

ACUTE PERICARDITIS

Etiology, Fibrinous 'Bread-and-Butter' Morphology, Suppurative Complications & Cardiac Tamponade

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Learning Objectives: Acute Pericarditis

Pathophysiology & Etiology

Define normal pericardial anatomy, fluid dynamics, and barrier functions.

Classify pericarditis into infectious, immunologically mediated, and secondary etiologies.

Contrast serous, fibrinous, purulent, hemorrhagic, and caseous exudates.

Morphology & Histology

Examine gross features of serous effusion, 'bread-and-butter' fibrinous pericarditis, and purulent pus.

Analyze microscopic features of neutrophilic infiltration, fibrin deposition, and granulation tissue.

Understand mechanisms of organization leading to adhesive or constrictive pericarditis.

Clinical Pathology

Identify classic clinical triad: Sharp pleuritic positional chest pain, friction rub, and diffuse ECG ST elevation.

Recognize life-threatening complications: Cardiac tamponade (Beck's triad, pulsus paradoxus).

Correlate pathology with clinical management, pericardiocentesis cytology, and pericardiectomy.

Normal Pericardial Anatomy & Physiologic Roles

Anatomical Architecture

Fibrous pericardium: Tough, noncompliant outer flask-like fibrous sac anchored to sternum and diaphragm.

Serous pericardium: Inner double-layered serosa composed of parietal layer and visceral layer (epicardium).

Pericardial cavity: Potential space between parietal and visceral serosa.

Pericardial Fluid Dynamics

Normal fluid volume: 15–50 mL of clear, pale-yellow ultrafiltrate of plasma.

Secreted and absorbed by continuous single layer of mesothelial cells lining the serosa.

Low protein content (<3 g/dL) and low specific gravity (<1.015).

Key Physiologic Functions

Provides mechanical lubrication, reducing friction during cardiac contraction and relaxation.

Restricts excessive acute cardiac dilation during hypervolemia or strenuous exertion.

Forms an anatomical barrier preventing contiguous spread of infection from lungs or pleural spaces.

Taxonomy & Causes of Acute Pericarditis

Etiologic Category	Primary Causative Agents / Disorders	Clinical Context & Presentation
Infectious: Viral (Most Common)	Coxsackieviruses A & B, Echovirus, Adenovirus, Influenza, EBV, CMV, SARS-CoV-2	Young adults; antedated by upper respiratory or gastrointestinal illness; self-limiting
Infectious: Pyogenic Bacteria	Staphylococcus aureus, Streptococcus pneumoniae, Gram-negative rods	Direct spread from pneumonia/empyema, bloodstream seeding, post-cardiac surgery
Infectious: Mycobacterial / Fungi	Mycobacterium tuberculosis, Histoplasma capsulatum, Coccidioides	Immunocompromised or endemic areas; chronic fibrocaseous exudate; constrictive risk
Immunologically Mediated	Post-MI Dressler syndrome, Rheumatic fever, SLE, Scleroderma, Drug hypersensitivity	Delayed autoimmune response weeks post-infarction; anti-heart antibodies; multisystem autoantibodies
Miscellaneous / Non-Infectious	Acute Myocardial Infarction (early episthenocardiac), Uremia, Malignancy, Radiation, Trauma	Uremic pericarditis in severe renal failure; direct tumor seeding (lung, breast, lymphoma)

Serous Pericarditis: Pathogenesis & Gross Pathology

Non-Infectious Inflammation

Characteristically produced by noninfectious inflammatory diseases.

Classical causes: Systemic Lupus Erythematosus (SLE), Rheumatic fever, Systemic Sclerosis (Scleroderma).

Also incited by uremia and contiguous irritation from adjacent pleural bacterial inflammation.

Exudate Characteristics

Fluid is clear, straw-colored, or pale yellow with low cell count.

Typically 50–200 mL volume accumulated slowly in pericardial sac.

Exudate contains moderate protein and sparse inflammatory cells without overt purulence.

Gross Serosal Appearance

Serosal membranes remain smooth, glistening, and transparent.

Mild epipericardial capillary engorgement and hyperemia.

Rarely organizes into dense fibrous adhesions; Typically resolves completely without residual scarring.

Serous Pericarditis: Histopathology & Neoplastic Effusions

Pathology Findings

Microscopic examination reveals mild inflammatory infiltrate confined primarily to epi-pericardial adipose tissue.

Infiltrate consists predominantly of mature lymphocytes, accompanied by scattered histiocytes and plasma cells.

Mesothelial lining cells may become plump and reactive (mesothelial hyperplasia).

Malignant neoplasms (metastatic lung cancer, breast carcinoma, malignant melanoma, lymphoma) frequently produce serous or serosanguinous effusions.

Tumor cells gain access to pericardium via retrograde lymphatic permeation or direct contiguous invasion.

Diagnostic confirmation requires cytologic examination of centrifuged pericardial tap fluid (centrifuged smear, cell block, immunohistochemistry).

Diagnostic Takeaway

Cytology Pearl: Cytologic examination of aspirated serous fluid in elderly patients is critical to rule out clinically occult metastatic adenocarcinoma.

Fibrinous & Serofibrinous Pericarditis: The Classic Presentation

Frequency & Dominance

Fibrinous and serofibrinous pericarditis represent the most frequent morphologic patterns of pericarditis.

Composed of serous fluid admixed with variable, often copious amounts of dense fibrinous exudate.

Fluid appears turbid, cloudy, and yellowish with suspended fibrin flakes.

Classic Etiologic Triggers

Acute transmural myocardial infarction (early epistenocardiac pericarditis within 2–4 days).

Post-myocardial infarction Dressler syndrome (autoimmune response arising 2–8 weeks post-MI).

Uremia in end-stage chronic kidney disease;
Routine post-cardiac surgery reaction.

Rheumatic carditis, systemic lupus erythematosus (SLE), blunt chest trauma, and mediastinal radiation.

Pathophysiologic Mechanism

Marked vascular permeability allows escape of high-molecular-weight fibrinogen into the pericardial space.

Procoagulant tissue factor converts fibrinogen into insoluble, polymerized cross-linked fibrin networks.

Fibrin coats parietal and visceral serosal membranes, creating rough, shaggy surfaces.

The 'Bread-and-Butter' Morphology of Fibrinous Pericarditis

Gross Appearance

Visceral and parietal pericardium lose their normal glistening, smooth transparency.

Serosal surfaces are covered by an irregular, thick, shaggy, yellowish-pink fibrinous exudate.

Pulling the two pericardial layers apart reveals rough, bristly, irregular projections.

Classically likened in pathological anatomy to the appearance of two slices of buttered bread separated from each other ('bread-and-butter pericarditis').

Microscopic Architecture

The epicardial surface is layered with an eosinophilic meshwork of dense, amorphous polymerized fibrin.

Contains an underlying acute inflammatory infiltrate with numerous neutrophils and extravasated red blood cells.

Epipericardial capillaries are markedly congested and surrounded by edema.

Overlying mesothelial cell layer is often completely denuded by the inflammatory process.

Clinical Hallmarks of Fibrinous Pericarditis

Pathology Findings

Pain: Sharp, retrosternal or precordial chest pain; Classically pleuritic (worse on inspiration, coughing).

Positional nature: Pain is distinctly position-dependent — characteristically exacerbated by lying flat (supine) and relieved by sitting up and leaning forward.

Radiation: Often radiates to the trapezius ridge (phrenic nerve innervation of the parietal pericardium).

Pericardial Friction Rub: Pathognomonic physical finding — superficial, scratching, high-pitched grating sound heard on auscultation.

Three components of friction rub: Atrial systole, ventricular systole, and early ventricular rapid filling.

Rub dynamics: Best heard at the left lower sternal border with the patient sitting forward; Transient and disappears if effusion separates the serosal layers.

Diagnostic Takeaway

Physical Exam Pearl: A friction rub requires contact between rough serosal layers; Accumulation of a large pericardial effusion causes the rub to disappear.

Purulent / Suppurative Pericarditis: Portals of Entry

Direct Contiguous Extension

Most common route: Direct spread from neighboring thoracic infections.

Bacterial empyema of the pleural cavity, lobar pneumonia, or suppurative mediastinitis.

Direct myocardial extension: Myocardial abscess or infected aortic root ring abscess in infective endocarditis.

Hematogenous Seeding

Bloodstream seeding from distant primary infections (bacteremia, sepsis).

Staphylococcus aureus furuncles, endocarditis, osteomyelitis, or intravenous catheter sepsis.

Streptococcus pneumoniae bacteremia.

Lymphatic Spread & Iatrogenic

Retrograde lymphatic spread from subdiaphragmatic or mediastinal infected lymph nodes.

Direct introduction during open cardiectomy, pericardiocentesis, or penetrating thoracic trauma.

Suppurative Pericarditis: Morphology & High Risk of Scarring

Gross Exudate

Pericardial cavity contains creamy, yellow-green pus, frequently up to 400–500 mL in volume.

Serosal surfaces are intensely reddened, granular, and lined by thick, opaque purulent exudate.

Exudate may extend into surrounding anterior mediastinal adipose tissue (mediastinopericarditis).

Microscopic Characteristics

Dense, sheets of viable and necrotic polymorphonuclear leukocytes (neutrophils).

Abundant cellular debris, fibrin meshworks, and bacterial colonies.

Underlying epicardium shows extensive necrosis and capillary proliferation (granulation tissue).

Organization & Constrictive Outcome

Complete resolution without scarring is extremely infrequent.

Intense inflammatory destruction triggers extensive fibroblastic proliferation and collagen synthesis.

Organization leads to dense fibrous obliteration of the pericardial space, frequently resulting in constrictive pericarditis.

Hemorrhagic Pericarditis: Etiologies & Diagnostic Tap

Gross Features

Exudate composed of frank blood or heavily blood-stained fluid admixed with fibrinous or purulent material.

Must be distinguished from iatrogenic bloody tap (which clots rapidly, whereas defibrinated hemorrhagic exudate does not).

May rapidly cause cardiac tamponade if accumulation occurs acutely.

Malignant Neoplasia

Spread of malignant neoplasms is the single most common cause of hemorrhagic pericarditis.

Metastatic lung carcinoma, breast adenocarcinoma, malignant melanoma, and hematologic malignancies (lymphoma, leukemia).

Cytologic examination of aspirated fluid demonstrates atypical malignant epithelial or lymphoid cells.

Non-Malignant Causes

Tuberculous pericarditis; Severe bacterial infections.

Underlying bleeding diathesis (severe thrombocytopenia, hemophilia, therapeutic anticoagulation).

Post-cardiac surgery bleeding; Acute type A aortic dissection leaking into pericardial sac.

Caseous (Tuberculous) Pericarditis: The Fibrocaseous Cascade

Pathology Findings

Caused by *Mycobacterium tuberculosis*, typically via retrograde spread from infected mediastinal lymph nodes.

Pericardial space contains thick, turbid fluid with prominent cheesy, white-yellow caseous necrotic material.

Histology: Classical necrotizing (caseating) granulomas composed of Langhans multinucleated giant cells, epithelioid macrophages, and peripheral rim of lymphocytes.

Acid-fast bacilli (AFB) demonstrated with Ziehl-Neelsen stain or mycobacterial PCR.

Tuberculous pericarditis is the single most common antecedent cause of chronic constrictive pericarditis in non-industrialized regions.

Fibrocaseous reaction triggers massive, dense, concentric fibrous obliteration and dystrophic calcification ('calcified armored heart' / *concretio cordis*).

Diagnostic Takeaway

Global Pathology Pearl: In TB-endemic regions, tuberculosis accounts for >70% of all cases of constrictive pericarditis.

Electrocardiographic Evolution in Acute Pericarditis

Stage 1: Acute Presentation

Widespread, diffuse concave-upward ST-segment elevation across virtually all limb and precordial leads (except aVR and V1).

Reciprocal ST depression restricted exclusively to lead aVR (and occasionally V1).

PR segment depression in leads II, aVF, and V4–V6 due to atrial subepicardial injury (highly specific).

Stage 2: Normalization

Occurs several days after initial presentation.

ST segments return to baseline; T waves remain upright or flatten.

PR segment depressions resolve.

Stage 3: T-Wave Inversion

Diffuse, symmetrical T-wave inversions across leads previously showing ST elevation.

Occurs without development of pathologic Q waves (crucial distinction from acute myocardial infarction).

Stage 4: Complete Resolution

T waves gradually normalize over weeks to months, or rarely persist as permanent flat/inverted T waves.

Differential Diagnosis: Acute Pericarditis vs Acute MI

Diagnostic Feature	Acute Pericarditis	Acute Myocardial Infarction (STEMI)
Chest Pain Quality	Sharp, pleuritic, positional; improved sitting forward	Crushing, heavy pressure/tightness; unaffected by position
Pain Radiation	Trapezius ridge (phrenic nerve C3–C5 distribution)	Left arm, neck, jaw, epigastrium (dermatomal T1–T4)
Friction Rub	Common, superficial, high-pitched scratching; 3 components	Uncommon in acute phase; may appear day 2–4 in transmural MI
ECG ST Elevation	Diffuse, concave-upward across multiple non-contiguous territories	Localized to specific anatomic vascular territory (e.g., II, III, aVF)
ECG Reciprocal Changes	Absent (only in lead aVR / V1)	Prominent reciprocal ST depression in opposite territory
PR Segment Changes	PR depression common (atrial subepicardial injury)	Normal PR segment (unless atrial infarction)
Pathologic Q Waves	Never form (superficial epimyocardial inflammation only)	Frequently develop over hours to days as transmural necrosis evolves
Cardiac Biomarkers	Normal or mild elevation (troponin elevated only if myopericarditis)	Marked, exponential elevation of cardiac troponin I/T and CK-MB

Cardiac Tamponade: Pathophysiologic Triad

Pericardial Compliance Curve

The normal pericardium is elastic up to 50 mL, but becomes acutely noncompliant at higher volumes.

Rapid accumulation of even 150–200 mL of fluid produces dramatic intrapericardial pressure spikes.

Slow accumulation (e.g., in uremia, myxedema) allows gradual stretching, accommodating up to 1000–1500 mL without tamponade.

Hemodynamic Collapse

Intrapericardial pressure exceeds normal diastolic filling pressures of right atrium and ventricle.

Severe compression prevents systemic venous return, drastically reducing ventricular end-diastolic volume.

Stroke volume and cardiac output plummet; Compensatory tachycardia and systemic vasoconstriction ensue.

Beck's Clinical Triad

1. Systemic Hypotension (decreased stroke volume).
2. Jugular Venous Distension (elevated systemic venous pressures, blunted y-descent).
3. Muffled / Distant Heart Sounds (fluid layer acoustically insulates cardiac sounds).

Pulsus Paradoxus: Mechanism & Clinical Measurement

Pathology Findings

Pulsus paradoxus is defined as an abnormally large inspiratory decrease in systemic systolic blood pressure (>10 mmHg).

Physiologic basis: During normal inspiration, negative intrathoracic pressure increases venous return to the right ventricle, causing slight RV expansion.

In cardiac tamponade, total cardiac volume is strictly constrained by the rigid, pressurized pericardial fluid shell.

Ventricular Interdependence: Increased RV filling during inspiration can only occur by pushing the interventricular septum leftward into the left ventricular cavity.

Leftward septal bowing compromises LV diastolic filling, reducing LV stroke volume and systolic pressure.

Measurement: Inflate blood pressure cuff above systolic pressure, deflate slowly; Note difference between first heard Korotkoff sounds and sounds heard continuously through respiration.

Diagnostic Takeaway

Clinical Diagnostic Pearl: Pulsus paradoxus is the most sensitive physical exam finding for detecting hemodynamic cardiac tamponade.

Outcomes & Complications of Acute Pericarditis

1. Complete Resolution

Most common outcome in acute viral and idiopathic pericarditis (>80% of cases).

Exudate is resorbed by lymphatics; Damaged mesothelium regenerates smoothly.

Minimal or no permanent hemodynamic impairment.

2. Recurrent Pericarditis

Occurs in 15–30% of patients following an initial episode of idiopathic pericarditis.

Mechanisms include incomplete viral clearance or secondary autoimmune activation.

Treated with colchicine and NSAIDs;
Refractory cases respond to IL-1 receptor antagonists (anakinra, rilonacept).

3. Chronic Constriction

Organization of suppurative, caseous, or hemorrhagic exudates triggers intense fibrosis.

Dense collagen and dystrophic calcification encase the heart in a rigid fibrous armor.

Results in chronic constrictive pericarditis with severe diastolic heart failure.

Clinical Case: Post-Cardiac Injury (Dressler) Syndrome

Pathology Findings

A 64-year-old male presents 4 weeks following an uncomplicated anterior STEMI with severe sharp chest pain.

Pain is worsened by deep breathing and lying supine; Dramatically relieved by sitting upright and leaning forward.

Physical examination: Temperature 38.3°C; Loud triphasic pericardial friction rub heard along left sternal border; No pulsus paradoxus.

Laboratory findings: Leukocytosis (14,500/mcL), ESR 85 mm/hr, hs-CRP 120 mg/L; Cardiac troponin flat/baseline.

Pathophysiology: Autoimmune reaction against cardiac antigens released during myocardial necrosis, forming circulating immune complexes.

Treatment & Outcome: Prompt symptom resolution following oral high-dose NSAIDs and colchicine.

Diagnostic Takeaway

Board Pearl: Dressler syndrome occurs WEEKS post-MI (autoimmune), whereas epistenocardiac pericarditis occurs DAYS 2–4 post-MI (direct transmural inflammation).

Master Summary: Acute Pericarditis Pathology

Etiologic Spectrum

Viral (coxsackie, echovirus) is most frequent in young adults; Uremia, post-MI, and SLE are key non-infectious causes.

Suppurative pericarditis results from direct extension (pneumonia, empyema); Hemorrhagic from metastatic malignancy.

Pathologic Hallmarks

Fibrinous 'bread-and-butter' exudate with dense polymerized fibrin meshworks.

Suppurative pericarditis produces creamy pus and high risk of constrictive scarring; Caseous from tuberculosis.

Clinical Essentials

Triad of positional pleuritic chest pain, friction rub, and diffuse concave ST elevation with PR depression.

Cardiac tamponade marked by Beck's triad and pulsus paradoxus (>10 mmHg inspiratory drop), requiring urgent drainage.

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Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

Echo-free space between parietal and visceral pericardium quantifies effusion volume.

Diastolic right ventricular anterior wall collapse confirms hemodynamic tamponade.

Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

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Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 37

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

Echo-free space between parietal and visceral pericardium quantifies effusion volume.

Diastolic right ventricular anterior wall collapse confirms hemodynamic tamponade.

Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 38

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

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Echocardiographic Correlation

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Therapeutic Principles

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Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 39

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

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Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 40

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

Echo-free space between parietal and visceral pericardium quantifies effusion volume.

Diastolic right ventricular anterior wall collapse confirms hemodynamic tamponade.

Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 41

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

Echo-free space between parietal and visceral pericardium quantifies effusion volume.

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Therapeutic Principles

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Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 42

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

Echo-free space between parietal and visceral pericardium quantifies effusion volume.

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Therapeutic Principles

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Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 43

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

Echo-free space between parietal and visceral pericardium quantifies effusion volume.

Diastolic right ventricular anterior wall collapse confirms hemodynamic tamponade.

Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 44

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

Echo-free space between parietal and visceral pericardium quantifies effusion volume.

Diastolic right ventricular anterior wall collapse confirms hemodynamic tamponade.

Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 45

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

Echo-free space between parietal and visceral pericardium quantifies effusion volume.

Diastolic right ventricular anterior wall collapse confirms hemodynamic tamponade.

Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 46

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

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Therapeutic Principles

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Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 47

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

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Therapeutic Principles

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Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 48

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

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Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 49

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

Echo-free space between parietal and visceral pericardium quantifies effusion volume.

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Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 50

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

Transthoracic echocardiography (TTE) serves as the primary non-invasive imaging modality.

Echo-free space between parietal and visceral pericardium quantifies effusion volume.

Diastolic right ventricular anterior wall collapse confirms hemodynamic tamponade.

Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 51

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

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Therapeutic Principles

First-line therapy: High-dose aspirin or NSAIDs combined with weight-adjusted colchicine for 3 months.

Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 52

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

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Therapeutic Principles

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Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 53

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

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Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 54

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

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Therapeutic Principles

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Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 55

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

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Therapeutic Principles

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Colchicine depolymerizes microtubules, disrupting neutrophil chemotaxis and the NLRP3 inflammasome.

Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 56

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

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Systemic corticosteroids reserved for refractory cases or specific rheumatologic indications.

Acute Pericarditis: Advanced Histopathology & SGD Module 57

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

Echocardiographic Correlation

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Acute Pericarditis: Advanced Histopathology & SGD Module 58

Microvascular Changes

Severe microvascular endothelial activation with PECAM-1 and E-selectin upregulation.

Submesothelial edema separates collagen bundles in the parietal pericardial layer.

Perivascular cuffing by CD4+ and CD8+ lymphocytes in viral and autoimmune variants.

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Acute Pericarditis: Advanced Histopathology & SGD Module 59

Microvascular Changes

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