clinic efficiency, and lastly, sending out letters to the patients periodically requesting that they respect their assigned clinic time.

Acknowledgements

The University Internal Medicine Research Foundation (UIMRF) provided Summer Student Research Funding for two of the authors of this paper (JW, PL). The authors would like to extend their gratitude to the cystic fibrosis clinic staff who were indispensable in the research process.

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Diagnose this

A 25 year-old male with chest pain, fatique, and altered sensation

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A 25-year-old male presents to the emergency department with symptoms of generalized weakness, tingling, and numbness on the dorsum of his left hand. On further questioning, he describes a three-day history of extreme fatigue with mild dizziness, and a two-day history of intermittent chest tightness and pressure. The patient's symptoms are not triggered by exertion, as he feels mild dyspnea even at rest, but they are worsened by it. He is a 3rd year university student, admits to a fair degree of school-related stress, and states his symptoms have interfered with his ability to attend classes this week. He has a 3-year history of smoking up to half a pack of cigarettes per day, has moderate social alcohol consumption, and denies any illicit drug use. He is otherwise healthy with no known medical conditions, no previous hospitalizations or surgeries, and is not on any medications.

On examination, the patient is a tall, thin male who appears pale and anxious. His vitals are: heart rate 95 beats per minute with frequent ectopic beats palpable, blood pressure 134/83 mmHg, respirations 24 per minute, and oxygen-saturation 100% on room air. His speech is slow and hesitant, but he is oriented and appropriate, and easily follows commands. His cardiorespiratory exam is normal, and neurological exam is positive for an essential tremor, but negative for any focal or lateralizing findings.

Screening investigations reveal the following:

TEST	RESULT	NORMAL VALUES
RBC	4.8 $x10^{12}$/L	4.4 – 5.7 $x10^{12}$/L
Hgb	149 g/L	140 – 174 g/L
Hct	0.47	0.42 – 0.52
Plt	382 $x10^{9}$/L	130 – 400 $x10^{9}$/L
WBC	8.02 $x10^{9}$/L	4.0 – 10.0 $x10^{9}$/L
Na	141 mmol/L	135 – 145 mmol/L
K	4.5 mmol/L	3.5 – 5.0 mmol/L
Cl	97 mmol/L	98 – 106 mmol/L
Urea	5.5 mmol/L	2.5 – 8.0 mmol/L
Creatinine	77 µmol/L	70 – 120 µmol/L
BGL (random)	5.1 mmol/L	< 6.5 mmol/L
TSH	2.6 µU/L	0.4 – 5.0 µU/L
EKG	102 bpm, sinus tachycardia with PACs, normal axis, normal intervals, no blocks	

Choose the most likely diagnosis after reviewing the case and investigations:

A. Arrhythmia
B. Hypoglycemia
C. Thyroid disorder
D. Hyperventilation
E. Anemia

Discussion

Ventilation is both autonomic and voluntary. Autonomic regulation is coordinated by the respiratory nuclei in the medulla and pons.[1,2] These nuclei receive and integrate input regarding serum pH (chemoreceptors in the brainstem, carotid artery, and aortic arch), cardiovascular demand (arterial baroreceptors), thoracic compliance (stretch fibers in the thoracic muscles), and pain.[1,2] Autonomic ventilation is increased by acidosis, PaO2 < 55 mmHg, PaCO2 ≥ 75 mmHg, hypotension, and pain.[1,2] Autonomic tachypnea (increased rate of ventilation) is triggered by stimulation of the vagus nerve (e.g. by irritation of J fibers during increased tidal ventilation, or C fibers by pulmonary inflammation or edema).[1,2] Voluntary control of breathing is modulated by the hypothalamus and typically increases in response to temperature and emotion. Voluntary control of breathing can supersede the autonomic controls (within limits).[1,2]

Hyperventilation is an increase in both depth and rate of ventilation that exceeds metabolic needs. Hyperventilation syndrome (HVS) is a term used to describe the diverse set of symptoms associated with these respiratory increases and their subsequent alkalosis.[3] Acute HVS can present in a wide variety of pathological disease scenarios (e.g. during a panic attack, asthma attack, chronic obstructive pulmonary disease exacerbation, or in the setting of acute coronary syndrome). Chronic HVS is present in up to 83% of patients with an anxiety disorder (in the absence of a panic attack), and up to 55% of asthmatic patients (in the absence of an asthma attack), and is idiopathic in up to 10% of cases.[4-7] In HVS, patients commonly have PaCO2 < 30 mmHg, causing decreased cerebral perfusion, and possibly hypophosphatemia and peripheral vasoconstriction.[3,8,9] In chronic HVS, there is evidence that peripheral chemoreceptors are hyposensitized to hypocapnia and hypersensitized to hypoxia, but that the breathing pattern is a learned behaviour.[3,10,11] A patient suffering from acute or chronic HVS may present with any of the following: poor concentration and memory, confusion, fatigue, weakness, dizziness, paresthesia, diaphoresis, palpitations, and chest pain.[9,11] Long-term HVS can lead to impaired quality of life, increased rates of chronic fatigue syndrome, right-to-left shunt, and sleep apnea.[7,12,13]

Management

HVS is a diagnosis of exclusion and there is no diagnostic criteria or gold-standard test for it. If there is a low index of suspicion for organic disease, routine investigation might include: oxygen saturation monitoring, point-of-care blood glucose, chest X-ray, or electrocardiogram.[9] In the appropriate clinical setting, the following tests may also be indicated: complete blood count, extended electrolytes, beta-human chorionic gonadotropin, thyroid stimulating hormone, arterial blood gas, D-dimer, or chest computed tomography with pulmonary embolism protocol.

Patients with HVS are often eager and relieved to know that there is nothing serious or life-threatening causing their symptoms. Primary management is to address any underlying psychiatric and/or respiratory conditions. Refractory or recurrent symptoms can often be successfully managed with behaviour modification consisting of breathing exercises with biofeedback.[3,10,11,14]

Summary

HVS is a condition with a highly variable presentation and can be easily confused with more potentially serious metabolic, cardiac, and neurological disorders. The primary role of the physician is to recognize the possibility of this condition when other causes have been ruled out, and to reassure and educate the patient.

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