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WHEN NSTEMI IS NOT CORONARY DISEASE: MINOCA REVEALING PHEOCHROMOCYTOMA

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INTRODUCTION

Pheochromocytoma is a catecholamine-secreting tumor with diverse cardiovascular manifestations, including myocardial infarction with non-obstructive coronary arteries (MINOCA). We report a case of biochemically confirmed pheochromocytoma initially presenting as non-ST-elevation myocardial infarction (NSTEMI), later reclassified as MINOCA.

CASE

A 62-year-old female with type 2 diabetes mellitus and hypertension was admitted with presumed NSTEMI and commenced on dual antiplatelet therapy. Further history revealed recurrent presyncope associated with paroxysmal headache, palpitations, and profuse diaphoresis. During admission, her blood pressure was markedly labile, ranging from 75/45 to 220/122 mmHg, raising suspicion of pheochromocytoma. Biochemical evaluation demonstrated markedly elevated 24-hour urinary normetanephrine of 36.19 µmol/day (reference 0–2.13) and methoxytyramine of 3.90 µmol/day (reference 0.10–1.79), consistent with catecholamine excess. Dedicated adrenal computed tomography identified a 3.8-cm right adrenal lesion with high attenuation (44 Hounsfield Unit [HU]), arterial enhancement (119 HU), and low washout (absolute 40%, relative 24%), without calcification or necrosis. Electrocardiography showed sinus rhythm with T-wave inversion in leads I, aVL, and V5–V6. Transthoracic echocardiography demonstrated a preserved left ventricular ejection fraction of 67% without regional wall-motion abnormalities. Coronary angiography subsequently showed normal coronary arteries, supporting a diagnosis of MINOCA likely secondary to pheochromocytoma-related catecholamine excess and hypertensive crisis. Antiplatelets were discontinued. She was commenced on α-blockade, with additional felodipine and low-dose β-blocker for blood pressure optimization, and subsequently underwent successful open right adrenalectomy. Postoperatively, she required transient inotropic support but was weaned within 36 hours.

CONCLUSION

Pheochromocytoma-associated MINOCA is uncommon but important to recognize. Catecholamine surges may cause myocardial injury through coronary vasospasm, myocardial oxygen supply-demand mismatch, and direct catecholamine-mediated cardiotoxicity. Recognition is crucial, as management differs fundamentally from atherosclerotic acute coronary syndrome and requires α-blockade before β-blockade.

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THE DOPAMINE DILEMMA: EXOGENOUS L-DOPA OR RARE DOPAMINOMA?

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INTRODUCTION

Dopaminomas or dopamine-secreting pheochromocytomas are rare neuroendocrine tumors that frequently lack the classic paroxysmal symptoms of catecholamine excess (headache, sweating, palpitation). Diagnosing these tumors is exceptionally challenging when biochemical markers—specifically markedly elevated urinary dopamine—are interpreted in patients receiving exogenous Levodopa (L-DOPA) therapy, as the intake obscures clinical significance.

CASE

A 76-year-old male with ischemic heart disease, stage 4 chronic kidney disease, and new-onset hypertension was admitted for acute cholecystitis. Imaging studies incidentally revealed bilateral adrenal masses and demonstrated lipid-rich characteristics upon further evaluation with computed tomography (CT) adrenal washout. Biochemical evaluation revealed extreme elevations in 24-hour urinary dopamine (47,534 nmol/24 hours; normal <3,237) and 3-methoxytyramine (18.93 µmol/24 hours; normal <2.60), whereas downstream urinary noradrenaline (5.0 nmol/24 hours; normal 71.5–505.3), adrenaline (4.0 nmol/24 hours; normal 9.2–122.3), normetanephrine (0.22 µmol/24 hours; normal 0.88–2.88), and metanephrine (0.25 µmol/24 hours; normal 0.33–1.53) levels were all significantly below their respective reference ranges. The diagnosis was complicated by the patient's concurrent use of L-DOPA/Benserazide for flupentixol-induced parkinsonism. L-DOPA is a direct precursor to dopamine; its administration can cause massive, false-positive elevations in urinary dopamine, mimicking a dopaminoma's biochemical signature. However, the unique combination of profoundly high dopamine alongside suppressed downstream catecholamines and metanephrines suggested a true dopamine-secreting tumor—potentially secondary to dopamine beta-hydroxylase (DBH) deficiency—rather than drug interference.