# Acute Transient Effusive-Constrictive Pericarditis 

Kazuhito Hirata, MD, ${ }^{\text {a }}$ Izumi Nakayama, MD, ${ }^{\text {b }}$ Minoru Wake, $\mathrm{MD}^{\text {a }}$

## ABSTRACT


#### Abstract

A 52-year-old female developed acute idiopathic pericarditis, which was complicated with tamponade. Constrictive physiology persisted after pericardiocentesis, and effusive-constrictive pericarditis (ECP) was diagnosed. Constrictive physiology improved in 10 days with anti-inflammatory therapy. This case was remarkable because it showed that ECP may present in an acute and reversible form. (Level of Difficulty: Beginner.) (J Am Coll Cardiol Case Rep 2019;1:616-21) © 2019 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).


Effusive-constrictive pericarditis (ECP) is characterized by pericardial constriction in the presence of pericardial effusion. Transient constrictive pericarditis (TCP) is a reversible form of constrictive pericarditis without progression to chronic constriction. This paper presents a clinically instructional case in which features of both ECP and TCP coexisted during the acute phase of idiopathic pericarditis.

## PRESENTATION

A 52-year-old woman attended the authors' emergency room with symptoms of dyspnea and orthopnea. Five days earlier, she had begun to experience

## LEARNING OBJECTIVES

- Effusive-constrictive pericarditis may present as an acute form.
- The features of both effusive-constrictive pericarditis and transient constrictive pericarditis may coexist in 1 patient.
- Early recognition and appropriate medical therapy of effusive-constrictive pericarditis may prevent progression to chronic constriction.
low-grade fever ( $37.5^{\circ} \mathrm{C}$ ) with malaise and coughing. A day before admission, she developed dyspnea then orthopnea on the day of admission. Her history was unremarkable.

Her vital signs on admission were as follows: blood pressure was $90 / 65 \mathrm{~mm} \mathrm{Hg}$; heart rate was 118 beats $/ \mathrm{min}$; and respiratory rate was 24 breaths/min. With $2 \mathrm{l} / \mathrm{min}$ nasal oxygen, her oxygen saturation was $93 \%$. The jugular venous pressure was elevated; her skin was cold, and her legs were swollen. Heart auscultation revealed no pericardial friction rub. Laboratory results were as follows: white blood cell count was $8,200 / \mathrm{ml}$; kidney function and electrolytes were normal, and liver enzymes were normal. Arterial blood gas analysis showed a pH of 7.46, a $\mathrm{Pco}_{2}$ of $22.6 \mathrm{~mm} \mathrm{Hg}, \mathrm{a}_{2}$ of 112 mm Hg , and a bicarbonate concentration of $16 \mathrm{mmol} / \mathrm{l}$. Her C-reactive protein level was $0.76 \mathrm{mg} / \mathrm{dl}$ (normal level: $<0.3 \mathrm{mg} / \mathrm{dl}$ ). Screening for collagen vascular disease yielded negative results.

An electrocardiogram revealed sinus tachycardia and diffuse ST-segment elevation, typical of acute pericarditis, and a chest radiograph showed cardiomegaly and pleural effusion. Echocardiography revealed moderate pericardial effusion and restricted

[^0]Manuscript received June 25, 2019; revised manuscript received August 9, 2019, accepted August 12, 2019.
wall motion of the right atrium and ventricle (Figures 1A and 1B, Videos 1 and 2).

## DIFFERENTIAL DIAGNOSIS

Acute pericarditis complicated with cardiac tamponade was suspected.

## INVESTIGATION AND MANAGEMENT

She was admitted to the intensive care unit and was given normal saline ( 21 in 2 h ) and dopamine ( $3 \mu \mathrm{~g} / \mathrm{kg} / \mathrm{min}$ ) for the hypotension. Blood pressure in the left radial arterial line was $70 / 54 \mathrm{~mm} \mathrm{Hg}$ (average $=60 \mathrm{~mm} \mathrm{Hg}$ ), with marked pulsus paradoxus (Figure 1C). Mitral inflow velocity was low, with respiratory variations (Figure 1D). Because of the hemodynamic instability, urgent pericardiocentesis was performed, draining 200 ml of straw-yellow exudative pericardial fluid in which neutrophils were dominant. Her blood pressure increased to $90 / 50 \mathrm{~mm} \mathrm{Hg}$ (average $=65 \mathrm{~mm} \mathrm{Hg}$ ), and her heart rate decreased to

90 beats/min. However, she remained hypotensive, with elevated jugular venous pressure (with prominent y descent). Echocardiography on day 2 confirmed there was no residual pericardial effusion and showed normal left ventricular systolic function (Figures 2A and 2B, Videos 3 and 4). The pulsus paradoxus had improved (Figure 2C); however, there was a respirophasic shift of the interventricular septum, and the respiratory variation of the mitral inflow velocity had increased to $32 \%$ (Figure 2D). Consistent with annulus reversus, the septal $\mathrm{e}^{\prime}$ velocity ( $8.7 \mathrm{~m} / \mathrm{s}$ ) was higher than the lateral $\mathrm{e}^{\prime}(8.3 \mathrm{~m} / \mathrm{s})$.

On day 4, the patient underwent cardiac catheterization. The right atrial pressure remained elevated ( 10 mm Hg ) with prominent y descent; the end diastolic pressures in the 4 cardiac chambers were nearly equalized, with the ventricular diastolic pressure wave form showing a dip-plateau pattern (Figure 3A). Simultaneous measurements of right and left ventricular pressures clearly showed ventricular interdependence, with a discordant pressure response to

(A, B) Echocardiography shows moderate pericardial effusion and restricted wall motion of the right atrium and ventricle. (A) Apical 4-chamber view. (B) Parasternal long-axis view. (C) An arterial pressure tracing shows pulsus paradoxus. (D) Initial diastolic mitral flow velocity. See Videos 1 and $2 . E=$ expiration; $I=$ inspiration; $L V=$ left ventricle; $R V=$ right ventricle.

respiration (Figure 3B). Computed tomography scanning revealed that the pericardium was thickened, with shaggy appearance (Figure 4A).

These findings were consistent with acute ECP. Cultures of the pericardial fluid were negative for organisms, and the cytologic examination was negative for malignancy. After ibuprofen ( $400 \mathrm{mg}, 3$ times daily) was administered, the patient's condition improved without requiring surgical pericardiectomy. Echocardiography on day 10 showed a marked improvement in the respirophasic shift of the interventricular septum and of the respiratory variation of the mitral inflow velocity (Figures 5A to 5C, Videos 5 and 6).

## DISCUSSION

This case illustrates acute ECP, which was also considered to be TCP because the constrictive physiology improved with non-steroidal anti-inflammatory drugs. Because the specific cause could not be
identified, the case was classified as acute idiopathic pericarditis (1); however, viral pericarditis was the most likely cause, given the preceding influenza-like symptoms and the pericardial fluid consistent with inflammatory exudate, with negative cultures and cytology.

ECP is characterized by the coexistence of tense pericardial effusion and constriction of the heart by the nonelastic pericardium (2-4). Usually, the diagnosis is based on the persistent restriction of diastolic filling (constrictive physiology) even after the clinical tamponade has been treated, as in the present case (2). Ventricular interdependence shown by echocardiography with Doppler imaging (2-4) or elevated 4-chamber end-diastolic pressure shown by cardiac catheterization is useful for diagnosis (2). ECP is rare, accounting for just $1.3 \%$ of all pericarditis (2), although the prevalence increases from $7.9 \%$ to $16 \%$ in patients with clinical tamponade (2-4). Common causes in developed countries include post-pericardiectomy, idiopathic, cardiac

FIGURE 3 Pressure Measurements, Day 4

(A)Simultaneous right and left heart pressure measurements. (B) Discordant respiratory variation of the left and right ventricular pressures (red and black arrows) suggests interventricular dependence. $\mathrm{Ao}=$ aorta; $\mathrm{PA}=$ pulmonary artery; $\mathrm{RA}=$ right atrium; $\mathrm{WP}=$ pulmonary capillary wedge pressure; other abbreviations as in Figure 1.
procedure-related, and malignancy (2-4). Purulent or tuberculous ECP is rare. The clinical course is generally subacute $(2,3)$. Its natural history depends on the cause $(2,3,5)$. Sagrista-Sauleda et al. (2) reported that approximately one-half of ECP cases required surgical pericardiectomy because of persistent constriction. Conversely, Kim et al. (3) recently reported a case series in which the course was more benign, rarely requiring pericardiectomy, which suggested that ECP is reversible.

TCP, first described by Sagrista-Sauleda et al. (6), is characterized by reversible constrictive physiology due to transiently thickened and inelastic pericardium caused by inflammation. These findings were observed in the present case. In a study by Sagrista-Sauleda et al. (6) of 177 cases of acute effusive idiopathic pericarditis, 16 subjects (9.8\%) developed TCP, which improved within 12 days to 10 months (mean: 2.7 months), with none developing chronic constriction during the mean

## FIGURE 4 Plain Computed Tomography Scans on Day 4 and at 6 months


(A) Day 4 shows the thickened pericardium (arrows). (B) At 6 months.

(A, B) Echocardiography (parasternal long-axis and short-axis views, respectively) shows marked improvement. (C) Variation in diastolic transmitral flow velocity is improved. See Videos 5 and 6.
follow-up period of 31 months. Among 212 cases of constrictive pericarditis, Haley et al. (7) reported 36 cases (17\%) of TCP which improved with medical therapy within an average of 8.3 weeks. The main causes were idiopathic or viral (41\%), postpericardiotomy ( $25 \%$ ), and collagen vascular disease $(14 \%)$. The authors reported that 8 of the 36 patients with TCP required pericardiocentesis (7), indicating that those cases had transient ECP, as did the present case.

## FOLLOW-UP

The patient in the present case was discharged to home on day 12. Her clinical condition remained stable at the 1-month and 1-year follow-up examinations, with no signs or symptoms of chronic constriction on echocardiography and computed tomography scans (Figure 4B).

## CONCLUSIONS

ECP may be acute and transient and represent a potentially reversible phase of the spectrum of constrictive pericarditis $(8,9)$. It can improve spontaneously, or it may progress to irreversible phase of constriction if untreated. Early recognition is important because anti-inflammatory therapy, in addition to cause-specific treatment, can prevent progression (9).

ACKNOWLEDGMENT The authors thank Enago Co. for the English language review.

ADDRESS FOR CORRESPONDENCE: Dr. Kazuhito Hirata, Division of Cardiology, Okinawa Chubu Hospital, 281 Miyazato, Uruma, Okinawa 904-2293, Japan. E-mail: kheart911@yahoo.co.jp.

## REFERENCES

1. Lange RA, Hillis LD. Acute pericarditis. N Engl J Med 2004;351:2195-202.
2. Sagrista-Sauleda J, Angel J, Sanchez A, Per-manyer-Miralda G, Soler-Soler J. Effusiveconstrictive pericarditis. N Engl J Med 2004;350: 469-75.
3. Kim KH, Miranda WR, Sinak LJ, et al. Effu-sive-constrictive pericarditis after pericardiocentesis: incidence, associated findings, and natural history. J Am Coll Cardiol Img 2018; 11:534-41.
4. Syed FF, Ntsekhe M, Mayosi BM, Oh JK. Effu-sive-constrictive pericarditis. Heart Fail Rev 2013; 18:2777-87.
5. Imazio M, Brucato A, Maestroni S, et al. Risk of constrictive pericarditis after acute pericarditis. Circulation 2011;124:1270-5.
6. Sagrista-Sauleda J, Permanyer-Miralda G, Can-dell-Riera J, Angel J, Soler-Soler J. Transient cardiac constriction: an unrecognized pattern of evolution in effusive acute idiopathic pericarditis. Am J Cardiol 1987;59:961-6.
7. Haley JH, Tajik AJ, Danielson GK, Schaff HV, Mulvagh SL, Oh JK. Transient constrictive pericarditis causes and natural history. J Am Coll Cardiol 2004;43:271-5.
8. Gentry J, Klein AL, Jellis CL. Transient constrictive pericarditis: current diagnostic
and therapeutic strategies. Curr Cardiol Rep 2016;18:41.
9. Cremer PC, Kumar A, Kontzias A, et al. Complicated pericarditis: understanding risk factors and pathophysiology to inform imaging and treatment. J Am Coll Cardiol 2016;68:2311-28.

KEY WORDS constrictive pericarditis, effusive-constrictive pericarditis, transient pericarditis, cardiac tamponade

APRENDIX For supplemental videos, please see the online version of this paper.


[^0]:    From the ${ }^{\text {a }}$ Division of Cardiology, Okinawa Chubu Hospital, Uruma, Okinawa, Japan; and the ${ }^{\text {b }}$ Intensive Care Unit, Okinawa Chubu Hospital, Uruma, Okinawa, Japan. The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

    Informed consent was obtained for this case.

