CASE REPORT

Cough as the sole manifestation of pericardial effusion

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SUMMARY

A 59-year-old woman with paroxysmal atrial fibrillation (AF) presented with severe non-productive cough, malaise, low-grade fever and AF flare-up 3 weeks following pulmonary vein isolation with radiofrequency catheter ablation. She denied chest pain or dyspnoea. Patient was haemodynamically stable. There was no pulsus paradoxus. Laboratories showed leucocytosis and elevated C-reactive protein. ECG showed sinus tachycardia. CT abdomen and pelvis showed a large pericardial effusion (PE). Shortly after admission, she developed AF with rapid ventricular response, responsive to intravenous amiodarone. Transthoracic echocardiogram revealed 2.4 cm posterior PE without tamponade physiology, non-amenable to pericardiocentesis via sub-xiphoid approach. Patient underwent left thorascopic pericardial window with removal of 250 cc bloody fibrinous fluid. Cough improved significantly and she was discharged on oral amiodarone and apixaban. Repeat CT chest after 2 weeks for recurrent cough showed a small PE, treated with oral prednisone for suspected postablation pericarditis, with complete resolution of cough. Amiodarone was stopped without recurrence of AF.

BACKGROUND

Pericardial effusion (PE) is characterised by accumulation of transudate, exudate or blood in the pericardial space,¹ which is typically caused by injury, inflammation or infection to the pericardium.² Chest pain and dyspnoea are the usual complaints for pericardial effusion.³ Cough as a presenting symptom is rare. We present a case of pericardial effusion with cough and generalised malaise as the only manifestations.

CASE PRESENTATION

A 59-year-old white woman with paroxysmal atrial fibrillation (AF)/flutter and well-controlled hypothyroidism underwent radiofrequency catheter ablation (RFCA) with isolation of pulmonary veins and cavotricuspid isthmus. She presented 3 weeks later with persistent non-productive cough, generalised malaise with decrease in oral intake and intermittent low-grade fever. She also reported flaring up of AF. She denied chest pain or dyspnoea.

She was sent by her primary care physician to the local emergency room for evaluation. Examination revealed afebrile status, sinus tachycardia and normal blood pressure without pulsus paradoxus.

Laboratories were remarkable for leucocytosis at 17 000 with leftward shift and elevated serum C-reactive protein (CRP) to 16.6 mg/dL. Serum troponin was undetectable. Twelve-lead ECG showed sinus tachycardia with low QRS voltage. CT abdomen and pelvis (figure 1) was done for concerns of gastrointestinal pathology causing abdominal discomfort and decreased oral intake, which showed an incidental finding of large pericardial effusion, predominantly posterior to the left ventricle. She was subsequently transferred to the cardiology service of our hospital for further evaluation. Shortly after admission, she developed AF with rapid ventricular response responsive to intravenous amiodarone.

Transthoracic echocardiogram (TTE) revealed 2.4 cm posterior pericardial effusion, without tamponade physiology. CT images and TTE were reviewed by an interventional cardiologist, cardiac surgeon and thoracic surgeon who all considered the pericardial effusion to be predominantly posterior to left ventricle, not easily amenable to percutaneous drainage by sub-xiphoid or periapical approaches. Patient underwent left video-assisted thorascopic surgery with pericardial window and drain placement into left pleural space. Bloody fibrinous fluid (250 cc) was drained from the pericardial space. Fluid cytometry revealed 12 000 white blood cells (WBCs), predominantly neutrophils, negative for malignant cells, with no microbial growth at 72 hours. Pathology demonstrated acute on chronic inflammation. TTE after the procedure demonstrated no residual effusion. There was significant improvement in cough. The pleural drain was removed after 2 days. She was discharged on oral amiodarone and apixaban.

Repeat CT chest after 2 weeks for recurrent cough showed a small PE. CRP was elevated to 14.4 mg/dL. She was treated with oral prednisone for suspected postablation pericarditis, with complete resolution of cough and major improvement in energy level and exercise capacity. Amiodarone was stopped without recurrence of AF.

INVESTIGATIONS

On/before admission:

- ▶ Laboratories: WBC 17 000 with leftward shift, elevated CRP at 16.6 mg/dL.
- ▶ CT abdomen and pelvis: large pericardial effusion, predominantly posterior.
- ▶ TTE: 2.4 cm posterior pericardial effusion, without tamponade physiology. Repeat TTE showed the same findings.

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Figure 1 CT abdomen and pelvis (transverse view) showing pericardial effusion as an incidental finding. The red arrow shows that the pericardial effusion is predominantly posterior to left ventricle, and thus not easily amenable to percutaneous drainage.

Two weeks after pericardiocentesis:

- ▶ Laboratories: elevated CRP at 14.4 mg/dL.
- ▶ CT chest: small pericardial effusion.

DIFFERENTIAL DIAGNOSIS

- ▶ Pneumonia
- ▶ Acute bronchitis
- ▶ Pulmonary vein stenosis
- ▶ Laryngitis
- ▶ Allergies
- ▶ Postnasal drips

OUTCOME AND FOLLOW-UP

Complete resolution of cough with normal exercise capacity. No recurrence of AF.

DISCUSSION

Classic symptoms and signs of mild to moderate PE without tamponade are pleuritic chest pain improved by sitting up or leaning forward, dyspnoea, pericardial friction rub and QRS alternans on ECG.^{3,4} Our patient presented with dry cough, generalised malaise and intermittent low-grade fever as the only manifestations, along with flare-ups of atrial fibrillation. PE was found as an incidental finding on CT abdomen and pelvis obtained for concern for gastrointestinal pathology.

Cough as a symptom in PE was first reported by Hancock in 1983.⁵ He described a 53-year-old woman who presented to the emergency room with progressive cough and dyspnoea of 2 weeks' duration, preceded by flu-like symptoms and intermittent pleuritic chest pain, identified as idiopathic acute pericarditis with effusion and cardiac tamponade based on physical examination, ECG and echocardiography findings.⁵ More recently in 2015, Hamiel *et al* reported a 75-year-old woman who presented with progressive cough, dyspnoea and fatigue, diagnosed with malignant pericardial effusion caused by small lymphocytic lymphoma.⁴ Dyspnoea accompanied cough in both case reports, unlike our patient.

The aetiology of PE can be classified into infectious, autoimmune, neoplastic, iatrogenic, traumatic and metabolic.⁶ Bloody

pericardial fluid was noted in our patient rather than frank bleeding expected with perforation, suggestive of postablation pericardial inflammation complicated by active oral anticoagulation. Also, PE was noted 3 weeks after AF ablation making cardiac perforation a less likely cause. No PE was noted on intra-cardiac echocardiogram during ablation. PE can be a common finding in hypothyroidism, but it was well controlled in our patient with thyroid replacement therapy.⁷

PE complicating RFCA in patients with arrhythmias is not rare.^{8,9} Lan *et al* showed that out of 1756 patients with arrhythmias undergoing RFCA, 27 (1.54%) developed iatrogenic pericardial effusion.⁸ In a study by Lellouche *et al*, up to 22% of patients who underwent AF ablation developed PE.⁹ However, these two studies mainly focused on PE within 24 hours of RFCA while our patient presented 3 weeks after AF ablation. Majority (83%) of PEs following AF ablation are located posteriorly as reported by Lellouche *et al*,⁹ as also seen with our patient. PE is more common following epicardial ablation, which is rarely used for AF.

Most PE following AF ablation are mild and asymptomatic, which can be managed with observation.⁹ As demonstrated by Chierchia *et al*, incidence of minor PE versus moderate PE was 13.8% vs 1.2%, with 1.2% incidence of tamponade¹⁰; and Lellouche *et al*, when none of the patients with PE (22%) had tamponade or required surgical drainage, and all of them had complete resolution of PE on repeat TTE in 1 month.⁹ Our patient presented with large PE with flaring of paroxysmal AF following AF ablation.

It is reasonable to consider Dressler's syndrome and/or post-cardiac injury syndrome (PCIS) as the cause of PE in our patient given her signs and symptoms of inflammation on presentation, elevated leucocytes in pericardial fluid and dramatic clinical improvement with resolution of cough following steroid therapy. There are several cases reported on Dressler's syndrome/PCIS following RFCA.^{11–15} Luckie *et al* stated that "any insult which causes myocardial, and in particular, pericardial injury, could potentially trigger Dressler's syndrome".¹¹ Our patient could have developed Dressler's syndrome as an immunological response to myocardial tissue destruction during RFCA or as a direct inflammatory response to ablation.¹¹ The former is more likely given delayed onset and atypical symptomatology of PE following AF ablation. However, we cannot completely rule out

Patient's perspective

I am very happy, I got my problem fixed. My heart is doing perfect now. I did have the problem with the heart procedure where there was fluid around my heart, but they are all fixed now. I am very happy with the outcome. Dr Gautam is a very nice doctor and I really like him.

Learning points

- ▶ Cough can be the sole manifestation of pericardial diseases without other classic symptoms like chest pain and shortness of breath.
- ▶ We should have a high clinical suspicion for pericardial effusion/pericarditis in patients presenting with unexplained cough after cardiac radiofrequency catheter ablation procedures, especially if accompanied by flaring up of arrhythmia.

the outside possibility of procedural cardiac perforation with slow oozing.

CONCLUSION

Cough and malaise are uncommon signs of pericardial inflammation or effusion, in the absence of classic symptoms like chest pain and shortness of breath. Despite rare occurrence, it is important to rule out PE and pericarditis in patients presenting with only cough after cardiac radiofrequency ablation procedures.

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