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Severe Obstructive Sleep Apnea Mimicking Pheochromocytoma or Paraganglioma in a Young Patient With Class III Obesity, Hypertension, and Elevated Metanephrines

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Female, 25-year-old
Final Diagnosis: Obstructive sleep apnea
Symptoms: Lower extremity edema • palpitation • shortness of breath
Clinical Procedure: —
Specialty: Cardiology

Objective: Unusual clinical course

Background: Severe obstructive sleep apnea and acute physiological stress can complicate interpretation of metanephrine testing for pheochromocytoma and paraganglioma (PPGL).

Case Report: A 23-year-old woman with class III obesity, previously diagnosed hypertension, and a maternal history of postpartum cardiomyopathy presented with edema, exertional dyspnea, palpitations, and severe hypertension. Echocardiography showed an ejection fraction of 30%, and chest computed tomography angiography identified subsegmental pulmonary emboli. Urinary metanephrines were reported as elevated during the acute illness, prompting evaluation for PPGL. Abdominal magnetic resonance imaging and reportedly normal repeat outpatient metanephrine testing reduced suspicion for PPGL, although quantitative repeat results and testing conditions were unavailable. Subsequent polysomnography demonstrated severe obstructive sleep apnea, with an apnea-hypopnea index of 46.8 events/hour and an oxygen saturation nadir of 77%. Continuous positive airway pressure (CPAP) was initiated alongside guideline-directed heart-failure treatment. Obstructive sleep apnea was considered a possible contributor within a multifactorial presentation; other endocrine causes of secondary hypertension were not documented as excluded. An ejection fraction of 40% was reported during cardiac evaluation, but its timing relative to CPAP initiation could not be established.

Conclusions: Severe obstructive sleep apnea should be considered in patients with obesity, hypertension, and witnessed apneas when metanephrine testing raises concern for PPGL. The biochemical findings require interpretation in the context of acute illness and appropriate reassessment after clinical stabilization. This case does not establish obstructive sleep apnea as the cause of the cardiac dysfunction or pulmonary emboli.

Keywords: Heart Failure, Systolic • Pulmonary Embolism • Sleep Apnea, Obstructive • Pheochromocytoma

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Introduction

Plasma free or urinary fractionated metanephrines are central to the biochemical evaluation of pheochromocytoma and paraganglioma (PPGL), but positive results require interpretation in the context of the magnitude of elevation and the testing conditions [1]. Severe obstructive sleep apnea (OSA) can be associated with sympathetic activation and elevated metanephrines [2]. We report a young woman with class III obesity, heart failure with reduced ejection fraction (HFrEF), pulmonary emboli, and elevated urinary metanephrines during acute illness who was subsequently diagnosed with severe OSA. The primary learning point is the diagnostic dilemma created by metanephrine elevation in this setting, where OSA and acute physiological stress may complicate evaluation for PPGL.

Case Report

A 23-year-old woman with class III obesity and hypertension diagnosed 1 year earlier presented to the emergency department with 1 week of progressive lower extremity edema, exertional dyspnea, and palpitations. Her history also included easy bruising and recurrent wrist and ankle sprains. She worked with young children and had recently returned from an out-of-state road trip. She denied sick contacts, fever, chills, chest pain, smoking, alcohol, illicit drug use, and oral contraceptive use. She reported significant stress at home. Her husband had observed frequent nocturnal breathing pauses. Her mother had developed postpartum cardiomyopathy after giving birth to the patient, and her brother had pes cavus and hypermobility. Further family history concerning young-onset hypertension, premature cardiovascular disease, other cardiomyopathy, or sudden cardiac death was unavailable.

At presentation, her heart rate was 132 beats/minute, respiratory rate was 24 breaths/minute, and blood pressure was 182/110 mm Hg. Laboratory findings included leukocytosis, acute kidney injury, baseline and 2-hour high-sensitivity troponin levels of 122 ng/L, brain natriuretic peptide of 531 pg/mL, and D-dimer of 1401 ng/mL. Chest radiography showed pulmonary edema. Chest computed tomography (CT) angiography demonstrated cardiomegaly, a small-to-moderate pericardial effusion measuring up to 2 cm, and right lower lobe subsegmental pulmonary emboli (**Figure 1**). The electrocardiogram showed sinus tachycardia with nonspecific T-wave abnormalities.

She was admitted to the intensive care unit with subsegmental pulmonary emboli and a diagnosis of non-ST-elevation myocardial infarction. Echocardiography demonstrated an ejection fraction of 30%, moderate mitral regurgitation, and severe left ventricular hypokinesis. Initial treatment included intravenous heparin, furosemide, and metoprolol succinate. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers were initially withheld because of acute kidney injury.



Figure 1. Computed tomography angiogram of chest revealing subsegmental pulmonary embolism (red square).

During hospitalization, thyroid-stimulating hormone was within normal limits, and a respiratory viral panel was positive for coronavirus. Urinary metanephrine was recorded as 424 mcg, normetanephrine as 866 mcg, total metanephrines as 1290 mcg, and norepinephrine as 171 mcg. These units are reproduced from the available case documentation; a collection duration was not specified for these analytes. Vanillylmandelic acid was recorded as 8.0 mg/24 hours. The metanephrine results were described as elevated during acute heart failure, pulmonary emboli, severe hypertension, and substantial physiological stress. The assay method, laboratory reference limits, and urinary creatinine or another collection-completeness measures were not specified in the available documentation. The degree of elevation relative to the applicable upper reference limits and collection adequacy could not be established. Lupus anticoagulant-related testing showed a partial thromboplastin time of 41 seconds, dilute Russell viper venom time of 64 seconds, and a positive hexagonal phase lupus anticoagulant test.

At discharge, her heart-failure regimen included sacubitril/valsartan, metoprolol succinate, spironolactone, and dapagliflozin, together with furosemide and hydralazine. She also received apixaban for pulmonary emboli and a wearable cardioverter-defibrillator.

Further cardiac evaluation demonstrated dilated cardiomyopathy, with an ejection fraction of 40% on cardiac MRI, and was reported to show no evidence of viral myocarditis. She was referred to a tertiary care center because of her age and

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the degree of cardiac dysfunction. Retroperitoneal ultrasound was reported to show no renal artery stenosis. Abdominal MRI with contrast did not identify a pheochromocytoma. Repeat outpatient metanephrine testing was reported as normal, and repeat rheumatologic and hypercoagulable testing, including lupus anticoagulant testing, was reported as negative. Quantitative repeat metanephrine results, specimen type, assay and sampling conditions, investigation dates, and the interval from resolution of the acute illness were unavailable. These findings reduced clinical suspicion for PPGL, but the reported repeat result could not be independently interpreted without those details.

Whether primary aldosteronism and hypercortisolism or Cushing syndrome were investigated could not be confirmed from the available documentation, and no test results were available. These conditions therefore cannot be regarded as excluded on the basis of the information presented.

Subsequent polysomnography demonstrated severe OSA, with an apnea-hypopnea index of 46.8 events/hour and an oxygen saturation nadir of 77%. CPAP was initiated in addition to guideline-directed heart-failure treatment. OSA was considered a possible contributor alongside class III obesity and severe hypertension. The patient was reported to be doing well and remained under outpatient monitoring. The intervals between initial investigations, abdominal imaging, repeat biochemical testing, polysomnography, and CPAP initiation were unavailable. Follow-up duration, blood pressure measurements, symptom trajectory, CPAP adherence, and timing of the reported 40% ejection fraction relative to CPAP initiation were also unavailable. The increase from the initial echocardiographic ejection fraction of 30% to the reported MRI value of 40% cannot be attributed specifically to CPAP.

Discussion

The central diagnostic issue was elevated urinary metanephrines in a young woman with severe OSA and acute cardiopulmonary illness. OSA-associated intermittent hypoxia and sympathetic activation can produce biochemical findings that overlap with PPGL. In a study by King et al, patients with OSA without PPGL had false-positive urinary metanephrine results more often than plasma results: 17/67 (25.4%) vs 2/67 (3.0%). False-positive urinary results were more common with severe OSA than with mild or moderate OSA: 13/34 (38.2%) vs 4/33 (12.1%) [2]. These findings support considering OSA as a confounder without assuming that an elevated result excludes a tumor.

Acute illness is a separate and important confounder in this case. The Endocrine Society PPGL guideline recognizes physiological stress during extreme illness as a potential cause of false-positive plasma or urinary metanephrine elevations and supports confirmatory testing after such confounders have resolved [1]. Its scientific statement on endocrine hypertension also cautions against interpreting testing obtained during critical illness without accounting for stress-mediated sympathetic activation [3]. The initial measurements here were obtained during acute heart failure, pulmonary emboli, severe hypertension, and acute kidney injury. Reassessment after clinical stabilization was therefore relevant. Reportedly normal outpatient results and negative abdominal imaging reduced suspicion for PPGL, but the absence of reference limits, collection details, actual repeat values, and recovery-to-testing intervals limits independent assessment. The relative contributions of OSA and acute illness to the biochemical elevation cannot be separated from this account.

The secondary-hypertension evaluation also defines the limits of interpretation. Current guidance supports screening for primary aldosteronism using aldosterone and renin, with attention to potassium and interfering medications [4]. Evaluation for Cushing syndrome is guided by clinical suspicion, including features unusual for age such as hypertension, rather than obesity alone [5]. This patient's young-onset hypertension and history of easy bruising support retaining hypercortisolism in the differential without establishing that diagnosis [5]. Hypercortisolism is relevant because it is associated with hypertension and increased venous thromboembolic risk [5,6]. Neither primary aldosteronism nor Cushing syndrome is documented as excluded here. Consequently, the reported evaluation does not establish OSA as the major explanation for the hypertension, cardiomyopathy, or pulmonary emboli.

OSA has been associated with pulmonary emboli in observational research, including the retrospective study by Seckin et al [7], but this association does not establish the cause of pulmonary emboli in this patient. Class III obesity, recent travel, the acute illness, and the initial lupus anticoagulant-related findings also require consideration. A negative repeat lupus anticoagulant result should be interpreted within its testing interval and anticoagulant context; those details are not reported here. The respiratory viral finding likewise does not by itself establish viral myocarditis, and the reported cardiac evaluation did not support that diagnosis.

Cardiac recovery must be interpreted in the context of combined management with CPAP and guideline-directed heart-failure therapy. The patient received sacubitril/valsartan, metoprolol succinate, spironolactone, and dapagliflozin, in addition to diuretic therapy and blood pressure management. Ejection fraction was reported as 30% on the initial echocardiogram and 40% on cardiac MRI. The interval between assessments and the relationship of the latter measurement to CPAP initiation

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are not established, and the measurements were obtained using different imaging modalities. The maternal history of postpartum cardiomyopathy is also relevant to the etiologic assessment; a familial contribution cannot be established or excluded from the limited history. Thus, any improvement cannot be assigned specifically to CPAP or used to demonstrate OSA as the sole or dominant reversible cause of cardiac dysfunction.

The educational value of this case lies in recognizing severe OSA when class III obesity, witnessed apneas, and hypertension coexist with metanephrine findings suggestive of PPGL. The demographic and cardiopulmonary features provide clinical context for that diagnostic dilemma. The case supports careful biochemical reassessment after stabilization and an appropriately broad differential; it does not demonstrate that OSA caused the metanephrine elevation, HFrEF, or pulmonary emboli. Limited documentation of laboratory reference limits, quantitative repeat testing, endocrine investigations, detailed chronology, and follow-up restricts independent biochemical interpretation and stronger etiologic or treatment-related conclusions.

Conclusions

This case supports considering severe OSA in a young patient with class III obesity, hypertension, and witnessed apneas when elevated metanephrines raise concern for PPGL. Acute physiological stress and OSA can complicate biochemical interpretation, making appropriately documented repeat testing after stabilization important. OSA remains a possible contributor within a multifactorial presentation. The biochemical documentation and secondary-hypertension evaluation limit etiologic certainty, and cardiac improvement cannot be attributed specifically to CPAP during concurrent heart-failure treatment.

Institution Where Work Was Done

Inspira Mullica Hill, Mullica Hill, NJ, USA.

Patient Consent

Consent was obtained by the patient for this report.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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