

Clinical Features of Post Cardiac Injury Syndrome Following Catheter Ablation of Arrhythmias: Systematic Review and Additional Cases



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Background	Post cardiac injury syndrome (PCIS) is a troublesome but not uncommon complication following catheter ablation of arrhythmias. We aimed to study the clinical features of ablation-associated PCIS.
Methods	For this purpose, we conducted a computerised literature search that identified 19 published cases, and we additionally included another 2 two new cases from our centres. Twenty-one (21) cases of PCIS following ablation were analysed.
Results	Among the 21 cases, PCIS most commonly occurred after atrial flutter/fibrillation (AFL/AF) ablation (71.4%), followed by atrioventricular re-entrant tachycardia (AVRT) ablation (9.5%), atrioventricular node (AVN) ablation (9.5%), atrioventricular nodal re-entrant tachycardia (AVNRT) ablation (4.8%) and ventricular tachycardia (VT) ablation (4.8%). Thirty-eight (38) per cent of PCIS was suggested to be secondary to cardiac perforation. Specific symptoms or features include pleuritic chest pain (76.2%), fever (76.2%), elevated markers of inflammation (76.2%), pericardial effusion (90.5%), pleural effusion (71.4%) and pulmonary infiltrates (28.6%). Interestingly, all the six cases with pulmonary infiltrates were following AFL/AF ablation (6/15, 40%). Serious clinical manifestations include cardiac tamponade, massive pleural effusion with hypoalbuminaemia and hyponatraemia, and massive pulmonary infiltrates with hypoxaemia. Notably, empiric antibiotic therapy was used in seven cases including five with pulmonary infiltrates but failed to work. No mortality occurred during a mean follow-up of 4.1 ± 5.3 (1 to 19) months.
Conclusions	Catheter ablation of AFL/AF was most commonly involved in ablation-associated PCIS. Pulmonary infiltrate is an important feature of PCIS following AFL/AF ablation and may be misdiagnosed as pneumonia. Although PCIS is troublesome and even dangerous, it does carry a benign prognosis.
Keywords	Post cardiac injury syndrome • Catheter ablation • Cardiac arrhythmias • Atrial fibrillation • Pulmonary infiltrates • Pericarditis

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Introduction

Postcardiac injury syndrome (PCIS) is an inflammatory syndrome characterised by pericarditis or pleuropericarditis following cardiac surgery [1,2], myocardial infarction [3], percutaneous coronary intervention [4], pacemaker implantation [5], and catheter ablation [6,7]. In past decades, catheter ablation has been increasingly used for the treatment of cardiac arrhythmias, including atrial flutter/fibrillation (AFL/AF), atrioventricular re-entrant tachycardia (AVRT), ventricular tachycardia (VT), etc. [8,9]. Meanwhile, PCIS has been frequently reported in patients with catheter ablation of cardiac arrhythmias. However, the clinical features of PCIS associated with catheter ablation are not fully understood. The primary aim of the present systematic review was to identify those clinical characteristics that could be associated with increased risk of PCIS, observations potentially useful for the clinical management of patients for whom PCIS may be considered.

Materials and Methods

Systematic Literature Search

We performed a systematic literature search for primary evidence in PubMed electronic database on 10 September 2017, by the combination of terminological (MeSH terms) and methodological search filters. The search was not limited by publication time. Key words used in electronic searching included “post cardiac injury syndrome” “postcardiotomy syndrome”; “Dressler’s syndrome”; “pericarditis”; “pericardial effusion”; “pleural effusion”; and “catheter ablation”. The reference lists of all relevant articles were also carefully checked.

Two reviewers independently and carefully reviewed the titles and abstracts. Only the articles that met the following criteria were included in this study: (1) Investigating the clinical features of post cardiac injury syndrome (PCIS); (2) Studies with catheter ablation-associated PCIS. Studies with the following criteria were excluded: (1) Not in English; (2) Review or only abstract; (3) Irrelevant or unavailability of data; (4) Studies with surgical ablation-associated PCIS. Thereafter, the two reviewers obtained the full texts of the studies. We retracted the relevant data including: types of arrhythmias, clinical features/manifestations, treatment and prognosis, etc.

New Cases and Cases’ Descriptions

Besides the cases already reported in the literature, the authors additionally included in the analysis new patients with PCIS after catheter ablation of arrhythmias that had been referred to their centres in the meantime.

Case 1

An 82-year-old male patient had persistent AF with a rapid ventricular rate, which was not well controlled by antiarrhythmic drugs. He was admitted for catheter ablation of AF. After obtaining the patient’s informed consent, radiofrequency catheter ablation of AF was performed as previously

described [8]. All four pulmonary veins were isolated and left atrial linear ablation was performed, including the roof line, anterior line and mitral isthmus line. No fractionated potential ablation was attempted. After intracardiac cardioversion, sinus rhythm was restored. Electrophysiological mapping confirmed the bidirectional conduction block between each pulmonary vein and left atrial. Ablation at the roof line and mitral isthmus line was also confirmed to achieve bidirectional block. Two (2) days after the procedure, the patient was discharged with bisoprolol, furosemide, eplerenone, and warfarin. Two (2) weeks later, the patient complained of poor appetite, fatigue and low-grade fever and he was readmitted. C-reactive protein value (90 mg/L, normal value 0–8 mg/L) and erythrocyte sedimentation rate (ESR, 32 mm/h, normal value 0–15 mm/h) were both elevated. The value of N-Terminal Pro-B-Type Natriuretic Peptide (NT-proBNP) was slightly increased (888.3 pg/ml, normal value 0–526 pg/ml). There were marked hyponatraemia (111 mMol/L, normal value 137–145 mMol/L) and hypoalbuminaemia (25 g/L, normal value 35–50 g/L). When breathing room air, the patient had serious hyoxaemia with arterial blood gas values as follows: pH, 7.54; PaO₂, 71.25 mmHg; PaCO₂, 38.7 mmHg. The chest X-ray was normal before the operation (Figure 1A). A chest computed tomography (CT) scan showed small pericardial effusion, bilateral effusion (massive effusion in left-side), and massive pulmonary infiltrates (Figure 1B). Echocardiogram showed a non-dilated left ventricular with an estimated ejection fraction of 68% and a small amount of pericardial effusion. The antibiotic (moxifloxacin) was given for the treatment of possible community-acquired pneumonia. The patient underwent left-side thoracentesis and a chest drain was placed. About 500–1,000 millilitres of straw-coloured fluid was drained per day and then the drainage tube was clamped. The pleural fluid analysis suggested an exudative fluid. Simultaneously, human serum albumin was administered (10 g per day). As for the marked hyponatraemia, the patient was given oral and intravenous sodium supplementation. After 16 days of the treatment, the total drainage amount was about 9,000 ml. However, the drainage of pleural effusion was still endless and could not be drained completely. The CT was rechecked and showed that there was still massive pleural effusion and the pulmonary infiltrate was partially resolved (Figure 1C). The diagnosis of post-cardiac injury syndrome (PCIS) was then considered. He was subsequently treated with prednisone (10 mg, bid) with rapid clinical improvement. Three (3) days later, there was no longer any drainage when a total of 2,000 ml pleural liquid was drained out and then the chest tube was removed. After 12 days corticotherapy, the chest CT displayed dramatic improvement with almost complete resolution of the massive pleural effusion and pulmonary infiltrates (Figure 1D).

Case 2

A 78-year-old male patient with persistent AF was intolerant to antiarrhythmic drugs and was referred for catheter ablation. After obtaining the patient’s informed consent,

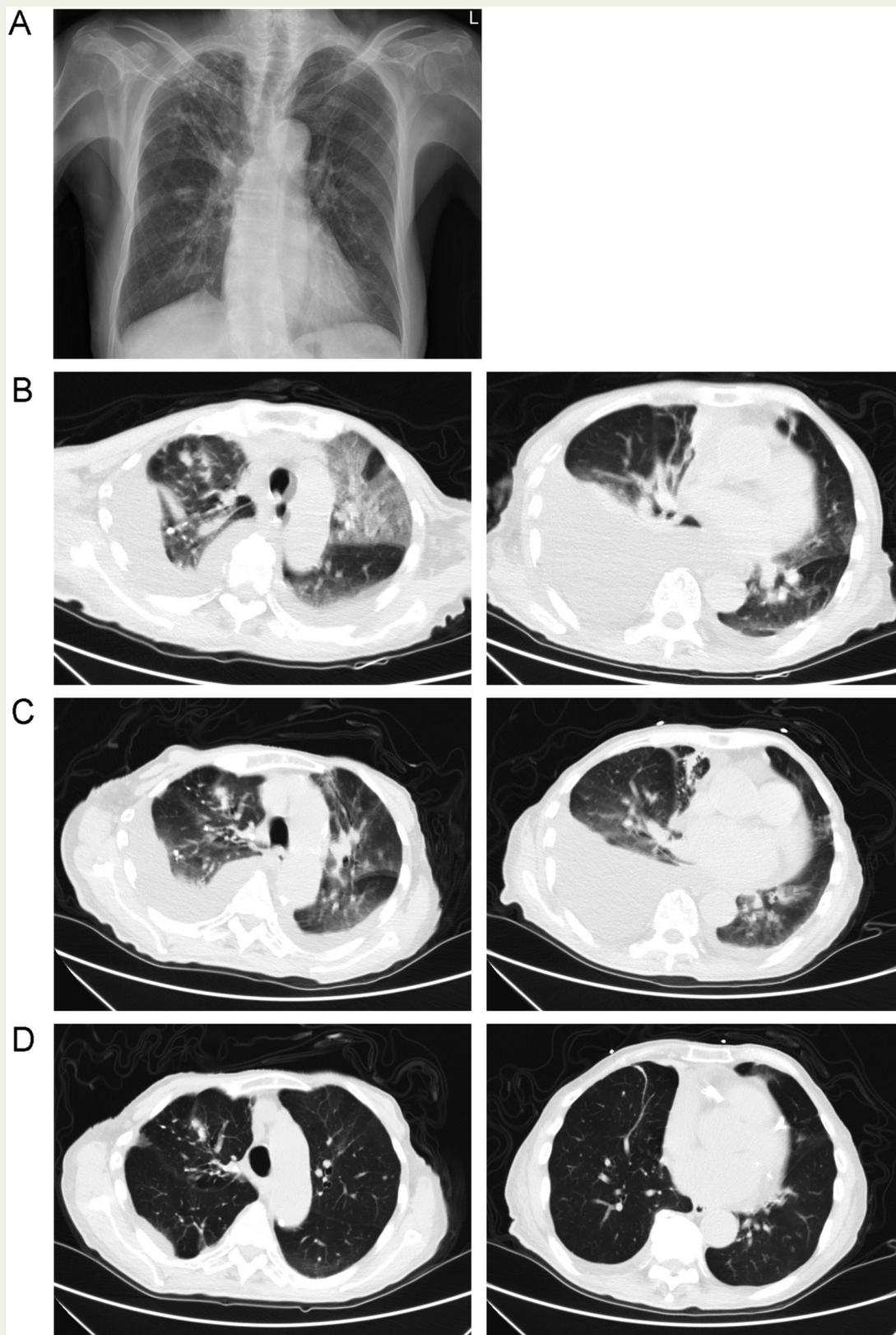


Figure 1 Representative images of the chest X-ray and computed tomography (CT) in case 1 with post cardiac injury syndrome after catheter ablation of atrial fibrillation. (A) Chest X-ray before catheter ablation. (B) CT images one month after the operation showed massive pulmonary infiltrates and pleural effusion. (C) Repeated CT images showed that there was still massive pleural effusion and the pulmonary infiltrates were partially resolved after antibiotic treatment and thoracentesis and chest drain. (D) Two (2) weeks after prednisone treatment and chest drain, the massive pleural effusion and pulmonary infiltrates were completely resolved.

radiofrequency catheter ablation of AF was performed consisting of circumferential pulmonary vein isolation and additional linear ablation (roof line, anterior line and mitral isthmus line). After the procedure, sinus rhythm was restored. On the third day post ablation, the patient complained of cough, dyspnoea and fever with a body temperature of 38.2 °C. The blood leukocyte count was $10.6 \times 10^9/L$ (normal 4.0–10.0 $\times 10^9/L$). C-reactive protein level was elevated to 95 mg/L (normal 0.0–8.0 mg/L). The value of NT-proBNP was slightly increased (531.2 pg/ml, normal value 0–526 pg/ml). The patient had serious hyoxaemia with PaO₂ of 61.5 mmHg when breathing room air. The chest radiograph showed massive pulmonary infiltrates and bilateral effusion which were not present before the operation (Figure 2A and 2B). A chest CT scan showed massive pulmonary infiltrates and bilateral effusion (Figure 2C). The sputum smears and cultures were all negative. Considering the possibility of pneumonia, the patient received empiric antibiotic therapy (Tienam) for one week. But he still had low fever (37.4–37.9 °C) and the CRP was still high. The diagnosis of PCIS was then considered. He was subsequently given anti-

inflammatory treatment with prednisone (10 mg, bid). His symptoms resolved soon after prednisone therapy and he was discharged 5 days later. After one month's therapy, the repeated CT scan showed almost complete resolution of the massive pulmonary infiltrates and pleural effusion (Figure 2D).

Statistical Analysis

Continuous variables were described as mean \pm standard deviation (SD) and categorical data as number and percentages.

Results

According to our search, 157 articles were initially identified between 1993 and 2017. After the screening of the articles, nine articles not in English were excluded. One-hundred-and-thirty-nine (139) articles that did not meet the inclusion criteria were also excluded. We did not include another two articles because of unavailability of required data. Finally, 12 articles were considered eligible for this systematic review

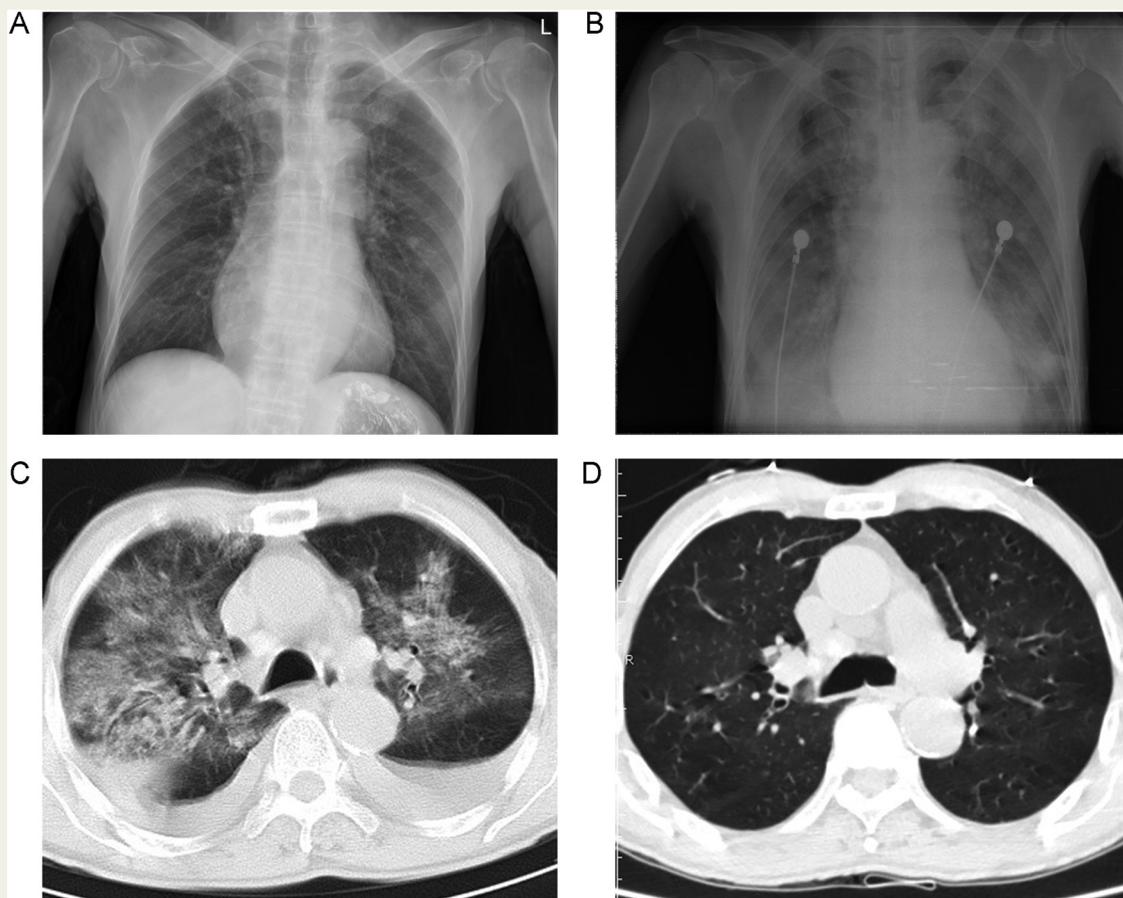


Figure 2 Representative images of the chest X-ray and computed tomography (CT) in case 2 with post cardiac injury syndrome after catheter ablation of atrial fibrillation. (A) Chest radiograph before catheter ablation. (B) Chest radiograph 2 days after operation showed massive pulmonary infiltrates and small pleural effusion. (C) CT Images 3 days after the operation showed massive pulmonary infiltrates and small pleural effusion in both sides. (D) One (1) month after prednisone treatment, the massive pulmonary infiltrates and small pleural effusion were completely resolved.

(Figure 3). Twenty-one (21) cases (19 cases from the 12 articles plus two new cases from our centres) were analysed. These cases (including our cases) reported on 11 women and 10 men. The mean age was 63 ± 13 years, ranging from 24 to 84 years.

The distribution of PCIS following catheter ablation of various arrhythmias is depicted in Figure 4. It can be seen that catheter ablation of AFL/AF was the most commonly involved (15/21, 71.4%), followed by AVRT ablation (2/21, 9.5%), AVN ablation (2/21, 9.5%), AVNRT ablation (1/21, 4.8%) and VT ablation (1/21, 4.8%).

A detailed list of clinical features at presentation is summarised in Table 1. A series of symptoms associated with PCIS usually developed within 1 day to 3 months after ablation, with a mean of 13 days post ablation. Most cases occurred in the first week (73.3%). The predominant symptoms include fever (76.2%, 16/21), chest pain (76.2%, 16/21), and dyspnoea (61.9%, 13/21) (Table 2). Most patients had pericardial effusion (19/21, 90.5%) and pleural effusion (15/21, 71.4%), usually mild and occasionally large. Another specific clinical sign of PCIS is elevated markers of inflammation (16/21, 76.2%). Pulmonary infiltrate is also an important clinical sign of PCIS and present in 28.6% cases (6/21). Interestingly, all the six cases with pulmonary infiltrates

occurred following catheter ablation of AFL/AF (6/15, 40%), but not in other arrhythmias ablation (0/6, 0%). In one case with PCIS following ablation of AF, pulmonary infiltrate was the only imaging manifestation without pleural and pericardial effusion [10].

Serious clinical manifestations were presented in six cases with PCIS (Table 3). Large pericardial effusion and delayed cardiac tamponade were developed in four cases (4/21, 19.0%), including one after AV node ablation and the other three after atrial fibrillation ablation. In one case from our centre, the patient had massive pleural effusion and pulmonary infiltrates combined with hypoalbuminaemia, hyponatraemia and hypoxaemia 2 weeks after catheter ablation of AF (Figure 1). In another case, the patient had massive pulmonary infiltrates combined with serious hypoxaemia 3 days after catheter ablation of AF (Figure 2).

Therapeutic strategies for PCIS were based on empiric anti-inflammatory therapy with nonsteroidal anti-inflammatory drugs (NSAID) and (or) corticosteroids. NSAIDs were administered in nine cases (42.8%) and prednisone in 10 cases (47.6%). For patients with moderate or massive pleural and pericardial effusion, pericardiocentesis and thoracentesis were needed. Among the 21 cases, five patients underwent pericardiocentesis (23.8%) and nine underwent

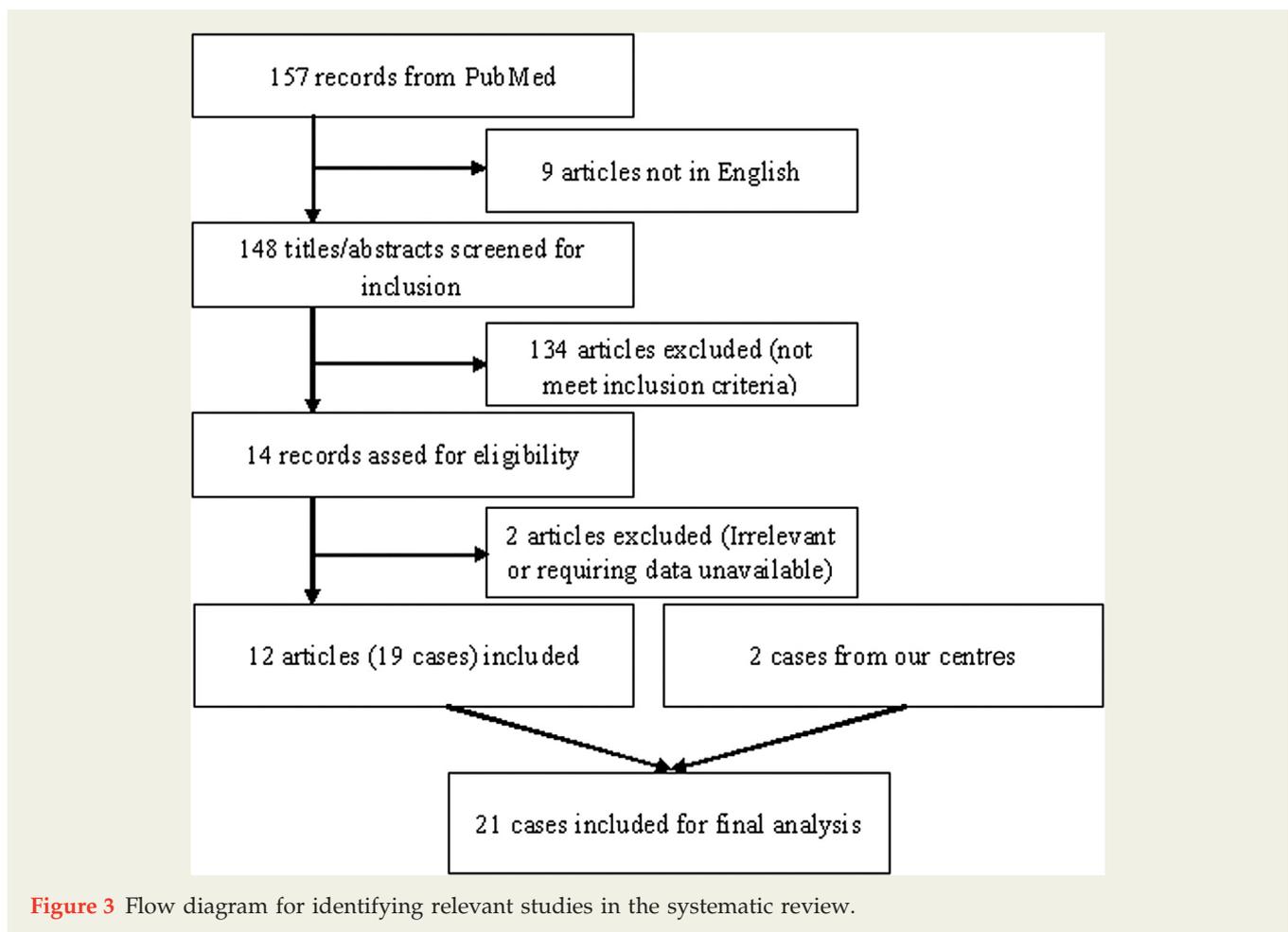


Figure 3 Flow diagram for identifying relevant studies in the systematic review.

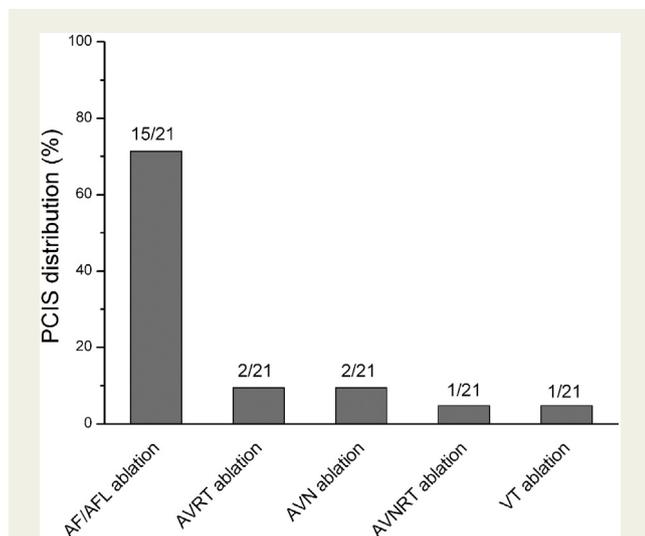


Figure 4 The distribution of PCIS following catheter ablation of various arrhythmias.

Abbreviations: PCIS, post cardiac injury syndrome; AF, atrial fibrillation; AFL, atrial flutter; AVRT, atrioventricular reentrant tachycardia; AVN, atrioventricular node; AVNRT, atrioventricular node re-entrant tachycardia; VT, ventricular tachycardia.

thoracentesis (42.8%). Notably, treatment with only pericardiocentesis and thoracentesis may not resolve pleural and pericardial effusion, which may recur after drainage. As shown in Figure 1, the patient with massive pleural effusion had undergone thoracentesis, but the drainage was still endless after 10 days with a total drainage amount of about 9,000 ml. However, there was no drainage 3 days after treatment with prednisone. Empirical antibiotic therapy was administered in seven cases including five with pulmonary infiltrates, but it failed to improve the patient's condition. Nevertheless, short-term treatment with corticosteroids effectively resolved the symptoms and the pulmonary parenchymal inflammation in these cases (Figures 1 and 2). No mortality occurred during a mean follow-up of 4.1 ± 5.3 (1 to 19) months.

Discussion

Post cardiac injury syndrome is suggested to be an immune-mediated inflammatory syndrome secondary to initial cardiac injury including the myocardium, pericardium, pleura and even the lung tissue [3,11]. It was most commonly reported in patients following cardiac surgery [1,12]. With the rapid increase in the number of patients with catheter ablation of cardiac arrhythmias in the past two decades, PCIS following catheter ablation was frequently reported [13–16]. However, the clinical features of PCIS following catheter ablation are not fully clarified. In this systematic review, we found that catheter ablation of AFL/AF was most commonly involved in ablation-associated PCIS (71.4%). The underlying mechanisms remain unclear. Koller

[17] suggested that the incidence of PCIS may correlate to the extent of myocardial injury and may be higher in procedures that involve larger areas of the myocardium. It is known that catheter ablation of AFL/AF, especially AF, usually causes extensive linear lesions of the atrial myocardium and even damages adjacent tissue, such as pericardium and pleura. By comparison, atrioventricular node ablation for controlling rapid ventricular response, slow-pathway ablation for treatment of AVNRT and accessory pathway ablation for AVRT usually cause spot-wise myocardium injury. Pericardial and (or) pleural inflammation may be triggered from myocardial injury or from direct thermal injury to the pericardium and (or) pleura. Obviously, catheter ablation of AFL/AF has a larger area of myocardium injury and has a higher probability of damaging the adjacent pericardium and pleura, and subsequently more easily triggers immune reactions. Thus, it is not surprising that most cases of ablation-associated PCIS (71.4%) are those with ablation of AFL/AF.

Another clinically significant observation is that pulmonary infiltrate was commonly seen in PCIS following catheter ablation of AFL/AF (40%), but not in that following ablation of other arrhythmias. This may correlate to lung tissue injury caused by catheter ablation of AFL/AF. Previous studies [18,19] have shown that catheter ablation of AF may cause damage to collateral structures including lung tissue, epicardial coronary arteries, nerve bundles, and oesophagus. In an animal study, Gerstenfeld [18] showed that pulmonary vein isolation caused visible damage to the lung overlying the left atrial wall. The lung tissue injury may trigger immune-mediated pulmonary infiltrates in predisposed individuals. Notably, the pulmonary infiltrates were usually misdiagnosed as pneumonia and treated empirically with antibiotics, especially when the pulmonary infiltrate was the only sign of PCIS [10]. In five cases with pulmonary infiltrates, empirical antibiotic treatment failed to improve the patient's condition. Thus, physicians should keep in mind that pulmonary infiltrate is an important clinical sign of PCIS following catheter ablation of AFL/AF. Once diagnosed as PCIS, anti-inflammatory therapy with NSAIDs and (or) corticosteroids, but not antibiotics should be administered.

In a few predisposed subjects, some rare and serious clinical manifestations may occur including cardiac tamponade, massive pleural effusion with hypoalbuminaemia and hyponatraemia, and massive pulmonary infiltrates with hypoxaemia. Under the circumstances, pericardiocentesis and (or) thoracentesis were needed. But the treatment was based on anti-inflammatory therapy with NSAIDs and (or) corticosteroids, and sometimes colchicine [20–23]. The hypoalbuminaemia and hyponatraemia may result from the large volumes of fluid loss and the patient's poor appetite.

Cardiac perforation was suggested to be an aetiology of PCIS [7]. It can cause injury to the pericardium. In this review, 8 of 21 cases were suggested to be secondary to cardiac perforation. It seems that cardiac perforation may play a role in ablation-associated PCIS.

Table 1 Clinical characteristics of all cases with post cardiac injury syndrome reported in the literature.

Reference/ year	Gender/ age	Ablation target	Day to PCIS	Dyspnoea	Fever	Chest pain	Pericardial effusion	Pleural effusion	Cardiac perforation	Cardiac Tamponade*	Pulmonary infiltrates	Elevated CRP/ESR /WBC	NSAID/ Steroids	Colchicine	Antibiotic therapy	Pericardiocentesis* /Thoracentesis	Follow- Up
Rovang [16], 1993	F,72	AVRT	1	Yes	Yes	Yes	Moderate	Yes	No	No	No	Yes	Yes/Yes	No	No	No/No	NA
Turitto [14], 1998	F, 84	AVNRT	3	No	Yes	No	No	Yes	Yes	No	No	Yes	No/No	No	No	No/Yes	2 mon
Turitto [14], 1998	F, 64	AVN	7	Yes	Yes	Yes	Yes	Yes	Yes	No	No	NA	No/Yes	No	No	No/Yes	4 mon
Wood [13], 2003	M, 54	AF	5	Yes	Yes	Yes	Yes	Yes	No	No	No	Yes	No/No	No	Yes	Yes/Yes	3 mon
Koller [17], 2004	F, 64	AFL	1	No	Yes	Yes	Small	Yes	No	No	Yes	Yes	Yes/Yes	No	Yes	No/Yes	NA
Kibos [15], 2006	F, 64	AVN	1	Yes	No	Yes	Massive	Yes	No	Yes	No	Yes	No/Yes	Yes	No	Yes/Yes	19 mon
Zheng [9], 2007	M, 73	VT	1	Yes	Yes	Yes	Yes	Yes	No	No	No	Yes	Yes/No	No	No	No/Yes	1 mon
Luckie [23], 2008	M,56	AF	56	Yes	Yes	Yes	Modearte	No	No	No	No	Yes	No/yes	Yes	No	No/No	4 mon
Goossens [22],2012	F, 68	AF	4	Yes	Yes	Yes	Massive	No	No	Yes	No	Yes	Yes/Yes	Yes	No	Yes/No	1 mon
Liu [7], 2013	F, 60	AF	NA	Yes	Yes	No	Yes	Yes	Yes	No	No	Yes	Yes/No	No	No	No/No	1 mon
Liu [7], 2013	F, 77	AF	NA	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes	No/Yes	No	Yes	No/Yes	1 mon
Liu [7],2013	M, 56	AF	NA	No	Yes	Yes	Yes	No	Yes	No	Yes	No	Yes/No	No	No	No/No	1 mon
Liu [7], 2013	M, 67	AF	NA	Yes	No	Yes	Yes	Yes	Yes	No	No	No	No/No	No	No	No/No	1 mon
Liu [7],2013	F, 62	AF	NA	No	No	Yes	Yes	No	Yes	No	No	No	No/No	No	No	No/No	1 mon
Liu [7], 2013	F, 67	AVRT	NA	No	Yes	Yes	Yes	Yes	Yes	No	No	No	No/No	No	No	No/No	1 mon
Yukumi [6], 2015	M, 24	AF	90	No	Yes	Yes	Small	Yes	No	No	No	Yes	Yes/No	Yes	Yes	No/Yes	11 mon
Torihashi [21],2015	M, 49	AF	1	Yes	No	Yes	Massive	No	No	Yes	No	Yes	Yes/No	No	No	Yes/No	12 mon
Torihashi [21],2015	M, 49	AF	1	No	No	Yes	Massive	Yes	No	Yes	No	Yes	Yes/No	No	No	Yes/No	12 mon
Han [10], 2016	F, 68	AF	21	Yes	Yes	Yes	No	No	No	No	Yes	Yes	No/Yes	No	Yes	No/No	1 mon
New case, 2017	M, 82	AF	14	No	Yes	No	Yes	Massive	No	No	Yes	Yes	No/Yes	No	Yes	No/Yes	1 mon
New case, 2017	M, 78	AF	3	Yes	Yes	No	Small	Small	No	No	Massive	Yes	No/Yes	No	Yes	No/No	1 mon

Abbreviations: M, male; F, female; AVRT, atrioventricular reentrant tachycardia; AF, atrial fibrillation; AFL, atrial flutter; AVN, atrioventricular node; AVNRT, atrioventricular node reentrant tachycardia; VT, ventricular tachycardia; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; NSAID, nonsteroidal anti-inflammatory drug; NA, not available.

*For cardiac tamponade and pericardiocentesis only resulting from PCIS, but not from cardiac perforation.

Table 2 Major symptoms and clinical features in reported cases.

Major symptoms and clinical features	Percentage
Pleuritic chest pain	76.2% (16/21)
Fever	76.2% (16/21)
Dyspnoea	61.9% (13/21)
Elevated markers of inflammation	76.2% (16/21)
Pericardial effusion	90.5% (19/21)
Pleural effusion	71.4% (15/21)
Pulmonary infiltrates	28.6% (6/21)

Table 3 Rare and serious clinical manifestations in reported cases.

Rare and serious clinical manifestations	Percentage
Massive pericardial effusion and delayed cardiac tamponade	19.0% (4/21)
Massive pleural effusion with hypoalbuminaemia and hyponatraemia	4.8% (1/21)
Massive pulmonary infiltrates with hypoxaemia	9.5% (2/21)

Conclusions

Ablation-associated PCIS most commonly occurred following catheter ablation of AFL/AF and was rarely seen in ablation of other arrhythmias. Pulmonary infiltrate was an important feature of PCIS following catheter ablation of AFL/AF. Rare and serious clinical manifestations may occur but the overall clinical course was benign.

Declarations of Interest

None.

Acknowledgements

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