IDCases 39 (2025) e02158

Contents lists available at ScienceDirect

IDCases

journal homepage: www.elsevier.com/locate/idcases

Case report

Influenza A Virus Complicated by Myopericarditis with Pericardial Effusion

Dhara Rana [a,*], Anson Marsh [a], Mahum Sami [a], Hiral Shukla [a], Dimitris Barbouletos [b], Nicholas Calder [a,c]

[a] Department of Internal Medicine, Inspira Medical Center Vineland, Vineland, NJ, USA
[b] Department of Cardiology, University of Pennsylvania, Vineland, NJ, USA
[c] Department of Internal Medicine, Cooper University Medical Center, Camden, NJ, USA

ARTICLE INFO

Keywords:
Influenza A
myopericarditis
Cardiac tamponade
Pericardial effusion
Oseltamivir
Colchicine

ABSTRACT

Influenza A viral infection classically presents as pulmonary manifestations which often require symptomatic management. It can rarely be complicated by pericarditis with concurrent pericardial effusion. We present a unique case of myopericarditis with a pericardial effusion caused by Influenza A. Our patient was presented with elevated troponin and BNP. Chest x-ray showed an enlargement of the cardiac silhouette and clear lungs. CT angiography was remarkable for pericardial effusion. An echocardiogram was performed which demonstrated mild concentric left ventricular hypertrophy with small to moderate circumferential pericardial effusion, and no echocardiographic signs of cardiac tamponade. The significance of our case makes clinicians aware that acute myopericarditis with concurrent pericardial effusion can lead to fatal complications such as cardiac tamponade or cardiogenic shock if left untreated. Early diagnosis and treatment as presented in our case could reduce the risk of such severe cardiac events from occurring.

Introduction

Influenza is a severe viral respiratory illness that accounts for up to 300,000 deaths annually worldwide [1]. Many patients with influenza A are asymptomatic. Some clinical presentations may vary from afebrile respiratory symptoms to febrile illness causing clinical manifestation in the lung, heart, brain, kidney, and liver [2]. Heart complications such as myocarditis are rare. In a large study of 625 patients from 20 institutions in the United States who presented with acute viral myocarditis, PCR of cardiac samples obtained from cardiac catheterization studies showed a viral genome in 238 (3 %) patients [3]. A total of 5 out of 238 (2 %) were found to have influenza A viral genome by PCR. Furthermore, viral myocarditis is commonly accompanied by some degree of pericarditis, which is referred to as “myopericarditis” or “perimyocarditis” interchangeably in clinical practice [4]. Here, we present a rare clinical manifestation of myopericarditis due to influenza A complicated with a pericardial effusion.

Case presentation

A 49-year-old female presented to the emergency department for generalized weakness, malaise, fatigue, and worsening dyspnea with exertion and pleuritic pain. The patient also endorsed intermittent fevers which have since been resolved. On admission, the patient’s blood pressure was 144/101 mmHg, heart rate 108 beats per minute, respiratory rate 22 breaths per minute, body temperature 36.6°C, and an oxygen saturation of 98 % on ambient air. Her physical exam was unremarkable.

The patient had no significant past medical history except for vitiligo. She denied any other autoimmune disease. However, seven days prior to this admission, the patient was seen at the emergency department with a fever, sore throat, and headache. On that visit, she tested positive for influenza A (QAIGEN QIAstat-Dx Respiratory Panel Plus) and was treated with one-time dexamethasone 10 mg oral, and acetaminophen 975 mg once, and subsequently discharged (Fig. 1).

On laboratory findings, the patient had elevated baseline troponin of 2454 ng/L, 2-hour troponin 2506 ng/L with delta of 53, elevated BNP 132 pg/L, as well as normal complete metabolic panel, ESR, CRP and

Abbreviations: EF, Ejection fraction; CV, cardiovascular; MI, myocardial infraction; ESR, erythrocyte sedimentation rate; CRP, c-reactive protein; TSH, thyroid stimulating hormone.

* Correspondence to: Department of Internal Medicine, Inspira Medical Center Vineland, 1505 West Sherman Avenue, Vineland, NJ 08360, USA.
E-mail address: ranad@ihn.org (D. Rana).

https://doi.org/10.1016/j.idcr.2025.e02158
Received 17 November 2024; Received in revised form 11 January 2025; Accepted 13 January 2025
Available online 14 January 2025

TSH. Complete blood count showed a decreased red blood count of 5.29 × 10^6/uL, and decreased hematocrit of 48.3 % with MCV 91.3 fL. Initial electrocardiogram showed sinus tachycardia with right axis deviation and low voltages with a ventricular rate of 108 and QTc 420 (Fig. 2). Chest x-ray showed an enlargement of the cardiac silhouette and clear lungs. CT angiography was remarkable for pericardial effusion.

A transthoracic echocardiography (TTE) was performed which demonstrated mild concentric left ventricular hypertrophy, ejection fraction (EF) of 65–70 %, small to moderate circumferential pericardial effusion, and no echocardiographic signs of cardiac tamponade (Fig. 3A). Using PCR QAIGEN QIAstat-Dx Respiratory Panel Plus, the serum virology was positive for influenza A (indicating subtype/serotype can be either H2N2, H5N3, or H10N7), but negative for influenza A serotypes H1, H1N1/pdm09, and H3. The patient also had positive IgG antibodies for CMV (CMV Ab IgG >10 unit/L), EBV (Nuclear Ab IgG 303 unit/L, Viral Ag VCA IgG 63.7 unit/L), and parvovirus-19 (IgG 6.5 unit/L), but negative IgM antibodies for those viruses indicating the patient had a prior infection.

The patient was started on antiviral therapy with oseltamivir 75 mg twice a day for 5 days, colchicine 0.6 mg twice daily for 30 days, and ibuprofen 600 mg three times a day for 5 days. Before discharge, a TTE demonstrated remarkably reduced pericardial effusion (Fig. 3B). The patient showed progressive clinical improvement.

At a one-month cardiology follow-up, the repeat TTE showed a significant reduction of pericardial effusion (Fig. 4). Clinically, the patient's dyspnea also had resolved.

Discussion

Influenza is a severe viral respiratory illness that accounts for up to 300,000 deaths annually worldwide [1]. While many cases of influenza A are asymptomatic, clinical presentations range from mild respiratory symptoms to severe febrile illness affecting multiple organs [2]. Heart complications such as myocarditis are rare [3].

Different influenza A serotypes are associated with causing myopericarditis. A literature review conducted by Radovanovic et al. showed a majority of the reported myopericarditis was due to influenza A followed within the influenza subtype no reported and influenza H1N1 [4]. In our study, the PCR that was used to detect the influenza A was the QIAstat-Dx Respiratory Panel Plus, which is a multiplex nuclei acid test intended to detect and identify multiple respiratory viral nucleic acid in nasopharyngeal swabs. This PCR tested for influenza A, influenza A H1, influenza A H3, influenza A H1N1/pdm09, and Influenza B. Our patient was only positive for influenza A with this test. Per the equipment manual, positive influenza A is indicative of either H2N2, H5N3, or H10N7 subtypes[5].

Influenza A-associated myopericarditis can be fatal leading to cardiogenic shock, cardiac tamponade, or death [4]. In a literature review, between 1951 and 2021, 58 of 75 cases of patients between 11 and 49 with either influenza A/B myopericarditis or isolated pericarditis initially presented with tachycardia, hypotension, chest pain and dyspnea [4]. The literature review also showed that in 63.8 % of the cases with myopericarditis patients had cardiogenic shock with or without cardiac tamponade[4]. While our patient was hypertensive and lacked generalized chest pain symptoms, she was tachycardic and dyspneic, which is considered atypical. Furthermore, our patient presented with pericardial effusion, which had not evolved to tamponade or cardiogenic shock. This may partly be due to the fact the patient returned to the emergency department after feeling dyspneic with exertion before symptoms worsened.

Moreover, according to the European Society of Cardiology, the diagnosis of myopericarditis is made clinically if a patient has defined criteria for acute pericarditis showing elevated biomarkers of myocardial injury without newly developed focal or diffused impairment of the left ventricular function on TTE [6]. Our patient presented with worsening shortness of breath, elevated cardiac markers (troponin baseline 2454 ng/L and troponin 2-hour 2506 ng/L), and echocardiography showing a pericardial effusion with an ejection fraction of 65–70 % making the presentation consistent with that seen in previous literature.

The management of myopericarditis is similar to pericarditis. Our patient was treated with colchicine 0.6 mg twice daily for 30 days, and ibuprofen 600 mg three times a day for 5 days. The literature recommends empirical anti-inflammatory therapies such as aspirin 1500–3000 mg/day or NSAIDs (ibuprofen 1200–2400 mg/day or indomethacin 75–150 mg/day) to control chest pain. In contrast, corticosteroids are prescribed as the second choice in cases with contraindication, intolerance, or failure of aspirin/NSAIDs [6]. In the setting of myopericarditis, it is recommended to reduce dosages of aspirin/NSAID because animal models of myocarditis have shown aspirin/NSAIDs to be non-efficacious and may enhance inflammation and thus increase mortality [6].

The question arises if the use of colchicine is an effective adjunct therapy to myopericarditis. According to the literature review of 78 cases of influenza A/B myopericarditis and isolated pericarditis between 1951 and 2021, 17.3 % of cases used NSAIDs, 16 % used corticosteroids and 10.7 % used colchicine. In another study of an experimental model using C57BL6/j mice of viral myocarditis secondary to coxsackievirus virus found that colchicine reduced cardiac markers from myocardial injury (ie troponin), improved left ventricular ejection fraction, and reduced inflammatory markers without exacerbation of the viral agent [7].

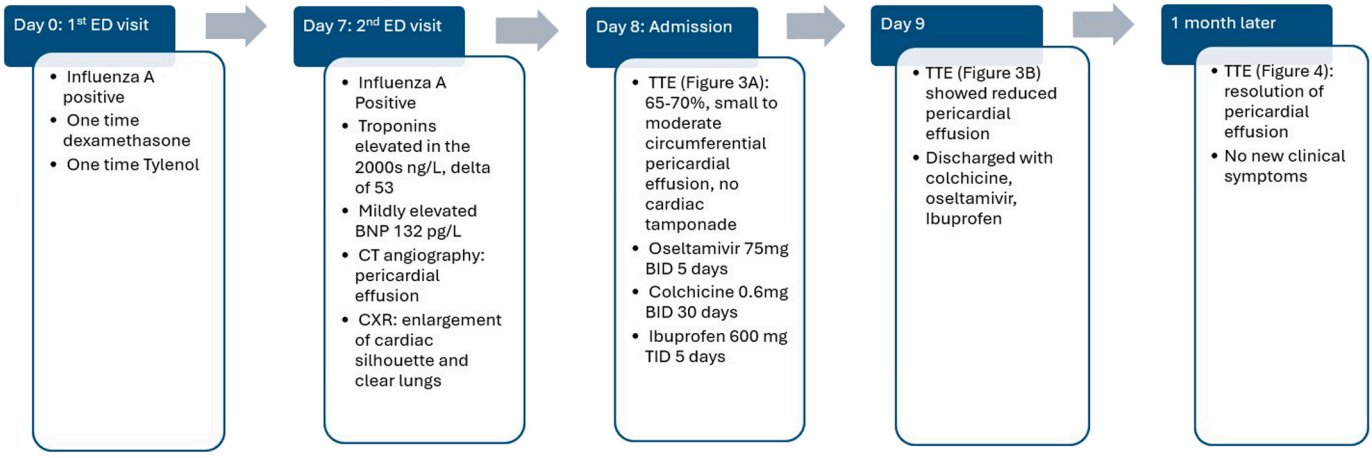

Fig. 1. Timeline of events.

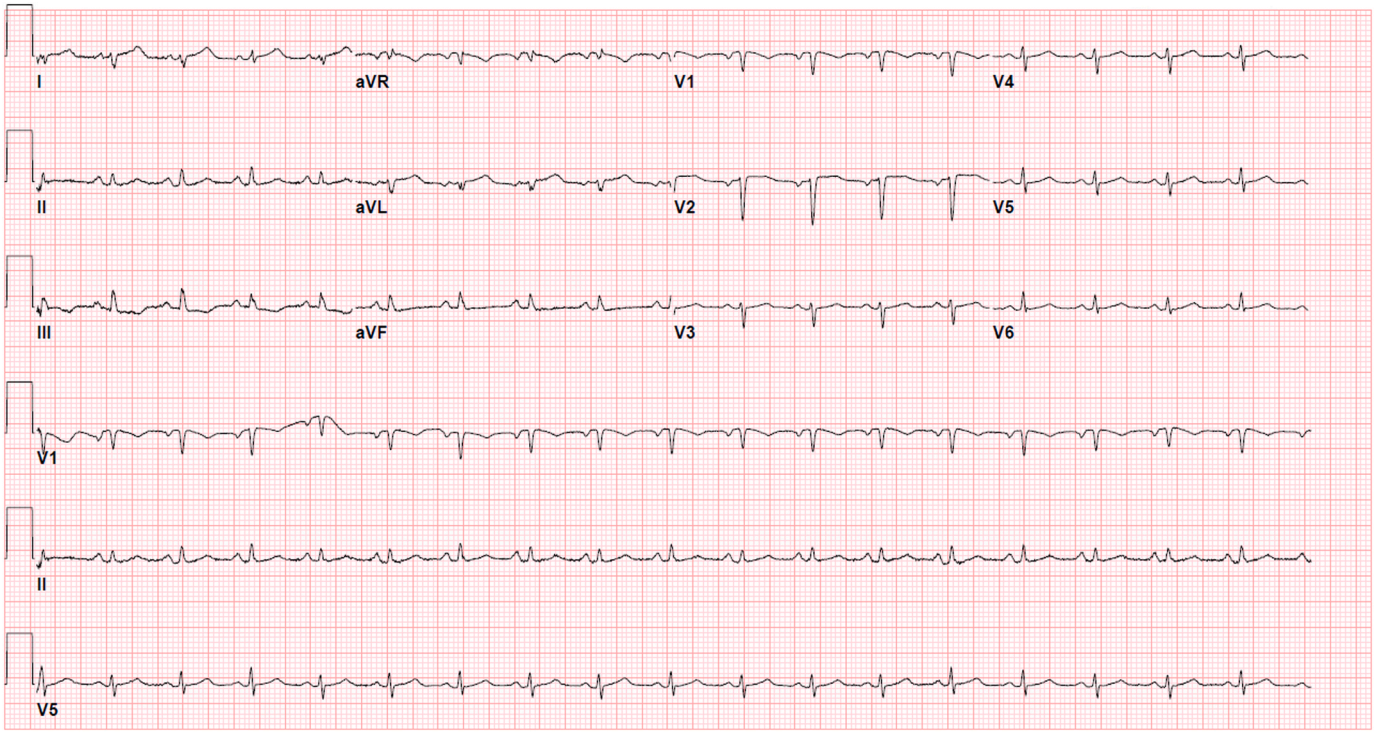

Fig. 2. EKG. Electrocardiogram demonstrating sinus tachycardia with right axis deviation and low voltages with a ventricular rate of 108 and QTc 420.

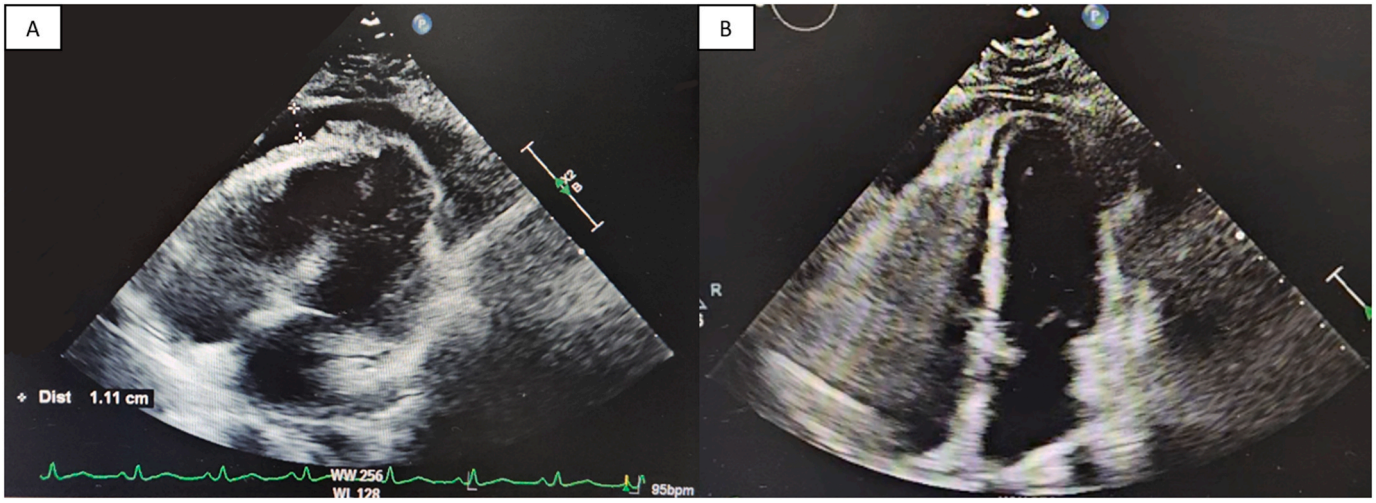

Fig. 3. A. Transthoracic echocardiography Prior to treatment the distance between the outer border of the apex and pericardium showed a small to moderate pericardial effusion. **B.** Transesophageal echocardiography 2 days later demonstrated reduction of pericardial effusion.

Our patient had influenza A which precipitated the patient's myopericarditis and was also treated with oseltamivir 75 mg twice a day for 5 days. In a literature review that investigated 78 cases of influenza, myopericarditis and pericarditis, antivirals such as oseltamivir (n = 33, 44 %), peramivir (n-6,8 %), or zanamivir (n = 3,5 %) were used in half of the cased and with four cases receiving a combination of two. In addition, a study investigated the efficacy and safety of oseltamivir in treating influenza-infected high-risk populations; those with chronic pulmonary or cardiac disease at risk of serious influenza-related complications such as bronchitis, asthma, pneumonia, and myocarditis. The study showed viral shredding was reduced by 70 % in oseltamivir-treated at-risk patients and reduced the duration of febrile illness, and individual influenza symptoms and respiratory complications [8]. The question begets whether cardiac complication can be reduced with addition of antiviral medication. A study conducted by Ward et al. established that oseltamivir treatment for influenza inpatient with cardiac history significantly reduced the risk of recurrent CV events such as (MI, angina pectoris, stroke, health failure and sudden cardiac death) [9]. With this information, if the patient was started on anti-viral treatment on her initial presentation to the ED, it may have prevented the further cardiac complications. However, when the patient presented during her initial presentation (day 0, see Fig. 1), she presented with generalized symptoms of fever, sore throat and headache without increased oxygen requirements with a positive PCT test for influenza A. Due to the ongoing symptoms she received a onetime dose of dexamethasone 10 mg oral solution, a one-time dose of Tylenol 975 mg, with discussion on symptom management in the outpatient setting and was discharged from the emergency department.

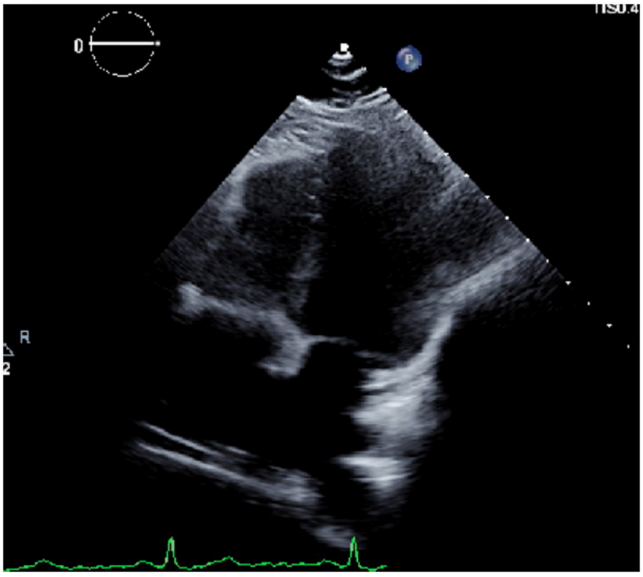

Fig. 4. Transthoracic echocardiography 1 month later demonstrated significant reduction of pericardial effusion.

Lastly, our patient was presented with concurrent pericardial effusion in the setting of influenza-associated myopericarditis. She had a TTE completed to rule out cardiac tamponade and to assess cardiac hemodynamics. Fortunately, our patient showed mild pericardial effusion which improved with medical treatment (Fig. 3A, B, Fig. 4). It is important to note that after 2 days there was some reduction on the second TTE (Fig. 3B), but findings on echocardiography are susceptible to user-variability and varies in measurement between echocardiographer and readers. Therefore, a repeat TTE in one month did show a more significant reduction compared to prior TTE (Fig. 4), which is reasonable to conclude that treatment contributed to clinical improvement. In addition, the patient was not given diuretics.

Cardiac tamponade is a rare manifestation of the disease process. The recommendation for pericardial effusion is target therapy for the etiology and aspirin/NSAIDs/colchicine is recommended when the pericardial effusion is associated with systemic inflammation [6].

Conclusion

Pericardial effusion associated with myopericarditis is a rare occurrence in those with influenza A. Our case report makes clinicians aware that acute myopericarditis with concurrent pericardial effusion can lead to fatal complications such as cardiac tamponade or cardiogenic shock. Early diagnosis and treatment as presented in our case could reduce the risk of such severe cardiac events occurring. It may be prudent to investigate further regarding cardiogenic shock or death stratification for patients with Influenza A-associated myopericarditis due to the high morbidity and mortality risk. In addition to classic pericardial treatment with aspirin/NSAIDS/colchicine, patients with influenza A infection should be placed on antiviral medications such oseltamivir as studies have shown to reduce pulmonary complications.

Ethical approval

Institutional review board approval did apply to this case

Sources of funding

None

Disclosures

The authors have nothing to disclose.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request

Author contribution

All authors were involved in patient care. DR and AM are first co-authors of the paper. All authors contributed to review of literature and manuscript writing.

Funding

No funding.

Author statement

Thank you for giving us an opportunity to revise our paper. All authors were involved in patient care. DR and AM are first co-authors of the paper. All authors contributed to review of literature and manuscript writing. All authors have seen and approve of the final manuscript version that is being submitted. This manuscript is original work that has not been submitted or published elsewhere.

CRediT authorship contribution statement

Dhara Rana: Writing – review & editing, Writing – original draft. **Dimitris Barbouletos:** Writing – review & editing. **Hiral Shukla:** Writing – review & editing. **Mahum Sami:** Writing – review & editing. **Anson Marsh:** Writing – review & editing, Writing – original draft. **Nicholas Calder:** Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Osterholm M.T.Preparing for the next Pandemic, Global Health, Routledge; 1120; 2020.
[2] Mamas MA, Fraser D, Neyses L. Cardiovascular manifestations associated with influenza virus infection. Int J Cardiol 2008;130(3):304–9.
[3] Bowles NE, Ni J, Kearney DL, Pauschinger M, Schultheiss H-P, McCarthy R, et al. Detection of viruses in myocardial tissues by polymerase chain reaction: evidence of adenovirus as a common cause of myocarditis in children and adults. J Am Coll Cardiol 2003;42(3):466–72.
[4] Radovanovic M, Petrovic M, Barsoum MK, Nordstrom CW, Calvin AD, Dumic I, et al. Influenza myopericarditis and pericarditis: a literature review. J Clin Med 2022;11 (14):4123.
[5] QIAGEN: QIAstat-Dx® Respiratory Panel Plus Instructions for Use. In.: QIAGEN GmbH; 2024: 70-74.
[6] Asteggiano R, Bueno H, Caforio AL, Carerj S, Ceconi C. 2015 ESC guidelines for the diagnosis and management of pericardial diseases–Web Addenda. Eur Heart J 2015.
[7] Pappritz K, Lin J, El-Shafeey M, Fechner H, Kühl U, Alogna A, et al. Colchicine prevents disease progression in viral myocarditis via modulating the NLRP3 inflammasome in the cardiosplenic axis. ESC Heart Fail 2022;9(2):925–41.
[8] Martin C., Mahoney P., Ward P.Oral Oseltamivir Reduces Febrile Illness in Patients Considered at High Risk of Influenza Complications, Elsevier, International Congress Series2001. 807811.
[9] Casscells SW, Granger E, Kress AM, Linton A, Madjid M, Cottrell L. Use of oseltamivir after influenza infection is associated with reduced incidence of recurrent adverse cardiovascular outcomes among military health system beneficiaries with prior cardiovascular diseases. Circ Cardiovasc Qual Outcomes 2009;2(2):108–15.