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Recommendations for the management of pericardial diseases

Priporočila za obravnavo bolnikov z boleznijo perikarda

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Abstract

The pericardium may be affected as an isolated process or as part of a systemic disease. In developed countries (with a low prevalence of tuberculosis), the most common aetiologies are presumed to be viral, immune-mediated forms (systemic inflammatory diseases, post-cardiac injury syndromes) and cancer. In clinical practice, there are few general presentations with distinctive signs and symptoms that are referred to as pericardial syndromes: pericarditis, isolated pericardial effusion, cardiac tamponade, constrictive pericarditis and pericardial masses and cysts. In the latest European Society of Cardiology guidelines for pericardial disease new diagnostic strategies have been proposed for the triage of patients with pericarditis and pericardial effusion, and specific diagnostic criteria have been established for acute and recurrent pericarditis. Moreover, new therapeutic strategies have emerged. The article covers a translation of ESC Guidelines on the Diagnosis and Management of Pericardial Diseases adapted to our situation.

Izvleček

Bolezni perikarda so lahko izoliran bolezenski proces, ali pa so del sistemskih bolezenskih stanj. V zahodnem svetu, kjer je prevalenca tuberkuloze nizka, je perikarditis najpogosteje povezan z virusnimi okužbami, avtoimunskimi procesi ali pa malignomi. Glede na klinično sliko lahko bolezni perikarda razdelimo v nekaj glavnih sindromov: perikarditis, izoliran perikardni izliv, tamponada srca, konstrikcijski perikarditis in mase/ciste v perikardu. Zadnje smernice Evropskega kardiološkega združenja prinašajo nekaj novosti na področju triažiranja in diagnosticiranja bolnikov z boleznimi perikarda, predvsem pa se jasneje opredelijo do strategij zdravljenja posameznih perikardnih sindromov. Članek povzema evropske smernice in vključuje prilagoditve za naše razmere.

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Foreword

In 2015, the European Society of Cardiology issued new guidelines for the treatment of patients with pericardial disease (1). According to the latest update from 2004 (2), they bring several new developments: triage of patients with acute

pericarditis and pericardial effusion according to the level of risk, criteria for diagnosis of acute and recurrent pericarditis, multimodal approach to diagnosis, treatment of pericarditis according to multicentre randomized results and recommendations for specific patient groups. This article contains a translation of European guidelines adapted to our conditions. The strength of the recommendations and the level of evidence are defined by pre-defined scales (Table 1 and Table 2).

The recommendations were approved and adopted by the Expert Council of Internal Medicine on 7 September 2020, and at the Main Expert Council of the Slovenian Medical Association on 8 September 2020.

Table 1: Classes of recommendations.

Classes of recommendations	Definition	Suggested wording to us
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, and effective.	Is recommended/ is indicated.
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness or efficacy of the given treatment or procedure.	
Class IIa	Weight of evidence/opinion is in favour of usefulness/efficacy.	Should be considered.
Class IIb	Usefulness/efficacy is less well established by evidence/opinion.	May be considered.
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.	Is not recommended.

Table 2: Levels of evidence.

Evidence level A	Data derived from multiple randomized clinical trials or meta-analyses.
Evidence level B	Data derived from a single randomized clinical trial or large non-randomized studies.
Evidence level C	Consensus of opinion of the experts and/ or small studies, retrospective studies, registries.

1 Introduction

Pericardial diseases may be either isolated disease or part of a systemic disease. Pericardial syndromes include different clinical presentations of pericardial diseases with distinctive signs and symptoms that can be grouped in specific 'syndromes'. The main pericardial syndromes are pericarditis, pericardial effusion, cardiac tamponade, constrictive pericarditis, and pericardial masses.

2 Epidemiology and aetiology

Epidemiological data on the incidence of pericardial disease are scarce. Acute pericarditis is the most common pericardial disease, with an incidence of 28 cases per 100,000 person per year and is the cause of emergency room admissions for chest pain in 5%. It is more common in men than in women (relative risk 2.0), especially in young adults. A simple aetiological classification for pericardial diseases is to consider infectious and non-infectious causes (Table 3). In the developed world, the most common causes of pericarditis are viruses, and in developing countries, tuberculosis (TB), often in comorbidity with a human immunodeficiency virus (HIV) infection. Autoimmune and neoplastic causes are also common in the developed world.

3 Pericardial syndromes

3.1 Acute pericarditis

Acute pericarditis is an inflammatory pericardial syndrome with or without pericardial effusion. Two of the four diagnostic criteria are required to make a clinical diagnosis (Table 4). Typical pericarditic chest pain occurs in 85-90% of cases. Pericardial friction rub is highly specific for pericarditis but is present in only up to 30% of cases. Characteristic

Table 3: Aetiology of pericardial disease.

A. Infectious causes
Viral (common): Enteroviruses (coxsackieviruses, echoviruses), herpesviruses (EBV, CMV, HHV-6), adenoviruses, parvovirus B19 (possible overlap with aetiologic viral agents of myocarditis).
Bacterial: *Mycobacterium tuberculosis* ((common, other bacterial rare), *Coxiella burnetii, Borrelia burgdorferi, Pneumococcus spp., Meningococcus spp., Gonococcus spp., Streptococcus spp., Staphylococcus spp., Haemophilus spp., Chlamydia spp., Mycoplasma spp., Legionella spp., Leptosira spp., Listeria spp., Providenca stuarti.*
Fungal (very rarely): *Histoplasma spp., Aspergillus spp., Blastomyces spp., Candida spp.*
Parasitic (very rarely): *Echinococcus spp., Toxoplasma spp.*
B. Non-infectious causes
Autoimmune (common): Systemic autoimmune and autoinflammatory diseases (systemic lupus erythematosus, Sjögren's syndrome, rheumatoid arthritis, scleroderma), systemic vasculitides (granulomatosis with polyangiitis, Churg-Strauss syndrome, Horton disease, Takayasu disease, Behcet syndrome), sarcoidosis, familial Mediterranean fever, inflammatory bowel disease, Still disease.
Neoplastic: Primary tumours (rare): mainly mesothelioma, sarcoma, fibrosarcoma, haemangioma, teratoma. Secondary metastatic tumours (common): lung cancer, breast cancer, lymphoma, leukaemia, melanoma.
Metabolic: uraemia, myxoedema, anorexia nervosa; other rare.
Traumatic and iatrogenic: Early onset (rare): • Direct injury: penetrating thoracic injury, aesophageal perforation. • Indirect injury: blunt chest injuries, radiation injury. Delayed onset (common): • Pericardial injury syndromes: Dressler's syndrome, postcardiotomy syndrome, posttraumatic pericarditis (including iatrogenic injuries after percutaneous cardiac interventions).
Drugs (rare): lupus-like disease (procainamide, hydralazine, methyldopa, isoniazid, phenytoin); cancer drugs (doxorubicin, daunorubicin, cytosine arabinoside, 5-fluorouracil, cyclophosphamide); penicillins (hypersensitivity eosinophilic pericarditis), amiodarone, methysergide, mesalazine, clozapine, minoxidil, dantrolene, practolol, phenylbutazone, thiazides, streptomycin, thiouracil, streptokinase, para-aminosalicylic acid, sulfasalazine, cyclosporine, bromocriptine, vaccines, GM-CSF, and anti-TNF drugs.
Other causes: Common: amyloidosis, aortic dissection, pulmonary arterial hypertension, chronic heart failure. Rare: congenital partial or complete absence of the pericardium.

EBV – Epstein-Barr virus; CMV – cytomegalovirus; HHV – human herpesvirus; spp – species; GM-CSF – granulocyte-macrophage colony-stimulating factor; TNF – tumour necrosis factor.

widespread ST-segment elevation and PR depression occur in 60% of cases in acute pericarditis. ECG changes reflect epicardial inflammation because the parietal layer of the pericardium is electrically inert. The characteristic four-stage development of ST/T changes (Figure 1) is present in 50% of patients and is affected by therapy. The main differential diagnoses according to ECG changes are acute coronary syndrome with ST-segment elevation and early repolarization. Pericardial effusion, usually minor, occurs in up to 60% of pericarditis cases. Additional symptoms and signs may be associated with the underlying disease that caused the pericarditis (e.g., infection, systemic connective tissue disease, neoplasm). The diagnosis is further supported by elevation of markers of

Table 4: Definition and diagnostic criteria for pericarditis.

Pericarditis	Definition and diagnostic criteria
Acute	**Two of the four diagnostic criteria are required to confirm a diagnosis:** 1. Pericarditic chest pain. 2. Pericardial rub. 3. New widespread ST-elevation or PR depression on ECG. 4. Pericardial effusion (new or worsening). **Additional supporting findings:** • Elevated markers of inflammation (CRP, ESR, white blood cell count). • Evidence of pericardial inflammation by an imaging technique (CT or CMR).
Incessant	Pericarditis lasting for > 4–6 weeks but < 3 months without remission.
Recurrent	Recurrence* of pericarditis after a documented first episode of acute pericarditis and a symptom-free interval of 4–6 weeks or longer.
Chronic	Pericarditis lasting for > 3 months.

*usually within 18 to 24 months; ECG – electrocardiogram; CRP – C-reactive protein; ESR – erythrocyte sedimentation rate; CT – computed tomography; CMR – cardiac magnetic resonance.

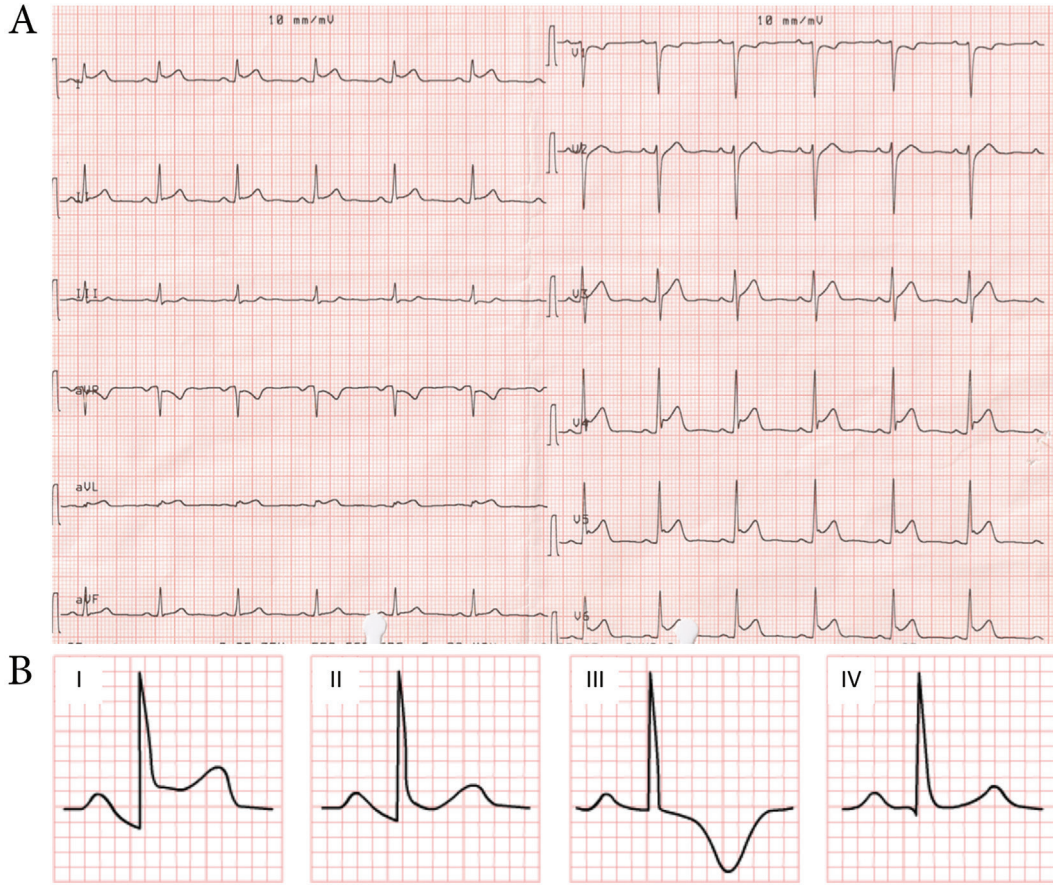

Figure 1: ECG in acute pericarditis (A) and characteristic four-stage development of ST segment/T-wave changes (B).

inflammation (e.g., C-reactive protein, CRP) and evidence of pericardial inflammation by computed tomography (CT) or cardiac magnetic resonance (CMR).

In the acute period of a few hours to a few days (stage I), widespread concave-up ST-segment elevation and PR depression are present. In leads V1 and aVR, the changes are reciprocal. In 1st – 2nd week (stage II), the ST-segment and

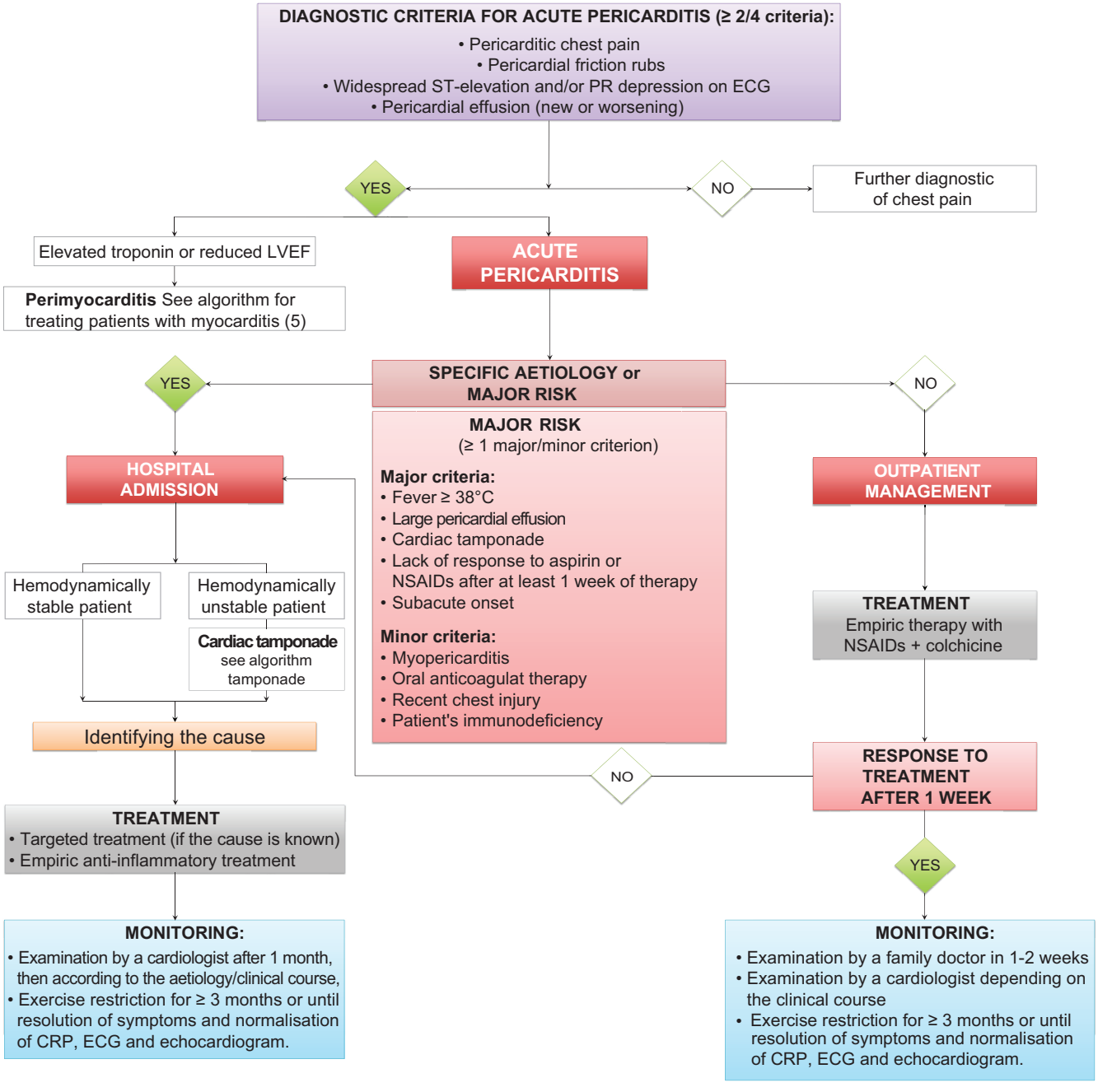

Figure 2: Clinical course of treatment of patients with suspected acute pericarditis.
ECG – electrocardiogram; LVEF – left ventricular ejection fraction; NSAIDs – non-steroidal anti-inflammatory drugs. The risk of developing complications in acute pericarditis is assessed by major criteria derived from meta-analyses, and minor criteria based on the consensus of experts.

Recommendations for the treatment of acute pericarditis.

Recommendations	[a]	[b]
Hospital admission is recommended in patients with acute pericarditis and a high risk of complications (at least one major or minor risk criterion[c]).	I	B
Outpatient management is recommended for low-risk patients with acute pericarditis.	I	B
Evaluation of response to anti-inflammatory therapy is recommended after 1 week.	I	B

[a]recommendation class; [b]evidence level; [c]Figure 2.

PR returns into isoline (pseudonormalisation). After week 3 (stage III), negative T waves appear, which persist or gradually normalize over several months (stage IV).

Due to the relatively benign course of the disease and the low yield of diagnostic tests, the cause of pericarditis does not always need to be identified, especially not in countries with low TB prevalence. The new guidelines recommend triage of patients according to the level of risk (Figure 2). Patients with suspected specific aetiology of the disease or at least one risk indicator for complications (3-4) need hospital treatment and causation. Low-risk patients are treated on an outpatient basis. We introduce experiential treatment with anti-inflammatory drugs and check the effect after one week. If there is no improvement (resolution of symptoms, normalization of CRP, reduction of the size of the pericardial effusion < 10 mm), hospitalization and additional diagnostics are required.

In all patients with suspected acute pericarditis, basic laboratory examinations, ECG, chest X-ray and transthoracic echocardiography are performed (Figure 3). Additional laboratory and imaging

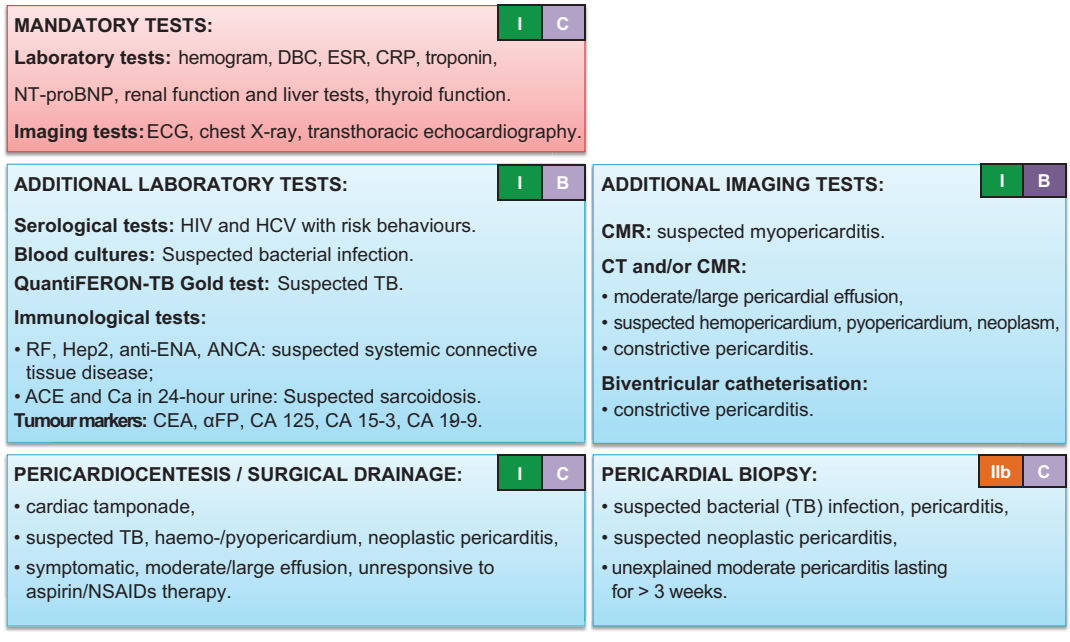

Figure 3: First and second level investigations for pericarditis.
DBC – differential blood count; ESR – erythrocyte sedimentation rate, CRP – C-reactive protein ; NT-proBNP – amino-terminal fragment of B-type natriuretic peptide; ECG – electrocardiogram; X-ray – radiograph; HIV – human immunodeficiency virus; HCV – hepatitis C virus; TB – tuberculosis; ACE – Angiotensin-converting enzyme; CEA – carcinoembryonic antigen; CT – computed tomography; CMR – cardiac magnetic resonance.

Table 5: Treatment of acute and recurrent pericarditis in adult patients.

Drug	Initial dosage	Duration of treatment	Tapering
Aspirin	500–1000 mg/6–8 hours	First episode: 1–2 weeks. Recurrence: 2–4 weeks to a few months.	Decrease dose by 250–500 mg per 1–2 weeks.
Ibuprofen	600 mg/8 hours		Decrease dose by 200–400 mg per 1–2 weeks.
Indomethacin	25–50 mg/8 hours		Decrease dose by 25 mg per 1–2 weeks.
Naproxen	500–1000 mg/12 hours		Decrease dose by 125–250 mg per 1–2 weeks.
Colchicine	0.5 mg/12 hours (≥ 70 kg) 0.5 mg/24 hours (<70 kg)	First episode: 3 months. Recurrence: ≥ 6 months.	Not mandatory, alternatively 0.5 mg every other day (< 70 kg) or 0.5 mg once (≥70 kg) in the last weeks last week only.
Methylprednisolone	0.2–0.4 mg /kg/24 hours	First episode: 2 weeks. Recurrence: 4 weeks	Dose > 40 mg: decrease by 8 mg per 1–2 weeks. Dose 40–20 mg: decrease by 4–8 mg per 1–2 weeks. Dose 20–12 mg: decrease by 2 mg per 2–4 weeks. Dose < 12 mg: decrease by 1–2 mg per 2–6 weeks.

tests are recommended if a specific disease aetiology is suspected (6,7). In particular, bacterial pericarditis, pericarditis in the context of systemic connective tissue diseases and neoplastic pericarditis should be excluded.

The basic non-pharmacological measures are rest and avoidance of physical exertion that goes beyond a sedentary lifestyle. In athletes, physical activity should be restricted for at least 3 months, and in others until resolution of symptoms and normalization of CRP, ECG and echocardiogram (in which case, it may take a shorter time).

Targeted treatment of acute pericarditis is possible with a known cause of the disease. In case of unknown aetiology, empiric treatment with anti-inflammatory drugs (Table 5) is introduced (8). Among nonsteroidal anti-inflammatory drugs (NSAIDs), ibuprofen or acetylsalicylic acid (aspirin) are most commonly used. The initial high doses are maintained until the symptoms disappear and the CRP normalizes for at least 1-2 weeks, after which they are gradually tapered. At the same time, gastroprotection with proton pump inhibitors is required. Concurrently, colchicine is introduced, which significantly improves the response to anti-inflammatory treatment and reduces the frequency of recurrences by half (9). Corticosteroids should be considered as a second option in patients with contraindications and failure of aspirin or NSAIDs (e.g., with severe renal impairment, recent gastric ulcer or gastrointestinal bleeding, concomitant anticoagulant treatments and with pregnancy). Corticosteroids, especially when used in high doses, favouring the chronic evolution of the disease and promoting drug dependence (10). Low to moderate doses of corticosteroids along with colchicine are recommended, which are to be reduced very slowly after CRP normalization. For a long-term use of corticosteroids, the addition of calcium, 1,200-1,500 mg per day, and vitamin D, 800-1,000 international units per day, is recommended.

Drug dosage for the elderly (age > 70 years): 50% reduction in colchicine dose; the lowest doses of aspirin/other NSAIDs.

Dosage with renal impairment:

- creatinine clearance 35–49 ml/min – colchicine 0.5 mg/24 hours;
- creatinine clearance 10–34 ml/min – colchicine 0.5 mg/48–72 hours, NSAIDs contraindicated;

Recommendations for the treatment of acute pericarditis.

Recommendations	[a]	[b]
Aspirin or NSAIDs are recommended as first-line therapy for acute pericarditis with gastroprotection.	I	A
Colchicine is recommended as first-line therapy for acute pericarditis as an adjunct to aspirin/NSAID therapy.	I	A
Serum CRP should be considered to guide the treatment length and assess the response to therapy.	IIa	C
Low to moderate-dose corticosteroids[c] should be considered for acute pericarditis in cases of contraindication/failure of aspirin/ NSAIDs, and when an infectious cause has been excluded, or when there is a specific indication such as autoimmune disease.	IIa	C
Exercise restriction should be considered until resolution of symptoms and normalization of CRP, ECG and echocardiogram; that is, for at least 3 months for athletes.	IIa	C
Corticosteroids are not recommended as first-line therapy for acute pericarditis.	III	C

[a]recommendation class; [b]evidence level; [c]added to colchicine; NSAIDs - non-steroidal anti-inflammatory drugs; CRP – C-reactive protein, ECG – electrocardiogram.

- creatinine clearance < 10 ml / min – colchicine, NSAIDs and aspirin are contraindicated.

Dosage with hepatic impairment: caution and possible dose adjustments.

Most patients with acute pericarditis have a good prognosis. The main complications are recurrence, cardiac tamponade, and constrictive pericarditis. Acute pericarditis recurs in 30% of cases within 18 months after the first episode. Cardiac tamponade is extremely rare in idiopathic or viral pericarditis, but is more common in neoplastic, TB, or purulent pericarditis. The risk of developing constrictive pericarditis is low (< 1%) in idiopathic pericarditis, moderate (2–5%) in immune-mediated or neoplastic pericarditis, and high (20–30%) in bacterial (especially purulent) pericarditis.

3.2 Incessant and chronic pericarditis

Incessant pericarditis indicates the duration of symptoms > 4–6 weeks without clear remission (Table 4). Chronic pericarditis indicates the duration of symptoms > 3 months.

3.3 Recurrent pericarditis

Recurrent pericarditis is a recurrence of the disease within 18–24 months after confirmed acute pericarditis, with intervening remission lasting ≥ 4–6 weeks (Table 4). The diagnosis of recurrent pericarditis is made on the basis of the same diagnostic criteria as for acute pericarditis. The frequency of the first recurrence after acute pericarditis is 115–30%, and subsequent recurrences may occur in patients not receiving colchicine in up to 50% (especially when treated with corticosteroids). A common cause of recurrence of pericarditis is insufficient treatment (underdoses of drugs, treatment time too short). The viral agent is proven in up to 20% of cases, and an autoimmune cause of the disease is also possible.

The instructions for exercise restriction with recurrent pericarditis are the same as with acute pericarditis. Recurrent pericarditis is treated with aspirin/NSAIDs and colchicine for a longer period than in the first episode (Table 5, Figure 4) (11). If there is no satisfactory improvement, we can try replacing the NSAID with another drug from the same group. If there is still no response, corticosteroids are added in small to moderate doses as the third drug. In patients who do not respond to conventional anti-inflammatory therapy, immunosuppressive or immunomodulatory therapy may be considered (12) (Table 6). In acute relapses, high doses of intravenous immunoglobulins for 5 days are successful (13). In recurrent, corticosteroid-dependent and colchicine resistant pericarditis (at least two recurrences of the disease after discontinuation of corticosteroids) an interleukin-1 antagonist

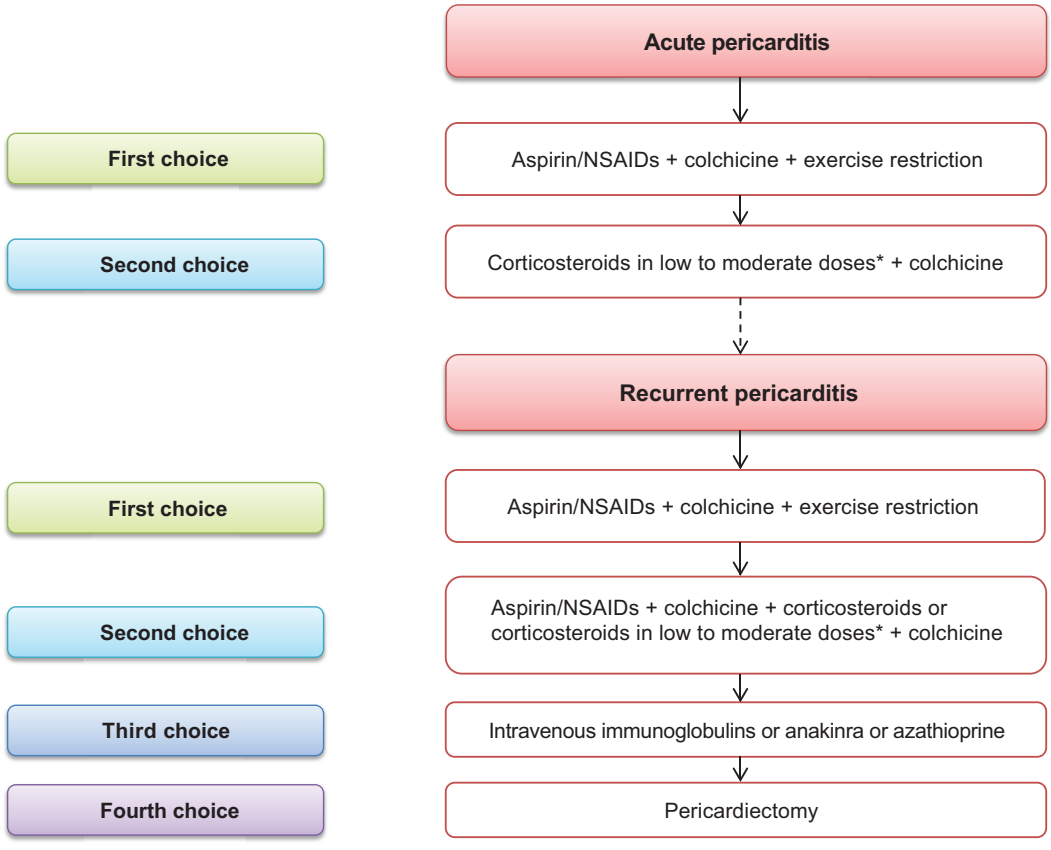

Figure 4: Therapeutic algorithm for acute and recurrent pericarditis.
NSAIDs – non-steroidal anti-inflammatory drugs; *in case of unresponsiveness or contraindications to aspirin/other NSAIDs and after the exclusion of infection.

Table 6: Immunosuppresant and biological drugs used in recurrent pericarditis.

Drug*	Initial dosage	Duration of treatment	Tapering
Azathioprine	Initial dosage: 1 mg/kg/day. Maintenance dose: 2–3 mg/kg/day	Several months.	Several months.
Intravenous immunoglobulins	400–500 mg/kg/day i.v. 5 days or 1 g/kg/day i.v. 2 days, possible repeated administration every 4 weeks.	5 days.	Not applicable.
Anakinra	1–2 mg/kg/day to 100 mg/day s.c.	Several months.	Several months.

*– level of evidence C, i.v. – intravenously, s.c. – subcutaneously.

(anakinra) may be considered; it rapidly alleviates symptoms and prevents recurrences (14). Azathioprine is more suitable in the chronic phase of the disease, as it can successfully replace long-term treatment with corticosteroids (15). Pericardiectomy is a method of choice if the drug treatment is not successful (16).

The prognosis of recurrent pericarditis is usually good. It does not depend on the number of recurrences of pericarditis, but on the aetiology of the disease. Cardiac tamponade is rare and usually occurs early in the disease. The risk of developing constriction is lower in recurrent pericarditis than in acute pericarditis (< 1%).

Recommendations for the management of recurrent pericarditis.

Recommendations	[a]	[b]
Aspirin and NSAIDs are mainstays of treatment and are recommended at full doses, if tolerated, until complete symptom resolution.	I	A
Colchicine use for 6 months is recommended as an adjunct to aspirin/NSAIDs.	I	A
Prolonged treatment with colchicine (12 to 24 months) is recommended with a more severe disease.	IIa	C
CRP dosage should be considered to guide the treatment duration and assess the response to therapy.	IIa	C
After CRP normalization, a gradual tapering of therapies should be considered, tailored to symptoms and CRP, stopping a single class of drugs at a time.	IIa	C
Drugs such as intravenous immunoglobulins, anakinra and azathioprine may be considered in cases of corticosteroid-dependent recurrent pericarditis in patients not responsive to colchicine.	IIb	C
Exercise restriction should be considered for non-athletes with recurrent pericarditis until symptom resolution and CRP normalization, taking into account the previous history and clinical conditions.	IIa	C
Exercise restriction for at least of 3 months should be considered for athletes with recurrent pericarditis until symptom resolution and normalization of CRP, ECG and echocardiogram.	IIa	C
If symptoms recur during therapy tapering, the management should consider not increasing the dose of corticosteroids to control symptoms, but increasing to the maximum dose of aspirin or NSAIDs and intravenously if necessary, adding colchicine and adding analgesics for pain control.	IIa	C
Corticosteroid therapy is not recommended as a first line-approach.	III	B

[a]recommendation class; [b]evidence level; NSAIDs – non-steroidal anti-inflammatory drugs; CRP – C-reactive protein, ECG – electrocardiogram.

Recommendations for the management of acute pericarditis with myocarditis.

Recommendations	[a]	[b]
In patients with pericarditis and suspected associated myocarditis, coronary angiography is required to rule out the acute coronary syndrome.	I	C
CMR is recommended to confirm myocardial involvement.	I	C
In patients with suspected myopericarditis, hospital treatment is required for diagnosis and monitoring.	I	C
In athletes and non-athletes with myopericarditis, a 6-month rest period and avoidance of physical exertion that exceeds a sedentary lifestyle is recommended.	I	C
Empirical anti-inflammatory drugs are allowed in myopericarditis to relieve chest pain in the lowest yet effective doses.	IIa	C

[a]recommendation class; [b]evidence level, CMR – cardiac magnetic resonance.

3.4 Pericarditis associated with myocardial involvement

Due to the close anatomical proximity and common aetiologies, pericarditis and myocarditis occur simultaneously in approximately 30% of cases (17). Overlapping forms may be encountered in clinical practice; myopericarditis indicates more severe pericardial involvement and perimyocarditis predominate myocardial involvement. Clinically, the syndromes are manifested by chest pain associated with other signs of acute pericarditis and elevated troponin.

Myopericarditis is when patients with unequivocal signs of pericarditis have elevated troponin I in the absence of newly developed focal or diffuse impairment of left ventricular function in echocardiography or CMR. Coronary angiography should be performed in order to rule out acute coronary syndrome (18).

Myocardial involvement is confirmed by CMR. In myopericarditis, due to cardiotoxicity, empirical anti-inflammatory drugs are allowed to relieve chest pain in the lowest, still-effective doses. In myopericarditis, there is insufficient evidence to support colchicine treatment.

3.5 Aspirin/other NSAIDs with concomitant antiplatelet or anticoagulant therapy

In patients with coronary heart disease or in patients already receiving acetylsalicylic acid, aspirin at doses up to 1,500 mg per day is recommended for inflammatory pericardial syndromes. In ischemic heart disease, NSAIDs other than Naprosyn are not recommended, because they increase the cardiovascular risk by 1.3-fold.

In patients treated with direct oral anticoagulant drugs, a specialist in an antithrombotic clinic should be consulted about the choice of anti-inflammatory drugs. Aspirin is not recommended for concomitant use with warfarin, unless otherwise indicated, e.g., after insertion of stents. In such cases, corticosteroids or other NSAIDs in lower doses are more commonly administered in addition to colchicine. According to literature, there is no strong evidence that concomitant anticoagulant treatment in acute pericarditis increases the risk of bleeding into the pericardium and cardiac tamponade (19). In contrast, anticoagulant treatment is a risk factor for tamponade in traumatic pericardial effusion.

3.6 Pericardial effusion

There is usually 10–50 ml of friction-reducing serous fluid between the visceral and parietal layers of the pericardium. Any pathological process in the pericardium may cause inflammation and increase the accumulation of fluid in the pericardial space (exudate). Another reason may be a decrease in pericardial reabsorption due to elevated systemic venous pressure as in congestive heart failure or pulmonary hypertension (transudate). The classification of pericardial effusion is shown in Table 7, and diagnostic examinations of pericardial fluid are shown in Table 8. In the developed world, pericardial effusion is often not aetiologically explained; the most common causes are infections with viruses (in 5–30%), neoplasms (in 10–25%), iatrogenic injuries (in 15–20%) and systemic connective tissue diseases (in 5–15%). In developing countries, TB infection is the most common cause of pericardial effusion.

Table 7: Classification of pericardial effusion.

Classification of pericardial effusion	
Progression	Acute (<1 week). Subacute (>1 week and <3 months). Chronic (>3 months).
Size	Minor (<10 mm). Moderate (10–20 mm). Large (>20 mm).
Distribution	Circumferential. Loculated.
Haemodynamic effect	No cardiac tamponade. Cardiac tamponade. Effusive–constrictive.
Composition	Transudate. Exudate.

The clinical presentation of pericardial effusion varies according to the speed of pericardial fluid accumulation. With rapid accumulation of fluid (e.g., injury, iatrogenic perforation), even a small amount of blood can increase the pressure in the pericardial space and cause cardiac tamponade. On the other hand, a slow accumulation of pericardial fluid allows the collection of a large effusion in days to weeks before a significant increase in pericardial pressure causes symptoms and signs. The most common symptoms

Table 8: Main analyses to be performed on pericardial fluid.

Analysis	Test	Aetiology
Biochemical tests	• Specific weight >1.015. • Proteins >30 g/L, punctate/serum ratio >0.5. • LDH >2 µCat/L, punctate/serum ratio >0.6. • Glucose, leukocytes.	Exudate.
Cytologic tests	• Neoplastic cells.	Neoplasm.
Biological markers	• CEA >5 ng/L or CYFRA 21–1 >100 ng/mL. • Adenosine deaminase > 40 U/L, interferon-gamma	Neoplasm.
PCR	• TB-PCR.	TB.
Microbiological tests	• Bacilli staining. • Aerobic and anaerobic cultures.	TB, other bacteria.

LDH – lactate dehydrogenase; CEA – carcinoembryonic antigen; CYFRA – cytokeratin; TB – tuberculosis; PCR – polymerase chain reaction.

are chest pain and dyspnea on exertion, but symptoms of pressure on surrounding structures (coughing, nausea, dysphagia, hoarseness, hiccups) and general symptoms such as fatigue and palpitations, may also be associated. A large proportion of patients with a small chronic pericardial effusion have no symptoms, so pericardial effusion may also be an accidental finding.

The diagnosis of pericardial effusion is performed by echocardiography, which also enables a semiquantitative assessment of the pericardial effusion size and its haemodynamic effects.. Hospitalization is recommended in patients with pericardial effusion and who are at a high risk for complications (Figure 2). CT and/or CMR are performed with small pericardial effusion and with concomitant myocarditis, and with moderate-to-large effusion with suspected hemopericardium, pyopericardium and pericardial effusion with malignancies (7).

Treatment of pericardial effusion should be targeted at the aetiology as much as possible. A simplified algorithm for pericardial effusion triage and management is shown in Figure 5 (20). If inflammatory signs are present, empiric

Recommendations for diagnosing pericardial effusion.

Recommendations	[a]	[b]
Hospitalization is recommended in patients with pericardial effusion and who are at a high risk for complications (≥1 major or minor criterion present[c]).	I	C
If pericardial effusion is suspected, chest X-ray and transthoracic echocardiography should be performed.	I	C
In patients with pericardial effusion, evaluation of systemic indicators of inflammation (e.g., CRP) is recommended.	I	C
CT or CMR are recommended in suspected localized pericardial effusion, pericardial thickening, pericardial masses, and associated thoracic anomalies.	IIa	C

[a]recommendation class; [b]evidence level; [c]Figure 2; CRP – C-reactive protein; X-ray – radiograph; CT – computed tomography; CMR – cardiac magnetic resonance.

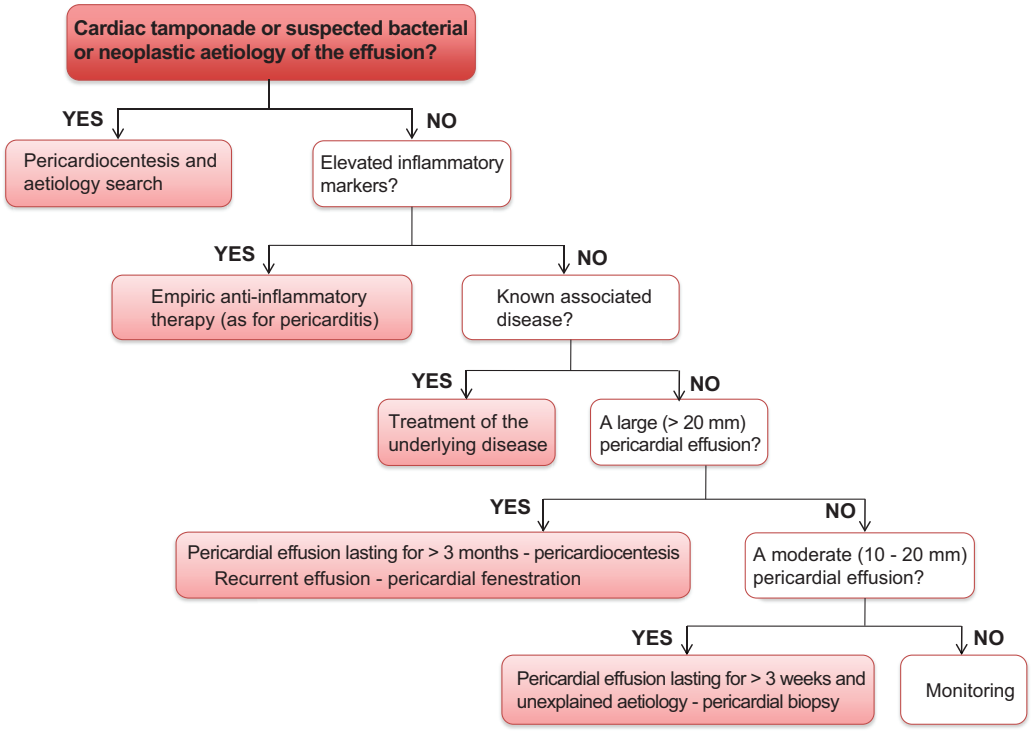

Figure 5: A simplified algorithm for pericardial effusion triage and management.

anti-inflammatory treatment is recommended (same as for pericarditis).

The prognosis of pericardial effusion is essentially related to the aetiology. Asymptomatic patients with small idiopathic pericardial effusion usually have a good prognosis of outcome and do not require specific monitoring. Moderate and especially large pericardial effusion may worsen and cause cardiac tamponade in 30–35% of cases (21). Patients with idiopathic moderate pericardial effusion should be echocardiographically monitored at 6 months, and those with large pericardial effusion at 3–6 months.

Recommendations for the therapy of pericardial effusion.

Recommendations	[a]	[b]
It is recommended to target the therapy of pericardial effusion at the aetiology.	I	C
Aspirin/NSAIDs/colchicine and treatment of pericarditis is recommended when pericardial effusion is associated with systemic inflammation.	I	C
Pericardiocentesis or cardiac surgery is indicated for cardiac tamponade or for symptomatic moderate to large pericardial effusions not responsive to medical therapy, and for suspicion of unknown bacterial or neoplastic aetiology.	I	C

[a]recommendation class; [b]evidence level; NSAIDs – non-steroidal anti-inflammatory drugs.

3.7 Cardiac tamponade

Cardiac tamponade is a life-threatening condition that occurs when pericardial effusion impairs diastolic filling of the heart till reducing cardiac output. Tamponade may be acute (e.g., with dissection of the initial part of the aorta), but it may also be a complication of a slowly increasing pericardial effusion. The causes of cardiac tamponade are described in Table 9. In the typical clinical presentation of a patient with cardiac tamponade, tachycardia, hypotension, and quieter heart tones stand out (the so-called Beck's triad), paradoxical heart rate

Table 9: Causes of cardiac tamponade.

Causes of cardiac tamponade
Common causes: • pericarditis, • tuberculosis, • iatrogenic complications, • trauma, • neoplasm.
Rare causes: • systemic connective tissue diseases (e.g., systemic lupus erythematosus, rheumatoid arthritis, scleroderma), • radiation-induced pericarditis, • epistenocardial pericarditis, • uraemia • aortic dissection, • bacterial infection, • pneumopericardium.

(pulse), elevated central venous pressure, low voltage with electrical alternans on ECG and increased cardiac silhouette on X-ray of the chest organs (with prolonged effusion). The severity of clinical and haemodynamic changes in a patient with cardiac tamponade depends in particular on the speed of fluid accumulation, on the compliance of the pericardium and the filling pressures of the cardiac cavities. Thus, the clinical picture is the most pronounced and the haemodynamic consequences are the most severe in patients with non-compliant pericardium when rapid fluid accumulation occurs. In this case, even small amounts of pericardial fluid can cause a fully developed clinical picture of tamponade and haemodynamic collapse of the patient.

Table 10: Echocardiographic signs of cardiac tamponade.

Echocardiographic feature
• inferior vena cava dilatation (diameter ≥2.5 cm) and <50% reduction of diameter with respiration, • early diastolic collapse of the right ventricle, • late diastolic collapse of the right atrium, • respiration related fluctuations in flow through the aortic valve, • paradoxical movement of the ventricular septum (towards the left ventricle during inspiration and towards the right ventricle during expiration), • a reduction in inflow velocity through the mitral valve by ≥30% in the first beat during inspiration and by ≥60% through the tricuspid valve in the first beat during expiration, • large pericardial effusion with swinging heart.

In a patient with suspected cardiac tamponade, the assessment of the clinical picture is crucial, and certain tests can be of additional help:

- The ECG may show a reduced voltage in all leads, which occurs because the pericardial fluid suppresses the myocardial electrical signal, and an electrical alternans caused by the swinging motion of the heart in the pericardial fluid.
- Transthoracic echocardiography is a key test in a patient with suspected cardiac tamponade. It can be performed bedside and in urgent/emergency situations, allowing immediate estimation of the size and location of the pericardial effusion and its haemodynamic consequences. The main echocardiographic signs of cardiac tamponade are listed in Table 10. Echocardiography is indispensable in the determination and performance of pericardiocentesis.
- CT or CMR are less useful in patients with cardiac tamponade due to limited availability and duration of examination. We perform them in the acute phase of treatment of a patient with cardiac tamponade only if the latter is a direct consequence of another acute disease that also endangers the patient (e.g., pericardial effusion with signs of tamponade and suspicion of type A aortic dissection).
- Cardiac catheterization is performed very rarely in patients with cardiac tamponade.

STEP 1 (SCORE THE AETIOLOGY)	
• malignant disease	2
• tuberculosis	2
• recent mediastinal irradiation	1
• recent viral infection	1
• recurrent pericardial effusion, previous pericardiocentesis	1
• chronic terminal renal failure	1
• immunodeficiency or immunosuppression	1
• hypothyroidism, hyperthyroidism	1
• systemic autoimmune disease	-1

STEP 2 (SCORE THE CLINICAL PRESENTATION)	
• dyspnoea, tachypnoea	1
• orthopnoea	3
• hypotension	0,5
• sinus tachycardia (in the absence of negative chronotropic drugs)	1
• oliguria	1
• oliguria	1
• pericardial chest pain	0,5
• pericardial friction rub	0,5
• rapid worsening of symptoms	2
• slow evolution of the disease	-1

STEP 3 (SCORE THE IMAGING)	
• cardiomegaly (chest X-ray)	1
• electrical alternans (ECG)	0,5
• microvoltage (ECG)	1
• large pericardial effusion (>20 mm)	3
• moderate pericardial effusion (10 – 20 mm)	1
• small pericardial effusion (<10 mm)	-1
• RA collapse for more than 1/3 of a cardiac cycle	1
• IVC dilated over 2,5 cm, inspiratory collapse <50%	1,5
• RV collapse	1,5
• LV collapse	2
• mitral/tricuspid valve respiratory flow variations	1
• swinging heart	1

TOTAL POINTS:

- **less than 6 points:** pericardiocentesis can be postponed for up to 12-24 hours,
- **6 points or more:** urgent pericardiocentesis (immediately after contraindications are ruled out)

CONTRAINDICATIONS FOR PERICARDIOCENTESIS:

- INR> 1.5 or use of NOACs,
- thrombocytopenia (<50 X 10^9/L),
- difficult to access effusion (usually posterior),
- response to aspirin/NSAID treatment.

URGENT SURGICAL MANAGEMENT (REGARDLESS OF THE SCORE):

Cardiac tamponade within:

- type A aortic dissection,
- ventricular free-wall rupture after myocardial infarction,
- recent severe chest trauma,
- iatrogenic hemopericardium when the bleeding cannot be controlled percutaneously.

Figure 6: Triage of cardiac tamponade proposed by the European Society of Cardiology Working Group on myocardial and pericardial diseases.

Legend: X-ray - radiograph; ECG - electrocardiogram; RA - right atrium; IVC - inferior vena cava; RV - right ventricle; LV - left ventricle; NOAC - direct oral anticoagulant drugs; NSAIDs – non-steroidal antirheumatic drugs.

Recommendations for diagnosis and treatment of cardiac tamponade.

Recommendations	[a]	[b]
In a patient with a clinical suspicion of cardiac tamponade, echocardiography is recommended as the imaging examination of choice to assess the size and location of the effusion and its haemodynamic effect.	I	C
Urgent pericardiocentesis or cardiac surgery is recommended to treat cardiac tamponade.	I	C
The time and the method of pericardial drainage are decided on individually according to the patient's clinical picture, haemodynamic condition and echocardiographic findings.	I	C
A triage system may be considered to guide the timing of pericardiocentesis (Figure 6).	IIb	C
The use of vasodilators and diuretics is not recommended in patients with cardiac tamponade.	III	C

[a]recommendation class; [b]evidence level.

In a patient with cardiac tamponade, pericardial drainage should be performed while ensuring sufficient filling pressures of the heart (intravenous fluid replacement). The latter can be performed percutaneously (pericardiocentesis), or surgically. Immediate pericardial drainage is required in patients who are haemodynamically unstable and in whom tamponade has developed as part of type A aortic dissection, free-wall cardiac rupture or iatrogenic injury. In other patients, a triage system may be considered to guide the timing of pericardiocentesis (Figure 6) (22).

3.8 Constrictive pericarditis

Constrictive pericarditis can occur as a result of any pericardial disease, but rarely as a result of recurrent idiopathic pericarditis (23). The risk of developing constrictive pericarditis is significantly related to the underlying cause of acute pericarditis. The risk of developing constrictive pericarditis is low (< 1%) in idiopathic pericarditis, moderate (2–5%) in immune-mediated or neoplastic pericarditis, and high (20–30%) in bacterial (especially purulent) pericarditis. In developed countries, the most common reported causes were viral (42–49%), post-cardiac surgery (11–37%), post-radiation therapy (9–31%), systemic connective tissue diseases (3–7%) and bacterial (3–6%). In developing countries, TB infection is still the leading cause of constrictive pericarditis. However, TB related constrictive pericarditis is also increasing in developed countries, probably at the expense of an increase in the population with HIV.

From the point of view of haemodynamics, a key change in constrictive pericarditis is the reduced diastolic function of the left and/or right ventricle. Thereby, in the classic clinical picture of constrictive pericarditis, there are signs of right-sided heart failure with relatively preserved systolic function of both ventricles. Patients with constrictive pericarditis report decreased physical performance, dyspnoea, flexo-dyspnoea, and peripheral oedema, ascites as well as pleural effusions may be found during a clinical examination. The delay between the acute phase of pericarditis and the development of signs of constriction is highly variable and largely depends on the cause of the disease.

The diagnosis of constrictive pericarditis is made on the basis of a combination

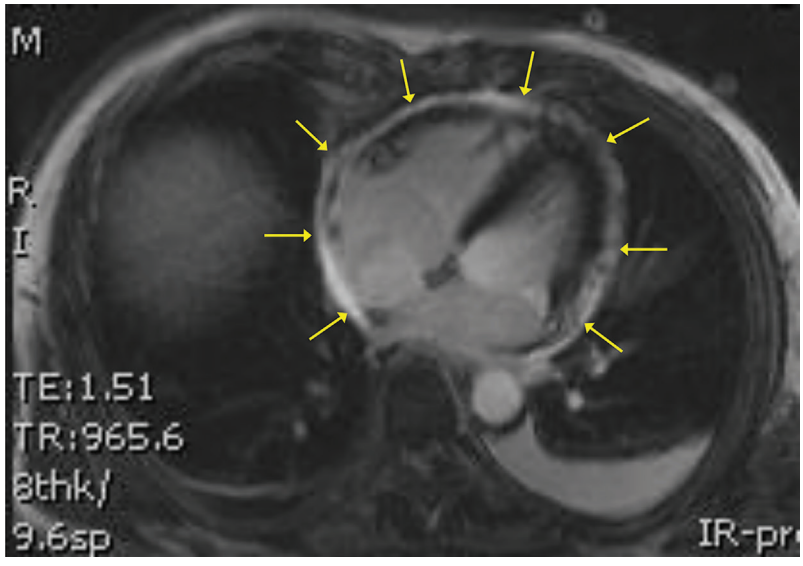

Figure 7: Cardiac magnetic resonance imaging (CMR) - a sequence for late gadolinium enhancement (LGE) for a newly diagnosed constrictive pericarditis. Yellow arrows indicate late gadolinium enhancement suggesting an active pericardial inflammation.

of the clinical picture of the predominant right-sided heart failure and evidence of an impaired diastolic filling due to pericardial constriction by one or more imaging methods including echocardiography, CT, CMR and cardiac catheterization (Figure 7, Figure 8). It is very important to be aware that in 20% of patients with constrictive pericarditis, the pericardial thickness may be normal, and that the absence of a thickened pericardium does not yet rule out the diagnosis. The main differential diagnosis of constrictive pericarditis is restrictive cardiomyopathy (Table 11). Within the general diagnosis of constrictive pericarditis, we distinguish three specific syndromes: transient constrictive pericarditis, constrictive pericarditis with associated pericardial

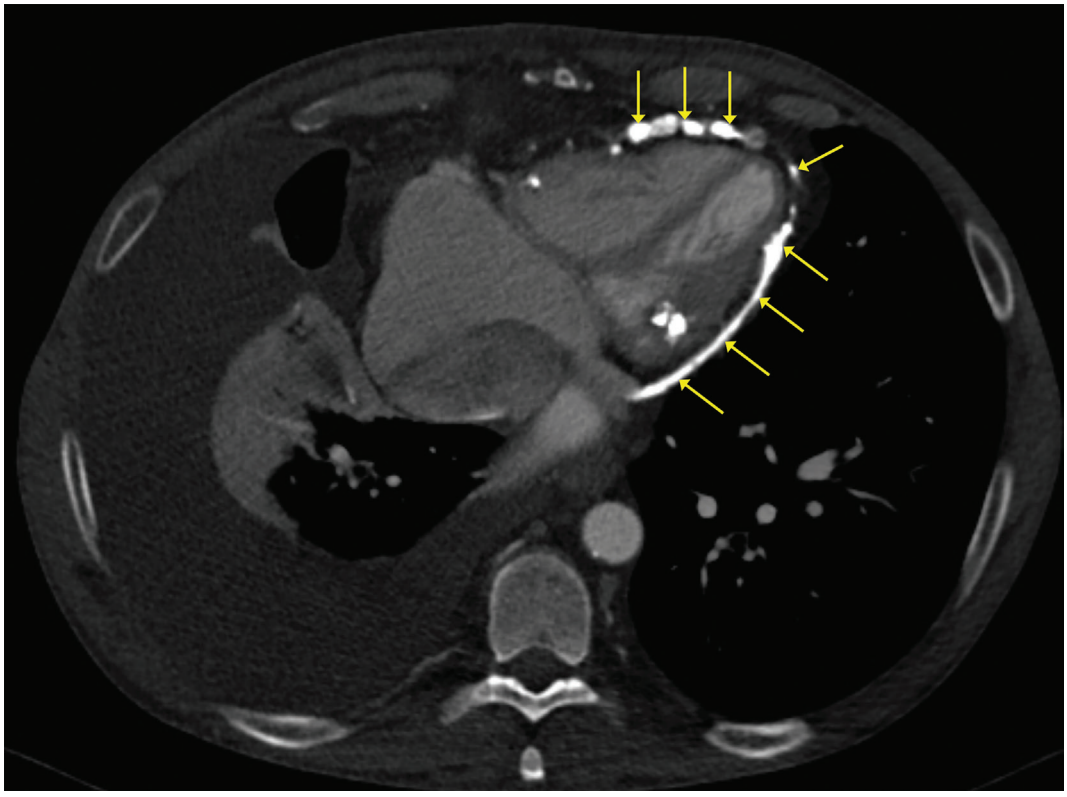

Figure 8: CT angiography of the heart in chronic constrictive pericarditis. Yellow arrows indicate a thickened and calcined pericardium.

Table 11: Constrictive pericarditis vs. restrictive cardiomyopathy: a brief overview of features for the differential diagnosis.

Diagnostic assessment	Constrictive pericarditis	Restrictive cardiomyopathy
Clinical signs:	• Kussmaul sign, a pericardial knock.	• Regurgitant murmur, Kussmaul sign, S3 (rare).
ECG:	• AF, low voltages, non-specific ST /T changes.	• AF, low voltage, left axis, broad QRS.
Chest X-ray:	• Pericardial calcifications (30% of cases).	• No pericardial calcifications.
Echocardiography:	• pericardial thickening and calcifications, • mild/moderate atrial enlargement • paradoxical movement and the 'septal bounce' of the ventricular septum, • respiratory variation of the mitral peak E wave velocity of >25% and variation in the pulmonary venous peak D wave flow velocity of >20%, • TDI: high e' sept ≥8,0 cm/s and low e' lat (e' sept/e' lat <1), • marked diastolic reversal of blood flow in the hepatic veins during expiration.	• normal appearance of the pericardium, • severe atrial enlargement, • the movement of the ventricular septum is not dependent on respiration, • respiratory changes in blood flow rate through the AV valves <15 %, • E/A ratio >2, short deceleration time, • TDI: e' sept <8 cm/s, • diastolic reversal of blood flow in the hepatic veins during expiration.
Invasive haemodynamics:	• the square root phenomenon, • equalization of right and left ventricular diastolic pressures, • marked ventricular interdependence.	• greatly elevated pulmonary pressure, • LVEDP > RVEDP for at least 5 mmHg.
CT/CMR:	• thickened pericardium ≥ 3 mm, • pericardial calcifications.	• thickness od the pericardium < 3 mm, • morphological and functional signs of myocardial damage on CMR.

AF – atrial fibrillation; X-ray – radiograph; CT – computed tomography; CMR – cardiac magnetic resonance; LV – left ventricle; RV – right ventricle; LVEDP – left ventricular end-diastolic pressure; RVEDP – right ventricular end-diastolic pressure, TDI – tissue Doppler imaging; AV valves – atrioventricular valves; e' sept – tissue doppler velocity of septal mitral annulus; e' lat – tissue doppler velocity of lateral mitral annulus; E/A ratio – transmitral E/A wave velocity ratio; 'septal bounce' – a sudden posterior displacement of the ventricular septum in early diastole during inspiration.

Table 12: Definitions and therapy of main constrictive pericardial syndromes.

Syndrome	Definition	Therapy
Transient constrictive pericarditis	Reversible pattern of constriction following spontaneous recovery or medical therapy.	Empiric anti-inflammatory treatment (2–3 months).
Effusive-constrictive pericarditis	Failure of the right atrial pressure to fall by 50% or to a level below 10 mmHg after pericardiocentesis. May be diagnosed also by non-invasive imaging.	• Pericardiocentesis followed by medical therapy. • Surgery for persistent cases.
Chronic constrictive pericarditis	Persistent signs of constriction 3–6 months after initial symptoms/signs.	• Surgical decortication. • Symptomatic therapy of heart failure and anti-inflammatory treatment in patients in whom surgery is too risky or if there is an overgrowth of the calcified pericardium into the myocardium.

Recommendations for management of constrictive pericarditis.

Recommendations	[a]	[b]
The main form of treatment for chronic constrictive pericarditis is pericardiectomy.	I	C
Medical therapy of specific pericarditis (i.e. tuberculous pericarditis) is recommended to prevent the progression of constriction.	I	C
Empiric anti-inflammatory therapy may be considered in cases with transient or new diagnosis of constriction with concomitant evidence of pericardial inflammation (CRP elevation or pericardial enhancement on CT/CMR).	IIb	C

[a]recommendation class; [b]evidence level; CRP – C-reactive protein; CT – computed tomography; CMR – cardiac magnetic resonance.

effusion, and chronic constrictive pericarditis. The main characteristics and treatment of these forms are presented in Table 12.

The final and the most effective form of treatment of chronic constrictive pericarditis is surgical treatment – pericardial decortication. Nevertheless, drug treatment is recommended for (24):

- specific forms of pericarditis when there is a targeted treatment (e.g., TB-pericarditis);
- transient constrictive pericarditis (Table 5), when an anti-inflammatory treatment can cure the disease without surgery;
- symptomatic treatment of the developed picture of heart failure.

3.9 Masses and cysts in the pericardium

Pericardial involvement in neoplastic diseases. Primary pericardial tumours, both benign (lipomas and fibroids) as well as malign (mesothelioma, sarcoma, fibrosarcoma, haemangioma, and teratoma), are very rare. Mesothelioma, the most common malignant tumour, is incurable. Pericardial metastases are more common in lung cancer (lymphangitic spread), breast cancer (hematogenous spread), lymphomas, and leukaemias. It can also be an overgrowth of the tumour from the surrounding structures (e.g., in oesophageal cancer). Pericardial involvement in neoplastic diseases can also be the result of radiation injury and opportunistic infections. It occurs clinically as pericarditis, pericardial effusion (usually moderate to large), effusive-constrictive pericarditis, or constrictive pericarditis. Diagnostic and therapeutic pericardiocentesis is required in all patients with suspected neoplastic pericardial effusion. The diagnosis is confirmed by evidence of neoplastic cells in the pericardial fluid or by pericardial biopsy (25). Evidence of tumour markers in the pericardial fluid is also helpful. Malignant pericardial effusion recurs after pericardiocentesis in more than 50% of cases. Several methods are available to prevent recurrence: intrapericardial treatment with cytostatic agents, sclerosing and cytotoxic agents (cisplatin in lung cancer, thiotepa in breast cancer), radiation (for radiosensitive tumours such as lymphomas and leukaemias), or surgical procedures (pericardiectomy, pericardial fenestration).

Pericardial cysts are rare masses in the mediastinum that are not associated with the pericardial space. They occur in 1 in 100,000 patients and represent 6% of the masses and 33% cysts in the mediastinum. Inflammatory cysts include pseudocysts and encapsulated pericardial effusion in rheumatic diseases, bacterial infections, injuries, and heart surgery. Echinococcal pericardial cysts most commonly originate from a rupture of a hydatid cyst in the liver or lung. The differential diagnosis includes localized pericardial effusion and neoplasms in the pericardium. In addition to a transthoracic ultrasound examination of the heart, there are methods of choice for the diagnosis of CT of the heart and/or CMR, which show the size and density of the cyst content and the surrounding

Recommendations for the diagnosis and management of neoplastic involvement of the pericardium.

Recommendations	[a]	[b]
Pericardiocentesis is recommended for cardiac tamponade to relieve symptoms and establish the diagnosis of malignant pericardial effusion.	I	B
Cytological analysis of pericardial fluid is recommended for the confirmation of the neoplastic pericardial disease.	I	B
Pericardial or epicardial biopsy should be considered for the confirmation of malignant pericardial disease.	IIa	B
Tumour marker testing should be considered for distinguishing malignant from benign effusions in pericardial fluid.	IIa	B
Systemic antineoplastic treatment is recommended in confirmed cases of neoplastic aetiology.	I	B
Prolonged pericardial drainage is required in patients with suspected or confirmed neoplastic pericardial effusion to prevent effusion recurrence and provide intrapericardial therapy.	I	B
Intrapericardial instillation of cytostatic/ sclerosing agents should be considered since it may prevent recurrences in patients with malignant pericardial effusion.	IIa	B
Intrapericardial cisplatin should be considered in pericardial involvement in the course of lung cancer and intrapericardial instillation of thiotepa should be considered in breast cancer pericardial metastases.	IIa	B
Radiation therapy is considered for pericardial effusion in the context of radiosensitive primary tumours such as e.g., with lymphoma and leukaemia.	IIa	B
Pericardiectomy is considered if pericardiocentesis is not feasible.	IIa	B
Percutaneous balloon pericardiectomy is considered to prevent recurrence of neoplastic pericardial effusion.	IIb	B
Pericardial window creation via left minithoracotomy may be considered in the surgical treatment of malignant cardiac tamponade.	IIb	B
Interventional techniques should consider seeding of neoplastic cells, patient prognosis and the overall quality of life of the patients.	IIa	C

[a]recommendation class; [b]evidence level.

structure. Cysts are usually clinically silent and are detected incidentally, but can cause chest discomfort, and palpitations with pressure. Symptomatic congenital and inflammatory cysts are treated by percutaneous aspiration. Surgical removal is recommended when the diagnosis is unclear and in case of recurrence.

4 Multimodal diagnosis of pericardial disease

In patients with pericardial diseases, a variety of invasive and non-invasive diagnostic methods can be used in the diagnostic procedure, and their choice depends on the initial clinical presentation, suspicious aetiology and the patient's hemodynamic stability. In the event of hemodynamic instability of the patient, it is important that the diagnosis and appropriate measures to stabilize the patient are performed simultaneously. The main imaging methods used in patients with pericardial diseases, their characteristics in individual disease states and a comparison between them are described in more detail in Table 13 and Table 14 (7).

5 Specific aetiology of pericardial syndromes

The recommended diagnostic tests for more common pericardial conditions in high-risk patients are shown in Table 15.

Viral pericarditis. In the developed countries, the most common causes

Table 13: Diagnostic contribution of the different imaging modalities in various pericardial diseases.

	Echocardiography	Computed tomography	Cardiac magnetic resonance
Acute pericarditis:	• the test results can be normal, • thickened and hyper-reflective pericardial layers, • variable amount of pericardial fluid ± intrapericardial fibrinous strands, • wall motion abnormalities in myopericarditis.	• thickened pericardial layers enhancing after contrast administration, • the size of pericardial effusion • ± intrapericardial fibrinous strands.	• thickened pericardial layers, • strong pericardial LGE following contrast administration, • the size of pericardial effusion ± fibrin fibrinous strands, • inspiratory septal flattening, • assessment of associated myocarditis.
Recurrent pericarditis:	• similar to that of acute pericarditis.	• similar to that of acute pericarditis, • uneven distribution of fibrous adhesions.	• similar to that of acute pericarditis, • uneven distribution of fibrous adhesions.
Chronic constrictive pericarditis:	• thickened and hyperechoic pericardium, • ± ascites, • ± pericardial effusion • atrial enlargement, • the paradoxical movement of the interventricular septum, • respiratory changes in flow rate through the mitral valve >25 %, • respiratory changes in flow rate in the pulmonary veins >20%, • marked diastolic reversal of blood flow in the hepatic veins during expiration, • systolic function may be normal for both ventricles.	• thickened pericardium ± pericardial calcification, • degrees of pericardial thickening, • changes are usually more pronounced at the base of the ventricle (RV > LV), atrioventricular grooves and atria, • expansion of calcifications into the adjacent myocardium, • compression of cardiac contents by rigid, deformated pericardium, • dilated atria, caval/hepatic veins, • contrast reversal in caval/hepatic veins in the expiration, • ± ascites, • ± pleural effusion.	• thickened pericardial layers, • changes are usually more pronounced at the base of the ventricles (RV > LV), AV grooves and atria, • pericardial LGE reflects residual inflammation, • compression of cardiac contents by rigid, deformed pericardium, • dilated atria, caval/hepatic veins, • contrast agent reverse flow in the venae cavae and hepatic veins in the expiration, • ± ascites, • ± pleural effusion, • assessment of increased ventricular interdependence.
Pericardial effusion:	• fluid accumulation in pericardial sac and/or pericardial sinuses, • pericardial echolucent space throughout cardiac cycle, • fluid distribution, • semi-quantitative assessment of effusion severity.	• volume and distribution of fluids in the pericardium, • pericardial width > 4 mm, • assessment of localized effusions, • attenuation values of pericardial effusion (HU may indicate the nature of the effusion (HU 0–20: simple effusion, HU >20: proteins/blood), • assessment of inflammation in the pericardium, • assessment of other thoracic structures.	• volume and distribution of fluids in the pericardium, • pericardial width > 4 mm, • assessment of localized effusions, • assessment of the nature of the pericardial effusion, • assessment of inflammation in the pericardium, • assessment of other thoracic structures.
Cardiac tamponade:	• assessment of pericardiocentesis management, • guide and monitoring pericardiocentesis, • re-evaluation for timing catheter removal.		

LGE – late gadolinium enhancement; LV – left ventricle; RV – right ventricle, AV grooves – atrioventricular grooves; HU – Hounsfield units.

Table 14: Comparison of non-invasive imaging methods in the treatment of pericardial disease.

	Echocardiography	CT	CMR
Technical aspect			
Availability	+++	++	+
Costs	low	middle range	high
Exam duration (min)	15–30	10	30–40
Safety	+++	+	++
Patient monitoring	+++	++	+/-
Pericardium			
Pericardial thickness	+/-	+++	+++
Calcifications	+	+++	+
Inflammation	+/-	++	+++
Adhesions	++	+	+++
Effusion detection	++	+++	+++
Effusion characterization	+	++	++
Pericardial massess	+	+/++	++/+++
Guiding/monitoring pericardiocentesis	+++	/	/
Cardiac morphology			
Tissue characterization	++	++	+++
Cardiac function			
Systolic function	+++	++	+++
Diastolic function	+++	/	++
Septal motion (coupling)	+++	+/-	+++
Respiratory changes	++	+/-	++

CT – computed tomography; CMR – cardiac magnetic resonance.

Recommendations for the diagnosis and treatment of viral pericarditis.

Recommendations	[a]	[b]
For the definite diagnosis of viral pericarditis, a comprehensive workup of histological, cytological, immunohistological and molecular investigations in pericardial fluid and peri-/epicardial biopsies should be considered.	IIa	C
Routine viral serology is not recommended, with the possible exception of HIV and HCV.	III	C
Corticosteroid therapy is not recommended in viral pericarditis.	III	C

[a]recommendation class; [b]evidence level; HIV – human immunodeficiency virus; HCV – hepatitis C virus.

of pericarditis are cardiotropic viruses (Table 3). They damage the pericardium through a direct cytolytic or cytotoxic effect (e.g., enteroviruses) or through an immune-mediated mechanism (herpes viruses). Serological tests for viral agents of pericarditis, except for HIV and HCV, are no longer performed routinely (26). Confirmed viral pericarditis would require extensive diagnostic treatment with histological, cytological, immunohistochemical, and molecular analysis of pericardial fluid and biopsy, which is rarely performed. In the absence of the above evidence, we speak of "alleged viral or. idiopathic pericarditis". A viral or idiopathic pericarditis is usually a self-limiting disease with a good response to experiential treatment with anti-inflammatory drugs. Corticosteroids are not recommended in viral pericarditis because they can reactivate a viral infection and cause chronic inflammation.

Bacterial pericarditis is rare in the developed world (Table 3), while TB infection is the most common cause of pericarditis in developing countries.

In the endemic region, **TB-pericarditis** is the cause of pericardial effusion in 50–70% of cases, and in HIV-infected patients, in more than 90% of cases. It can manifest clinically as pericardial effusion, effusive-constrictive pericarditis, and constrictive pericarditis. It affects men more often. 6 months

Table 15: The recommended diagnostic tests for more common pericardial conditions.

Clinical condition	Blood tests	Imaging tests	Pericardial fluid tests	Other
Suspected autoimmune cause:	• ANA, ENA, ANCA; • ACE and Ca in 24-hour urine (sarcoidosis?), • Ferritin (Still's disease?).	• PET (Horton's disease, Takayasu arteritis, sarcoidosis?).		• Consultation with a rheumatologist, • Eosinophilia (Churg-Strauss syndrome?), • Ulcerations (Behçet's disease?) • Dry eyes (Sjögren's syndrome, sarcoidosis?), • Macroglossia (amyloidosis?).
Suspected tuberculosis:	• QuantiFERON test.	• Chest CT.	• BBacilli staining, • Cultures, • TB-PCR, • ADA, IFN–gamma.	
Suspected neoplastic disease:	• Tumour markers*.	• Chest and abdomen CT, • Considered PET.	• Cytology, • CEA >5 ng/ml, • CYFRA21–1 >100 ng/ml.	• Considered pericardial biopsy.
Suspected viral infection:	• PCR for cardiotropic virusesa, • Serology: HIV, HCV.		• PCR: enteroviruses, adenoviruses, Parvovirus B19, EBV, CMV, HHV-6.	• Consultation with an infectiologist when the tests are positive.
Suspected bacterial infection:	• Blood cultures, • Coxiella burnetii, Borrelia burgdorferi serology.	• Chest CT.	• Aerobic and anaerobic cultures.	• Considered pericardial biopsy.
Chronic pericardial effusion:	• TSH, • Renal retention.			• Tests for tuberculosis and malignancies.
Suspected constriction:	• NT-proBNP.	• CMR, CT, biventricular catheterisation of the heart.		• Tests for TB.

* CA 125 is often elevated in serous effusions; PCR – polymerase chain reaction; ADA – adenosine deaminase, IFN – interferon; HIV – human immunodeficiency virus; HCV - hepatitis C virus; EBV – Epstein-Barr virus; CMV – cytomegalovirus; HHV-6 – human herpes simplex virus; CEA – carcinoembryogenic antigen; CYFRA – cytokeratin; TSH – thyroid stimulating hormone; NT-proBNP – amino-terminal fragment of B-natriuretic peptide; CT – computed tomography; CMR – cardiac magnetic resonance; PET – positron emission tomography.

after diagnosis, mortality is 17–40%. If TB-pericarditis is suspected, pericardiocentesis is essential. The diagnosis of TB-pericarditis is confirmed if tuberculosis bacilli are detected in the pericardial fluid or pericardial biopsy by culturing, or DNA with the help of PCR. The diagnosis of TB-pericarditis is likely with elevated markers in the pericardial fluid (ADA test, interferon-γ assay), with evidence of active TB of other organs (positive cultures from faeces, urine, gastric aspirate and lymphadenopathy on chest radiograph) or in response to antitubercular treatment in the endemic area. Extrapulmonary TB is treated with

Recommendations for the diagnosis and treatment of tuberculous pericarditis and effusion.

Recommendations	[a]	[b]
Diagnostic pericardiocentesis should be considered in all patients with suspected tuberculous pericarditis.	IIa	C
In tuberculous pericarditis, the use of intrapericardial urokinase may reduce the risk of constriction onset.	IIb	C
In patients living in non-endemic areas, empiric antituberculosis treatment is not recommended when systematic investigation fails to yield a diagnosis of tuberculous pericarditis.	III	C
In patients living in endemic areas, empiric antituberculosis chemotherapy is recommended for exudative pericardial effusion, after excluding other causes.	I	C
In tuberculous pericarditis, corticosteroids can only be introduced in HIV-negative patients.	IIb	C

[a]recommendation class; [b]evidence level; HIV – human immunodeficiency virus.

rifampicin, isoniazid, pyrazinamide, and ethambutol for 2 months, followed by isoniazid and pyrazinamide for 4 months. A serious complication is the development of constrictive pericarditis, which usually occurs within 6 months. The incidence of constrictive pericarditis decreased to 17–40% after the introduction of rifampicin (27). The use of intrapericardial urokinase and 6-week treatment with high-dose corticosteroids in HIV-negative patients also further reduce the risk of developing constriction.

Purulent pericarditis is rare and occurs in less than 1% of cases. The most common causative agents are streptococci, staphylococci, and pneumococci with associated empyema (50%) or pneumonia (30%). It is a life-threatening condition that is fatal if not treated in time. With decisive treatment, patient survival is 85%. When purulent pericarditis is suspected, urgent pericardiocentesis, which is both diagnostic and therapeutic, should be performed. Purulent pericardial effusion is characterized by a low ratio of glucose in pericardial fluid and serum (mean 0.3) and an increased number of leukocytes with predominant neutrophils (mean 2.8/μL, 92% of neutrophils). The pericardial fluid is sent

Recommendations for the treatment of constrictive tuberculous pericarditis.

Recommendations	[a]	[b]
Standard antituberculosis drugs for 6 months is recommended for the prevention of tuberculous pericardial constriction.	I	C
Pericardiectomy is recommended if the patient's condition is not improving or is deteriorating after 4–8 weeks of antituberculosis therapy.	I	C

[a]recommendation class; [b]evidence level.

Recommendations for the diagnosis and treatment of purulent pericarditis.

Recommendations	[a]	[b]
When purulent pericarditis is suspected, urgent pericardiocentesis should be performed.	I	C
Pericardial fluid should be sent for microbiological tests, and blood culture samples should be taken.	I	C
Effective pericardial drainage is crucial in the treatment of purulent pericarditis.	I	C
Intravenous antibiotics are recommended for the treatment of purulent pericarditis.	I	C
Subxiphoid pericardiotomy and pericardial lavage can also be considered.	IIa	C
Intrapericardial thrombolysis is recommended for localized pericardial effusion.	IIa	C
Pericardiectomy is to be considered with adhesions, localized or dense purulent effusion, recurrences and tamponade, persistent infection, and constriction.	IIa	C

[a]recommendation class; [b]evidence level.

for microbiological examination. In the treatment of purulent pericarditis, urgent pericardiocentesis with drainage and the introduction of an intravenous antibiotic before the isolation of individual infections are crucial (28). Subxiphoid pericardiotomy and pericardial lavage can also be considered.

Pericarditis in end-stage renal disease (29) can occur in three different forms: uremic pericarditis before or within 8 weeks of kidney transplantation, dialysis pericarditis (≥ 8 weeks after the start of dialysis treatment), and rarely, constrictive pericarditis. Pericardial involvement as part of end-stage renal disease is most commonly manifested as acute pericarditis (due to accumulation of toxic metabolites), chronic pericardial effusion (due to chronic hypervolemia), and rarely as constrictive pericarditis. Pericarditis as part of renal failure is characterized by the absence of chest pain and typical changes in the ECG. Pericardial effusion is often haemorrhagic due to uremic coagulopathy, so caution should be exercised in anticoagulant treatment before initiating dialysis. Uremic pericarditis is treated with dialysis, which is intensified if necessary. In case of poor responsiveness, pericardiocentesis, or drainage are recommended. NSAIDs and corticosteroids are also advised in unresponsive patients, while colchicine is contraindicated.

Recommendations for the diagnosis and treatment of pericarditis in renal failure.

Recommendations	[a]	[b]
Dialysis should be considered in uraemic pericarditis.	IIa	C
When patients with adequate dialysis develop pericarditis, intensifying dialysis should be considered.	IIa	C
If there is no improvement with dialysis treatment, pericardiocentesis and/or pericardial drainage is required.	IIb	C
NSAIDs and corticosteroids (systemic or intrapericardial) may be considered when intensive dialysis is ineffective.	IIb	C
Colchicine is contraindicated in patients with pericarditis and end-stage renal disease.	III	C

[a]recommendation class; [b]evidence level; NSAIDs – non-steroidal anti-inflammatory drugs.

Pericardial involvement in systemic connective tissue diseases may present as pericarditis or a clinically silent pericardial effusion. Rarely, the first occurrence of systemic connective tissue disease reflects the level of activity of the underlying disease. It is common in systemic lupus, Sjögren's syndrome, rheumatoid arthritis, scleroderma, systemic vasculitis, Behçet's disease, sarcoidosis, and in chronic inflammatory bowel disease (Table 3). It can occur with comorbid myocarditis, which should be ruled out. Recurrences are common (30). Treatment is aimed at alleviating the underlying systemic disease, with the cooperation of specialist rheumatologists (12). A special group are patients with recurrent fever or autoinflammatory disease. It is an inherited disease characterized by a mutation in genes that regulate an inflammatory response, independent of T lymphocytes or antibodies. The most common autoinflammatory syndromes are familial Mediterranean fever (serositis episodes last 1–3 days) and recurrent, TNF receptor-associated fever syndrome (relapses last several weeks). We consider autoinflammatory disease with pericarditis in the family history of with recurrent fevers, poor responsiveness to colchicine, and the need for immunosuppressive therapy. The disease is proven by genetic testing. Interleukin 1 (anakinra) antagonist has been shown to be effective in treatment (14).

Post-cardiac injury syndrome includes post-myocardial infarction pericarditis (Dressler's syndrome), post-pericardiotomy syndrome and post-traumatic pericarditis (either iatrogenic or not). The main triggers of inflammation are bleeding into the pericardium and incision

Table 16: Diagnostic criteria for post-cardiac injury syndromes.

Two of the five diagnostic criteria are required to confirm a diagnosis
Fever without alternative cause.
Pericarditic or pleuritic chest pain.
Pericardial or pleural friction rubs.
Pericardial effusion.
Pleural effusion with elevated CRP.

CRP – C-reactive protein.

of the pleura. The immune-dependent mechanism of inflammation is supported by a latent period generally of a few weeks until the apperance of the furst manifestations and a good response to anti-inflammatory treatment with the possibility of recurrences. Two of the five diagnostic criteria are required for diagnosis (Table 16). Treatment of post-cardiac injury syndrome is based on empiric anti-inflammatory treatment. The scheme is similar to that of acute pericarditis (Table 5).

Recommendations for the treatment and prevention of the post-cardiac injury syndrome.

Recommendations	[a]	[b]
Aspirin is recommended as a first choice for anti-inflammatory therapy of post-myocardial infarction pericarditis and those patients already on antiplatelet therapies.	I	C
Colchicine added to aspirin or NSAIDs should be considered for the therapy of the post-cardiac injury syndrome, as in acute pericarditis.	IIa	B
Colchicine should be considered after cardiac surgery using weight-adjusted doses (i.e. 0.5 mg once for patients ≤70 kg and 0.5 mg twice daily for patients > 70 kg) for 1 month for the prevention of post-cardiac injury syndrome if there are no contraindications and it is tolerated.	IIa	A
Due to the risk of progression to constrictive pericarditis, patients with post-cardiac injury syndrome should be monitored by echocardiography every 6–12 months.	IIa	C

[a]recommendation class; [b]evidence level; NSAIDs – non-steroidal anti-inflammatory drugs.

It is important to distinguish postcardiotomy syndrome from isolated pleural or pericardial effusion, which occurs as a mechanical complication after heart surgery. Evidence of inflammatory activity is crucial. Anti-inflammatory treatment of pericardial effusion without signs of inflammation is not recommended. A small, clinically silent pericardial effusion usually resorbs within 7-10 days. The development of cardiac tamponade in the first few hours after surgery is due to bleeding into the pericardium and requires surgical revision. Colchicine at low doses, adjusted to the patient's body weight, has been shown to be successful in preventing the development of post-cardiotomy syndrome after heart surgery (31). The prognosis of patients with post-cardiac injury syndrome is relatively good; recurrence occurs in < 4%, cardiac tamponade in < 2%, and constrictive pericarditis in 3% of cases (32).

Post-myocardial infarction pericarditis may occur early (after 2–3 days, epistenocardial pericarditis) or late (after 1–2 weeks, Dressler's syndrome). In the period of primary percutaneous interventional interventions on coronary arteries, the occurrence of early and late pericarditis following AMI is rare (< 1%). Diagnosis and treatment of epistenocardial pericarditis are the same as for acute pericarditis, and of Dressler's syndrome the same as for post-cardiac injury syndrome. With concomitant pericardial effusion > 10 mm, subacute heart rupture should be ruled out. Although the occurrence of pericarditis following AMI indicates a more extensive myocardial necrosis or a late coronary reperfusion, is not associated with poorer outcome prognosis in patients.

Traumatic pericardial effusion and hemopericardium. Percutaneous surgery on the heart (percutaneous surgery on the coronary arteries, pacemaker insertion, radiofrequency ablation)

Recommendations for the treatment of traumatic pericardial effusion and hemopericardium.

Recommendations	[a]	[b]
Urgent imaging technique (transthoracic echocardiogram or CT) is indicated in patients with a history of chest trauma and systemic arterial hypotension.	I	B
Immediate thoracotomy is required for cardiac tamponade due to penetrating chest injury.	I	B
In the setting of aortic dissection with hemopericardium, controlled pericardial drainage of very small amounts of the hemopericardium should be considered to temporarily stabilize the patient in order to maintain blood pressure at about 90 mmHg.	IIa	C
Pericardiocentesis as a bridge to thoracotomy may be considered in cardiac tamponade due to penetrating trauma to the heart and chest.	IIa	B

[a]recommendation class; [b]evidence level; CT – computed tomography.

can cause perforation of the coronary artery or cardiac cavity with the development of hemopericardium and cardiac tamponade. Immediate ultrasound diagnostics and drainage are crucial. Hemopericardium can also occur with penetrative and blunt chest injuries. If aortic dissection with tamponade occurs, drainage of a small amount of hemopericardium is required to temporarily stabilize the patient and maintain systolic blood pressure around 9 mmHg (33).

Radiation-induced pericarditis is most commonly caused by irradiation due to Hodgkin's lymphoma, breast cancer, and lung cancer. Pericarditis with or without effusion usually occurs early after irradiation, and in 20% of cases, even up to two years later. Radiation-induced pericarditis clinically manifests as effusive-constrictive or constrictive pericarditis (34). Pericardial effusion after irradiation is usually large, haemorrhagic, and prone to fibrous adhesions. Radiation-induced pericarditis is treated with pericardiectomy, which is associated with an increased risk of concomitant radiation-induced myocardial damage.

Recommendations for treatment of radiation-induced pericarditis.

Recommendations	[a]	[b]
To prevent radiation-induced injury to the pericardium, irradiation with the lowest volume and absorption dose is recommended whenever possible.	I	C
Radiation-induced pericarditis is treated with pericardiectomy, which presents a risk of concomitant radiation-induced myocardial damage.	IIa	B

[a]recommendation class; [b]evidence level.

Chylopericardium is caused by the accumulation of lymph fluid in the pericardial space. It is a rare disorder of the lymphatic system, which can be primary (rare) but usually secondary to thoracic duct injury. Chylothorax is also often concomitant. Injury or damage to the thoracic duct can result from trauma, heart surgery, radiotherapy, subclavian vein thrombosis, infection (especially TB), neoplasms of the mediastinum, and acute pancreatitis. CT with or without contrast in combination with lymphangiography shows the site of injury or obstruction. Chylopericardium is characterized by a milky pericardial effusion with elevated triglyceride concentration > 500 ng/dl, cholesterol/triglyceride concentration ratio < 1, negative cultures, and predominant lymphocytes. Cardiogenic complications include cardiac tamponade, acute pericarditis, and chronic constrictive pericarditis. Chylopericardium is treated with drainage, parenteral nutrition and octreotide (100mcg subcutaneously, 3 times daily for 2 weeks). Pericardiocentesis is often not sufficient and surgical pericardiectomy and treatment of the underlying disease are required.

Pericarditis and drug-induced pericardial effusion are usually rare (Table 3). Damage to the pericardium can be caused by inhalation of polymers, serum sickness after the use of blood products

Recommendations for the diagnosis and treatment of chylopericardium.

Recommendations	[a]	[b]
Chylopericardium is characterized by a milky pericardial effusion with high triglyceride concentration >500 ng/dl, cholesterol/triglyceride concentration ratio <1, negative cultures, and predominant lymphocytes.	I	C
With large symptomatic pericardial effusion due to chylopericardium, pericardial drainage and parenteral nutrition should be considered.	IIa	C
If conservative treatment does not reduce pericardial drainage and the course of the thoracic duct is known, surgical treatment is recommended.	IIa	C
Treatment with octreotide (100 mcg s.c., 3 times daily for 2 weeks), which reduces lymph formation, can also be considered.	IIb	C

[a]recommendation class; [b]evidence level; s.c. – subcutaneously.

or serums of foreign proteins, snake and reptile venoms, reactions to foreign substance by direct introduction of substances into the pericardium (talc, magnesium sulphate), use of silicone and tetracyclines and iron exposure with beta thalassemia and asbestos. The treatment is symptomatic.

Pericardial effusion in metabolic and endocrine diseases. The most common metabolic cause of pericardial disease is hypothyroidism (35). With hypothyroidism, pericardial effusion occurs in 5–30% of cases. The effusion is usually large, but rarely causes cardiac tamponade. Diagnosis is thought of at high TSH values, bradycardia, and low voltage of QRS complexes on the ECG.

Pericardial effusion is relatively common in pulmonary arterial hypertension (in 25–30%), is usually small and rarely causes haemodynamic impairment. Pericardial effusion is a consequence of right ventricular failure. As a result, the pressure in the right ventricle, right atrium, and coronary sinus increases. Filtration and lymphatic obstruction are increased, leading to pericardial effusion (36). The presence of pericardial effusion in pulmonary arterial hypertension is associated with a poorer outcome in patients.

Signs of cardiac tamponade may be blurred with severe pulmonary arterial hypertension. Due to the high pressure in the right cavities, they remain dilated during the cardiac cycle, and in contrast, diastolic collapse of the left atrium may occur. The paradoxical movement of the interventricular septum is usually preserved.

6 Pericardial diseases in pregnancy

The most common form of pericardial involvement in pregnancy is hydropericardium (40%). It is usually a minor benign pericardial effusion that occurs in the third trimester and does not require treatment. Treatment of pericarditis during pregnancy and lactation is shown in Table 17 (37). Classic NSAIDs are allowed in the first and second trimesters. After the 20th week of gestation, all NSAIDs (except acetylsalicylic acid ≥ 100 mg daily) are prohibited because they can cause constriction of ductus Botalli and impair foetal kidney function. The lowest effective dosing of corticosteroids (with calcium and vitamin D substitutes) that can be used throughout pregnancy and while breastfeeding is recommended. Paracetamol and proton pump inhibitors are allowed during pregnancy, abut colhicine is contraindicated.

7 Interventions and surgical treatment

The aetiology of pericardial disease often remains unexplained due to the infrequent use of invasive diagnostic methods. The gold standard is still **surgical pericardial drainage and biopsy** with a subxiphoid approach. **Pericardioscopy** is a percutaneous invasive method

Table 17: Treatment of pericarditis during pregnancy and breastfeeding is shown.

Drug	Pregnancy		After childbirth
	prior to the 20th week	after 20 weeks	During breastfeeding
Aspirin, 500–750 mg/8 hours	First choice.	Must be avoided.	Preferably avoided.
NSAID (ibuprofen, indomethacin, naproxen)	Allowed.	Must be avoided.	Allowed.
Paracetamol	Allowed.	Allowed.	Allowed.
Methylprednisolone 2–8 mg/daily	Allowed.[a]	Allowed.[a]	Allowed.[a]

NSAIDs - non-steroidal anti-inflammatory drugs, [a]in combination with NSAIDs, only 10% of the active metabolite can reach the foetus.

(subxiphoid approach) that allows direct examination of the pericardial space, tissue- and fluid sampling, and intrapericardial treatment (38). **Pericardiocentesis** should be ultrasound guided with US, and haemodynamic and ECG monitoring should be provided. Complications occur in 4–10% and depend on the mode of monitoring, circumstances (urgent vs. elective pericardiocentesis) and operator experience (Table 18). Pericardiocentesis is additionally risky in laterally- or posteriorly localized, minor pericardial effusion. **Intrapericardial treatment.** With prolonged pericardial drainage, intrapericardial administration of drugs can affect the underlying disease (e.g., the introduction of cisplatin in lung cancer, thiotepa in breast cancer and triamcinolone in uremic pericarditis). **Pericardial fenestration** is usually a surgical procedure that allows for communication and drainage of pericardial fluid into the pleural space. It can also be performed percutaneously between thoracoscopy and balloon pericardiectomy. The main indications for the procedure are recurrent large pericardial effusion and cardiac tamponade, if the risk associated with pericardiectomy is high or if it is a palliative procedure to shorten the life expectancy of the patient. In recurrent pericardial effusion, balloon pericardiotomy is also considered, which creates a pericardial-pleural-abdominal window to drain the effusion. **Pericardiectomy** is the main treatment for constrictive pericarditis. The purpose of surgical decortication is to remove as much of the constrictive parietal and visceral pericardium as possible, which requires a sternotomy. If the pericardium is highly calcinated and adherent, smaller isles of the pericardial tissue may still remain after the procedure. Due to the possibility of haemorrhagic complications, surgical readiness with the possibility of immediate cardiopulmonary bypass is required.

Table 18: Complications of pericardiocentesis.

Complications of pericardiocentesis
Malignant heart rhythm disorders.
Damage to the epicardial arteries, right ventricle and liver.
Haemorrhage: hemopericardium, hemoperitoneum, hematoma of the liver.
Air embolism.
Pseudoaneurysm of the right ventricle.
Fistula between right ventricle and abdomen.

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