Case Report

Prolonged Sinus Pause with Sleep Apnea Syndrome: A Case Report

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Abstract:

Cardiac rhythm during sleep is influenced by the autonomic nervous system and various pathological states. Most arrhythmias that occur during sleep are detected incidentally on Holter monitoring, and are in fact benign. However, sometimes they may be an important clue to an underlying disorder requiring further investigation and treatment. Efforts should be given to recognize factors predisposing to sleep-related arrhythmias; successful management of an underlying disorder can prevent and treat potentially dangerous cardiac rhythms. Here we describe a case of obstructive sleep apnea syndrome presenting with prolonged pause on Holter monitoring, which was corrected by continuous positive airway pressure (CPAP) therapy.

Key words: Arrhythmias, Cardiac, Sleep apnea syndromes, CPAP ventilation.

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Introduction:

Human beings spend approximately one-third of their lives asleep[1]. Although generally considered to be a peaceful stable period for the body and mind to rest and heal, sleep in fact represents a complex dynamic process involving dynamic changes in physiological state. Cardiac arrhythmia during sleep is common due to sleep-state dependent autonomic nervous system activity, especially in patients with cardiovascular diseases. Most arrhythmias that occur during sleep are detected incidentally on Holter monitoring and are benign, however, 15% of fatal ventricular arrhythmias still occur in sleep resulting in sudden nocturnal death.[1] Bradyarrhythmia, sinus pauses > 2 seconds and atrioventricular (AV) conduction delays commonly occur during sleep in young and healthy individuals specially more frequently in young athletes and less frequently in those over 80 years of age. In a series, long sinus pauses upto 9 seconds, were reported in 4 healthy athletes without cardiac disorders.[1] It is important to recognize predisposing factors to sleep-related arrhythmia in order to prevent and treat potentially dangerous cardiac rhythm. In patients with significant obstructive sleep apnea (OSA), even more dramatic conduction blockade can occur, and ventricular asystole of up to 15.6 seconds was described.[2]

Case report:

A 55-year-old obese hypertensive lady was referred to a cardiologist to the National Institute of Cardiovascular Diseases (NICVD), Dhaka for permanent pacemaker implantation for bradyarrhythmia. She had body mass index (BMI) 36 kg/m^2, disturbed sleep at night with snoring, choking and recurrent arousal. She also had daytime fatigue and somnolence. She complained of episodes of palpitation at night, but no syncopal attack or chest pain consistent with coronary artery disease. She had no history of thyroid disease. Her pulse was 68 bpm, regular and BP 140/90 mmHg. Other systemic examination findings were unremarkable. Investigation findings were: Hb 11.6 gm/dl, ESR 20 mm in 1st hour, WBC 7000/cmm, RBS 5.2 mmol/l, serum creatinine 0.7 mg/dl, serum Na^+ 143 meq/l, serum K^+ 4.6 meq/l, and serum TSH 1.45 µIU/ml. Her ECG showed sinus rhythm, with heart rate 62 bpm. Echocardiography revealed features of right-sided pressure and volume overload and moderate pulmonary HTN (PASP 50 mmHg). The polysomnogram showed apnea index of

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74.9, lowest desaturation 57% during rapid eye movement (REM) sleep. The associated ECG showed multiple significant Pause (longest >6 secs) and brady events (minimum heart rate 12 bpm) (Figure 1), and maximum pause coincided with maximum desaturation. Her coronary angiogram was normal (Figure 2). Electrophysiological (EP) study was done to exclude sinus node disease and revealed normal findings. Considering the clinical scenario and investigation findings, a diagnosis of OSA was made. Along with life style measures, she was offered continuous positive airway pressure (CPAP) ventilation during sleep. She responded well and her symptoms during sleep improved remarkably. Repeated polysomnography and Holter monitoring were done 8 weeks later; where her oxygen saturation was maintained above 94% throughout the night and there were few insignificant brady events at night. (Figure3)

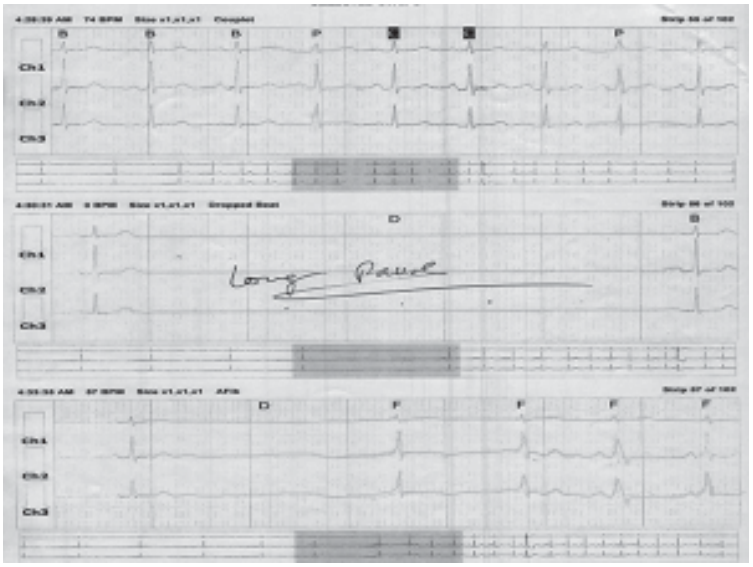

Fig.-1: *24-hour Holter ECG before CPAP therapy showing significant pause >6 seconds.*

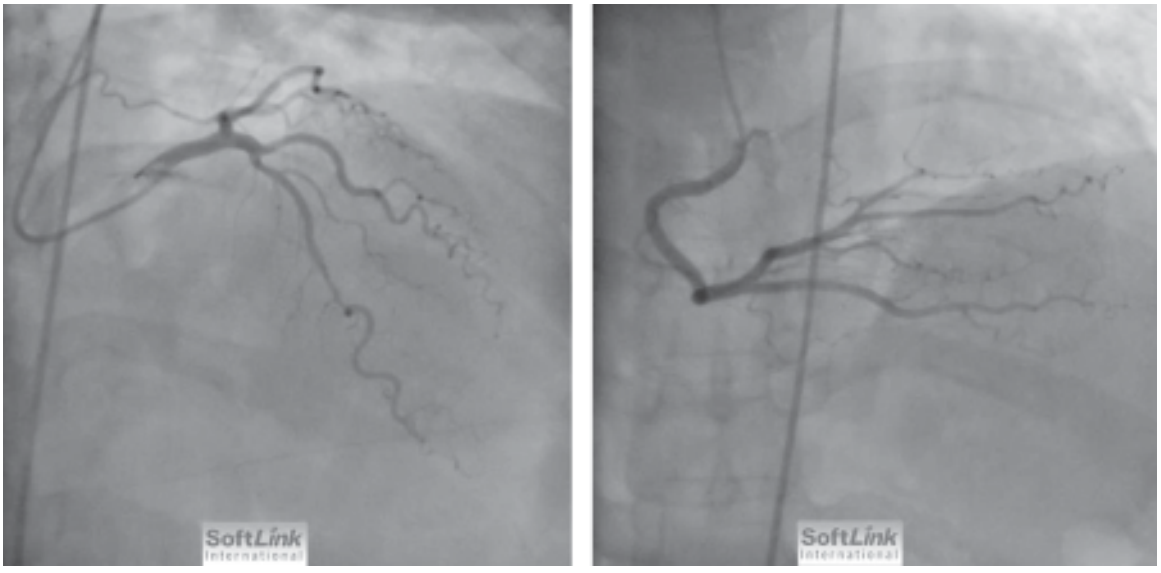

Fig.-2: *Coronary angiogram showing normal finding.*

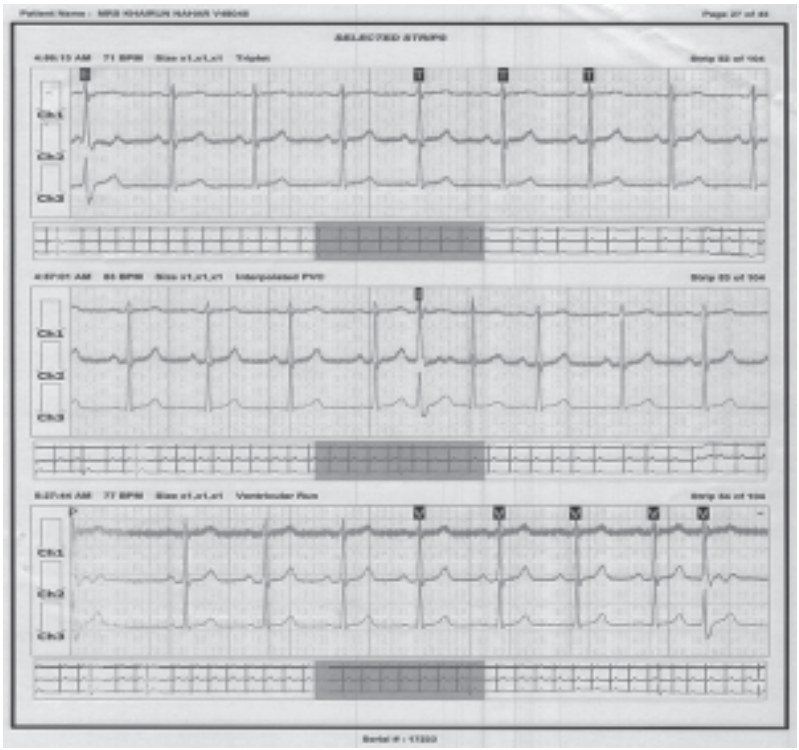

Fig.-3: *24-hour Holter ECG after CPAP therapy showing no significant pause.*

Discussion:

Cardiac rhythm is controlled by the sympathetic and parasympathetic system output during sleep. The autonomic nervous system in turn is affected by circadian rhythm and sleep states. Non rapid eye movement (NREM) sleep is accompanied by an increase in parasympathetic and a decrease in sympathetic tone. REM sleep is associated with decreased parasympathetic and variable sympathetic tone. Parasympathetic tone predominates at night since NREM sleep occupies 80% of total sleep time. Thus bradycardia and conduction delays commonly occur during sleep.[3]

In patients with OSA, repetitive pharyngeal collapse, resultant hypoxemia and arousals cause disturbances in autonomic system output. Arousals from NREM sleep can cause surges in sympathetic output with tachyarrhythmia as well as rise in blood pressure. Prolonged apnoea and hypoxemia is postulated to cause reflex increase in cardiac vagal tone, slowing cardiac conduction.[3] Thus either tachyarrhythmia or bradyarrhythmia may occur, depending on predominant autonomic system output. Becker et al. reported oxygen desaturation to 72% as a precondition to heart block.[4] Koehler and colleagues[2] did not find a threshold arterial oxygen saturation (SaO_2) below which conduction block occurred, with overall 56% of bradyarrhythmias occurring below saturation of 72%. Interestingly, the same study also showed 87% of sinus arrest occurring below oxygen saturation of 72%. The combination of apnea and hypoxemia is required to produce significant bradycardia.[4,5]

In the classic study by Guilleminault et al. involving 400 patients with OSA, 48% had significant nocturnal arrhythmia with 18% bradyarrhythmia, 11% sinus arrest, and 8% AV block.[6] A more recent study by Abe and colleague involving 1350 patients with OSA diagnosed by polysomnography demonstrated significant increase in incidence of sinus bradycardias (12.5% with OSA vs. 2.3% in normal subjects, $p=0.036$) and sinus pause (8.7% with OSA vs. 2.3% in normal subjects, $p<0.001$).[7] Roche et al. noted increase in prevalence of nocturnal paroxysmal asystole in patients with OSA as well as increase in prevalence of sinus bradycardias and pauses in association with increase in severity of OSA as measured by apnoea hypo-apnoea index and oxygen desaturation.[8]

CPAP therapy has been demonstrated to significantly reduce sleep related bradyarrhythmia, sinus pause and the increased risk of cardiac death.[9,10] In the Abe study,

sinus bradycardia and sinus pauses were reduced by CPAP therapy. No pacemakers were needed for treatment of bradyarrhythmia after CPAP therapy.[7] In the Becker study[4], 17 patients with OSA without established cardiac disease or conduction disorder were given CPAP therapy, 16 had resolution of bradyarrhythmia. Koehler's study[2] showed improvement and resolution of bradyarrhythmia in 12 out of 16 patients with 4 patients requiring pacemaker placement. Of the 4 requiring pacemaker, 1 was non-compliant with CPAP therapy and 3 had persistent sinus pauses >5 seconds despite effective CPAP treatment. Harbison reported resolution of bradyarrhythmia with CPAP treatment alone in all of 6 patients with sinus pause/AV block.[11] Thus in patients with bradyarrhythmias who are at risk for OSA, overnight polysmnography should be performed prior to pacemaker implantation, especially in younger individuals without underlying cardiac disease. Permanent pacemakers should be considered if significant bradyarrhythmia or pause persists after adequate treatment trial with CPAP therapy.

Our patient has no underlying conduction disorder and coronary artery disease which were excluded by EP study and coronary angiogram. The case presented here highlights the importance of recognizing the association between OSA and bradyarrhythmia. Since the bradyarrythmia and pause related to OSA are often relieved by CPAP, one option would be to treat such a patient with CPAP, combined with careful monitoring Holter ECG. Compliance with CPAP is variable and if life threatening bradyarrythmia and significant pause is present despite CPAP and are also for patients who have persistent sympatho-vagal imbalance and haemodynamic fluctuations resulting in day-time bradyarrhythmia - placement of permanent pacemaker may be preferred.

Disclosure: None.

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