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Takotsubo Syndrome in the Setting of Pneumonia and Suspected Chronic Obstructive Pulmonary Disease: A Therapeutic Dilemma

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Abstract

Takotsubo syndrome (TTS) is characterized by transient left ventricular systolic dysfunction, typically involving the apical region, and is often associated with physical stress. The clinical management of TTS in patients with concomitant respiratory disease can be challenging because of the therapeutic dilemma it presents. We report a case of TTS in the setting of pneumonia and suspected chronic obstructive pulmonary disease (COPD). The patient had a 100-pack-year smoking history and presented with fever (38.9°C), tachycardia (110 beats/min), and hypoxemia (oxygen saturation 89% on room air). TTS was initially suspected based on routine electrocardiography revealing ST-segment elevation and was subsequently confirmed by echocardiography. A transdermal β_1-blocker (bisoprolol) was administered for one week to control heart rate and attenuate adrenergic effects during the acute phase of TTS. After cardiac function improved, bisoprolol was discontinued, and an inhaled β_2-agonist (olodaterol) was initiated to treat respiratory symptoms in the setting of suspected COPD. This sequential treatment was well tolerated in this patient, without deterioration in cardiac or respiratory status. However, further evidence is needed to determine the safety and appropriateness of this treatment approach in other patients.

Categories: Pharmacology, Cardiology, Pulmonology
Keywords: echocardiography diagnosis, myocardial scintigraphy, smoking habits, β2-adrenergic agonist, β-blocker treatment

Introduction

Takotsubo syndrome (TTS) is characterized by transient left ventricular (LV) systolic dysfunction, typically involving the apical region, and predominantly affects older women. It is often associated with physical or emotional stress [1]. β-blockers are frequently used in the acute management of TTS [2], partly because of their presumed ability to attenuate sympathetic activation, although the underlying pathophysiological mechanisms and clinical benefits of β-blocker therapy remain incompletely understood [3-5].

In patients with known or suspected chronic obstructive pulmonary disease (COPD), β-blocker therapy may raise concerns about adverse respiratory effects [6]. Nonselective β-blockers, such as propranolol, may induce bronchoconstriction through β_2-receptor blockade and worsen airflow limitation [7], whereas selective β_1-blockers, such as bisoprolol, are generally preferred in patients with COPD, although they should be used with caution. Conversely, β_2-agonists, although essential for bronchodilation, may cause cardiovascular effects, including tachycardia and increased myocardial oxygen demand, which may be of particular concern in patients with TTS. Therefore, the management of patients with TTS and concomitant COPD presents a therapeutic dilemma.

Here, we report a case of TTS in the setting of pneumonia and suspected COPD in which a selective β_1-blocker and a β_2-agonist were used sequentially without deterioration in cardiac or respiratory status.

Case Presentation

A 78-year-old woman with a 100-pack-year smoking history was referred to our hospital for worsening dyspnea. On presentation, she was in a tripod position with marked dyspnea. She had experienced exertional dyspnea for several years and reported poor appetite and weight loss. Her cough and dyspnea had progressively worsened over the preceding two months, and she had been unable to smoke for one week before presentation because of her respiratory symptoms. She had no previous medical history and had never sought medical attention. Her body temperature was 38.9°C, blood pressure was 146/84 mmHg, heart rate was 110 beats/min, and oxygen saturation was 89% on room air. Her body weight was 32 kg. No peripheral edema or jugular venous distension was observed. Crackles were audible over the left lower lung field. Pneumonia was suspected, and she was urgently admitted with oxygen supplementation at 2 L/min.

Laboratory tests showed elevated inflammatory markers (white blood cell count, 11,800/μL; C-reactive protein, 15.73 mg/dL) but no anemia (Table *1*).

Variable	Day 1	Day 2	Day 7	Reference range
Blood count				
White blood cell count (×10^3/μL)	11.8	NA	6.5	3.3-8.6
Red blood cell count (×10^6/μL)	5.10	NA	4.04	3.86-4.92
Hemoglobin (g/dL)	14.9	NA	11.8	11.6-14.8
Platelet count (×10^3/μL)	256	NA	413	158-348
Blood chemistry				
Aspartate aminotransferase (U/L)	29	25	21	13-30
Alanine aminotransferase (U/L)	16	15	16	7-23
Lactate dehydrogenase (U/L)	199	189	159	124-222
Creatine phosphokinase (U/L)	64	56	24	41-153
Albumin (g/dL)	3.7	NA	2.9	4.1-5.1
Blood urea nitrogen (mg/dL)	26	NA	10	8-20
Creatinine (mg/dL)	0.62	NA	0.53	0.46-0.79
Sodium (mmol/L)	133	NA	142	138-145
Potassium (mmol/L)	3.6	NA	4.4	3.6-4.8
Chloride (mmol/L)	94	NA	107	101-108
C-reactive protein (mg/dL)	15.73	NA	4.46	<0.14
NT-proBNP (pg/mL)	3,476	NA	NA	<126
Troponin I (pg/mL)	150.2	NA	NA	0-26.2
Arterial blood gas				
pH	NA	7.441	7.443	7.350-7.450
$PaCO_2$ (mmHg)	NA	36.1	38.1	35.0-45.0
PaO_2 (mmHg)	NA	92.2	80.9	83.0-108
HCO_3^- (mmol/L)	NA	24.2	25.7	21.2-27.0

TABLE 1: Laboratory data

Arterial blood gas measurements were obtained with supplemental oxygen at 2L/min on day 2 and on room air on day 7.

NT-proBNP: N-terminal pro-B-type natriuretic peptide; NA: not available.

Chest radiography showed an infiltrative shadow in the left lower lung field, partially overlapping the cardiac silhouette (Figure *1A*, arrows). The lungs appeared vertically elongated, with a narrow cardiac silhouette. Chest computed tomography confirmed the infiltrative shadow in the left lower lobe (Figures *1B*, *1C*, arrows). Based on these findings, she was diagnosed with pneumonia.

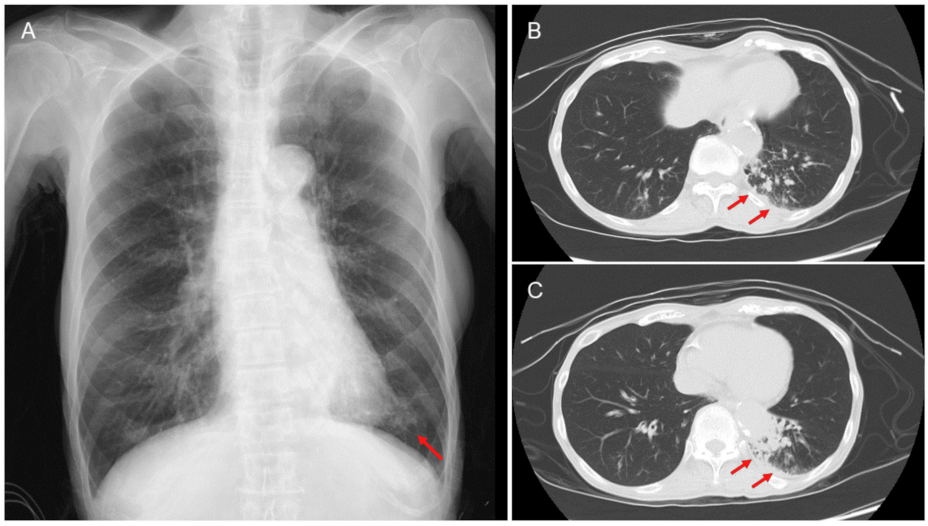


FIGURE 1: Chest radiography and computed tomography

Chest radiography showed an infiltrative shadow in the left lower lung field, partially overlapping the cardiac silhouette (A, arrows). Chest computed tomography confirmed the infiltrative shadow in the left lower lobe (B and C, arrows).

Although she did not report chest pain, routine electrocardiography (ECG) unexpectedly revealed ST-segment elevation in leads II, III, aV_F, $-aV_R$, and V_3-V_5 [8], with a maximum ST-segment elevation of 2 mm in lead V_3 (Figure 2, arrows). Terminal T-wave inversion was also noted in leads V_3-V_5, with a corrected QT (QTc) interval of 450 ms.

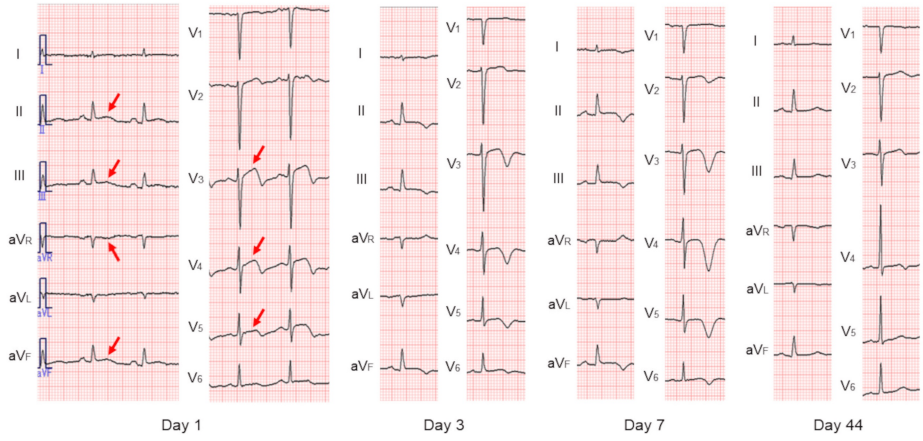


FIGURE 2: Serial electrocardiograms

Routine electrocardiography (ECG) on admission unexpectedly revealed ST-segment elevation in leads II, III, aV_F, $-aV_R$, and V_3-V_5 (arrows), with a maximum ST-segment elevation of 2 mm in lead V_3. Terminal T-wave inversion was also noted in leads V_3-V_5, with a corrected QT (QTc) interval of 450 ms. ST-segment elevation resolved, whereas T-wave inversion and QTc interval prolongation became more pronounced over one week. T-wave inversion and QTc interval prolongation had almost completely resolved by post-discharge day 44.

ECG recordings were obtained at a paper speed of 25 mm/s and an amplitude calibration of 10 mm/mV.

ECG: electrocardiography; QTc: corrected QT.

Additional laboratory tests showed elevated N-terminal pro-B-type natriuretic peptide and troponin I levels of 3,476 pg/mL and 150.2 pg/mL, respectively. Echocardiography revealed a small area of akinesis confined to the apical tip (Figure 3, arrows), with hyperkinesis of the basal segments. The LV ejection fraction was preserved at 70%, with no evidence of left ventricular outflow tract (LVOT) obstruction or mitral regurgitation. Based on the clinical course, TTS associated with respiratory failure was suspected.

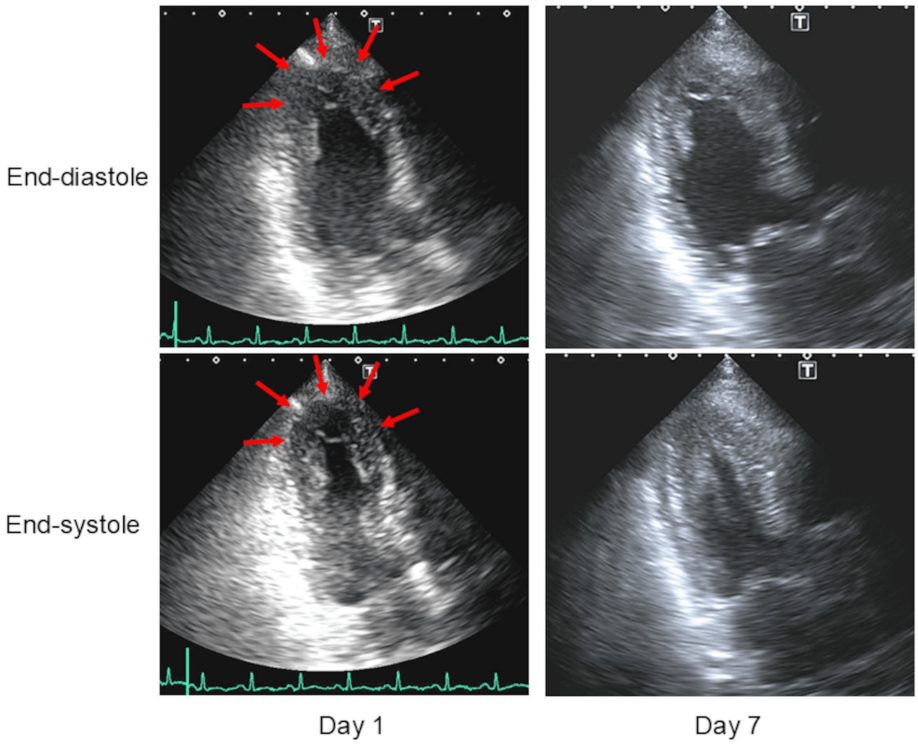


FIGURE 3: Serial echocardiographic images

Echocardiography revealed a small area of akinesis confined to the apical tip (arrows), with hyperkinesis of the basal segments. Follow-up echocardiography on hospital day 7 confirmed complete resolution of apical akinesis.

Images were obtained from the apical view.

Intravenous ceftriaxone (2 g/day) was initiated for pneumonia, and acetaminophen was administered as needed for fever. Azilsartan (10 mg/day) was initiated for elevated blood pressure, while transdermal bisoprolol (2 mg/day) was initiated for elevated heart rate. Following treatment initiation, both blood pressure and heart rate improved (Figure *4*). Because COPD was suspected based on her smoking history and chest radiographic findings, her respiratory status was closely monitored after bisoprolol initiation.

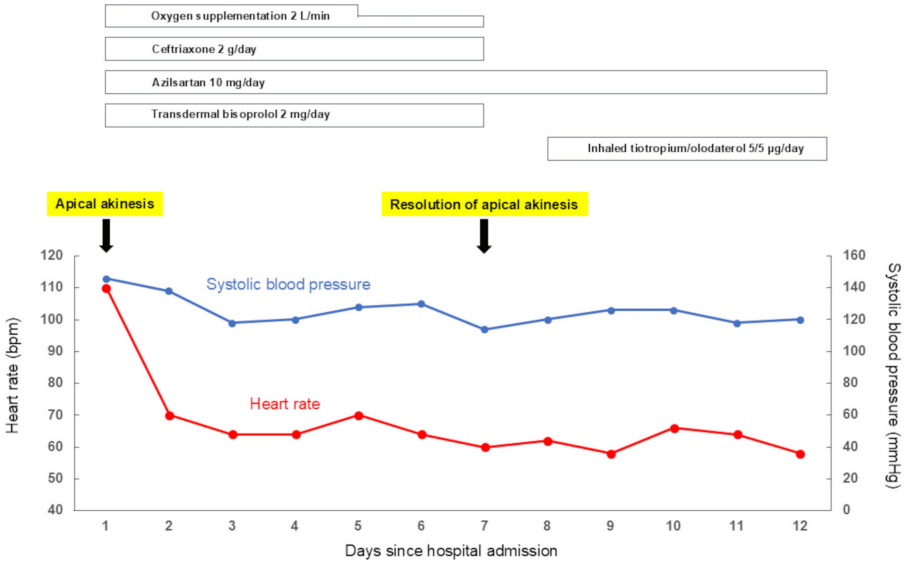


FIGURE 4: Clinical course

Transdermal bisoprolol (2 mg/day) was initiated on admission, resulting in prompt improvement in heart rate. Inhaled olodaterol (5 μg/day) was initiated on hospital day 8, after which the patient's dyspnea improved. The sequential use of transdermal bisoprolol followed by inhaled olodaterol was well tolerated under careful monitoring, without deterioration in cardiac or respiratory status.

Day 1 was defined as the day of hospital admission, and subsequent days were numbered consecutively.

As her pneumonia and respiratory status improved over the course of one week, supplemental oxygen and ceftriaxone were discontinued. ST-segment elevation resolved, and T-wave inversion became more pronounced on serial ECGs. The QTc interval progressively prolonged from 450 ms on hospital day 1 to 492 ms on day 3 and 505 ms on day 7 (Figure *2*). Follow-up echocardiography on hospital day 7 confirmed complete resolution of apical akinesis (Figure *3*), with an LV ejection fraction of 68% and no evidence of LVOT obstruction or mitral regurgitation. Bisoprolol was subsequently discontinued. On hospital day 8, dual-isotope myocardial scintigraphy using thallium-201 (^{201}Tl) and iodine-123 (^{123}I)-β-methyl-p-iodophenyl-pentadecanoic acid (BMIPP) demonstrated a marked perfusion-metabolism mismatch, with normal ^{201}Tl uptake and severely reduced ^{123}I-BMIPP uptake in the corresponding apical region (Figure *5*, arrows) [9]. These findings were consistent with the clinical course of TTS.

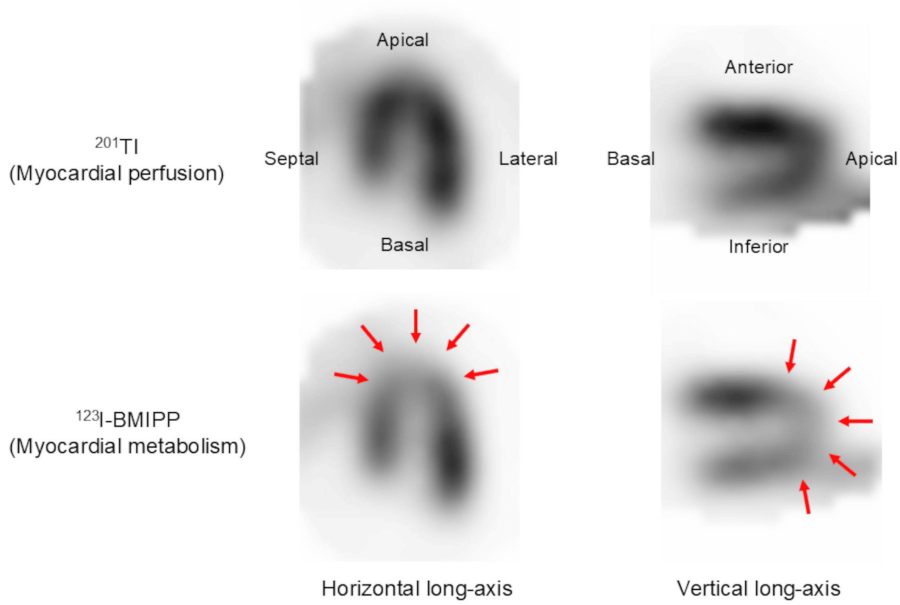


FIGURE 5: Dual-isotope myocardial scintigraphy

Dual-isotope myocardial scintigraphy using thallium-201 (^{201}Tl) and iodine-123 (^{123}I)-β-methyl-p-iodophenyl-pentadecanoic acid (BMIPP) demonstrated a marked perfusion–metabolism mismatch, with normal ^{201}Tl uptake and severely reduced ^{123}I-BMIPP uptake in the corresponding apical region (arrows).

On hospital day 8, spirometry showed an FEV_1/FVC ratio of 66%, indicating airflow obstruction consistent with COPD. Inhaled tiotropium/olodaterol (5/5 μg/day) was initiated. The patient's exertional dyspnea improved without clinically significant tachycardia or deterioration in cardiac function. Her clinical condition remained stable, and she was discharged on hospital day 12.

On post-discharge day 44, she remained free of recurrent symptoms and continued to abstain from smoking. T-wave inversion had almost completely resolved, and the QTc interval had returned to 450 ms (Figure 2).

Discussion

In this report, we describe a case of TTS in the setting of pneumonia and suspected COPD. Transdermal bisoprolol, a selective β1-blocker, was initiated during the acute phase of TTS. Inhaled olodaterol, a β_2-agonist, was subsequently initiated during the early recovery phase of TTS, after resolution of apical akinesis and identification of airflow obstruction, which raised suspicion of COPD. Although LV wall motion had recovered by the time olodaterol was introduced, ECG abnormalities, including QTc prolongation, persisted. Both therapies were administered for their respective indications without deterioration in cardiac or respiratory status.

Although the pathophysiology of TTS remains unclear, excessive sympathetic stimulation and catecholamine-mediated myocardial injury are thought to play important roles [1]. β-blockers are therefore frequently used in the acute phase to attenuate adrenergic effects [2-5]. TTS can be complicated by life-threatening events, including LVOT obstruction [2] and cardiac rupture [10,11]. Kumar et al. reported that cardiac rupture was associated with a higher double product, higher LV peak systolic blood pressure, persistent ST-segment elevation, and less frequent use of β-blockers, suggesting a potential role of excessive sympathetic activity and LV wall stress in its development [11]. In the present case, bisoprolol was initiated immediately after admission for marked tachycardia and ST-segment elevation in the setting of pneumonia, fever, and respiratory failure. During bisoprolol treatment, no cardiac adverse events occurred, and its negative chronotropic effect may have contributed to reducing cardiac workload.

The use of β-blockers in patients with COPD has traditionally raised concerns because blockade of β_2-receptors may cause bronchoconstriction and worsen airflow limitation. However, accumulating evidence suggests that cardioselective β-blockers can be used in patients with COPD without clinically significant adverse effects on respiratory status when there is a clear cardiovascular indication [6,12]. COPD does not necessarily contraindicate β-blocker therapy, although the degree of β_1 selectivity and the severity of airflow limitation should be considered. In the present case, transdermal bisoprolol was selected because of its relative β_1 selectivity in the setting of suspected COPD. In addition, the transdermal formulation allowed drug delivery to be discontinued by removing the patch if respiratory deterioration occurred, although

removal would not immediately reverse the pharmacological effects of bisoprolol that had already been absorbed. Respiratory status was closely monitored after initiation and remained stable throughout treatment. Bisoprolol was continued until recovery of cardiac function was confirmed one week later.

Notably, COPD has been reported in approximately 10-22% of patients with TTS [13,14]. COPD, its exacerbations, and associated respiratory stress may promote the development of TTS through inflammatory responses, hypoxia, and a catecholamine surge. However, TTS may be overlooked in patients presenting primarily with respiratory symptoms. In the present case, ECG abnormalities led to its recognition on admission for pneumonia. Thus, routine ECG assessment may be worth considering in patients admitted for noncardiac conditions, as concomitant cardiac conditions, including TTS, may occur even in the absence of prominent cardiac symptoms. COPD has been associated with an increased risk of TTS recurrence in observational studies [15]. A previous case report described a patient who experienced four episodes of TTS over a five-year period in association with COPD exacerbations [16]. In this context, preventing COPD exacerbations and maintaining adequate control of respiratory symptoms are important. In the present case, tiotropium/olodaterol could be introduced during the early recovery phase of TTS with close monitoring, without clinically significant tachycardia or deterioration in cardiac status. Our case suggests that inhaled β_2-agonist therapy may be considered when clinically indicated during the early recovery phase of TTS, with close monitoring of cardiac and respiratory status.

There are several limitations to this case. First, this is a single case, and the severity of both TTS and COPD varies among patients, limiting the generalizability of these findings. Therefore, treatment decisions should be individualized, and the sequential use of selective β_1-blockers and β_2-agonists requires careful monitoring and further clinical evidence. Second, the effect of bisoprolol on cardiac function is difficult to distinguish from the natural course of recovery from TTS. Third, because tiotropium and olodaterol were initiated simultaneously while pneumonia was resolving, the improvement in dyspnea could not be attributed specifically to olodaterol. Furthermore, the lack of follow-up spirometry precluded an objective assessment of pulmonary function after initiation of tiotropium/olodaterol therapy. Finally, given her pneumonia, impaired respiratory status, and poor nutritional status associated with marked weight loss, coronary angiography was not performed, and concomitant obstructive coronary artery disease could not be excluded. Nevertheless, the diagnosis of TTS was supported by the clinical course, the characteristic serial changes in ECG findings and LV wall motion, and the myocardial perfusion-metabolism mismatch on scintigraphy. In particular, the rapid resolution of apical akinesis and the subsequent evolution and resolution of ECG abnormalities were consistent with the typical clinical course of TTS.

Conclusions

In conclusion, this case highlights the importance of considering TTS in patients presenting with respiratory disease, even in the absence of prominent cardiac symptoms. In this patient, sequential treatment with transdermal bisoprolol, followed by inhaled olodaterol, was well tolerated, without deterioration in cardiac or respiratory status, under careful monitoring. Further evidence is needed to determine the safety and appropriateness of this treatment approach in other patients.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Satoshi Kurisu, Hitoshi Fujiwara

Acquisition, analysis, or interpretation of data: Satoshi Kurisu, Hitoshi Fujiwara

Drafting of the manuscript: Satoshi Kurisu

Critical review of the manuscript for important intellectual content: Satoshi Kurisu, Hitoshi Fujiwara

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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