

Special Edition
"Revisiting Physical Diagnosis in
Respiratory Medicine"

Case Report

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Cheyne-Stokes Respiration Revisited: Clinical Clue to the Diagnosis for Acute Exacerbation of Congestive Heart Failure

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CASE REPORT

A 72-year-old man was admitted to the hospital to initiate chemotherapy for pleomorphic lung carcinoma (T4N0M1a, stage 4 cancer). He had a history of chronic systolic heart failure with severe mitral regurgitation diagnosed five years prior to this admission who had been receiving cardiac resynchronization therapy (CRT). He had complained of nocturnal dyspnea, especially when lying flat, as well as of dyspnea on exertion. A few weeks prior to his admission, his dyspnea worsened to the point that he could not even walk a few steps, demonstrating a rapid deterioration of performance status (PS).

On examination, he was in mild distress, with a performance status of 3 out of a possible score of 5, defined as capable of only limited self care; confined to bed or chair more than 50 percent of waking hours.¹ His vital signs were normal, except for his sinus tachycardia, with an elevated rate of 108 beats per minute. His oxygen saturation while breathing ambient air was above 90%. However, he was later noted to experience nocturnal desaturation as his oxygen saturation dropped to 80-85% at night while he was sleeping. His liver function tests (LFTs) were significant for aspartate aminotransferase (AST) 51 international units per litre (IU/L), alanine aminotransferase (ALT) 359 IU/L, alkaline phosphatase (ALP) 360 IU/L, and γ -glutamyl transpeptidase (γ -GTP) 125 IU/L.

On the third day of the admission, he complained of increased shortness of breath at night. On examination, he was in respiratory distress and tachypnic. The body temperature was 37.3 °C, blood pressure of 94/70 mm Hg, pulse of 118 beats per minute, respiratory rate of 28 breaths per minute, and an oxygen saturation of 85% while he was breathing ambient air. He had cyclic crescendo-decrescendo breathing, which was accompanied by apnea and desaturation, and his apnea lasted for about 10 seconds per cycle, consistent with Cheyne-Stokes respiration (CSR) (Video 1). Although no apparent adventitious cardiac sounds were noted, his jugular venous pressure was slightly elevated to 10 cm H₂O with cold extremities, as well as marked elevation of serum brain natriuretic peptide (BNP) level (1274 pg/mL). He was thus diagnosed with acute congestive heart failure (CHF) with a congestive liver and was treated with oral furosemide (20 mg per day). His dyspnea, nocturnal hypoxemia, and CSR, as well as abnormal LFTs, improved over the following few days, and he was transferred to the sub-acute care facility where he continued to receive further therapy.

CSR has been recognized in 30-50% of patients with chronic CHF² and the prevalence is much higher when CHF is more severe.^{3,4} In general, hypoxemia in patients with CSR is

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Video 1: Breathing pattern of patient with cheyne-stoke respiration.

mild (oxygen saturation is usually above 80-85%). Some articles report that the presence of CSR is a poor prognostic factor for CHF,^{5,6} but conflicting data exists that would indicate otherwise.⁷ A CSR is not only a predictor of poor prognosis of CHF, but CSR may worsen the heart failure itself. Therefore, timely diagnosis and intervention, such as diuresis and/or bilevel positive airway pressure (BiPAP), are essential. In this case, it was with the careful bedside examination to observe this breathing pattern and with careful examination of neck veins that we detected acute worsening of congestive heart failure, since there was no apparent adventitious cardiac sound noted. This case reminds us of the importance of careful inspection at the bedside, and of the fact auscultation of the chest is not the only part of the physical examination to detect heart failure.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

CONSENT

The authors obtain written informed consent from the patient for submission of this manuscript for publication.

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