

Case Report

Difficulties in Identification of Sleep Disordered Breathing in an Outpatient Clinic for Heart Failure— A Case Study

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Abstract

Sleep disordered breathing (SDB) is prevalent in patients with heart failure (HF). The clinical signs of newly diagnosed HF and untreated SDB may overlap and patients in need of SDB treatment can therefore be difficult to identify in patients participating in disease management programmes (DMP). The aim was to describe the care process of two patients with HF involved in a DMP, focusing on the difficulties to identify and initiate treatment of SDB. A prospective case study design was used to follow one male (70 yrs) and one female (74 yrs) patient during 18 months at a Swedish University hospital. It took 5 to 10 months from diagnosis of HF until optimal treatment was reached for their heart conditions and 12 to 17 months until SDB was treated. None of the patients complained of poor sleep, but suffered from fatigue. In the male SDB was detected by the wife's complaints of her husband's snoring, apnoeas and restless sleep. In the female, SDB was detected after a detailed assessment of fatigue which was shown to be sleepiness. After optimal treatment of HF but before initiation of SDB treatment both cases cardiac function improved. For the female case improvements also were found in the blood pressure. SDB treatment improved fatigue in both patients. Initiation of HF treatment and self-care routines, as well as identification of SDB is complex and time consuming. Treatment of HF and SDB can improve sleep, cardiac function as well as disturbing associated symptoms.

ABBREVIATIONS

ACE-I: Angiotensin Converting Enzyme Inhibitors; AHI: Apnea Hypopnea Index; ARB: Angiotensin Receptor Blockade; ASV: Adaptive Servo Ventilation; CSA: Central Sleep Apnea/Cheyne Stoke Respiration; CPAP: Continuous Positive Airway Pressure; DMP: Disease Management Programme; ESS: Epworth Sleepiness Scale; HF: Heart failure; IHD: Ischemic Heart Disease; LVEF: Left Ventricular Function; NT proBNP: N Terminal Fragment proBrain Natriuretic Peptide; NYHA: New York Heart Association Class; MRA: Mineralocorticoid Receptor Antagonists; ODI: Oxygen Desaturation Index; OSA: Obstructive Sleep Apnea; SDB: Sleep Disordered Breathing; TIA: Transient Ischemic Attack.

INTRODUCTION

Patients with heart failure (HF) are often elderly and

suffer from multiple comorbidities that have to be considered when initiating treatment regimen for HF [1]. At least half of the patients have slept disordered breathing (SDB), including obstructive sleep apnea (OSA) and central sleep apnea/Cheyne Stoke respiration (CSA) [2,3]. For the patient, as well as the healthcare professional, SDB can be a challenge because of sometimes overlapping symptoms that can be difficult to detect, e.g., the association between SDB and self reported sleepiness is weak in HF [4,3].

Adherence to a complex pharmacological and non pharmacological regimen is embedded in the care of patients with chronic heart failure (HF). Pharmacological treatment does not only mean initiation of, but also an aim to reach optimal doses of angiotensin converting enzyme inhibitors (ACE-I) or angiotensin receptor blockade (ARB), beta blockers, and

mineralocorticoid receptor antagonists (MRA). In case of volume overload addition of a diuretic is necessary [1]. An optimally treated HF may have important implications for SDB. This since CSA may occur as a consequence of an impaired left ventricular function (LVEF). Thus an optimally treated HF may therefore also decrease the severity of SDB [5,6]. Non pharmacological treatment means performance of an array of self care activities, such as to develop routines to take medication and recognition of symptoms and side effects of drugs, life style changes, as well as to make decisions on how to cope with changes in symptoms (e.g., a decision to take an extra diuretic or not) [7,8] To help the patient to cope with all these challenges participation in disease management programmes (DMP) therefore is recommended [1]. DMP:s are often based on a collaboration between HF nurses, cardiologists and other health-care profession, has been shown to improve HF patients self care behaviour, prognosis and quality of life [9]. More over DMP for HF has been shown to decrease hospital readmissions and is associated with favorable cost outcomes [10].

Detection of SDB and the timing of its treatment during the course of a DMP can therefore be challenging, but pivotal, since some studies suggests more cardiac readmissions [11] and deaths from SDB in HF [12, 13] whereas other studies does not [14]. Recommended treatment for OSA is continuous positive airway pressure (CPAP), which has shown to reduce blood pressure and improve LVEF [6]. In CSA, after pharmacological treatment has been optimized, CPAP or adaptive servo ventilation (ASV) is available treatment options [11]. ASV may be preferable since it, compared to CPAP, seem to be a more effective way to suppress CSA, improve quality of life and cardiac function [15]. However, treatment with CPAP or ASV can be demanding, and non adherence is a well know problem [11]. SDB treatment may improve the situation, but adds at the same time further complexity, especially in newly diagnosed patients who has to adapt to a new life situation. Knowledge about difficulties associated with identification of SDB and treatment initiation can therefore be useful for healthcare personnel working in DMP. The aim of this case study was to describe the care process of two patients with HF involved in a DMP, focusing on the difficulties to identify and initiate treatment of SDB.

CASE PRESENTATION

The context for the cases

Two patients with newly diagnosed HF from a University hospital in the southeast of Sweden were followed during 2012/2013 from diagnosis until they were considered to be optimally treated. The DMP at the Department of Cardiology consisted of four HF nurses working in close collaboration with six cardiologists, two physiotherapists, one dietician and one social worker. The routines are based on the European Society of Cardiology guidelines recommendation for drug titration and self care education [1]. The number of patient visits to the nurse led HF clinic exceeds 2000 per year. Each appointment is approximately 30 minutes and includes an examination of the patients' subjective and objective status, titration of drugs, and detection of possible side effects, as well as self care education. The number of visits and the time between the visits depends on the individual patients' situation and status. When the patients is

considered as optimally treated a decision is made if the future follow up should be at the HF clinic or in primary care.

SDB full night poly graphic respiratory recordings were performed according to standard routines at the Department of Clinical Neurophysiology. The recording consisted of nasal airflow, pulse oximetry, respiratory movements and body position. Apneas and hypopneas were manually scored divided by estimated sleep time and gave the apneahypopnea index (AHI). An oxygen desaturation index (ODI) was calculated in the same manner based on desaturations of $\geq 4\%$. Respiratory event scorings were based on American Academy of Sleep Medicine criteria [16]. The Epworth sleepiness scale (ESS) was used to measure excessive daytime sleepiness. The total score range from 0-24 points, with a cut off of >10 indicating excessive daytime sleepiness. SDB and insomnia symptoms were measured by a clinical tool developed at the sleep laboratory including 20 items. The patients grade their symptoms on a scale ranging from no problems (0), to very great problems (4). The study was approved by all responsible physicians at the different clinics and both patients provided informed consent. The study was conducted in accordance with the Helsinki declaration.

Case A

Initial hospital admission: Case A is a married 70 years old male patient with newly diagnosed HF. The HF was detected during care for a Transient Ischemic Attack (TIA) at the Department of Neurology (Table 1). The patient had been a smoker for 50 years, but had no previous diseases. After four days, fully recovered from his TIA, the patient was transferred to the Department of Cardiology because of an echocardiography showing severely impaired systolic function (i.e., LVEF 28%). At the Department of Cardiology HF treatment was initiated (i.e., ACE-I, Beta blocker, and MRA, Acetylsalicylic acid and lipid lower agents). To identify possible causes for the impaired systolic function a polyclinic coronary angiography was planned. A follow up visit at the HF clinic was scheduled after 3 weeks.

Care process: Data from the care-process during the 12 months follow up period are presented in (Table 2). During the six visits (i.e., four to the HF nurse and two to a cardiologist) the patients NYHA class was unchanged. Blood pressure and NT-proBNP were followed consecutively. Blood pressure remained stable whereas NT-proBNP decreased from 1080 ng/L (initial visit) to 650 ng/L after 13 months in relation to dose titration. LVEF improved from 28% to 45% after 12 months. Since the patient suffered from cough treatment with ACE-I had to be substituted to full dose of ARB (i.e., Candesatan 32 mg). The patient also suffered from cold fingers (i.e., symptoms of Raynaud's disease) and the Beta blockade had to be adjusted (i.e., decreased to 10% of the optimal dose). Side effects improved. Twelve months after diagnose the patient had optimal doses of ARB and MRA.

Three months after the HF had been diagnosed the patient suffered another ischemic stroke and was treated during five days at the department of Neurology. Warfarin treatment was initiated and the patient was referred home with no persisting neurological symptoms. A three month follow up to a neurologist was planned. After approximately three and a half month a

Table 1: Demographic and clinical data of the cases at the first admission and after 12 months.

	Case A At discharge after initial admission	Case A 12 months	Case B At discharge after initial admission	Case B 12 months
Gender	Male		Female	
Age, years	70	71	74	75
Livingalone (yes/no)	No	No	Yes	Yes
Smoking	Yes	No	No	No
Co-morbidities				
Ischemicheartdisease	No	No	No	No
Hypertension	No	No	Yes	Yes
Cerebrovasculardisease	Yes	Yes	No	No
PulmonaryDisease	No	No	No	No
Pacemaker	No	No	Yes	Yes
Laboratory data				
LVEF %	28%	45%	45-50%	No data
NT-proBNP, ng/L	1170 (<900)	650	4690 (<900)	1200
Creatinine, μmol/L	80 (137-145)	91	63 (45-90)	96
Potassium, mmol/L	4.4 (3.5-4.4)	4.0	3,4 (3.5-4.4)	4.2
Cloride, mmol/L	141 (60-105)	142	139 (137-145)	143
Corpulm-x ray	No	No	Yes	No
Bloodpressure mm/hg	130/70	125/73	150/80	130/60
Medication				
ACE/ARB, mg	Enalapril 5 mg	Candersatan 32 mg	Candersatan 4 mg	Candersatan 32 mg
Betablocker, mg	Metroprolol 150 mg	Bisprolol 1.25 mg	Metroprolol 25 mg	Metroprolol 150 mg
Diuretics, mg	Loopdiuretic 40 mg	Loopdiuretic 40 mg	Loopdiuretic 30 mg	Loopdiuretic 40 mg
MRA, mg	25 mg	25 mg	No	25 mg
ASA, mg	75 mg	No	No	No
Simvastine, mg	40 mg	40 mg	No	No
Warfarine	No	Yes	No	No

Table 2: Overview of care during the follow up period for the male patient (Case A) with heart failure.

Initial hospital admission		19 days	1 month	3 months	3.5 months	4 months
<i>Treated at dept. of neurology for ischemic stroke (4 days). Treatment?</i>	<i>Treated at dept. of Cardiology (3 days).</i>	<i>First visit HF nurse. Feeling well, no signs of fluid retention. Weight 79 kg. No documentation of sleep.</i>	<i>Second visit HF nurse. Stable HF, no side effects of drugs. No documentation of sleep.</i>	<i>Treated at dept. of Neurology for new stroke (5 days). Initiation of Warfarine.</i>	<i>Pol. visit to dept. of Cardiology. Coronary-angiography shows no IHD. Diagnosed With dilated cardiomyopathy.</i>	<i>Third visit HF nurse. No signs of HF, stopped smoking and gained weight (85 kg), severe problems with cough and cold fingers. No documentation of sleep.</i>
Referral for further treatment at the dept. of Cardiology.	Referral for follow up at the HF clinic.	NYHA II BP 130/70 NT-proBNP 1080 ng/L ACE-I increased to 10 mg.	NYHA I-II BP 128/60 NT-proBNP 1000 ng/L ACE-I increased to 15 mg.			NYHA II BP 110/60 NT-proBNP 780 ng/L Fulldose ACE-I. Change to ARB. Change of beta blocker.

5 months	6 months	8 months	10 months	11 months	12 months	13 months
<p>First visit cardiologist. Feeling well, no signs of HF. No severe side effects.</p> <p>NYHA II</p> <p>BP 129/82</p> <p>NT-proBNP 570 ng/L.</p> <p>Current doses:</p> <p>ARB 32mg</p> <p>B-blocker 1.25 mg</p> <p>MRA 25 mg</p> <p>Furosemide 40 mg</p>	<p>Visit to neurologist. Fatigue and restless sleep according to wife.</p> <p>Referred for SDB investigation.</p>	<p>Visit to sleep lab. Home based polygraphy. AHI 16, mostly CSA. ESS 11.</p> <p>Neurophysiologists sends referral for CSA treatment at the sleep lab.</p>	<p>Fourth visit HF nurse. Feeling vital, minor problems with lightheadedness. No signs of fluid retention according to ultrasound assessment. No sleeping problems.</p> <p>Weight 79 kg.</p> <p>NYHA II</p> <p>BP 125/73</p> <p>NT-proBNP 1090 ng/L</p> <p>Referral for echocardiography and visit to cardiologist.</p> <p>No more planned visits to HF nurse.</p>	<p>First visit CPAP nurse at the sleep lab.</p> <p>Information about CSA and AVS treatment.</p> <p>Mask adaptation and test of the treatment at the lab.</p> <p>Receives ASV device and mask to try at home.</p>	<p>Second visit CPAP nurse at the sleep lab.</p> <p>Adherence 83%</p> <p>AHI 1.6</p> <p>ESS 6</p>	<p>Second visit cardiologist. Feeling well, no signs of fluid retention.</p> <p>No documentation on assessment of sleep, nor effects of ASV treatment.</p> <p>NYHA II</p> <p>NT-proBNP 650 ng/L</p> <p>LVEF=45%</p> <p>Current doses:</p> <p>ARB 32mg</p> <p>B-blocker 1.25 mg</p> <p>MRA 25 mg</p> <p>Furosemide 40 mg</p> <p>Planned for referral to primary care.</p>

Table 3: Overview of care during the follow up period for the female patient (Case B) with heart failure.

Initial hospital admission	20 days	2 months	3 months	3.5 months	6 months	7 months
<p>Treated at the dept. of internal medicine for pulmonary oedema (3days).</p> <p>Referral for further treatment at the dept of Cardiology and HF clinic.</p>	<p>First visit cardiologist.</p> <p>Improved HF status, but still dyspnea and fatigue. No pitting oedema. No documentation of sleep.</p> <p>NYHA III</p> <p>BP 150/80</p> <p>NT-proBNP 3600 ng/L</p> <p>ARB increased to 12 mg.</p> <p>Furosemide 40 mg.</p>	<p>First visit HF nurse.</p> <p>Stable dyspnea and fatigue. New pitting oedema.</p> <p>Impaired adherence to Furosemide.</p> <p>Skeptical to dose titration.</p> <p>No nighttime dyspnoea.</p> <p>Weight 87 kg</p> <p>NYHA III</p> <p>BP 185/85</p> <p>NT-proBNP 3540 ng/L</p> <p>ARB increased to 20 mg. Furosemide increased to 80 mg.</p>	<p>Second visit HF nurse.</p> <p>Stable HF status, but still fatigued.</p> <p>Decrease of pitting oedema. Improved adherence to Furosemide.</p> <p>Weight 87 kg</p> <p>NYHA III</p> <p>BP 155/80</p> <p>NT-proBNP 1420 ng/L</p> <p>Increase ARB to 32 mg.</p> <p>Flexible dose of Furosemide 40/80 mg.</p>	<p>Third visit HF nurse.</p> <p>Unchanged HF status. Further improvement of adherence to Furosemide. Sleep not documented.</p> <p>Weight 85.5 kg</p> <p>NYHA III</p> <p>BP 140/75</p> <p>NT-proBNP 1010 ng/L</p> <p>Full dose ARB (32 mg)</p> <p>Betablocker increased to 50 mg</p>	<p>Fourth visit HF nurse.</p> <p>Unchanged HF status. Minor pitting oedema left leg.</p> <p>No signs of fluid retention according to ultrasound assessment.</p> <p>Improved appetite. Sleep not documented.</p> <p>Weight 89.5 kg</p> <p>NYHA III</p> <p>BP 120/60</p> <p>B-blocker increased to 75 mg.</p> <p>Furosemide increased to 80 mg during 4 days.</p>	<p>Fifth visit HF nurse.</p> <p>Unchanged HF status. No signs of fluid retention according to ultrasound assessment.</p> <p>Weight 89 kg</p> <p>NYHA III</p> <p>BP 125/75</p> <p>NT-proBNP 3420 ng/L.</p> <p>B-blocker increased to 125 mg. Flexible dose of Furosemide 40/80 mg.</p>
8 months	9 months	10 months	11 months	12 months	13 months	17 months
<p>Sixth visit HF nurse.</p> <p>Unchanged HF status.</p> <p>Weight 90 kg.</p> <p>NYHA III</p> <p>BP 140/60</p> <p>NT-proBNP 2140 ng/L</p> <p>B-blocker increased to 200 mg.</p> <p>Flexible dose of Furosemide 40/80 mg.</p>	<p>Seventh visit HF nurse.</p> <p>Unchanged HF status.</p> <p>Minor problems with lightheadedness.</p> <p>Weight 90 kg.</p> <p>NYHA III</p> <p>BP 128/60</p> <p>NT-proBNP 2370 ng/L</p> <p>Full dose B-blocker.</p>	<p>Eighth visit HF nurse.</p> <p>Increased problems with lightheadedness.</p> <p>NYHA III</p> <p>BP not measured.</p> <p>NT-proBNP 2250 ng/L</p> <p>B-blocker decreased to 150 mg.</p> <p>MRA initiated 25 mg.</p>	<p>Ninth visit HF nurse.</p> <p>Minor improvement of dyspnoea, still fatigued. Improved lightheadedness.</p> <p>Feeling sad. Sleep not documented.</p> <p>Weight 91 kg.</p> <p>NYHA III</p> <p>BP 100/70</p> <p>NT-proBNP 1060 ng/L.</p>	<p>Tenth visit HF nurse.</p> <p>Improved HF status. Mentions daytime sleepiness and snoring.</p> <p>Discussion about possible SDB investigation.</p> <p>Weight 91 kg.</p> <p>NYHA III</p> <p>BP 130/60</p> <p>NT-proBNP 1200 ng/L</p>	<p>Eleventh visit HF nurse.</p> <p>Problems with daytime sleepiness.</p> <p>Referral for SDB investigation.</p> <p>NYHA III</p> <p>BP 118/55</p> <p>NT-proBNP 1270 ng/L</p> <p><u>Current doses:</u></p> <p>ARB 32mg</p> <p>B-blocker 150 mg</p> <p>MRA 25 mg</p> <p>Furosemide 40/80 mg</p>	<p>Visit sleep lab.</p> <p>Polygraphy.</p> <p>AHI 24, mostly OSA. ESS 9.</p> <p>Neurophysiologist sends referral for CPAP treatment at the sleep lab.</p>

18 months	19 months	20 months
<p><i>Twelfth visit HF nurse.</i> Stable HF status, some nocturnal awakenings and daytime sleepiness.</p> <p>NYHA III BP 122/58 NT-proBNP 1120 ng/L. Referred to GP with current treatment: ARB 32mg B-blocker 150 mg MRA 25 mg Furosemide 40/80 mg</p>	<p><i>First visit CPAP nurse at the sleep lab.</i></p> <p>Information about OSA and CPAP treatment. Mask adaptation and test of the treatment at the lab. Receives CPAP device and mask to try at home.</p>	<p><i>Second visit CPAP nurse at the sleep lab.</i></p> <p>Feeling dizzy after CPAP initiation. BP 120/55 Adherence 89% ESS 9 AHI 5.7</p>

polyclinic coronary angiography showed no ischemic heart disease (IHD). Dilated cardio myopathy was identified as the aetiology for HF. The patient did not spontaneously mention any sleep problems during any of the visits to the HF clinic.

Identification and treatment initiation of sleep disordered breathing: SDB was suspected for the first time at the 6-month visit to the neurologist when the wife mentioned that the patient was a heavy snorer, had frequent apneas and suffered from a restless sleep (Table 2). However, the patient himself denied sleep problems. The neurologist referred the patient to a sleep registration. At the Department of Neurophysiology two months later a poly graphy showed a position dependent CSA with an AHI of 16 (i.e., supine AHI of 38). The patient scored only mild excessive daytime sleepiness (i.e., ESS score of 11), but indicated that he frequently suffered from non-restorative sleep and daytime sleepiness on the sleep laboratory's clinical tool. The neurologist informed the patient by letter that treatment with ASV should be initiated shortly at the Department of Neurophysiology. Three months later treatment was initiated by a CPAP nurse during two visits. AHI decreased to 1.6 and the ESS score improved to 6 and the patient felt better. Objective adherence to treatment was good with 80% of the nights showing an ASV use >4 hours (i.e., data derived from the device). A twelve months follow-up visit was planned to the CPAP nurse at the sleep clinic.

Case B

Initial hospital admission: Case B is a divorced 74 years old female patient with newly diagnosed HF (Table 1). The HF was detected during a care episode for pulmonary oedema successfully treated by CPAP at the Department of Internal Medicine. Previously, the patient had hypertension and a pacemaker (i.e., due to AV-block III), but no IHD. She also suffered from impaired mobility due to osteoarthritis in her knees. Initially an x-ray showed pulmonary oedema and an enlarged heart. An echocardiography performed after three days showed a hypertrophic heart and mildly impaired systolic function (i.e., LVEF 45%) and moderate mitral valve insufficiency. At the Department of Internal Medicine HF treatment was initiated (i.e., a low dose of ARB and Furosemide). The patient was referred to the HF clinic and met a cardiologist after 20 days who increased the ARB dose.

Care process: Data from the care process during the 18 months follow up period are presented in (Table 3). During thirteen visits to the HF clinic (i.e., eleven to the HF nurse and two to a cardiologist) the patients NYHA class was unchanged,

however, the HF nurse perceived the symptoms to be slightly improved during the care process. Blood pressure was somewhat unstable, but decreased from 150/80 to 130/60 after 12 months. NT-proBNP fluctuated, but decreased from 3600 ng/L to 1200 ng/L after 12 months. During the care process the HF nurse had to motivate the patient to accept initiation and titration of HF medication. One reason was that the patient felt that she had to consume too much medicine. Another reason was incontinence problems associated with an increased dosage of Furosemide. The problems were solved by letting the patient use a flexible Furosemide dose (i.e., 40 mg one day, 80 mg the next day). During the titration of Beta blockers the weight and NT-proBNP increased. The patient suffered from dizziness and the Beta blockade was decreased to 150 mg (i.e., 75% of optimal dose). Initiation of Spironolactone after 10 months decreased NT-proBNP by 50% to 1060 ng/L. However, despite an almost optimal HF treatment (i.e., ARB 100% of optimal dose, Beta blockade 75% of optimal dose) and a relatively balanced NT-proBNP at 12 months the patient still complained of fatigue.

Identification and treatment initiation of sleep disordered breathing: Further assessment by the HF nurse (i.e., questions about sleepiness and snoring) at the 12-month follow up indicated a possible SDB and the patient was after a discussion with a cardiologist referred for a sleep registration. At the Department of Clinical Neurophysiology one month later, the poly graphy showed a position dependent OSA with an AHI of 24 (i.e., supine AHI of 44). The patient scored no excessive daytime sleepiness (i.e., ESS score of 9), but indicated on the sleep laboratory's clinical tool that she frequently suffered from non restorative sleep and daytime sleepiness. The sleep physician recommended that treatment with CPAP should be initiated at the Department of Neurophysiology. Two months later treatment was initiated by a CPAP nurse during two visits. AHI decreased to 5.7, the Epworth sleepiness scale was unchanged, but fatigue decreased. Objective adherence to treatment was good with 89% of the nights showing a use >4 hours (i.e., derived from the device).

DISCUSSION

This case study has shown that it can be difficult to identify patients with SDB and that the initiation of HF treatment and self care routines is complex and time consuming. Communication between the patient and the nurse is pivotal to identify SDB and to avoid a prolonged phase of treatment initiation. Titration of HF drugs to reach optimal doses and an adherent behaviour requires

that the nurse carefully monitor signs and symptoms, as well as laboratory data. Due to different types of side effects the nurse has to adjust doses in collaboration with the cardiologist.

It took 5 to 10 months from diagnosis of HF until optimal treatment was reached and 12 to 17 months until SDB was treated. Causes for the relatively long time to reach optimal doses were related to both pathophysiological and behavioural aspects of side-effects caused by HF drugs. The male patients suffered from cough and cold fingers, whereas the female patient suffered from dizziness and incontinence problems that caused non adherence. As a consequence, the different drugs had to be adjusted, or changed, in several steps requiring more frequent follow up visits. The clinical assessment by the nurse and an evaluation of data regarding blood pressure, weight and blood samples guided the drug titration. Interestingly, in the female case NT-proBNP and weight increased from the third to the fifth visit to the HF nurse during the titration of Beta blocker (i.e., Metoprolol 25 mg to 125 mg in three steps). No signs of fluid retention were, however, identified. Recent studies have shown that titration of Beta blockers can increase levels of brain natriuretic peptides without causing decompensation [17]. Knowledge of facilitators and barriers that has an impact on adherence in clinical situations is important for nurses and other healthcare personnel. Side-effects of treatment are common barriers to adherence in HF. Information, support and reflective listening are some facilitators that can promote adherence to self care [18]. Increased knowledge and understanding for the treatment can act as an invitation for the patient to take an active role in the decision making regarding their own treatment, and thus lead to increased adherence [18]. The support delivered by the HF nurse during the initial and middle phase of the care process may have helped the female patient to understand the purpose of the medication. This and a flexible dose of diuretics promoted a sense of participation in the decisions of her own treatment and hence an acceptance to take diuretics. Healthcare personnel can balance between different ways to educate and motivate patients about vital decisions. Either a paternalistic decision making, an appropriate information giving, a concealed persuasion or a complete handover of the decision to the patient can be used [19]. Shared decision making highlights the importance of patient participation during deliberated and determined decisions seems to be a useful way of acting [20, 21]. In both cases there were problems to identify SDB and the treatment initiation took relatively long time (i.e., 7-10 months after optimal HF doses were established). To identify patients with a possible SDB earlier in the care process in a DMP, healthcare professionals therefore have to work consciously and structured. General recommendations for how to find patients in need of sleep evaluation and potential treatment for SDB have been published by [22]. The primary step in the recommendations of American Academy of Sleep Medicine is based on a routine health examination, patient complaints and characteristics of SDB (e.g., restless sleep, daytime sleepiness), as well as an evaluation of occurrence of comorbidities associated with high risk of having SDB (i.e., obesity and hypertension). Based on this, the male patient was slightly overweight whereas the female patient was obese and had hypertension. Both patients initially denied sleep problems on specific questions, but suffered from fatigue. However, it is should be remembered

that approximately 40-50% of the patients with OSA also suffer from fatigue [23]. This increases the risk for SDB to remain undetected, since healthcare professionals most probably will consider fatigue to be a symptom of HF. Another problem is that fatigue and sleepiness are often used interchangeably, but differ conceptually. Sleepiness is often operationalized as the propensity to fall asleep, whereas fatigue involves mental and physical components and overlap with other constructs, such as lack of energy, tiredness and lack of strength [24]. Assessing if the patient is sleepy or suffer from fatigue can therefore provide important information. Later on the female patient admitted sleepiness when more specific questions regarding her complaints of fatigue were asked by the HF nurse. The ESS did only identify mild daytime sleepiness in the male patient. In the other questionnaire used at the sleep clinic both patients, however, indicated non restorative sleep. The ESS can be difficult to use in patients with HF, since measurement problems have been detected regarding some of the items, especially in female patients [25]. The importance of asking the "right questions" and having a spouse that informs about e.g., snoring and apneas can therefore be seen as important. Another cause that the SDB related symptoms were not highlighted in both cases might be that the patients did not consider them as HF related issues. An increased awareness concerning new/unknown clinical signs and symptoms that can be collected/measured in a DMP setting may help healthcare personnel to identify those who are in need of SDB evaluation/treatment at an earlier stage. Simple two channel devices can be used as a complement to clinical assessments and specific instruments for sleep problems such as the Pittsburgh Sleep Quality Index [26] or the Berlin sleep apnea questionnaire [27].

CPAP or ASV treatment for HF patients may not only relieve SDB but has also been found to improve cardiac function and/or treat possible underlying causes, such as hypertension [6,28,29]. This suggests that CPAP/ASV treatment may be initiated early in the care process at a DMP. However, adherence can be difficult to establish at the same time in two new and complex treatments [18]. Furthermore, an optimally treated HF with improved LVEF can from a pathophysiological perspective decrease the severity of SDB [5,6]. When this is achieved initiation of treatment with e.g., CPAP or ASV may not be necessary, or if still needed (e.g., if the patient suffers from sleepiness) initiation might be easier. This means that healthcare personnel should initially focus on optimization of treatment (i.e., titration of HF drugs, cardiac resynchronization therapy), symptom relief, and self care education [6]. One may consider that initiation of SDB treatment late in the care-process can hinder improvements in HF. In the present study cardiac function as measured by echocardiography and/or NT-proBNP improved for both patients' before treatment of SDB was established. For the female patient blood pressure also improved before SDB treatment. Hard endpoints should not be the only treatment incitements for SDB. Of importance is that both cases suffered from fatigue and sleepiness after that the HF drugs were optimally titrated which improved after treatment of SDB had been initiated. Considering adherence and quality of life, one might think that if a patient is not suffering from severe SDB (e.g., with an obvious impact on cardiac function and/or daily functioning) the care-process should follow the ESC guidelines [1].

If a sleep problem still can be suspected after optimal treatment is established, sleep should be evaluated in several steps. In the initial step, the clinical assessment should include specific sleep related questions focusing on the occurrence of fatigue, sleepiness, non restorative sleep, snoring and witnessed apneas. Patients indicating sleep problems should in a second step be given the possibility to respond to e.g. Pittsburgh Sleep Quality Index [26] or the Berlin sleep apnea questionnaire [27]. Sensitivity and specificity can, however, be a problem, and simple objective recording devices for sleep apnea detection can therefore be added as a second step. Patients with a high suspicion of SDB should be referred to a sleep clinic for further investigation and possible treatment initiation. CPAP/ASV treatment is associated with adherence problems. It may therefore be favourable that the patients are informed by the healthcare personnel at the DMP about the treatment options of SDB and which affects that may be expected before referral is done. This may help the HF patient to prepare for and to make a deliberated and determined decisions regarding treatment of SDB.

CONCLUSION

The care process in a DMP is complex and time consuming. Individualized optimal HF treatment requires careful monitoring and a close collaboration between the health care professional and the patient. A patient centred care and good communication is pivotal to educate and motivate the patient to be adherent to HF treatment, as well as to increase the possibility to identify symptoms caused by SDB in need of specific treatment.

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REFERENCES

- McMurray JJ, Adamopoulos S, Anker SD, Auricchio A, Bohm M, Dickstein K, et al. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC. *Eur Heart J*. 2012; 33: 1787-847.
- Oldenburg O, Lamp B, Faber L, Teschler H, Horstkotte D, Töpfer V. Sleep disordered breathing in patients with symptomatic heart failure: a contemporary study of prevalence in and characteristics of 700 patients. *Eur J Heart Fail*. 2007; 9: 251-257.
- Johansson P, Arestedt K, Alehagen U, Svanborg E, Dahlstrom U, Brostrom A. Sleep disordered breathing, insomnia, and health related quality of life -- a comparison between age and gender matched elderly with heart failure or without cardiovascular disease. *Eur J Cardiovasc Nurs*. 2010; 9:108-1017.
- Redeker NS, Muench U, Zucker MJ, Walsleben J, Gilbert M, Freudenberger R, et al . Sleep disordered breathing, daytime symptoms, and functional performance in stable heart failure. *Sleep*. 2010; 33: 551-560.
- Lamba J, Simpson CS, Redfearn DP, Michael KA, Fitzpatrick M, Baranchuk A. Cardiac resynchronization therapy for the treatment of sleep apnoea: a meta-analysis. *Europace*. 2011; 13: 1174-1179.
- Khayat R, Small R, Rathman L, Krueger S, Gocke B, Clark L, et al. Sleep-Disordered Breathing in Heart Failure: Identifying and Treating an Important but Often Unrecognized Comorbidity in Heart Failure Patients. *Journal of Cardiac Failure*. 2013; 19: 431-44.
- Dickstein K, Cohen-Solal A, Filippatos G, McMurray JJV, Ponikowski P, Poole-Wilson PA et al. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008: The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association of the ESC (HFA) and endorsed by the European Society of Intensive Care Medicine (ESICM). *European Journal of Heart Failure*. 2008; 10: 933-989.
- Lainscak M, Blue L, Clark AL, Dahlström U, Dickstein K, Ekman I, et al. Self-care management of heart failure: practical recommendations from the Patient Care Committee of the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail*. 2011; 13: 115-126.
- Strömberg A, Mårtensson J, Fridlund B, Levin LA, Karlsson JE, Dahlström U. Nurse-led heart failure clinics improve survival and self-care behaviour in patients with heart failure: results from a prospective, randomised trial. *Eur Heart J*. 2003; 24: 1014-1023.
- Maru S, Byrnes J, Carrington MJ, Stewart S, Scuffham PA. Systematic review of trial-based analyses reporting the economic impact of heart failure management programs compared with usual care. *Eur J Cardiovasc Nurs*. 2014.
- Khayat R, Abraham W, Patt B, Brinkman V, Wannemacher J, Porter K, et al. Central sleep apnea is a predictor of cardiac readmission in hospitalized patients with systolic heart failure. *J Card Fail*. 2012; 18: 534-540.
- Wang H, Parker JD, Newton GE, Floras JS, Mak S, Chiu KL, et al. Influence of obstructive sleep apnea on mortality in patients with heart failure. *J Am Coll Cardiol*. 2007; 49: 1625-1631.
- Ancoli-Israel S, DuHamel ER, Stepnowsky C, Engler R, Cohen-Zion M, Marler M. The relationship between congestive heart failure, sleep apnea, and mortality in older men. *Chest*. 2003; 124: 1400-1405.
- Roebuck T, Solin P, Kaye DM, Bergin P, Bailey M, Naughton MT. Increased long-term mortality in heart failure due to sleep apnoea is not yet proven. *Eur Respir J*. 2004; 23: 735-740.
- Kasai T, Kasagi S, Maeno K-i, Dohi T, Kawana F, Kato M, et al. Adaptive Servo-Ventilation in Cardiac Function and Neurohormonal Status in Patients With Heart Failure and Central Sleep Apnea Nonresponsive to Continuous Positive Airway Pressure. *JACC: Heart Failure*. 2013; 1: 58-63.
- Medicine AAOs. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep*. 1999; 22: 667-689.
- Luchner A, Behrens G, Stritzke J, Markus M, Stark K, Peters A, et al. Long-term pattern of brain natriuretic peptide and N-terminal pro brain natriuretic peptide and its determinants in the general population: contribution of age, gender, and cardiac and extra-cardiac factors. *Eur J Heart Fail*. 2013; 15: 859-867.
- Siabani S, Leeder SR, Davidson PM. Barriers and facilitators to self-care in chronic heart failure: a meta-synthesis of qualitative studies. *Springerplus*. 2013; 2: 320.
- Shaw D, Elger B. Evidence-based persuasion: an ethical imperative. *JAMA*. 2013; 309: 1689-1690.
- Braddock CH 3rd, Fihn SD, Levinson W, Jonsen AR, Pearlman RA. How doctors and patients discuss routine clinical decisions. Informed decision making in the outpatient setting. *J Gen Intern Med*. 1997; 12: 339-345.

21. Elwyn G, Frosch D, Thomson R, Joseph-Williams N, Lloyd A, Kinnersley P, et al. Shared decision making: a model for clinical practice. *J Gen Intern Med.* 2012; 27: 1361-1367.
22. Epstein LJ, Kristo D, Strollo PJ Jr, Friedman N, Malhotra A, Patil SP, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med.* 2009; 5: 263-276.
23. Mills PJ, Kim JH, Bardwell W, Hong S, Dimsdale JE. Predictors of fatigue in obstructive sleep apnea. *Sleep Breath.* 2008; 12: 397-399.
24. Bardwell WA, Ancoli-Israel S, Dimsdale JE. Comparison of the effects of depressive symptoms and apnea severity on fatigue in patients with obstructive sleep apnea: a replication study. *J Affect Disord.* 2007; 97:181-186.
25. Ulander M, Arestedt K, Svanborg E, Johansson P, Broström A. The fairness of the Epworth Sleepiness Scale: two approaches to differential item functioning. *Sleep Breath.* 2013; 17: 157-165.
26. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res.* 1989; 28:193-213.
27. Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Ann Intern Med.* 1999; 131: 485-491.
28. Arzt M, Floras JS, Logan AG, Kimoff RJ, Series F, Morrison D, et al. Suppression of central sleep apnea by continuous positive airway pressure and transplant-free survival in heart failure: a post hoc analysis of the Canadian Continuous Positive Airway Pressure for Patients with Central Sleep Apnea and Heart Failure Trial (CANPAP). *Circulation.* 2007; 115: 3173-80.
29. Momomura S. Treatment of Cheyne-Stokes respiration-central sleep apnea in patients with heart failure. *J Cardiol.* 2012; 59: 110-116.

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