JCEM Case Reports, 2025, **3**, luaf024
https://doi.org/10.1210/jcemcr/luaf024
Advance access publication 10 February 2025
Case Report

Acute Myocardial Infarction With Nonobstructive Coronary Arteries Across Endocrine Disorders: A Case Series

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Abstract

The endocrine and cardiology interface is particularly interesting and important. This is a case series of 3 endocrine diseases presenting with acute myocardial infarction without obstructive coronary arterial diseases (MINOCA). The diagnosis was Cushing's disease due to pituitary microadenoma, adrenal pheochromocytoma, and primary hyperaldosteronism due to unilateral adrenal hyperplasia. All patients were managed conservatively and improved considerably following the management of underlying endocrine and cardiovascular diseases. A spectrum of classical endocrine diseases can present with MINOCA, and the underlying diagnosis is often overlooked. Patients without traditional risk factors for MINOCA should undergo hormonal workup to reveal the underlying diagnosis.

Key Words: myocardial infarction, Cushing's syndrome, primary hyperaldosteronism, pheochromocytoma, MINOCA

Introduction

Several endocrine diseases can present with cardiovascular emergencies. However, the relationship of endocrine diseases with myocardial obstruction with no coronary artery obstruction (MINOCA) is rarely described. MINOCA is characterized by myocardial ischemia/infarction with normal coronary arteries in coronary angiography [1]. The European Society of Cardiology (ESC) diagnostic criteria [2] of MINOCA requires fulfillment of the following criteria: (1) the presence of universal acute myocardial infarction criteria; (2) nonobstructive coronary arteries on angiography, defined as no coronary artery stenosis ≥50% in any potential infarct-related arteries; and (3) no clinically overt specific cause for the acute presentation like myocarditis or embolism. MINOCA has a prevalence of 5% to 8% of patients undergoing cardiac catheterization for myocardial infarction (MI) [3-5]. MINOCA patients are at risk of developing long-term complications, including recurrent MI and stroke [6]. Nevertheless, as the ESC document mentions, MINOCA is a working diagnosis, requiring a search for the underlying cause.

In clinical endocrinology, pheochromocytoma can present with various cardiovascular manifestations, including acute coronary syndrome, cardiomyopathy, or arrhythmia [7]. MINOCA has been reported in pheochromocytoma [8], Graves' disease [9], hypothyroidism following treatment with levothyroxine [10], and bilateral pheochromocytoma in the background of multiple endocrine neoplasia type 2 [11]. However, Cushing's syndrome [12] (CS) and primary hyperaldosteronism [13] are 2 endocrinological entities that are rarely reported.

Case Presentation

Case 1

A 30-year-old mother of 2 children was found to have elevated blood pressure 5 years ago during her last childbirth. She had oligomenorrhoea and weight gain along with hypertension, requiring antihypertensives even after 1 year of the pregnancy. She received a 6-month course of antitubercular therapy after 2 years of the pregnancy, as she developed moderate ascites with septations, pleural effusion, and pedal edema. Weight gain persisted, and she experienced progressive skin darkening and face rounding. She presented with an episode of acute severe chest pain and attended the emergency room (ER) in July 2019. She had an acute ST elevation in the electrocardiogram (ECG) with raised serum troponin (see Table 1). She had regional wall motion abnormality (RWMA) in echocardiography at the anterior wall and ventricular septum and ejection fraction (LVEF) of 35%. She underwent computed tomography (CT) -coronary angiogram and conventional arterial angiography of the coronary arteries immediately after the episode. Notably, both were normal, showing no stenosis in the coronaries. Diabetes mellitus was detected this time.

Case 2

A 56-year-old male presented with acute chest pain, breathlessness, and palpitation in the ER. He had a previous history of hypertension and type 2 diabetes. His ECG revealed sinus tachycardia and ST segment depression in V4-V6 and inferior lead (II, III, aVF). Echocardiography revealed mild global

Received: 6 October 2024. **Editorial Decision:** 23 January 2025. **Corrected and Typeset:** 10 February 2025

Table 1. Summary of the principal cardiological findings among the patients

Attributes	Case 1	Case 2	Case 3
Age (years)	30	56	26
Sex	Female	Male	Male
Clinical presentation	Acute chest pain, respiratory distress	Chest pain, respiratory distress, palpitations	Acute chest pain and shock
ECG	ST-elevation in anterior lead V1-V6	Sinus tachycardia, ST-depression from lead V4-V6 and inferior leads, NSTEMI	Extensive ST-elevation in the antero-lateral leads V1-V6 and I-III
LVF at presentation	Yes	Yes	Yes
Troponin I (0-0.04 μg/L)	Elevated[a]	2.0 μg/L	Elevated[a]
CK-MB (0-24 IU/L)	NA	70.3 IU/L	30 IU/L
CAG	Normal[b]	Normal	Proximal left anterior descending artery block 30-50%. Other vessels were normal
2D-ECHO at presentation	Hypokinesia at the anterior wall and ventricular septum LVEF = 35%	Global hypokinesia with akinetic inferior wall LVEF = 40%	Global hypokinesia, mild TR right ventricular systolic pressure 41 mm Hg LVEF = 20%
Thrombolysis	Yes	Not done	Not done
Time to reach endocrinological diagnosis (months)	7	1	During workup for hypertension/ hypokalemia
Final diagnosis	Cushing's disease due to pituitary microadenoma	Right-sided adrenal pheochromocytoma	Primary hyperaldosteronism due to unilateral (left) adrenal hyperplasia

Abbreviations: CAG, coronary angiography; ECG, electrocardiogram; ECHO, echocardiography; LVEF, left ventricular ejection fraction; LVF, left ventricular failure; NA, not available; NSTEMI, non-ST segment elevation myocardial infarction; TR, tricuspid regurgitation.

[a]As measured qualitatively by rapid kits.

[b]Including both coronary angiography and computed tomography-angiography.

hypokinesia and RWMA in the inferior myocardial wall (akinetic) with an LVEF of 40%. His troponin I and CK-MB were elevated (see Table 1). An initial diagnosis of non-ST segment elevation MI was made in the ER. He also had normal coronaries in the coronary angiogram.

Case 3

A 26-year-old male with severe hypertension since 24 years of age was suspected of having primary hyperaldosteronism due to ongoing hypokalemia. His initial blood pressure was 180/130 mm Hg. He developed sudden chest pain in the hospital ward during evaluation of hypertension. ECG showed extensive anterior wall ST elevation (V1-V5 chest leads), and echocardiography showed severe left ventricle (LV) global hypokinesia with LVEF of 20%. He was immediately taken for coronary angiography, showing a mild proximal left anterior descending artery block of 30% to 50%. In contrast, other vessels (including the right coronary artery, left circumflex coronary artery, and left main coronary artery) were normal. A follow-up Tc-99 m methoxy isobutyl isonitrile myocardial perfusion imaging scan pre- and postadenosine infusion showed a dilated LV cavity and global hypokinesia without any evidence of stress-induced ischemia or scar in any of the LV myocardial segments. There was septal hypertrophy.

Diagnostic Assessment

Case 1

On further evaluation, the patient was found to have skin hyperpigmentation, proximal muscle weakness, centralized obesity, skin thinning, and facial plethora without other features like cushingoid striae or bruises. Her biochemical evaluation revealed ACTH-dependent CS [basal cortisol 38.9 μg/dL (1075 nmol/L) (normal range 5-25 μg/dL; 138-690 nmol/L), midnight cortisol 19.2 μg/dL (530 nmol/L) (normal <7.5 μg/dL (awake); < 207 nmol/L), overnight 1-mg dexamethasone suppression test: 26 μg/dL (717 nmol/L) (normal < 1.8 μg/dL; < 50 nmol/L), ACTH 197 pg/mL (43.4 pmol/L) (normal range 5-46 pg/mL; 1.1-10 pmol/L)]. She had a pituitary microadenoma (5 × 5 mm) on the right side in dynamic contrast magnetic resonance imaging (MRI).

Case 2

Further hormonal evaluation done for persistent hypertension revealed elevated plasma-free metanephrine [252 pg/mL (1277 pmol/L) (normal <65 pg/mL; < 330 pmol/L; ELISA)] and nor-metanephrine [218 pg/mL (1190 pmol/L) (normal <196 pg/mL; < 1070 pmol/L; ELISA)]. His plasma renin activity was 26.4 ng/mL per hour (26.4 μg/L/hr) (normal range 0.7-3.3 ng/mL/hr; 0.7-3.3 μg/L/hr) and plasma aldosterone level was 7.18 ng/dL (199 pmol/L) (normal range 7-30 ng/dL; 190 to 830 pmol/L). Thus, a presumptive diagnosis of pheochromocytoma/paraganglioma (PPGL) was made. A CT scan of the abdomen showed a well-defined heterogenous hypoattenuating lesion with postcontrast heterogenous enhancement in the right suprarenal region, suggestive of pheochromocytoma.

Case 3

Hormonal evaluation showed plasma renin activity of 0.57 ng/mL/hr (0.57 μg/L/hr) (normal range 0.7-3.3 ng/mL/hr; 0.7-3.3 μg/L/hr) and plasma aldosterone of 12.8 ng/dL (355 nmol/L) (normal range 7-30 ng/dL; 190 to 830 pmol/L),

and the aldosterone renin ratio was 22 (normal < 20), suggestive of primary hyperaldosteronism. Due to persistent severe hypertension requiring 4 different antihypertensives, a confirmatory saline loading test was avoided. His CT abdomen revealed a bulky left adrenal gland (8 mm), suggestive of unilateral hyperplasia.

Treatment

Case 1

At the initial presentation of MINOCA, the patient was thrombolyzed in the ER with a diagnosis of acute ST-segment elevation MI, and the ECG changes were normalized after 1 hour. She underwent transsphenoidal removal of the microadenoma after stabilization. Histopathology was consistent with adenoma, which stained diffusely positive for ACTH.

Case 2

At the initial presentation, the patient was managed medically with low molecular weight heparin, dual antiplatelet (aspirin and clopidogrel), statins (atorvastatin 80 mg), β-blockers, and antihypertensives. Later, once pheochromocytoma was diagnosed, he underwent right adrenalectomy after adequate α and β blockade along with reasonable control of hypertension. His postoperative histopathology showed encapsulated neoplasm with polygonal cells with abundant eosinophilic cytoplasm arranged in a nested pattern, and the periphery was surrounded by the sustentacular cells. Immunohistochemistry was positive for synaptophysin and chromogranin, which confirmed a pheochromocytoma.

Case 3

The patient was stabilized with medical management, including dual antiplatelet therapy (aspirin and clopidogrel) and antihypertensives, including spironolactone, carvedilol, and prazosin. After stabilization, he underwent a left adrenalectomy. Postoperative biopsy showed prominent hyperplasia of zona glomerulosa constituting about 25% to 30% of the adrenal gland with no evidence of atrophy of zona fasciculata and zone reticularis, suggesting adrenal hyperplasia.

Outcome and Follow-up

Case 1

Postoperatively, the patient was in remission and is currently on hydrocortisone replacement without other pituitary hormonal involvement. She developed an occipital infarct on postoperative day 4, which recovered partially with conservative management with low molecular weight heparin. Magnetic resonance angiography of the brain and neck revealed no abnormality during this postoperative period. Currently, she is in remission from Cushing's disease, and no further episodes of a cardiovascular event were noted at the 2-year follow-up.

Case 2

A repeat echocardiography after 1 week of the tumor removal showed marked improvement in the LV function with an ejection fraction of 68%. The patient's perioperative period was uneventful, and after 1 year of follow-up, he is doing well without the requirement of any medications.

Case 3

In this case, the perioperative period was also uneventful, and ejection fraction improved. Moreover, currently, the patient requires only a single antihypertensive. His latest echocardiography was normal, and he was clinically stable at 6 months of follow-up.

Discussion

Here we describe 3 cases of MINOCA presented to our hospital within a span of 2 years. The workup revealed 3 different endocrine disorders: ACTH-dependent Cushing's disease due to pituitary microadenoma, adrenal pheochromocytoma, and primary hyperaldosteronism due to unilateral adrenal hyperplasia. Thus, it signifies the importance of appropriate endocrine workup to identify the underlying diagnosis in a case of MINOCA. However, this observation does not suggest causality without robust, large-scale data.

MINOCA may be differentiated into 2 distinct patterns based on the findings on the left ventricular angiography, namely, the "epicardial pattern" and the "microvascular pattern." The epicardial causes are coronary plaque rupture, coronary vasospasm, and coronary dissection. Other microvascular causes implicated in MINOCA are coronary microvascular vasospasm, myocarditis, Takotsubo cardiomyopathy (TS), and coronary thromboembolism [1]. All these underlying etiologies of MINOCA are relevant to endocrinological conditions, particularly pheochromocytoma and CS.

PPGL can have ECG changes resembling ST segment elevated MI and result from the catecholamine surge. PPGL can present with either acute coronary syndrome (ACS), myocarditis, dilated cardiomyopathy, or the rare TS [7]. Though ACS is an established complication of PPGL, MINOCA has been described rarely. Melson et al had recently reported a 79-year-old woman who presented with acute chest pain and was diagnosed as having MINOCA based on ECG findings and elevated cardiac enzymes and normal coronaries in the coronary angiogram [8]. Later on, she was found to have right-sided adrenal pheochromocytoma. Thus, this case and ours point toward the importance of working up patients of MINOCA for the underlying cause. However, in our case, the persistence of hypertension triggered the workup for pheochromocytoma. It is important to consider the differential diagnosis of myocarditis and TS while diagnosing MINOCA in any patient with pheochromocytoma [7]. Pheochromocytoma patients can have catecholamine-induced myocarditis, often detected by cardiac MRI in the form of focal fibrosis or areas of myocarditis [14]. These MRI changes can be confused with TS sometimes. Unfortunately, in case 2, cardiac MRI was not available to verify the presence of myocarditis. Giavarini et al showed a prevalence of 11% of catecholamine-induced cardiomyopathy (including 6 cases of TS) in their series of 140 patients. They found that this type of cardiomyopathy is frequently reversible after surgery [15]. TS can mimic ACS and is characterized by typical left ventricular ballooning due to localized RWMA [16]. TS has multiple triggers, and catecholamine excess is 1 of them.

TS can be reversible within hours to weeks [16]. TS associated with pheochromocytoma can involve global LV dysfunction and have a high complication rate [17]. TS patients usually have normal coronaries, and cardiac imaging with either echocardiography or cardiac MRI is invaluable in diagnosing the apical ballooning of the LV [7]. In our case, no

echocardiographic features of TS were found. The prevalence of TS in PPGL varies, as 1 study found 4 out of 152 cases of secreting PPGL with TS [18]. Thus, pheochromocytoma diagnosis must be considered in an appropriate clinical context, like persistent severe hypertension, episodic symptoms of dizziness, and palpitations in MINOCA. However, the guideline does not consider routine workup for pheochromocytoma [19].

MINOCA presentation in a CS patient is infrequent or may be underdiagnosed. One case has been reported with MINOCA [12]. In our patient, features like central obesity, knuckle hyperpigmentation, and hypertension were evident, but the diagnosis was delayed. CS is a known hypercoagulable state and confers a significant risk of thrombotic episodes [20]. The reported coagulation abnormalities include elevation of coagulation factors VIII, IX, and XI, the von Willebrand factor [20, 21]. There is an increase in plasminogen activator inhibitor-1, suggesting impaired fibrinolysis [22]. Traditional risk factors like diabetes, insulin resistance, hypertension, and hypokalemia increase cardiovascular risk [23]. A recent study [24] of 208 patients with CS showed a risk of 20% for the development of a thrombotic event. Notably, among the thrombotic episodes, 21% had MI, but no MINOCA was reported. Coronary microvascular reserve and performance are impaired in CS [25]. Impaired coronary circulation in CS was found in the absence of overt ischemic cardiac disease without any changes in the epicardial coronary arteries [25]. Abnormalities in the coronary microvasculature and a hypercoagulable state played a perfect foil for developing MINOCA.

Primary hyperaldosteronism is a risk factor for the development of cardiovascular comorbidities, including MI, systolic dysfunction, and atrial fibrillation, among others [26]. A case of coronary embolism without significant stenosis and few TS had been depicted with primary hyperaldosteronism [13, 27, 28]. Chronic aldosterone excess can lead to vasoconstriction of both medium-sized coronaries and coronary microvasculature through its action on the vascular smooth muscle through suppression of sarcoplasmic reticulum calcium adenosine triphosphatase [29]. LV remodeling due to increased aldosterone can further elevate the risk [27].

We describe 3 endocrinologic diseases associated with MINOCA, including classical CS, pheochromocytoma, and primary hyperaldosteronism due to unilateral adrenal hyperplasia. Appropriate hormonal workup and prompt management can lead to improved diagnosis and treatment.

Learning Points

- Prioritize attention for patients with ACS who present without traditional risk factors for possible endocrine causes.
- Evaluate endocrine causes in cases of MINOCA to uncover potential underlying conditions.
- Cushing's disease, primary hyperaldosteronism, and pheochromocytoma may be associated with MI without evident coronary involvement in angiography.

Acknowledgments

We thank the Department of Cardiology of JIPMER for their prompt help in managing the cases. We also thank Dr. Somdatta Giri for his contribution in preparing the manuscript.

Contributors

All authors made individual contributions to authorship. J.P.S. conceived the idea and A.R. wrote the first draft. A.R., D.P., and C.M. were involved in the diagnosis and management of the patient and data acquisition. J.P.S., S.K.K., and D.B.N. were responsible for reviewing and editing the manuscript. All authors reviewed and approved the final draft.

Funding

No public or commercial funding.

Disclosures

None declared.

Informed Patient Consent for Publication

Signed informed consent was obtained directly from all 3 patients.

Data Availability Statement

Original data generated and analyzed during this study are included in this published article.

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