American Journal of Case Reports

ISSN 1941-5923
© Am J Case Rep, 2014; 15: 159-162
DOI: 10.12659/AJCR.890357

Received: 2014.01.13
Accepted: 2014.02.03
Published: 2014.04.19

New insights into the management of rhythm and conduction disorders after acute myocardial infarction

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1 Lyudmila Korostovtseva
ABCDEF 1 Yurii Sviryaev
CDE 1 Nadezhda Zvartau
BCD 2 Tatiana Druzhkova
CDG 3 Viktor Tikhonenko
DEG 3 Alexandra Konradi

1 Department of Hypertension, Federal Almazov Medical Research Centre, St, Petersburg, Russian Federation
2 1st Department of Cardiology, Federal Almazov Medical Research Centre, St. Petersburg, Russian Federation
3 Department of Functional Diagnostics, Federal Almazov Medical Research Centre, St. Petersburg, Russian Federation

Corresponding Author: Lyudmila Korostovtseva, e-mail: lyudmila_korosto@mail.ru and korostovtseva@almazovcentre.ru

Patient: Male, 53
Final Diagnosis: Myocardial infarction
Symptoms: Chest pain • tachycardia
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Challenging differential diagnosis

Background: Comorbidities, including obesity and sleep-breathing disorders, can adversely influence outcomes in acute myocardial infarction (AMI), and should be considered in diagnosis and treatment administration.

Case Report: The case demonstrates the difficulties of treating a middle-aged Caucasian patient with multiple comorbidities that could be overcome by a personalized approach and evaluation of concomitant sleep-breathing disorders (by polysomnography study). Diagnosis and treatment of sleep apnea by positive airway pressure (PAP therapy) played a pivotal role in heart rate and rhythm control.

Conclusions: In this case, effective PAP therapy enabled titration of antiarrhythmic drugs (to maximal doses) to achieve heart rate control and to eliminate severe ventricular tachyarrhythmias and contributed to the better recovery in a post-AMI patient with left ventricular systolic dysfunction.

MeSH Keywords: Acute Coronary Syndrome • Sleep Apnea Syndromes • Comorbidity • Tachycardia

Full-text PDF: http://www.amjcaserep.com/download/index/idArt/890357

1326 — 1 22

Background

In up to 20% of cases, acute myocardial infarction (AMI) is complicated by cardiac rhythm and conduction disturbances, leading to higher mortality rates [1,2]. Other factors and co-morbidities can cause or exacerbate rhythm and conduction disturbances and can adversely influence outcome and determine treatment. Therefore, these factors and comorbidities should be considered during the analysis of the clinical case. We present a case of an obese patient with a Q-wave myocardial infarction complicated by both cardiac rhythm and conduction disturbances that were alleviated by a successful complex treatment.

Case Report

The patient was a 53-year-old obese (body mass index, BMI 46.6 kg/m^2) Caucasian male with a history of long-term smoking, untreated essential hypertension, and with a family history of hypertension. He was admitted to the hospital with Q-wave myocardial infarction of the anterior wall and apex 5 days after symptoms onset (sudden fatigue, no typical chest pain reported). Troponin I was elevated to 0.83 ng/ml upon admission (normal <0.5) and subsequently decreased to 0.63 and 0.32 ng/ml at 6 and 12 h after admission. ECG on admission showed tachysystolic atrial fibrillation (AF) with heart rate (HR) 165 bpm, complete left bundle branch block (CLBBB) of unknown duration, and left ventricular hypertrophy. Cardiac ultrasound examination revealed severe dilation of all cardiac chambers; severe asymmetrical concentric left ventricular (LV) myocardial hypertrophy (MMI 326 g/m^2, relative wall thickness – 0.46); akinesis of the interventricular septum, apex, and lower wall of LV; ejection fraction (EF) 36%; and pulmonary systolic pressure 50 mm Hg.

The patient was stable upon admission; therefore, he was treated conservatively in the acute care unit with low molecular weight heparin, low-dose aspirin, clopidogrel, angiotensin-converting enzyme inhibitors (ACEi), beta-blockers, diuretics (torsemide, spironolactone), statins, gastroprotectors, and vitamin K antagonists titration under international normalized ratio (INR) control, and low fat and high fiber diet. Rhythm control was attempted with the use of amiodarone, but was ineffective. Beta-blockers (starting dose of metoprolol succinate 25 mg twice daily) were used for HR control.

At that point, 12-lead ECG monitoring showed AF as basic rhythm with mean HR 133 bpm in daytime (range 41–157) and 129 bpm during sleep (range 44–156); 1 paroxysm of non-sustained ventricular tachycardia (NSVT) and atrioventricular (AV)-conductive disorder (5 pauses >2000 msec) occurred at night. Therefore, further titration of beta-blockers was not possible.

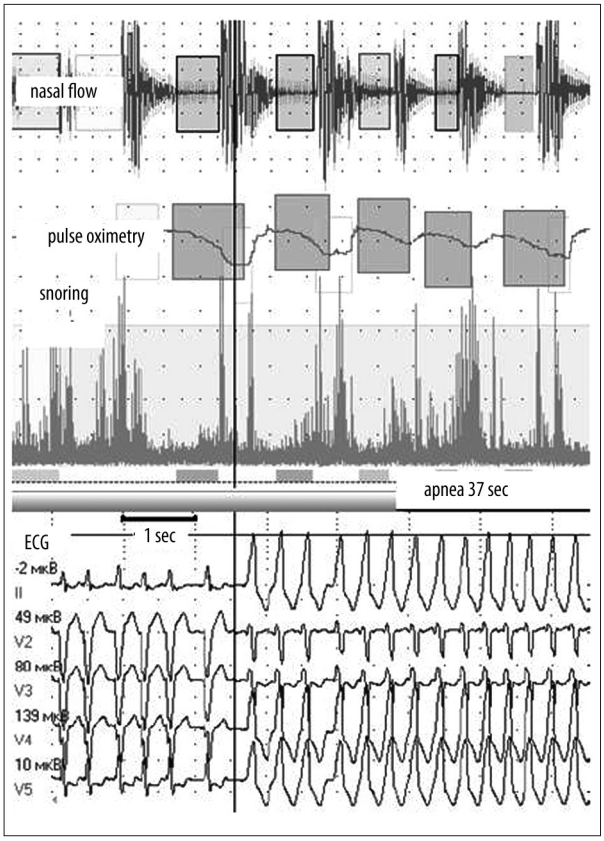

Figure 1. A paroxysm of non-sustained ventricular tachycardia associated with an episode of obstructive sleep apnea.

In view of the patient's severe ventricular arrhythmia, coronary arteriography was carried out and revealed proximal 70% and medial eccentric sub-occlusion of LAD. There were no lesions of LCx and RCA. PTCA and implantation of 2 non-drug-eluting stents in LAD were performed.

Twelve-lead ECG monitoring after successful revascularization showed 3 episodes of NSVT during sleep, although the ischemic nature of the rhythm and conduction disorders was controlled. The question of pacemaker implantation was raised [4–6].

Due to complaints of heavy snoring, daytime somnolence (12 scores on Epworth sleepiness scale), and daytime fatigue, the patient was referred to sleep study (cardiorespiratory monitoring) according to the guidelines and algorithm offered by Flemons (2002) [3,4]. Apnea-hypopnea index (AHI) was 62 episodes/h of sleep, and the mean and lowest O2 saturation levels were 87.7% and 69.4%, respectively. Thus, severe obstructive sleep apnea (OSA) was verified. Simultaneous ECG recording showed that NSVT were associated with apnea episodes (Figure 1). This suggested sleep apnea as the cause of the conduction and rhythm disorder.

Positive airway pressure (PAP) therapy was started with an automatic continuous PAP (autoCPAP) followed by bilevel PAP

replacement (BiPAP therapy, inspiratory pressure was set at 13 cm H_2O based on the mean autoCPAP pressure level, expiratory pressure – 9 cm H_2O) due to the presence of hypoventilation and low tolerance. Under BiPAP therapy, there was a successful attempt to increase the doses of antiarrhythmic drugs (beta-blocker+amiodarone) aimed at mild HR control. Metoprolol was titrated up to 100 mg twice daily after 6 months. Other treatments were: amiodarone 200 mg (5 days per week) for ventricular heart rhythm disturbances, zofenopril 7.5 mg twice daily, torsemide 5 mg, spironolactone 25 mg, low-dose aspirin 100 mg, clopidogrel 75 mg, simvastatin 10 mg, and warfarin 5 mg (under control of international normalized ratio, due to increased risk of stroke: CHA2DS2-VASc =3 scores, HAS-BLED =1 score [4]).

The patient showed high compliance to the prescribed treatment, including BiPAP therapy and life-style recommendations. According to the diaries and the data recorded by the PAP device, the patient did not miss a single night of device use, and mean time of PAP usage was 6.0 h. Subjectively, the patient noticed improvement of his general health, he did not complain of tachypnea or chest pain, did not have edema, and no symptoms reappeared during physical rehabilitation. Tiredness and daytime somnolence reduced significantly (a score of 5 on the Epworth sleepiness scale), and subjective exercise tolerance increased. On physical examination at 6 months, BMI decreased to 40.1 kg/m^2 (21 kg loss), respiration rate was 18 per min, HR – 90 bpm, BP – 120/66 mm Hg, and there was no edema.

ECG monitoring with simultaneous cardiorespiratory recording demonstrated AF, significant reduction of HR [mean HR at daytime – 90 bpm (74–114), at night – 85 bpm (76–97)], and *no conduction disturbances or ventricular arrhythmias*. Sleep apnea was effectively controlled: AHI and desaturation indices were 7 and 6 episodes per h of sleep, and mean and lowest O2 saturation level were 93.5% and 84.6%, respectively. Cardiac ECHO at 6 months showed a decrease in the LV volume (EDV – 183 ml, ESV – 108 ml) and an increase of the EF to 41%.

The outcome at the end of observation was alive-improved.

Discussion

The approach to the treatment of NSVT depends on the presence of underlying structural heart disease, for example, CAD in this case. NSVT in patients with known heart disease is associated with an increased risk of sudden death [1, 8] and requires treatment. As the time of AF onset was unknown in this case, electrical cardioversion was not indicated [5]. AV-node destruction and pacemaker implantation (CRT despite CLBBB was excluded due to EF>35% [5–7]) could be considered a possible approach for HR control that could enable therapy optimization, but AV-node destruction is irreversible, and should not be considered if other opportunities have not been tested. Moreover, in this particular case the patient refused pacemaker implantation.

Obstructive sleep apnea (OSA) is associated with an increased rate of cardiovascular morbidity and mortality [9–12]. Sleep-breathing disorders are known to be a trigger factor for both benign and life-threatening arrhythmias [13,14]. In a prospective study, patients with sleep-disordered breathing were shown to have 4-times higher odds of appropriate defibrillator activation for ventricular arrhythmias compared to subjects without sleep apnea, and most of them occurred at night [14]. Nocturnal onset of rhythm disturbances might be considered an indication for a sleep study with subsequent therapeutic intervention if necessary. However, although there are many predisposing factors linking sleep-disordered breathing and cardiac rhythm disturbances, including hypoxemia [15], increased oxidative stress [16], sympathetic hyperactivity, and arterial hypertension [17,18], the evidence on the effect of PAP therapy on survival is controversial [19–22], thus requiring further research. In our patient, a complex approach and PAP therapy administration helped to overcome the unfavorable drug-disease interaction and to control heart rhythm disturbances.

Conclusions

This clinical case demonstrates the difficulties of treating a patient with multiple co-morbidities. However, these difficulties can be overcome by a personalized approach. In an obese patient with CAD, AMI, and AF, sleep apnea can exacerbate the underlying condition, causing rhythm and conductive disturbances that could be successfully prevented by effective PAP therapy. In this case, effective BiPAP therapy enabled titration of antiarrhythmic drugs (to maximal doses) to achieve HR control and to eliminate severe ventricular tachyarrhythmias in a post-AMI patient with LV systolic dysfunction.

Acknowledgements

The authors thank Professor Eugene Shlyakhto, MD, PhD, Academician of the Russian Academy of Medical Sciences, Director of Federal Almazov Medical Research Centre (St Petersburg, Russia) for general support. We also would like to thank the patient and his family for the consent to publish these data.

References:

1. Van de Werf F, Bax J, Betriu A et al: Management of acute myocardial infarction in patients presenting with persistent ST-segment elevation: The Task Force on the management of ST-segment elevation acute myocardial infarction of the European Society of Cardiology. Eur Heart J, 2008; 29(23): 2909–45
2. Gang UJ, Jøns C, Jørgensen RM et al: Clinical significance of late high-degree atrioventricular block in patients with left ventricular dysfunction after an acute myocardial infarction – a Cardiac Arrhythmias and Risk Stratification After Acute Myocardial Infarction (CARISMA) substudy. Am Heart J, 2011; 162: 542–47.
3. Ward Flemons W: Obstructive sleep apnea. N Engl J Med, 2002; 347: 498–504
4. Epstein LJ, Kristo D, Strollo PJ Jr et al: Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clin Sleep Med, 2009; 5: 263–76
5. Camm AJ, Kirchhof P, Lip GY et al: Guidelines for the management of atrial fibrillation: The Task Force for the Management of Atrial Fibrillation of the European Society of Cardiology (ESC). Eur Heart J, 2010; 31(19): 2369–429
6. Vardas PE, Auricchio A, Blanc JJ et al: Guidelines for cardiac pacing and cardiac resynchronization therapy The Task Force for Cardiac Pacing and Cardiac Resynchronization Therapy of the European Society of Cardiology. Developed in Collaboration with the European Heart Rhythm Association. Eur Heart J, 2007; 28(18): 2256–95
7. Dickstein K, Cohen-Solal A, Filippatos G et al: ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008. Eur Heart J, 2008; 29(19): 2388–442
8. Harkness JR, Morrow DA, Braunwald E et al: Myocardial ischemia and ventricular tachycardia on continuous electrocardiographic monitoring and risk of cardiovascular outcomes after non-ST-segment elevation acute coronary syndrome (from the MERLIN-TIMI 36 Trial). Am J Cardiol, 2011; 108: 1373–81
9. Peker Y, Carlson J, Hedner J: Increased incidence of coronary artery disease in sleep apnoea: a long-term follow-up. Eur Respir J, 2006; 28: 596–602
10. Yaggi HK, Concato J, Kernan WN et al: Obstructive Sleep Apnea as a Risk Factor for Stroke and Death N Engl J Med, 2005; 353: 2034–41
11. Korostovtseva LS, Sviryaev YV, Zvartau NE et al: Prognosis and cardiovascular morbidity and mortality in prospective study of hypertensive patients with obstructive sleep apnea syndrome in St Petersburg, Russia. Med Sci Monit, 2011; 17(3): CR146–53
12. Parati G, Lombardi C, Narkiewicz K: Sleep apnea: epidemiology, pathophysiology, and relation to cardiovascular risk. Am J Physiol Regul Integr Comp Physiol, 2007; 293: R1671–83
13. Mehra R, Benjamin EJ, Shahar E et al: Association of nocturnal arrhythmias with sleep-disordered breathing: The Sleep Heart Health Study; Sleep Heart Health Study. Am J Respir Crit Care Med, 2006; 173: 910–16
14. Zeidan-Shwiri T, Aronson D, Atalla K et al: Circadian pattern of life-threatening ventricular arrhythmia in patients with sleep-disordered breathing and implantable cardioverter-defibrillators. Heart Rhythm, 2011; 8: 657–62
15. Shepard JW Jr, Garrison MW, Grither DA et al: Relationship of ventricular ectopy to nocturnal oxygen desaturation in patients with chronic obstructive pulmonary disease. Am J Med, 1985; 78: 28–34
16. Schulz R, Mahmoudi S, Hattar K: Enhanced release of superoxide from polymorphonuclear neutrophils in obstructive sleep apnea. Impact of continuous positive airway pressure therapy. Am J Respir Crit Care Med, 2000; 162: 566–70
17. Narkiewicz K, Somers VK: Sympathetic nerve activity in obstructive sleep apnoea. Acta Physiol Scand, 2003; 177: 385–90
18. Lavie P, Herer P, Peled R et al: Mortality in sleep apnea patients: a multivariate analysis of risk factors. Sleep, 1995; 18: 149–57
19. Doherty LS, Kiely JL, Swan V, McNicholas WT: Long-term effects of nasal continuous positive airway pressure therapy on cardiovascular outcomes in sleep apnea syndrome. Chest, 2005; 127: 2076–84
20. Cassar A, Morgenthaler TI, Lennon RJ et al: Treatment of obstructive sleep apnea as associated with decreased cardiac death after percutaneous coronary intervention. J Am Col Cardiol, 2007; 50: 1310–14
21. Martínez-García MA, Campos-Rodríguez F, Soler-Cataluña JJ et al: Increased incidence of nonfatal cardiovascular events in stroke patients with sleep apnoea: effect of CPAPtreatment. Eur Respir J, 2012; 39: 906–12
22. Barbé F, Durán-Cantolla J, Sánchez-de-la-Torre M et al., Spanish Sleep And Breathing Network: Effect of continuous positive airway pressure on the incidence of hypertension and cardiovascular events in nonsleepy patients with obstructive sleep apnea: a randomized controlled trial. JAMA, 2012; 307: 2161–68