysed by Gram, acid-fast and fungal staining, with the following sampling of pericardial fluid and blood cultures with antibiotic sensitivity testing.

### *Treatment*

Bacterial pericarditis requires percutaneous pericardiocentesis or surgical drainage with catheter maintenance for at least 3-4 days in dependence on the fluid amount.

Antibacterial systemic treatment is to be initiated fast. Intrapericardial administration of antibiotics is useful. Frequent rinsing of the pericardial cavity with streptokinase or urokinase may liquefy the purulent exudate, but in patients with thick purulent effusions, surgical drainage through subxiphoid approach is preferred.

Pericardectomy is indicated in patients with dense adhesions, thick purulent effusions which are local, recurrence of tamponade, progression to constriction.

In this form of pericarditis, the mortality remains high regardless the combined treatment.

### **Tuberculous pericarditis**

Tuberculous pericardial infection may be spread through blood, lymph and direct contact, the foci are frequently located in peritracheal,

peribronchial and mediastinal lymph nodes. The clinical picture is varied consisting of acute pericarditis with or without pericardial effusion, as well as cardiac tamponade, acute or chronic constrictive pericarditis or pericardial calcifications. It has a slow onset, with fever, perspiration, dyspnea, cough, moderate pain the anterior chest level.

### *Diagnosis*

Quiet cardiac sounds, pericardial friction rub, turgescient jugulars, hepatomegaly, ascites, edema are determined on physical examination.

Chest radiographic examination may sometimes denote old tuberculous pulmonary lesions, pleural effusion, cardiomegaly.

The diagnosis of tuberculous pericarditis can be made on the basis of history of pulmonary tuberculous infection, identification of *Mycobacterium tuberculosis* in the pericardial fluid and/or presence of specific tuberculous lesions are revealed by at pericardial biopsy (caseous granulomas).

The analysis of pericardial fluid shows high protein concentrations, elevated specific density, increased leukocytes, increased adenosine deaminase activity and elevated gamma interferon concentration or pericardial lyzozime. Pericardial biopsy reveals caseous granulomas or pathogen organisms. Positive intradermal tuberculin reaction is of limited value in adults. QuantiFERON-TB test quantifies interferon gamma as a marker of cell-mediated immunity activated in tuberculous infection.

Pericardiocentesis is indicated in diagnostic and therapeutic purposes. Pericardial biopsy allows a rapid diagnosis with a higher sensibility than pericardiocentesis.

*The treatment* should be initiated early, immediately as the etiologic diagnosis is established.

- *Four antituberculous drugs* are given for two months:

Rifampicin 600 mg/day

Isoniazid 300 mg/day

Pyrazinamide 15-30 mg/kg of body weight/day

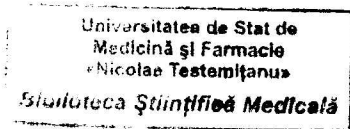
Ethambutol 15-25mg/kg of body weight/day,

- Then *two tuberculostatics* for 4 months

Rifampicin

Izoniazid

715758



The association of corticosteroids to treatment remains controversial. Tuberculostatic treatment combined with steroids accelerates the disappearance of symptoms, reaccumulation of pericardial fluid. If administered, prednisolone has to be in relatively high doses (1-2 mg/kg of body weight/day), 5-7 days, with progressive reduction in 6-8 weeks.

- *Pericardiectomy with drainage* is indicated in the case of tamponade recurrences in 4-6 weeks

- *Pericardectomy* is indicated in case of constriction or in effusive constrictive form.

Tuberculous pericarditis may regress to total resorption, evolve in chronic pericarditis or chronic pericardial constriction.

### **Fungal pericarditis**

Fungal pericarditis commonly occurs in immunocompromised persons, after heart surgery, fungal endocarditis in patients with valvular prosthesis, fungal pulmonary infections.

*Clinical picture* comprises all types of pericardial involvement, in some cases with the association of pulmonary, meningeal, myocardial and endocardial -valvular phenomena.

*Diagnosis* of fungal pericarditis is made on the basis of the analysis of culture, fluid and/or pericardial tissue histologic examination, complement fixation tests, precipitation tests and serum antifungal antibodies dosing.

*Antifungal treatment* is indicated in the case of documented fungal pericarditis, using ketoconazole, itraconazole, amphotericin B and liposomal amphotericin B. Corticosteroids and NSAIDs can be added to the treatment with antifungal medication. Pericardiocentesis is indicated in the case of cardiac tamponade occurrence. Fungal constrictive pericarditis will require pericardectomy.

Non-histoplasmosis fungal pericarditis prognosis is severe, with subsequent evolution to constrictive pericarditis.

In the course of histoplasmosis pericarditis does not need antifungal therapy, except disseminated histoplasmosis. It responds to treatment with NSAIDs administered for 2-12 weeks. Pericardiocentesis is necessary in large effusions or tamponade. The disease is benign in general.

Sulfonamides are the drugs of choice for nocardiosis. A combined treatment of three antibiotics including penicillin is to be given in actinomycosis.

### **Neoplastic pericarditis**

The most common causes of secondary malignant tumours are lung cancer, breast cancer, gastrointestinal carcinoma, malignant melanoma, sarcoma, lymphomas and leukemias. Primary malignant pericardial tumours occur relatively rarely. Metastasis at the pericardial level may occur by hematogenous or lymphatic spread. Neoplastic pericardial effusion is frequently hemorrhagic and may produce cardiac tamponade.

#### *Clinical manifestations*

Neoplastic pericarditis develops asymptotically, especially when the accumulation of the fluid is gradual.

The onset of symptoms - dyspnea, orthopnea, cough, chest pain, tachycardia, dysphagia, jugular turgescence, quiet heart sounds, fatigue is noted when the amount of fluid exceeds 500 ml. Pulsus paradoxus, hypotension, cardiogenic shock and paradoxical movement of jugular venous pulse are important signs of cardiac tamponade.

*The diagnosis* is made by the confirmation of the presence of malignant infiltration at the pericardial sac level.

In case of neoplasm, the pericardium involvement may be secondary to radiation, chemotherapy or infections. The diagnosis of neoplastic pericarditis is based on chest roentgenogram, echocardiography, CT and MRI, which reveal mediastinal widening, hilar masses and pleural effusion. Analyses of pericardial fluid, pericardial and epicardial biopsy show the presence of malignant cells and/or tumoral markers.

*Treatment* of neoplastic pericarditis implies the performance of pericardiocentesis that is indicated in cardiac tamponade.

In pericardial effusions without tamponade with suspected neoplasm the following is done:

- systemic antineoplastic treatment
- pericardiocentesis to relieve symptoms and make a diagnosis
- intrapericardial instillation of cytostatics/sclerosing agents
- pericardial drainage is to be done (if the technically possible) for all patients with large pericardial effusions
- prevention of recurrences may be achieved by intrapericardial instillation of cytostatics (Cisplatin, Thiotepa), sclerosing



agents (Tetracycline, Doxycycline, Bleomycin) or immunomodulators.

- Radiation therapy is very efficient in the control of malignant pericardial effusions in patients with radiosensitive tumours such as lymphomas and leukemias.
- In case pericardiocentesis cannot be performed, subxiphoid pericardiotomy is indicated.
- In recurrent pericarditis not responding to the above-mentioned strategies and with a favorable prognosis, a pleuro-pericardial direct connection may be performed by percutaneous balloon pericardiotomy (however it takes a risk of neoplastic dissemination) or extensive pericardectomy.

### **Pericarditis in renal failure**

Renal failure is a common cause of pericardial diseases. Two forms are described:

1) *Uremic pericarditis* – found in 6-10% of patients with severe renal failure (acute or chronic) prior to dialysis initiation or immediately thereafter, distinguished by large pericardial effusions.

2) *Dialysis-associated pericarditis* – found in up to 13% of patients on chronic hemodialysis program and occasionally in patients with peritoneal hemodialysis due to inadequate dialysis or fluid overload.

Toxic nitrogen metabolites, viral and bacterial infections, autoimmune mechanisms, secondary hyperparathyroidism are involved in the development of mechanisms. Pericarditis may be fibrinous, frequently with haemorrhagic effusion, sometimes with signs of cardiac tamponade.

*Clinical manifestations* depend on the stages of renal failure, on the performance of renal dialysis and presence of cardiac tamponade. These may include fever, pleuritic chest pain, leukocytosis and pericardial rub, however many patients may be asymptomatic. As a result of autonomic nervous system impairment in uremic patients, tamponade is not associated with tachycardia (HR 60-80 beats/min), even in conditions like fever and hypotension. The clinical picture may be worsened by the presence of anemia induced by resistance to erythropoietin.

ECG records do not show typical diffuse segment ST and T-wave elevations. Histopathological examination shows dense adhesions bet-

ween the thickened pericardial membranes („bread and butter” appearance).

The *treatment of pericarditis in renal failure* is managed by *renal dialysis* or intensification of dialysis sessions. Heparin-free haemodialysis is indicated in order to avoid haemopericardium. If it is not possible or if patients are resistant to haemodialysis, peritoneal dialysis may be performed. Intensified dialysis usually determines recovery of pericarditis within 1-2 weeks without any haemodynamic disturbances.

*NSAIDs* association may ameliorate symptoms. In large symptomatic effusions, intrapericardial instillation of *corticosteroids after pericardiocentesis or subxiphoid pericardiotomy* (Triamcinolone hexacetonide 50mg every 6 hours for 2-3 days) may be useful. In large chronic effusions or tamponade resistant to dialysis, *pericardiocentesis with prolonged drainage* is required. *Pericardectomy* is indicated only in refractory severely symptomatic patients.

### **Pericarditis in myocardial infarction**

There are two distinguished forms of postinfarction pericarditis:

- 1) „*early*” form, epistenocardic pericarditis
- 2) “*delayed*” form („Dressler’s syndrome”)

*Epistenocardic pericarditis* occurs in the first 1-3 days, maximally in a week after the onset of myocardial infarction. It is caused by transmural necrosis with inflammation which implies the adjacent visceral and parietal pericardium. The involvement of pericardium correlates with dimensions and location of infarction.

This form of pericarditis may develop painlessly and does not influence the prognosis. The appearance of pericardial friction rub associated with a larger effusion requires limitation of anticoagulant therapy. In some cases a certain amount of fluid may persist for many months.

*The treatment of choice of epistenocardic pericarditis* is the following:

Ibuprofen or aspirin 650mg every 4 hours for 2-5 days. Other non-steroidal antiinflammatory drugs influence cicatrization, which is a risk for infarction zone thinning.

Corticosteroid therapy can be used only in the case of refractory symptomatology, but it may delay myocardial infarction healing.

**Dressler's syndrome** occurs from 10 days to 2 months after myocardial infarction onset. The development mechanism is autoimmune. Pericardial inflammation is diffuse, but it is not located in the myocardial infarction region, unlike early postinfarction pericarditis.

**Clinical manifestations** include precordial pain intensified on breathing or movement, fever, pericardial and pleural friction rub. Cardiac tamponade occurs rarely, being favored by anticoagulant therapy. Pericardial postinfarction effusion > 10mm is more frequently associated with haemopericardium.

ECG records show changes of ST segment and T-wave, which are difficult to differentiate from myocardial infarction. Myocardial infarction pericarditis is characterized by a slow evolution or normalization of T-waves that were inverted. Small effusions, more rarely of a larger size, may be noted at echocardiographic examination.

### **Treatment**

In the presence of a major pericardial effusion, hospitalization is necessary, NSAIDs and aspirin relieve the symptomatology. Colchicine is efficient. Prednisone (40-60 mg/day with dose tapering and treatment stop in 7-10 days) may be administered to treat resistant patients or in recurrences.

Acute cardiac tamponade can be caused by the rupture of the LV free wall in the first 3-4 days after the onset of myocardial infarction. Urgent surgical treatment is a life saving for patients.

### **Postpericardiotomy pericarditis**

Postpericardiotomy pericarditis occurs within days-months after heart surgery, pericardial incisions or cardiac trauma. Cardiac tamponade after open heart surgery is more common after valvular surgery than after coronary artery by-pass grafting, caused by preoperative use of anticoagulants. Constrictive pericarditis may develop after heart surgery.

It is similar to postinfarction myocardial syndrome, however postpericardiotomy syndrome generates a greater acute autoimmune reaction with the formation of antiheart antibodies (antisarcolemmal and antifibrillary) as a response to myocardial lesion.

### **Clinical features**

Systemic inflammatory response is by fever, mild leukocytosis, whereas pleuropericardial one is characterized by chest pain. ECG shows acute pericarditis changes in 50% of cases. Echocardiography

may show small or moderate amounts of fluid. Chest radiological examination detects pleural effusions, sometimes pulmonary infiltrates.

### **Treatment**

• NSAIDs are of choice, being efficient in the first 48 hours after the beginning

• *Colchicine*

The treatment should be administered for several weeks-months, even after the disappearance of effusion.

• *Corticotherapy* is indicated in patients with severe, resistant symptoms, or recurrences. Oral corticosteroid treatment is administered for a long term (3-6 months)

• *Pericardiocentesis performance* and triamcinolone intrapericardial instillation (300 mg/m<sup>2</sup>).

• *Repeated surgery and pericardectomy* are rarely needed.

### **Post-traumatic pericarditis**

Post-traumatic pericarditis may be caused by accidental and iatrogenic wounds.

*Thoracic trauma* may be penetrating and blunt, may determine myocardial contusion with haemopericardium, cardiac rupture, pericardial rupture or partial mediastinal and pleural heart prolapse.

*Iatrogenic tamponade* develops more frequently in the case of percutaneous mitral valvuloplasty, during or after transseptal puncture. Perforation of atrial-free wall is possible, which induces chest pain and tamponade signs. Rescue pericardiocentesis is required.

Perforation or transection of the coronary artery may occur during percutaneous coronary angioplasty, which may rarely lead to significant pericardial bleeding and acute or subacute cardiac tamponade, that can be treated by implantation of a membrane-covered graft stent.

If the haemorrhage cannot be stopped, surgical treatment is necessary.

Iatrogenic tamponade may occur through myocardial perforation during endomyocardial biopsy of the LV or RV, by insertion of a catheter-electrode in case of temporary or permanent pacing, by epicardial electrodes or in the case of automated defibrillator implantation. Causes of haemopericardium and iatrogenic tamponade may be diagnostic and therapeutic procedures, such as endoscopic sclerotherapy for esophageal varices, esophagoscopy, implantation of a central venous catheter.

## **Autoreactive pericarditis and pericarditis in systemic autoimmune diseases**

Autoreactive pericarditis may be defined in the case of a proved systemic or loculated autoimmune response at pericardial level, granted that other specific pericardial etiologies are excluded.

Diagnosis of autoimmune pericarditis may be made in the presence of the following:

- 1) increased number of lymphocytes and mononuclear cells  $> 5000/\text{mm}^3$  or the presence of antibodies against heart muscle tissue (antisarcolemma) in the pericardial fluid
- 2) signs of inflammation on epicardial/endomyocardial biopsy by  $\geq 14 \text{ cells}/\text{mm}^2$
- 3) exclusion of active viral infection in pericardial fluid and epicardial/endomyocardial biopsies (no virus isolation, no IgM titer antibodies against cardiotropic viruses, CRP analysis is negative for main cardiotropic viruses)
- 4) exclusion by CRP and/or of bacterial infections
- 5) absence of neoplastic infiltration in pericardial fluid analysis and biopsies
- 6) exclusion of systemic, metabolic disorders, including uremia.

Autoimmune pericarditis can be associated to rheumatoid polyarthritis, systemic lupus erythematosus, systemic sclerosis, polymyositis/dermatomyositis, mixed connective tissue disease, seronegative spondyloarthropathies and vasculitides.

If pericardial effusions reduce gradually and clinical features disappear spontaneously or due an antiinflammatory treatment, a protocol of invasive diagnosis may not be needed.

The treatment of pericarditis should focus on disappearance of symptoms, pericardial fluid and implies a proper treatment of the main disease. If necessary, intrapericardial treatment with triamcinolone is very efficient with a low incidence of side effects.

### **Radiation – induced pericarditis**

Radiation – induced pericarditis may develop during the treatment or within up to 15-20 years after radiation. There are three groups of factors implied in the occurrence of pericarditis:

- 1) therapeutic including total radiation dose, radiation exposed measurements, nature of radiation source, duration of therapy;
- 2) tumoral histologic type, evolutive moment;
- 3) host with own immune system.

In some cases during the treatment or later on, an acute pericardial inflammatory reaction with fibrinous accumulations, serous, sero-haemorrhagic or haemorrhagic effusion develops. Then, pericardial thickening may develop, with installment of effusive-constrictive form or occurrence of constrictive pericarditis (in 20% of patients).

Inflammatory and fibrous processes may extend to the myocardial level provoking interstitial myocarditis.

In some cases of pericardiocentesis with the examination of the pericardial fluid Echocardiography, CT, MRI are necessary for making a *diagnosis*. The analysis of pericardial fluid and pericardial biopsy are useful in the differential diagnosis between radiation - induced pericarditis and neoplastic pericarditis.

The *treatment* has to be adapted to clinical, anatomical and haemodynamic forms. Asymptomatic patients with little pericardial fluid have be examined periodically. Pericarditis with moderate pericardial effusion may be treated with non-steroidal and steroid antiinflammatory agents. Pericardiocentesis with diagnostic purposes and tamponade reduction is to be performed in cardiac tamponade and effusive-constrictive pericarditis. In cases of recurrent tamponade, pericardiotomy with drainage and left pleural window are performed. Pericardectomy is indicated in constrictive pericarditis, but it is associated with higher postoperative mortality due to myocardial fibrosis.

#### **Drug- and toxin-related pericarditis**

Pericardial reactions appear rarely due to drug administration or contact with toxic substances. The most frequent causes of drug- and toxin-related pericarditis are:

1. drugs which induce lupus syndrome: Procainamide, Hydralazine, Isoniazid,  $\alpha$ -Methyldopa, Mesalazine, Hydantoins
2. drugs which induce hypersensitivity reaction: Penicillin, Tryptophan, Cromolyn sodium
3. drugs inducing idiosyncratic or hypersensitivity reaction: Methysergide, Minoxidil, Bromocriptine, Phenylbutazone, Amiodarone, Thiazides, Sulfamides, Streptokinaze

4. immunosuppressors and cytostatics: Cyclophosphamide, Cyclosporine, 5-Fluorouracil, Anthracycline derivatives (Doxorubicin, Daunorubicin)
5. serum sickness: foreign antisera, blood products
6. venom: scorpion
7. reactions to substances with direct pericardial application: sclerosant drugs (Tetracycline), Talc
8. anticoagulant and fibrinolytic agents.

Mechanisms and clinical and anatomical variants may be varied. Their differentiation from other etiologic forms of pericarditis is often difficult. The treatment of drug-related pericarditis implies discontinuation of etiologic agent and symptomatic therapy.

#### **Pericardial effusion in pregnancy**

There is no evidence that pregnancy modifies the sensibility to pericardial diseases, nevertheless small clinically non-significant pericardial effusions occur in 40% of healthy pregnant women.

Most pericardial diseases are treated as in nonpregnant women. Caution is necessary in the case of high-dose aspirin which may lead to premature closure of the ductus arteriosus, as well as in the case of colchicine that is contraindicated in pregnancy. Pericardiocentesis, preferably with echocardiography guidance, is indicated in tamponade and/or if infectious etiology is suspected. Pericardiotomy and pericardectomy may be performed if necessary.

#### **Pericarditis associated to hypothyroidism**

Pericarditis associated to hypothyroidism occurs in 5-30% of patients with hypothyroidism. The fluid accumulation is slow, tamponade occurs rarely. Clinical manifestations may include bradycardia, low-voltage of the QRS and T-wave flattening or inversion in the ECG record. Radiologic examination may determine cardiomegaly. Echocardiography may reveal pericardial effusion, these being associated with a history of thyroid dysfunction, myopathy, ascites and uveal edema. The diagnosis of hypothyroidism is based on serum levels of thyroxin and thyroid stimulating hormone(TSH).

Therapy with thyroid hormones decreases pericardial effusion.

## OTHER PERICARDIAL DISEASES

### **Cholesterol pericarditis**

Chylopericardium may be due to obstruction or post-traumatic lesion of the thoracic duct or after a surgical intervention, tuberculosis, cancer, lymphangiomatosis, sometimes being congenital (thoracic duct atresia) or idiopathic.

The pericardial fluid is sterile, opalescent, with a milky white appearance, and microscopically detected fat droplets. The chilous nature of the fluid is confirmed by its alkaline reaction and specific gravity between 1010 and 1021, Sudan III stain for fat, elevated concentrations of triglycerides (5-50g/l) and protein (22-60 g/l). Enhanced CT alone or combined with lymphography may identify the location of the thoracic duct and its lymphatic connections to the pericardium. Pericardiocentesis with the analysis of the fluid is also useful.

The treatment is administered in dependance on the etiology and amount of accumulated chylous fluid: diet, pericardiocentesis, thoracic duct ligation, pericardio-peritoneal shunting by means of pericardial window and pericardectomy.

### **Pericardial cysts**

Pericardial cysts represents a rare anomaly, localized more frequently on the right side. Cysts may be both unilocular and multilocular with the dimensions between 1-5 mm. These may be congenital, inflammatory (of rheumatic, bacterial, particularly tuberculous, traumatic and surgical etiology) or they may be of a parasitogenic origin (Echinococcus). Frequently cysts are asymptomatic, but they may cause chest pain, dyspnea, cough or palpitations. Chest X-ray (detects as oval, homogeneous opacities, usually at the right cardiophrenic angle), echocardiography, CT, MRI and if required – guided pericardial puncture are useful in diagnostics. In many cases the treatment is not needed. Whenever necessary, the treatment involves percutaneous aspiration and sclerosis with ethanol. In some cases surgical treatment is required. Surgical excision of echinococcal cysts is not recommended, unlike percutaneous aspiration and instillation of ethanol or silver nitrate after pre-treatment with Albendazole.



### **Congenital pericardial absence**

Congenital pericardial absence may be partial, more frequently on the left (70%), rarely on the right (17%) or even less rarely it may be total. This anomaly is usually associated with other cardiac congenital diseases (approximately 30% of patients).

The most frequent clinical manifestations are pains, palpitations, dyspnea, syncopes, sometimes sudden death caused by coronary artery compression due to herniation of the LA, left atrial appendage or LV due to the defect or torsion of large vessels because of excessive heart mobility.

In the absence of pericardium the heart shift to the left hyperdynamic apex impulse, systolic murmur are detected during the physical examination.

A radiologic examination reveals the heart shift to the left heart, pulmonary artery prominence, with a clear space between the aorta and pulmonary artery or between the left hemidiaphragm and inferior heart border.

Right axis deviation, pulmonary P waves, sometimes a hemiblock of the right bundle branch are determined electrocardiographically.

Echocardiography distinguishes RV dilation, interventricular septum paradoxal movement, excessive posterior movement of the LV wall. In partial pericardial absence on the left side is noted a herniation of the LV, LA or the left atrial appendage. If the anomaly is on the right a herniation of the RA or RV is noted.

The confirmation of diagnosis requires the performance of magnetic resonance imaging, that shows a pronounced left and posterior heart lateralization, interposition of the pulmonary parenchima between the ascending aorta and pulmonary artery trunk, as well as between the diaphragm, descending aorta and diaphragmatic heart border, the contact between the left atrial appendage and descendent aorta, the absence of the parietal pericardium between the epicardial and mediastinal fat.

In some cases of partial pericardial absence, surgery is necessary to remove heart strangulation and compression. A primary closure of the defect can be performed, as well as a partial pericardectomy, left atrial appendage extirpation, pericardioplasty with the parietal pleura. Associated cardiac anomalies, if needed, are to be corrected concomitantly.

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## CONTENTS

List of abbreviations.....	3
Definition and classification of pericarditis.....	4
Chronic pericarditis.....	4
Etiological forms of pericarditis.....	14
Other pericardial diseases.....	27
Bibliography.....	29