Obesity Hypoventilation Syndrome and Long-Term Sequelae of COVID-19 Pneumonia

Brian Burlew, MD

Lehigh Valley Pulmonary Critical Care

1

Case Report

- GH, 63 yo male smoker (60 pk-yrs), OSA with prior failure of CPAP therapy presents with panic attack after 3 persons close to him including best friend died in a several-day period
- Also upset that power recliner chair that was to arrive that day had been delayed and wouldn't arrive until late November
- No hx of COPD although term mentioned to him in the past
- No inhalers or home O2
- No prior PFT

2

Case Report

- PMH: CHF, spinal stenosis, GERD, Hyperlipidemia, HTN, DM, prior CVA
- P&SH: Married, but separated. Wife still looks after him. Disabled truck driver. No EtOH
- PE: Morbidly obese 6'1", 419# (BMI>50), HR 58, BP 144/71, RR 19, O2 sat RA rest 91%
- Decreased BS throughout with a prolonged expiratory phase, RRR, 1+ brawny LE edema
- CXR, portable – hard to interpret, ProBNP 519
- ABG 7.30/66/99/32/97% on 5L NC, COHB 6.5%
- SARS Coronavirus 2 negative, serum HCO3 34, Cr 1.55
- Prior polysomnogram: 4/2/18: AHI 75.5 events/hr, Nadir SpO2 78%

3

Obesity Hypoventilation Syndrome

- Defined as the presence of awake alveolar hypoventilation in an obese individual which cannot be attributed to other conditions
- Associated with increased cardiovascular morbidity and mortality

4

Risk Factors

- Obesity (BMI>30 kg/m2)
- Prevalence as high as 50% with severe obesity (BMI>50)
- Risk factors in obese patients may include: . . significant increase in waist:hip ratio (central obesity)
- . Reduced lung function due to obesity
- . Reduced inspiratory muscle strength
- . Severe obstructive sleep apnea (AHI>50events/hr.)
- . Male gender not a risk factor, unlike OSA
- . Patients usually present in the 5th and 6th decades of life

5

Clinical Manifestations

- Nonspecific and reflect manifestations of obesity and coexistent OSA (present in 90% of pts with OHS) or of OHS-related complications, such as pulmonary HTN
- Hypersomnolence, loud snoring, choking during sleep, resuscitative snorting, fatigue, impaired concentration and memory, small oropharynx and thick neck. 70% of patients have severe OSA (AHI>30 events/hr
- 10% of OHS pts without OSA more likely to be older females

6

Clinical Manifestations

- Many pts present with chronic stable symptoms or chronic hypercapnic respiratory failure
- 1/3 present with acute on chronic respiratory failure, prompting hospital admission
- Women may present later than men
- Often misdiagnosed with COPD or asthma, in spite of non-obstructive spirometry
- Daytime hypoxemia and significant sustained reductions in overnight oximetry, uncommon in OSA or obesity alone
- Right heart failure (DOE, elevated JVP, hepatomegaly, edema)

7

Laboratory Tests

- Elevated Serum bicarbonate (>27 mEq/L, nonspecific and not 100% sensitive either
- Hypercapnia (pCO_2 of > 45mmHg on ABG or > 50mmHg on VBG when awake and on RA
- Hypoxemia (pO_2 < 70mmHg) – usually present, A-a gradient classically normal, severe nocturnal desaturation is also common (O_2 sat < 80%)
- Polycythemia – late manifestation and uncommon
- Pulmonary function tests, more common to see restriction than in eucapnic obese pts but normal PFT's do not exclude diagnosis. Severe restriction is uncommon
- Imaging – cardiomegaly due to RVH, elevation of diaphragms, RVE on EKG or echo

8

Diagnostic Approach

- Establish awake hypoventilation in obese pt in absence of other causes of alveolar hypoventilation
- Sleep study with continuous nocturnal CO2 monitoring is gold standard and to see whether hypoventilation is associated with OSA
- In pts presenting with acute on chronic hypoventilation, Rx comes first with firm diagnosis later

9

Diagnosis (cont)

- Suspect in obese pts, particularly with AHI>60, unexplained awake O2 sats < 94% or overnight nadir < 80%
- Unexplained DOE
- Symptoms and signs of pulm HTN and/or R-sided heart failure
- Facial plethora/polycythemia
- Elevated serum bicarbonate

10

Exclusions of other causes of hypercapnia

- Consider COPD, restrictive lung disease (neuromuscular weakness, ILD, chest wall disease), hypothyroidism, electrolyte disturbances (hypophos, hypomag, rarely hypermag, hypokalemia, hypercalcemia)and chronic sedative/opiate use
- CBC to check for polycythemia suggestive of chronic hypoxemia
- TFTs as low T4 can be associated with decreasing chemo-responsiveness, may cause OSA (due to macroglossia or upper airway dilator muscle dysfunction, or by causing myopathy or neuropathy affecting respiratory muscles)
- PFTs including inspiratory and expiratory pressures (NIF and MEF)
- Imaging, including possible sniff test
- Tox screen, CK or aldolase to look for myositis

11

Treatment

- First line therapy is noninvasive positive airway pressure together with weight loss
- Nocturnal ventilation should not wait for pt attempts to lose weight
- CPAP usually 1st place to start for pts with coexistent OHS and OSA (90% of pts) with BiPAP with back-up rate for those who fail
- OHS with sleep-related hypoventilation generally managed with initial BiPAP
- Weight loss program – improves alveolar ventilation, reduces risk of cardiorespiratory complications (Pulm HTN and LV dysfunction), lowers AHI, improves nocturnal O2 saturation and improves pulmonary function

12

Treatment (cont)

- Bariatric surgery – pts should continue PAP therapy pre- and post-op
- Tracheostomy – may be effective by itself in pts with coexistent OSA, but generally will still need nocturnal ventilation
- Medication – wt loss meds usually not enough, wt loss of 5-10kg over 3-12 months before plateau achieved
- Neutral/harmful treatments – O2 alone, respiratory stimulants (progestins and acetazolamide)
- Supportive therapies – avoidance of alcohol, sedatives, muscle relaxants and treatment of comorbid conditions (COPD, hypothyroidism)

13

Prognosis

- High morbidity and mortality in untreated patients
- Main cause of death is cardiovascular disease
- Even when treated, mortality in those with OHS is worse than those with OSA alone
- Hospitalization rates higher in OHS than in eucapnic obese individuals and more likely to need ICU management, intubation and require long-term care at d/c
- Presence of awake hypoxemia at diagnosis and during PAP therapy associated with poor prognosis

14

Reference

- UpToDate. Clinical manifestations and diagnosis of obesity hypoventilation syndrome: Piper A, Yee B. Aug 2020

15

Pulmonary Complications of COVID-19

16

Case Report

- YB, 55 yo African-American female from Philadelphia (essential worker) developed cough, SOB and pneumonia and tested + for SARS Coronavirus 2
- Required intubation/mechanical ventilation, went on to require trach and PEG and then transferred to LTAC for weaning
- After 2 ½ months on mechanical ventilation, able to liberate from mechanical ventilation
- Mental status excellent, but couldn't tolerate speaking valve over a smaller tracheostomy tube nor tube capping

17

Case Report

- ENT eval revealed no vocal cord issues and bronchoscopy revealed severe tracheal stenosis, explaining inability to tolerate speaking valve/trach capping
- Transferred to UPenn where interventional bronchoscopist dilated proximal trachea with balloon and she was then able to be decannulated

18

COVID-19 STATISTICS

- Severe respiratory symptoms in 14% of cases – dyspnea, hypoxia or > 50% lung involvement on imaging
- Critical illness in 5% of pts – respiratory failure, shock or multiorgan system dysfunction
- Case fatality rate of 2.3%, all among critically ill pts (49% of those)
- Most common complications included ARDS, secondary infections, acute cardiac injury, hypoxic encephalopathy, acute kidney injury, shock and acute liver injury
- Unexpectedly high prevalence of thromboembolic disease and pulmonary hypertension

19

What Can We Expect

- Victims of COVID-19 can have no sequelae, mild sequelae or very severe sequelae
- Cough and SOB extremely common
- CXR's should be checked at a minimum 12 weeks after a pt is diagnosed with pneumonia
- PFT's should be checked in patients with ongoing SOB and w/u for thromboembolic disease should ensue

20

Late COVID-19 Complications

- Severity of initial disease predicts likelihood of severe sequelae
- Pts with milder disease may still develop thromboembolic complications in the recovery period
- Cardiac disease can also develop later and can consist of MI, cardiomyopathy or dysrhythmia
- Asthmatic state not uncommon after COVID-19 as with other viruses (flu, rhinovirus, RSV)

21

Lingering Pulmonary Effects in Hospitalized Patients

- Pulmonary radiographic abnormalities completely resolved in 53% during 3rd week after hospital discharge
- Remaining patients had residual CT abnormalities
- Younger age associated with better outcomes
- Study of 110 discharged patients (19 had severe disease) – half had decreased DLCO on PFTs

22

Lingering Effects in Mild Disease

- Referred to as “long COVID”
- UK study (phone app) found 10% have symptoms beyond 3 weeks, while some persist for months
- US study (telephone interviews) 14-21 days after positive test (outpatients) – 94% still experienced at least 1 symptom and 35% of those stated they had not returned to their usual state of health. Symptoms least likely to have resolved included cough (43%), fatigue (35%) and dyspnea (29%). 90+% of outpatients with influenza recover within 2 weeks

23

Lingering Effects in Mild Disease

- Post-acute COVID defined as symptoms > 3 weeks
- Chronic COVID beyond 12 weeks
- Common pulmonary symptoms are cough, breathlessness, “lung burn”

24

COVID-19 Complications

- ARDS survivors will commonly have problems such as neuromuscular disease from prolonged immobility, neuromuscular blocking agents and steroids as well as fibrotic lung disease with reduced diffusing capacity on PFT
- Pulmonary HTN usually develops while critically ill with necessity to r/o PE, vasodilator meds usually start in ICU and will usually need to be continued post-d/c
- Long-term mechanical ventilation via tracheostomy quite common in critically ill pts and associated with usual risks of pneumonia, line sepsis, GI bleeding, encephalopathy, renal failure
- Airway complications such as tracheal stenosis or laryngeal stenosis/vocal cord dysfunction may not be discovered until later at time of attempt to remove tracheostomy tube

25

References

- Sun P, et al. J. Clinical characteristic of hospitalized patients with SARS-CoV-2 infection. J Med Virol. 2020:92(6):612-617.
- Liu, et al. The pulmonary sequelae in discharged patients with COVID-19: a short-term observational study. Respiratory Research (2020) 21: 125.
- Mo X, Jian W, Su Z, et al. Abnormal pulmonary function in COVID-19 patients at time of hospital discharge. Eur Respir J 2020;55.

26

References

- Greenhalgh T, Knight M, A'Court C, Buxton M, Husain L., Management of post acute COVID-19 in primary care. BMJ 2020;370:m3026
- Tenforde MW, Kim SS, Lindsell C, et al., IVY Network Investigators, CDC COVID-19 Response Team, IVY Network Investigators. Symptom duration and risk factors for delayed return to usual health among outpatients with COVID-19 in a multistate health care systems network – United States, March-June 2020. MMWR Morb Mortal Wkly Rep2020:69:993-8
- UpToDate: Coronavirus disease 2019 (COVID-19): Critical Care and Airway Management Issues. Anesi G. Aug 2020

27