Postcardiac Injury Syndrome After Cardiac Surgery

An Evidence-Based Review

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Abstract: Postcardiac injury syndrome (PCIS) serves as a comprehensive term encompassing a spectrum of conditions, namely postpericardiotomy syndrome, postmyocardial infarction (MI) related pericarditis (Dressler syndrome), and post-traumatic pericarditis stemming from procedures like percutaneous coronary intervention or cardiac implantable electronic device placement. These conditions collectively give rise to PCIS, triggered by cardiac injury affecting pericardial or pleural mesothelial cells, leading to subsequent inflammation syndromes spanning from uncomplicated pericarditis to substantial pleural effusion. A thorough literature search conducted on MEDLINE/PubMed utilizing search terms including "postacute cardiac injury syndrome," "postcardiac injury syndrome," "postcardiotomy syndrome," "postpericardiotomy syndrome," and "post-MI pericarditis" was instrumental in collating pertinent studies. To encapsulate the amassed evidence, relevant full-text materials were meticulously selected and amalgamated narratively. The pathophysiology of PCIS is proposed to manifest through an autoimmune-mediated process, particularly in predisposed individuals. This process involves the development of anti-actin and antimyosin antibodies after a cascade of cardiac injuries in diverse forms. Treatment strategies aimed at preventing recurrent PCIS episodes have shown efficacy, with colchicine and nonsteroidal anti-inflammatory drugs, including ibuprofen, demonstrating positive outcomes. Conversely, corticosteroids have exhibited no discernible benefit concerning prognosis or recurrence rates for this ailment. In summary, PCIS serves as a unifying term encompassing a spectrum of cardiac injury-related syndromes. A comprehensive review of relevant literature underscores the autoimmune-mediated pathophysiology in susceptible individuals. The therapeutic landscape involves the proficient use of colchicine and Nonsteroidal anti-inflammatory drugs to deter recurrent PCIS episodes, while corticosteroids do not appear to contribute to improved prognosis or reduced recurrence rates. This nuanced understanding contributes to an enhanced comprehension of PCIS and its multifaceted clinical manifestations, potentially refining its diagnosis and management.

Key Words: postcardiac injury syndrome, pericarditis, myocardial infarction, autoimmune-mediated, colchicine

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Pericarditis, which is the inflammation of the pericardium, can lead to a condition known as postcardiac injury syndrome (PCIS).[1] This syndrome encompasses various conditions such as Dressler syndrome [postmyocardial infarction (MI)-related pericarditis], postpericardiotomy syndrome, and post-traumatic pericarditis.[2] The term "postcardiotomy syndrome" has been replaced by "postpericardiotomy syndrome" due to evidence indicating that even minor injuries to the pericardium can trigger the syndrome, not just those involving direct manipulation of cardiac structures. This can include procedures like percutaneous coronary interventions (PCI), placement of cardiac implantable electronic devices (CIEDs), or radiofrequency ablation.[3] These scenarios share a common trigger of insult to the pericardium and/or pleura, leading to a pleuropericardial syndrome involving pericarditis and pleural effusion. The pericardium is a relatively avascular structure that provides support to the heart, preventing excessive movement within the mediastinum, and helps prevent over-dilation of the cardiac chambers during diastole.[3] Neuroreceptors within the pericardium play a role in regulating heart rate and blood pressure during inspiration. The serous fluid present in the pericardial sac facilitates smooth movement of the heart within the thorax.[4,5] The pericardium is composed of fibrous (parietal) and serosal (visceral) layers that originate from mesodermal germ cells during embryonic development. The fibrous pericardium anchors the heart within the thorax, connecting to the lungs, cervical fascia, esophagus, coronary arteries' adventitia, and the descending aorta.[3] PCIS was first described by Dressler[6] as a complication of acute MI. The exact incidence of PCIS is uncertain due to heterogeneity in post-MI patients. In the era before reperfusion therapies, 1 study estimated a 3% incidence of PCIS in acute MI patients, while another study found no cases in their cohort.[7] With the advent of primary PCI and emergency coronary artery bypass graft surgery, the incidence of PCIS appears to have decreased. For example, 1 study of MI patients treated with thrombolysis reported only 1 case of PCIS out of 201 patients.[8] Another study involving patients with acute ST-elevation MI treated with primary PCI noted only a single case of late-onset pericarditis (Dressler syndrome) out of 743 patients.[9] However, after cardiac surgery, PCIS has been reported in a higher number of cases, though the incidence varies among different cohorts. Incidence rates of 15% and 9% were reported in 2 studies of patients undergoing coronary artery bypass graft surgery.[10,11] A Finnish registry of cardiothoracic surgery patients demonstrated a 1.7% incidence of PCIS.[12] In the context of CIED implantation, PCIS occurs in less than 5% of cases,[13] with 1 study reporting an incidence of 0.1%.[14] Pericardial complications occur in about 0.5% of PCI cases,[15] and the overall risk for pericardial complications and other percutaneous cardiac procedures is around 1%.[15,16] In the 21st century, particularly in developed countries, the aging population, along with expanding indications for percutaneous cardiac interventions and cardiac surgery, has underscored the importance of understanding the mechanisms and pathophysiology of PCIS.[17] The objective of this review is to provide a comprehensive update on the diagnosis, treatment, prognosis, and prevention of PCIS in a narrative format. This review aims to synthesize existing knowledge with current evidence to shed light on the complex nature of PCIS and its implications for clinical practice.

METHODS

To compile a comprehensive understanding of the topic, a systematic search was conducted using the MEDLINE/PubMed database, spanning the years 1956 to 2023. This search aimed to

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identify potentially relevant articles for inclusion in this review. The selected articles were integrated coherently and narratively to summarize the available evidence. The search strategy encompassed various relevant terms, including "postacute cardiac injury syndrome," "postcardiac injury syndrome," "postcardiotomy syndrome," "post-pericardiotomy syndrome," and "post-MI pericarditis." This approach ensured a thorough examination of the existing literature related to the subject. In addition to primary research articles, this review also incorporated conference proceedings and the latest guidelines from prominent organizations such as the European Society of Cardiology and the American Heart Association. These guidelines provide valuable insights and recommendations that contribute to a well-rounded understanding of the field. To ensure a comprehensive coverage of the topic, various types of studies were included, such as randomized controlled trials, observational studies, case reports, and reviews. By systematically compiling and integrating these diverse sources of information, this review aims to provide an up-to-date and comprehensive overview of the state-of-the-art understanding, investigation, and management of PCIS.

PATHOPHYSIOLOGY

The underlying mechanisms driving PCIS remain incompletely understood, and its pathophysiology has evolved. The earliest conceptualization of PCIS was proposed by Dressler in 1956, who suggested an auto-antigens-mediated hypersensitivity reaction following MI.[7] This process was believed to lead to myocardial necrosis, subsequently triggering pericarditis and pleuropericardial effusions in susceptible individuals. However, as subsequent reports emerged linking PCIS to valve replacement surgeries and congenital heart defect corrections, the understanding of its etiopathogenesis expanded.[18,19] Contemporary hypotheses regarding PCIS center around autoimmune processes. According to the current model, an autoimmune response is initiated when the pericardial and pleural mesothelial cells are damaged.[10,20] This damage can result from various factors such as myocardial necrosis, surgical trauma, blunt thoracic trauma, or iatrogenic harm to the pericardium.[19] Even minimally invasive procedures like percutaneous cardiovascular interventions (eg, PCI) and the placement of CIEDs can contribute to pericardial and pleural damage. When combined, these forms of damage can trigger the release of inflammatory cytokines, culminating in a hyper-inflammatory state that predisposes certain individuals to develop PCIS.[20] The potential for pericardial damage exists in invasive procedures that involve the manipulation of vessels and cardiac structures.[21] Notably, PCIS has been observed after blunt trauma, such as road traffic accidents, as well as sharp trauma like a bullet or stab wounds.[22,23] The autoimmune hypothesis is bolstered by the detection of anti-actin and antimyosin antibodies in patients undergoing cardiac surgery. Several studies have demonstrated elevated levels of these antibodies after cardiac surgical procedures, particularly among patients who later developed PCIS.[24,25] However, the exact role of these autoantibodies in pericardial and vascular injury is not fully elucidated. Insights into the autoimmune nature of PCIS emerged from studies examining antiheart antibodies. In 1 study comparing children who underwent cardiac surgery, those who developed PCIS exhibited increased levels of antimyocardial antibodies, indicating myocardial injury.[26] Similar findings were observed in adults undergoing cardiac surgery, though the precise significance of these antibodies in pericardial and vascular injury remains uncertain. Another study investigated antiheart antibodies in patients before and after cardiac surgery. Out of a group of 20 patients, 3 who developed PCIS became seropositive for antiheart antibodies within 2 weeks of the procedure.[27] This timeline suggests a potential link between antibody development and the onset of PCIS. Furthermore, the role of perioperative viral infections is being explored as a potential contributor to PCIS. Seasonal variation in PCIS incidence, coupled with elevated viral titers and antiheart antibodies, suggests a potential association between viral infections and PCIS.[28] However, the exact causal relationship requires further investigation. Interestingly, PCIS has been observed in immunosuppressed children following orthotopic cardiac transplant, implying that autoimmune processes might not be the sole driver of the syndrome in all cases.[29] In light of these complex interactions, ongoing research is needed to establish a comprehensive model for the underlying mechanisms and pathogenesis of PCIS. Figure 1 shows the major causes of PCIS.

CLINICAL PRESENTATION AND DIAGNOSTIC MODALITIES

The clinical presentation of PCIS can overlap with symptoms of acute pericarditis or pleural effusion arising from other clinical contexts. Key features that define PCIS include a predisposition to cardiac injury resulting from previous pericardial or myocardial invasion, as well as manipulation of the pleural cavity.[10] Moreover, there exists a variable latency period ranging from weeks to months between the initial injury and the emergence of pleural or pericardial effusion. Despite this variability, the clinical manifestation and course of PCIS resemble those of acute cardiac injury.[15,17] In a registry involving 360 patients, 15% developed PCIS.[10] The majority of these cases (79.6%) presented within the first month, with 13% in the second and 7.4% in the third month. Specific symptoms and signs included pleural effusion (92.6%), pericardial effusion (88.9%), elevated inflammation markers (74.1%), pleuritic chest pain (55.6%), fever (53.7%), and pericardial rub (32.3%).[30] While rare, complications such as chylothorax and hydropericardium can occur, particularly in children undergoing extensive surgical interventions for complex congenital heart diseases. The diagnostic criteria for PCIS lack standardization and often involve a process of exclusion. Initial investigations typically encompass inflammatory markers like erythrocyte sedimentation rate, C-reactive protein, troponin T or I, and complete blood count. Elevated levels of these markers are commonly observed in both acute pericarditis and PCIS (over 83% of patients), underscoring their potential utility in suspecting and monitoring PCIS.[31] Troponin T or I levels may be increased in cases involving epicardial or myocardial injury during acute events, although their specificity is limited, particularly in the context of acute MI.[32,33] Chest X-rays can reveal an enlarged cardiac silhouette in the presence of pericardial effusion or tamponade, aiding in the exclusion of alternative diagnoses for chest pain or dyspnea.[34] When diagnosing PCIS, it is important to consider other potential causes such as malignancy, uremia, and connective tissue disorders. The electrocardiogram (ECG) plays a pivotal role in distinguishing active pericarditis from PCIS. ECG changes characteristic of pericarditis include diffuse ST-segment depression and PR segment elevation in different stages.[34] However, these stages are usually absent in PCIS and instead manifest as nonspecific ST-T changes or pseudo-infarct patterns. Transthoracic echocardiography (TTE) is a readily available bedside diagnostic tool for assessing cardiac anatomy, function, and hemodynamics. It offers precise quantification for guiding pericardiocentesis in cases of pericardial effusion.[35,36] Nevertheless, TTE has limitations in visualizing pericardial anatomy effectively. Advanced cardiac imaging modalities like computed tomography and cardiac magnetic resonance provide detailed insights into complex cardiac structures, pathology, and function. They can distinguish between acute and chronic pericarditis and are particularly useful for detecting underlying inflammatory processes.[37–39] However, these modalities are less suitable for hemodynamically unstable patients, those with renal dysfunction, pregnancy, or arrhythmias, due to the need for prolonged supine positioning and breath-hold sequences, along with exposure to ionizing radiation.[40]

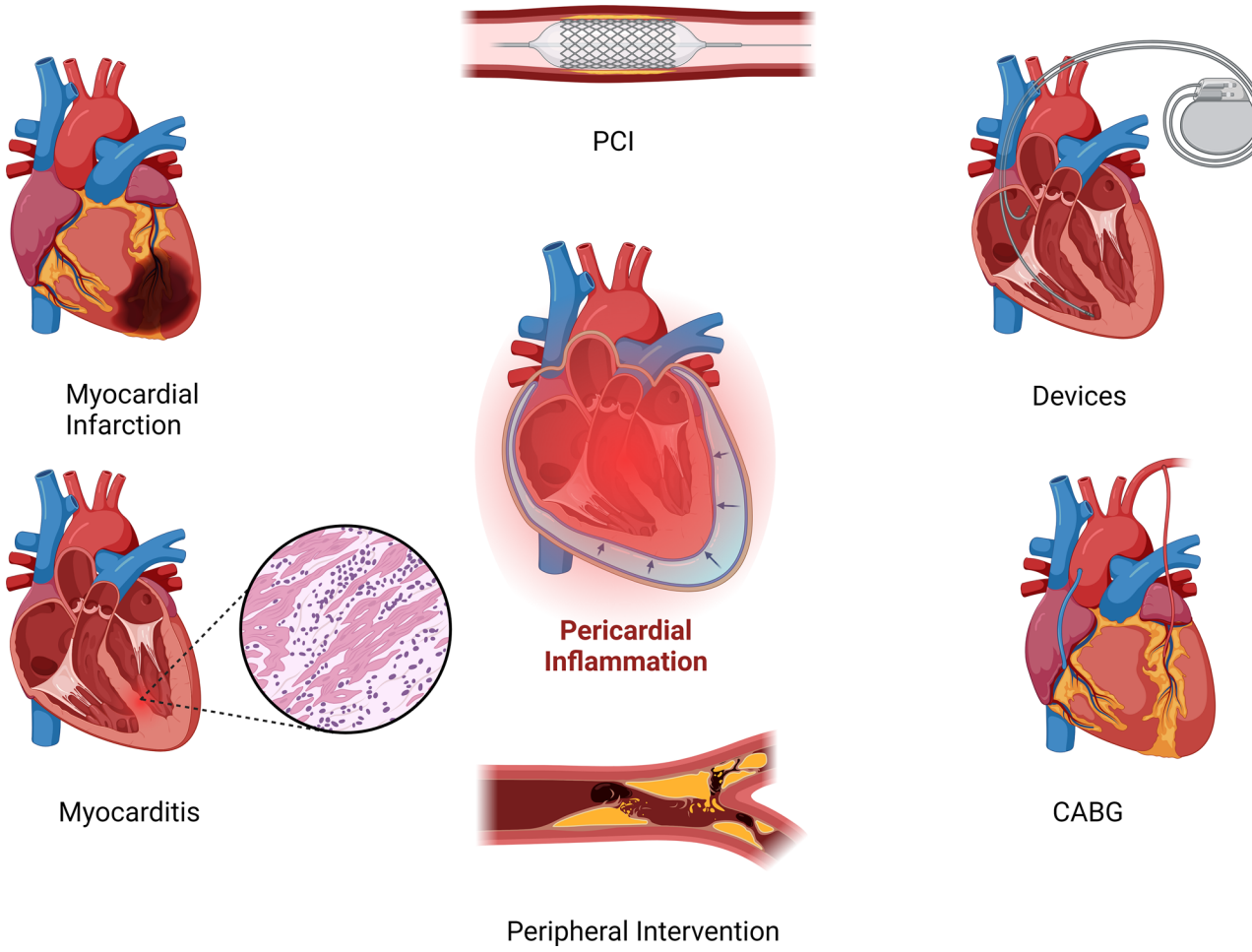

FIGURE 1. Causes of PCIS. PCIS indicates postcardiac injury syndrome.

TREATMENT AND PREVENTION

The treatment approach for PCIS aligns with the recommendations outlined in the 2015 European Society of Cardiology guidelines for the diagnosis and management of pericardial diseases.[34] The primary therapeutic goal in managing PCIS is to mitigate pericardial inflammation to alleviate symptoms and facilitate the resolution of the disease state. Patients with any clinical presentation exhibiting at least 1 predictor of poor prognosis [such as fever >38°C, symptoms lasting several days, large pericardial effusion >20 mm, and nonresponsiveness to nonsteroidal anti-inflammatory drugs (NSAIDs)] should be treated within a hospital setting. A crucial nonpharmacological recommendation is the restriction of intense physical activity for a period ranging from 3 to 6 months, depending on whether the patient is an athlete or a nonathlete. This recommendation continues until symptoms subside and inflammatory markers (erythrocyte sedimentation rate, C-reactive protein, ECG, and TTE) return to normal.[34] NSAIDs constitute the first-line treatment strategy for PCIS. Aspirin, due to its dual anti-inflammatory and antiplatelet effects, is the NSAID of choice for patients with concurrent acute ischemic heart disease. In PCIS, aspirin is administered at a dosage of 750–1000 mg every 8 hours, and indomethacin is given at a dosage of 600 mg every 8 hours for weeks to months until symptom resolution or normalization of inflammatory markers. The dosage should be tapered by 200–400 mg over 2 weeks. Colchicine, known for its potent anti-inflammatory effects in ischemic heart disease, is recommended for use in MI-associated pericarditis and PCIS.[34] For patients with resistance or contraindication to NSAIDs, colchicine is advised at a dosage of 0.5 mg every 12 hours for weeks to months, and tapering is not mandatory upon symptom resolution. In cases of resistant or recurrent pericarditis, corticosteroids (prednisolone 0.2–0.5 mg/kg/d) are employed as second-line agents.[41] However, corticosteroid use should be reserved for severe nonresponders to NSAIDs, as these agents can promote chronicity and have a range of side effects.[42] Immunomodulators like anakinra and azathioprine are recommended for corticosteroid-dependent PCIS that does not respond to colchicine. As a last resort, pericardiectomy may be considered for select patients if medical therapy proves unsuccessful.[43,44] Comparative investigations have been conducted to evaluate various treatment options for PCIS. The Colchicine for Prevention of Postpericardiotomy Syndrome and Postoperative Atrial Fibrillation trial, published in 2014, enrolled 360 patients undergoing cardiac surgery.[45] The trial demonstrated that colchicine reduced the incidence of PCIS by half compared to the placebo group as a primary outcome (9% vs 21%; $P = 0.002$). In contrast, 2 trials found no efficacy of aspirin and corticosteroids in preventing PCIS.[46,47] However, further research is necessary to assess the outcomes of aspirin in reducing the incidence of PCIS in patients following procedures such as PCI, pacemaker implantation, and valvular surgery. Generally, the prognosis for PCIS is favorable for most patients, with a reported recurrence rate of 10% to 15%.[48] Nonetheless, there is a 2%

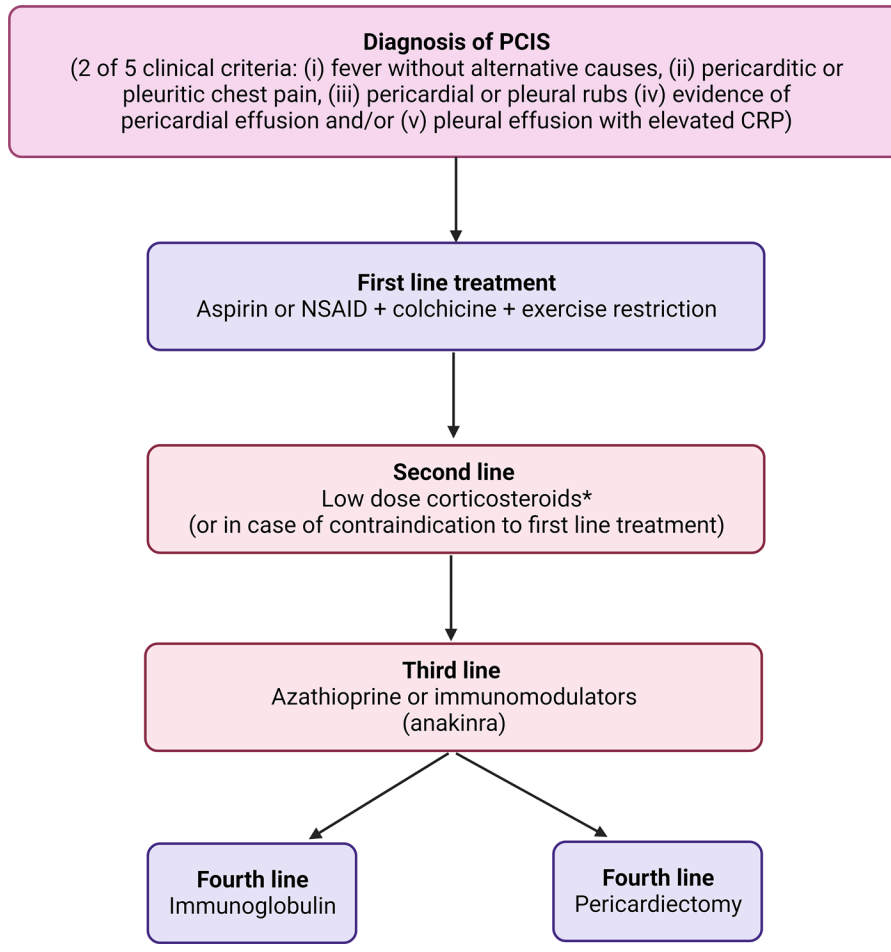

FIGURE 2. Algorithm for treatment of PCIS. PCIS indicates postcardiac injury syndrome.

risk of developing constrictive pericarditis. Therefore, patients with PCIS should undergo longer follow-up periods, spanning several years, to monitor their condition.[49] Figure 2 delineates the treatment algorithm for PCIS.

CONCLUSION

In conclusion, PCIS encompasses a range of pleuropericardial inflammation and effusion syndromes arising from traumatic mesothelial cell injury to the pericardium or pleural cavity. The triggers for PCIS, as highlighted in this review, encompass a spectrum of procedures, including PCI, cardiac CIED implantation, and cardiac surgical interventions. This syndrome can manifest in diverse forms, ranging from uncomplicated pericarditis to complex cases involving pleuropericarditis, tamponade, or massive pleural effusion. The underlying mechanism driving PCIS appears to involve an immune-mediated response, often affecting individuals with a predisposition to the disease. The intricate interplay between tissue damage, inflammation, and autoimmune processes contributes to the pathogenesis of PCIS. Therapeutically, the cornerstone of management lies in an anti-inflammatory approach, employing NSAIDs, colchicine, and restricted physical activity as first-line interventions. Colchicine, in particular, has demonstrated efficacy and safety in preventing the recurrence of PCIS, offering a promising avenue for long-term management. However, it's important to acknowledge that a small percentage of individuals may experience disease relapse, and in some cases, progression to constrictive pericarditis. Despite the potential complexities of PCIS, the overall prognosis is favorable, and reassurance plays a pivotal role in addressing the concerns of affected individuals. A recurrence rate of 10% to 15% underscores the benign nature of the syndrome. However, due to the rare possibility of developing constrictive pericarditis, a sustained, longer-term follow-up spanning several years is essential to monitor and manage potential complications. In an era marked by advancing medical interventions and surgical techniques, an understanding of PCIS and it is evolving treatment strategies becomes increasingly vital. While our comprehension of the syndrome's pathophysiology has grown, ongoing research is necessary to refine diagnostic criteria, optimize treatment protocols, and further illuminate the underlying mechanisms. By continuing to delve into the complexities of PCIS, we can provide more effective care and enhance the quality of life for individuals affected by this intriguing and clinically relevant syndrome.

REFERENCES

1. Imazio M, Hoit BD. Post-cardiac injury syndromes. An emerging cause of pericardial diseases. *Int J Cardiol*. 2013;168:648–652.
2. Patel ZK, Shah MS, Bharucha R, et al. Post-cardiac injury syndrome following permanent dual-chamber pacemaker implantation. *Cureus*. 2022;14:e21737.

3. Verma BR, Montane B, Chetrit M, et al. Pericarditis and post-cardiac injury syndrome as a sequelae of acute myocardial infarction. *Curr Cardiol Rep*. 2020; 22:127.
4. Kostreva DR, Pontus SP. Pericardial mechanoreceptors with phrenic afferents. *Am J Physiol*. 1993;264 :H1836–H1846.
5. Pickar JG. Chemical stimulation of cardiac receptors attenuates locomotion in mesencephalic cats. *J Appl Physiol (1985)*. 1997;83 :113–119.
6. Dressler W. A post-myocardial infarction syndrome; preliminary report of a complication resembling idiopathic, recurrent, benign pericarditis. *J Am Med Assoc*. 1956;160 :1379–1383.
7. Welin L, Vedin A, Wilhelmsson C. Characteristics, prevalence, and prognosis of postmyocardial infarction syndrome. *Br Heart J*. 1983;50:140–145.
8. Shahar A, Hod H, Barabash GM, et al. Disappearance of a syndrome: Dressler's syndrome in the era of thrombolysis. *Cardiology*. 1994;85:255–258.
9. Imazio M, Negro A, Belli R, et al. Frequency and prognostic significance of pericarditis following acute myocardial infarction treated by primary percutaneous coronary intervention. *Am J Cardiol*. 2009;103:1525–1529.
10. Imazio M, Brucato A, Rovere ME, et al. Contemporary features, risk factors, and prognosis of the post-pericardiotomy syndrome. *Am J Cardiol*. 2011;108:1183–1187.
11. van Osch D, Dieleman JM, Bunge JJ, et al; Dexamethasone for Cardiac Surgery Study Group. Dexamethasone for cardiac surgery study group: risk factors and prognosis of postpericardiotomy syndrome in patients undergoing valve surgery. *J Thorac Cardiovasc Surg*. 2017;153:878–885.e1.
12. Beohar N, Davidson CJ, Weigold G, et al. Predictors of long-term outcomes following direct percutaneous coronary intervention for acute myocardial infarction. *Am J Cardiol*. 2001;88:1103–1107.
13. Levy Y, Shovman O, Granit C, et al. Pericarditis following permanent pacemaker insertion. *Isr Med Assoc J*. 2004;6:599–602.
14. Gatzoulis K, Archontakis S, Tsiachris D, et al. Post-cardiac injury syndrome after permanent electronic cardiac device implantation. Incidence, presentation, management and long-term prognosis. *Int J Cardiol*. 2014;174:163–164.
15. Yang KP, Yu W-C, Lu T-M. Acute pericarditis after percutaneous coronary intervention mimicking inferolateral ST-elevation myocardial infarction. *J Invasive Cardiol*. 2013;25:E27–E29.
16. Rodevič G, Budrys P, Davidavičius G. Acute pericarditis after percutaneous coronary intervention: a case report. *Medicina (Kaunas)*. 2021;57:490.
17. Lim HK, Bae YP, Lee BD, et al. A case of postcardiac injury syndrome presenting as acute mediastinitis. *Korean Circ J*. 2009;39:288–291.
18. Janton OH, Glover RP, O'Neill TJ, et al. Results of the surgical treatment for mitral stenosis; analysis of one hundred consecutive cases. *Circulation*. 1952;6:321–333.
19. Ito T, Engle MA, Goldberg HP. Postpericardiotomy syndrome following surgery for nonrheumatic heart disease. *Circulation*. 1958;17:549–556.
20. Finkelstein Y, Shemesh J, Mahlab K, et al. Colchicine for the prevention of postpericardiotomy syndrome. *Herz*. 2002;27:791–794.
21. Velander M, Grip L, Mogensen L. The postcardiac injury syndrome following percutaneous transluminal coronary angioplasty. *Clin Cardiol*. 1993;16:353–354.
22. Tabatznik B, Isaacs JP. Postpericardiotomy syndrome following traumatic hemopericardium. *Am J Cardiol*. 1961;7:83–96.
23. Burgwardt K, Smally AJ. Postpericardiotomy syndrome following minimally invasive coronary artery bypass. *J Emerg Med*. 1998;16:737–739.
24. De Scheerder I, De Buyzere M, Robbrecht J, et al. Postoperative immunological response against contractile proteins after coronary bypass surgery. *Br Heart J*. 1986;56:440–444.
25. Nussinovitch U, Shoenfeld Y. The clinical and diagnostic significance of anti-myosin autoantibodies in cardiac disease. *Clin Rev Allergy Immunol*. 2013;44:98–108.
26. Engle MA, Gay JB, O'Loughlin WA, et al. Viral illness and the postpericardiotomy syndrome. A prospective study in children. *Circulation*. 1980;62:1151–1158.
27. Hoffman M, Fried M, Jabareen F, et al. Anti-heart antibodies in postpericardiotomy syndrome: cause or epiphenomenon? a prospective, longitudinal pilot study. *Autoimmunity*. 2002;35:241–245.
28. Tsang TS, Barnes ME, Hayes SN, et al. Clinical and echocardiographic characteristics of significant pericardial effusions following cardiothoracic surgery and outcomes of echo-guided pericardiocentesis for management: mayo clinic experience, 1979–1998. *Chest*. 116:322–331.
29. Cabalka AK, Rosenblatt HM, Towbin JA, et al. Postpericardiotomy syndrome in pediatric heart transplant recipients. immunologic characteristics. *Tex Heart Inst J*. 1995;22:170–176.
30. Cabalka AK, Rosenblatt HM, Towbin JA, et al. Postpericardiotomy syndrome in pediatric heart transplant recipients. Immunologic characteristics. *Tex Heart Inst J*. 1995;22:170–176.
31. Imazio M, Brucato A, Adler Y. Is it possible to prevent the post-pericardiotomy syndrome?. *Int J Cardiol*. 2012;159:1–4.
32. Bonnefoy E, Godon P, Kirkorian G, et al. Touboul Serum cardiac troponin I and ST-segment elevation in patients with acute pericarditis. *Eur Heart J*. 2000;21:832–836.
33. Köhler I, Saraiva PJ, Wender OB, et al. Behavior of inflammatory markers of myocardial injury in cardiac surgery: laboratory correlation with the clinical picture of postpericardiotomy syndrome. *Arq Bras Cardiol*. 2003;81:279–290.
34. Adler Y, Charron P, Imazio M, et al; ESC Scientific Document Group. 2015 ESC guidelines for the diagnosis and management of pericardial diseases: the task force for the diagnosis and management of pericardial diseases of the European Society of Cardiology (ESC)Endorsed by: the European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J*. 2015;36:2921–2964.
35. Ikäheimo M, Takkunen J. Postpericardiotomy syndrome diagnosed by echocardiography. *Scand J Thorac Cardiovasc Surg*. 1979;13:305–308.
36. Tamarappoo BK, Klein AL. Post-pericardiotomy syndrome. *Curr Cardiol Rep*. 2016;18:116.
37. Bogaert J, Francone M. Cardiovascular magnetic resonance in pericardial diseases. *J Cardiovasc Magn Reson*. 2009;11:14.
38. Verhaert D, Gabriel RS, Johnston D, et al. The role of multimodality imaging in the management of pericardial disease circ. *Circ Cardiovasc Imaging*. 2010;3:333–343.
39. Rajiah P, Kanne JP. Computed tomography of the pericardium and pericardial disease. *J Cardiovasc Comput Tomogr*. 2010;4:3–18.
40. Caraiani C, Petresc B, Dong Y, et al. Contraindications and adverse effects in abdominal imaging med. *Med Ultrason*. 2019;21:456–463.
41. Sevuk U, Baysal E, Altindag R, et al. Role of methylprednisolone in the prevention of postpericardiotomy syndrome after cardiac surgery. *Eur Rev Med Pharmacol Sci*. 2016;20:514–519.
42. Imazio M, Brucato A, Cumetti D, et al. Corticosteroids for recurrent pericarditis: high versus low doses: a nonrandomized observation. *Circulation*. 2008;118:667–671.
43. Lazaros G, Imazio M, Brucato A, et al. Anakinra: an emerging option for refractory idiopathic recurrent pericarditis: a systematic review of published evidence. *J Cardiovasc Med (Hagerstown)*. 2016;17:256–262.
44. Imazio M, Lazaros G, Picardi E, et al. Intravenous human immunoglobulins for refractory recurrent pericarditis: a systematic review of all published cases. *J Cardiovasc Med (Hagerstown)*. 2016;17:263–269.
45. Imazio M, Brucato A, Ferrazzi P, et al; COPPS-2 Investigators. Colchicine for prevention of postpericardiotomy syndrome and postoperative atrial fibrillation: the COPPS-2 randomized clinical trial. *JAMA*. 2014;312:1016–1023.
46. Gill PJ, Forbes K, Coe JY. The effect of short-term prophylactic acetylsalicylic acid on the incidence of postpericardiotomy syndrome after surgical closure of atrial septal defects. *Pediatr Cardiol*. 2009;30:1061–1067.
47. Mott AR, Fraser CD, Kusnoor AV, et al. The effect of short-term prophylactic methylprednisolone on the incidence and severity of postpericardiotomy syndrome in children undergoing cardiac surgery with cardiopulmonary bypass. *J Am Coll Cardiol*. 2001;37:1700–1706.
48. Imazio M, Hoit BD. Post-cardiac injury syndromes. An emerging cause of pericardial diseases. *Int J Cardiol*. 2013;168:648–652.
49. Sabzi F, Faraji R. Predictors of post pericardiotomy low cardiac output syndrome in patients with pericardial effusion. *J Cardiovasc Thorac Res*. 2015;7:18–23.