Comorbidity in the obese patient: the paroxysmal Pickwickian with pulmonary peculiarities and poor pump performance

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Abstract

Obesity is a growing epidemic worldwide and presents an important challenge to health care providers as a result of its association with chronic health conditions and early mortality. Treatments aim to target obesity itself, and the comorbid complications that may arise from it. Early diagnosis and treatment are paramount. However, symptoms are often multifactorial in etiology, and can be difficult to dissociate from one another. Furthermore, diagnostic tests and interventions can also be particularly challenging in this patient population. ■ Heart Metab. 2010;48:28–31.

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Introduction

The growing level of obesity in developed countries is expected to fuel a surge in cardiovascular diseases over the next few decades, with significant increases in arterial hypertension, coronary artery disease, heart failure, and stroke (Table I) [1]. Early disease detection and prompt intervention are of particular importance in this high-risk group. However, accurate symptom assessment can be challenging at times, driven by the presence of several, and potentially contributory, comorbidities, in addition to the recognized practical limitations in available diagnostic tests. We present a case report of a patient with obesity under multidisciplinary care for symptoms of breathlessness, in whom many of these issues arise.

Case report

A 58-year-old lady was referred for further assessment of exertional breathlessness by her endocrinologist in January 2008. Her medical history at this time was remarkable for morbid obesity (body mass index [BMI] 41.1 kg/m² and waist circumference 110 cm), cerebrovascular disease, hypothyroidism, tablet-controlled diabetes mellitus, and previous venous thromboembolism, for which she was receiving lifelong anticoagulation agents. The patient was a lifelong smoker. She also complained of morning headaches, peripheral edema, and daytime somnolence (Epworth sleepiness score 19).

Initial investigations revealed a resting arterial oxygen pressure of 7.5 kPa, with compensated hypercapnia (pCO₂ 8.1 kPa, HCO₃⁻ 32 mmol/L, pH 7.36).
A high apnea–hypopnea index on overnight oximetry suggested superimposed obstructive sleep apnea. There was marked restriction on pulmonary function testing (forced vital capacity [FVC] 1.36 L) consistent with her obesity, but also obstruction to flow (forced expired volume in 1 s [FEV1] 0.8 L, FEV1/FVC = 0.59) with moderate reversibility on salbutamol challenge. Pulmonary embolism was excluded by computed tomography pulmonary angiography. The patient was hypertensive, with a deranged lipid profile. Echocardiography was limited by poor windows related to body habitus. Transthoracic imaging with intravenous contrast suggested preserved biventricular systolic function, diastolic impairment (E/E' 15), bi-atrial dilatation, and increased pulmonary artery pressures (mean 58 mm Hg).

Loop diuretics and an angiotensin-converting enzyme (ACE) inhibitor were commenced in addition to her regular medications. The patient was established on domiciliary continuous positive airways pressure (CPAP) with supplemental oxygen. These interventions helped considerably with her fatigue, peripheral fluid retention, and blood pressure control. Her BMI reduced modestly to 38.9 kg/m2 in combination with targeted anti-obesity medication under close outpatient surveillance by the endocrinologists. However, she required admission to hospital on several occasions throughout 2008 for infective exacerbations of her airways disease and fluid retention. Furthermore, she remained very limited by persistent exertional dyspnea.

Towards the end of 2008, it became apparent that the patient was experiencing paroxysms of atrial fibrillation with rapid ventricular conduction, which was considered contributory to her continuing symptoms (Figure 1). She was reluctantly commenced on amiodarone therapy, given her background airways fragility and underlying thyroid dysfunction. Coincidentally, she developed left bundle branch block (QRS duration 142 ms) at this time and described intermittent chest pains unrelated to her arrhythmia. Repeat contrast echocardiography demonstrated delayed septal contraction, consistent with her conduction disease, and a marked deterioration in her cardiac function, with a calculated ejection fraction now of 30%. This was confirmed on a myocardial perfusion scan, together with probable anterolateral ischemia on dobutamine stress testing, although again there was considerable signal attenuation and artefact as a result of body habitus. Her weight and pulmonary congestion made it very difficult for her to lie flat, but coronary angiography performed with CPAP support and light sedation via the radial approach reassuringly demonstrated normal anatomy.

By autumn of 2009, the patient’s BMI was static at 38 kg/m2, but she was not considered fit for bariatric surgery. She had required several admissions for optimization of her ventilatory support earlier in the year. CPAP therapy was converted to non invasive ventilation because of poorly controlled hypercapnia (Figure 2) and she was commenced on home nebulizers. Her arrhythmia burden was increasing in frequency and severity, with very poor rate control during atrial fibrillation despite the addition of high-dose digoxin and a calcium channel antagonist. Her ejection fraction (in sinus rhythm) remained impaired on optimal ACE inhibition and the addition of a potassium-sparing agent to her diuretic regimen. Her medical care at this stage was coordinated predominantly between respiratory, cardiology, and endocrine services, with cross-disciplinary support throughout from physiotherapy, occupational therapy, and social services. There was additional input from psychiatry and palliative care because of her deteriorating quality of life.
In early 2010, the patient’s condition had degenerated to sustained atrial fibrillation and she was really quite limited in simple activities. She was not considered fit for general anesthetic for external cardioversion, and therefore underwent internal cardioversion under local anesthetic and sedation, with a reasonable improvement in her breathing. Her arrhythmia is anticipated to return, however, and her systolic ventricular function remains markedly depressed. She is being considered for permanent pacing (with an additional left ventricular lead for cardiac resynchronization) and atrioventricular node ablation as a safer alternative to pulmonary vein intervention for control of the atrial fibrillation and heart failure.

Comorbid disease in the obese patient

The prevalence of comorbid disease is significantly increased in overweight and obese individuals [2]. In some cases, it is difficult conclusively to dissociate cause from effect. However, as active weight loss reduces this increased risk to health, it is likely that...
obesity contributes directly towards the pathogenesis in many of these disease processes [3–7]. This case report demonstrates some of the complexities in the management of comorbid conditions in the obese patient, and also highlights the importance of a multidisciplinary approach to health care provision in this population. With increasing levels of obesity worldwide, this is, unfortunately, set to become an all too familiar scenario for clinicians.

**REFERENCES**


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**Case report**

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Figure 2. Overnight oximetry recording. Oxygen saturation (SpO2), carbon dioxide pressures (PCO2), and heart rate (bpm) were monitored transcutaneously. Continuous positive airways pressure therapy (with 2 L oxygen) was commenced during the study (red arrow). The presence of atrial fibrillation accounts for the marked heart rate variability. Mean arterial SpO2 remained low throughout the night and PCO2 climbed progressively. As a result of these findings, the patient was converted from CPAP to non invasive ventilation.