Case Report

Change in type of sleep-disordered breathing from predominant central to obstructive sleep apnea following coronary artery bypass grafting

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ARTICLE INFO

Article history:

Received 13 March 2017

Received in revised form 18 May 2017

Accepted 26 May 2017

Keywords:

Sleep-disordered breathing

Central sleep apnea

Heart failure

Coronary artery bypass graft

ABSTRACT

We report the case of a 67-year-old overweight man with reduced left ventricular ejection fraction (LVEF) due to myocardial infarction. He had an implantable cardioverter defibrillator (ICD) for frequent episodes of ventricular tachyarrhythmia and was initiated into adaptive-servo ventilation therapy for severe central sleep apnea (CSA), which was not suppressed by continuous positive airway pressure (CPAP). Since he still had several episodes of appropriate ICD therapies, coronary angiogram was performed, and severe three-vessel disease was found. He then underwent coronary artery bypass grafting (CABG). After CABG, his LVEF did not improve (from 29 to 25%); however, the B-type natriuretic peptide level decreased (from 560 to 330 pg/mL). Although the apnea-hypopnea index did not change (49.4 before and 55.1/h after CABG), his CSA converted to predominant obstructive sleep apnea accompanied by a shortening of the lung-to-finger circulation time (from 43 to 29 s) 2 weeks after CABG, which was completely suppressed by CPAP.

<Learning objective: Central sleep apnea (CSA) can be alleviated by treatments for heart failure. However, limited data are available regarding resolution of CSA after coronary revascularization. We describe a case whose predominant CSA converted to obstructive sleep apnea after surgical coronary revascularization without any improvements in systolic function. Coronary revascularization can alleviate CSA possibly through altering congestive status and may unmask obstructive phenotype in patients with severe coronary artery disease.>

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Introduction

Sleep-disordered breathing (SDB) is one of the common complications of cardiovascular diseases, such as heart failure (HF) [1]. Two different types can coexist in patients with HF: obstructive sleep apnea (OSA), which arises from an upper airway collapse and central sleep apnea (CSA), which results from a central respiratory drive reduction [1]. CSA appears to be secondary

to HF and occurs when the partial arterial pressure of carbon dioxide (PaCO_2) falls below the apnea threshold in association with hyperventilation, which chronically occurs in association with pulmonary congestion and increased chemosensitivity in some HF patients. Besides, delayed transmission in arterial blood gas alterations because of impaired cardiac output causes ventilatory overshoot/undershoot, which typically manifests as Cheyne-Stokes respiration (CSR) [1].

The presence of CSA is reported to be associated with mortality in HF patients [1]. However, in patients with HF, such CSA may be alleviated by the initiation of a specific therapy [2] and surgical approaches for HF, including mitral valve repair or heart transplantation [3,4]. However, limited information is available regarding the severity of SDB and SDB phenotype changes after coronary revascularization for severe coronary artery disease

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[5,6]. Here, we describe a case of a patient with HF in association with an old myocardial infarction (OMI) and CSA, whose residual myocardial ischemia was found to be caused by three-vessel coronary artery disease and whose predominant CSA converted to an OSA phenotype following coronary artery bypass grafting (CABG).

Case report

A 67-year-old overweight (body mass index, 26 kg/m²) man with a history of hypertension, type II diabetes, chronic kidney disease, and paroxysmal atrial fibrillation had been diagnosed with HF with severely impaired left ventricular (LV) ejection fraction (LVEF) (29%) due to OMI in the antero-septal and inferior walls. He had an implanted cardioverter defibrillator (ICD) because of frequent episodes of sustained ventricular tachycardia (VT) without indications for cardiac re-synchronization therapy. He had also been diagnosed with severe CSA with a CSR pattern in association with a prolonged lung-to-finger circulation time (LFCT); thus, an adaptive servo-ventilation (ASV) for severe CSA was initiated because his CSA had not been alleviated by continuous positive airway pressure (CPAP) therapy. Even after ICD implantation and ASV initiation, he still had episodes of appropriate ICD therapies. Thus, coronary angiogram was performed, and he was found to have severe three-vessel disease with chronic total occlusion in the left anterior descending artery, as well as 75% stenosis in the high lateral branch of the dominant circumflex artery and right coronary artery. Since it was suggested that his frequent episodes of VT could be associated with residual ischemia due to severe three-vessel disease, urgent surgical management was elected.

An on-pump CABG was performed, combined with mitral valve plasty, cryoablation, and left atrial appendagectomy for coexisting mild mitral regurgitation (MR) and paroxysmal atrial fibrillation. The postoperative course was uneventful, and only non-sustained VT events were documented.

The preoperative transthoracic echocardiographic data showed an impaired LVEF, dilated LV and left atrial volume with high LV filling pressure determined by the ratio of early diastolic peak LV

Table 1

Changes in body weight, heart rhythm, echocardiographic data, BNP, arterial blood gas data, and polysomnographic parameters.

	Before CABG	2 weeks after CABG	27 months after CABG
Body weight, kg	65	63	61
Heart rhythm	Pacing	Pacing	Pacing
LVEF, %	29	25	25
RVSP, mmHg	45	30	26
LVEDV, mL	181	110	131
LVESV, mL	128	80	96
LAESV, mL	123	51	107
E/e'	37.9	18.6	21.7
MR grade	Mild	Trivial	Trivial
Plasma BNP level, pg/mL	560	330	292
PaCO ₂ , Torr	33	37	35.2
Slow-wave sleep, % of TST	0	1.2	0
REM sleep, % of TST	8.2	6.4	0
Supine sleep, % of TST	95.6	100	87.3
Arousal index, events/h	55.2	53.3	46.5
Total AHI, events/h	49.4	58.0	46.2
Obstructive AHI, events/h	4.3	44.1	31.3
Central AHI, events/h	45.1	13.9	14.9
Minimum SO ₂ , %	76.0	75.0	78.0
LFCT, s	43	29	31.8

CABG, coronary artery bypass surgery; LVEF, left ventricular ejection fraction; RVSP, right ventricular systolic pressure; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LAESV, left atrial end-systolic volume; E/e', Early diastolic peak LV pulse Doppler inflow velocities (E)/peak early diastolic mitral septum and lateral annular tissue Doppler velocities (e'); MR, mitral regurgitation; BNP, B-type natriuretic peptide; PaCO₂, partial pressure of arterial carbon dioxide; TST, total sleep time; REM, rapid eye movement; AHI, apnea-hypopnea index; SO₂, oxyhemoglobin saturation; LFCT, lung-to-finger circulation time.

pulse Doppler inflow velocities (E)/peak early diastolic mitral septum annular tissue Doppler velocities (e'), pulmonary hypertension determined by the estimated right ventricular systolic pressure (RVSP), and mild MR (Table 1). On polysomnography without any treatment for SDB, severe predominant CSA in association with a prolonged LFCT was observed (Table 1 and Fig. 1). The blood gas analysis before polysomnography showed a preserved oxygenation with hypocapnea (Table 1).

Fig. 1.

Representative raw wave forms from the first polysomnography before CABG. There are four central apneas and subsequent waxing and waning hyperventilation. During the central apneas, movements of the chest and abdomen are absent. Duration from the onset of the first breath terminating the apnea to the nadir of the subsequent dip in the SO₂ measured at the finger indicates the LFCT, which is considerably long (average of 10 consecutive apnea-hyperpnea cycles during the first episode of stage 2 sleep, 43 s).

CABG, coronary artery bypass surgery; EOG, electro-oculogram; EMG, submental electromyogram; EEG, electroencephalogram; ECG, electrocardiography; LFCT, lung-to-finger circulation time; SO₂, oxyhemoglobin saturation.

Fig. 2.

Representative raw wave forms from the first polysomnography 2 weeks after CABG. There are four obstructive apneas. During the obstructive apneas, out-of-phase movements of the chest and abdomen are observed. LFCT is shorter than that before CABG (average of 10 consecutive apnea-hyperpnea cycles during the first episode of stage 2 sleep, 29 s).

CABG, coronary artery bypass surgery; EOG, electro-oculogram; EMG, submental electromyogram; EEG, electroencephalogram; ECG, electrocardiography; LFCT, lung-to-finger circulation time; SO₂, oxyhemoglobin saturation.

Two weeks after the operation, echocardiography and overnight polysomnography without any treatment for SDB were repeated. In the follow-up echocardiogram, despite a decreased LVEF, reductions in LV volumes, left atrial volume, LV filling pressure, pulmonary hypertension, and MR grades were observed (Table 1). In his second polysomnography, his CSA converted to predominant OSA in association with a shorter LFCT (Table 1 and Fig. 2), although his apnea-hypopnea index (AHI) had slightly increased. Blood testing revealed a decreased plasma B-type natriuretic peptide (BNP) level and increased PaCO₂. There were no changes in medications at the time of polysomnography before and after CABG.

Because of persistent severe OSA, ASV was substituted by CPAP. Data downloaded from the CPAP machine showed a sufficient suppression of apneas and hypopneas even while on a lower CPAP level (i.e. 4–8 cmH₂O). Thus, the patient continued to use auto CPAP of 4–8 cmH₂O.

The patient underwent the 3rd polysomnography without CPAP 27 months after undergoing CABG, and the results showed that severe OSA and shortened LFCT were still present (Table 1).

Discussion

Although it has been suggested that SDB, particularly CSA, might diminish significantly following surgical procedures such as cardiac transplantation [3] or valve repair [4], only one report showed that CSA with a CSR pattern had been completely resolved after CABG with improved LV function, although there was a lack of polysomnography assessment [6].

Our case is slightly different from the previous case; although coronary revascularization by CABG provided rapid resolution of CSA, predominant OSA with a similar SDB severity existed following CABG. Thus, we found that an obvious change from predominant CSA to OSA was accompanied by a shortening of the LFCT following CABG. These findings are of particular interest, since in HF patients with ischemic cardiomyopathy, the presence of SDB is associated with an increased risk of long-term mortality [7]. This suggests the potential of SDB as a therapeutic target, but treatment options for SDB, particularly for CSA in patients with HF, have not yet been established [8]. Therefore, in HF patients with ischemic cardiomyopathy, if indicated, CABG may be another

means for alleviation of CSA, and presumably improve patients' long-term prognosis. However, despite the resolution of CSA, our case still had severe OSA following CABG.

These findings suggest an important caution for taking care of SDB following CABG in HF patients with ischemic cardiomyopathy. It should be also taken into account that refractory CSA prior to surgical revascularization in HF patients with ischemic cardiomyopathy may be easily managed by CPAP following CABG.

One possible elucidation for such change of SDB type without reduction in the SDB severity may include increased proportions of sleep time in the supine position or rapid eye movement sleep during the second polysomnography. However, our patient showed a similar proportion of sleep time in the supine position and shorter rapid eye movement sleep. Several reports have suggested that a reduction in CSA severity and a shift of SDB type occurred following heart transplantation [3] and following mitral valve repair [4]. These reports suggest that an immediate improvement in hemodynamics can resolve CSA. LFCT, which is suggestive of an increased forward cardiac output, was shortened in our case.

Another mechanism of conversion of SDB types might be the improvement of pulmonary congestion in association with LV diastolic dysfunction following resolution of residual ischemia by CABG. It was known that ischemia in the myocardium can cause LV diastolic dysfunction [9], and its resolution can improve such diastolic dysfunction, consequently reducing the LV filling pressure [10]. In our case, reductions in the parameters of LV filling pressure and BNP level were observed. In addition, an increased PaCO₂, which is suggestive of hyperventilation resolution in association with pulmonary congestion, was also observed. These suggest that the chronic hyperventilatory state of the patient had improved following CABG and that his preexisting obstructive phenotype may have become prominent. Thus, his SDB converted from predominant CSA to OSA because his ventilation improved and his preexisting upper airway obstruction had been unmasked [4].

Pharyngeal edema in association with a postoperative state may affect predominant OSA. However, considering the fact that in the 3rd polysomnography, performed 27 months after CABG, our patient still had severe OSA, a similar degree to that seen in the 2nd polysomnography, therefore, this is not the case in this patient.

Conclusion

Despite unclear specific mechanisms, the present case highlights the existence of patients whose SDB type may change and whose therapy for SDB has to be reconsidered following CABG.

Conflict of interest

Drs Takatoshi Kasai, Shoko Suda, Hiroki Matsumoto, Nanako Shiroshita, Mitsue Kato, and Fusae Kawana are affiliated with a department endowed by Philips Respironics, ResMed, Teijin Home Healthcare, and Fukuda Denshi. H. Daida received manuscript fees, research funds, and scholarship funds from Kirin Co. Ltd., Kaken Pharmaceutical Co., Ltd., Abbott Japan Co., Ltd., Astellas Pharma Inc., AstraZeneca K.K., Bayer Yakuhin, Ltd, Boston Scientific Japan K. K., Bristol-Myers Squibb, Daiichi Sankyo Company, MSD K.K., Pfizer Inc., Philips Respironics, Sanofi K.K., and Takeda Pharmaceutical Co. Ltd. The other authors have no conflict of interest.

Acknowledgments

This work is partly supported by a Grant-in-Aid for Scientific Research (C) [Grant Number 26507010; by a grant to the Respiratory Failure Research Group from the Ministry of Health, Labor and Welfare of Japan; by a Health, Labour and Welfare Sciences Research Grant, Research on Region Medical from the Ministry of Health, Labor and Welfare of Japan.

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