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CASE REPORT

Resting Dyspnea, Regional Wall Motion Abnormality, and Normal Coronary Angiography in a Hypertensive Patient: A Diagnostic Dilemma

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Abstract

Background: Progressive dyspnea in a hypertensive patient with echocardiographic regional wall motion abnormality frequently raises suspicion of obstructive coronary artery disease and may lead to invasive coronary evaluation. However, regional wall motion abnormality is not always synonymous with stentable epicardial coronary disease, particularly in patients with long-standing hypertension, chronic alcohol exposure, and renal dysfunction.

Case Presentation: A 55-year-old male graphic designer with long-standing systemic hypertension, chronic alcohol consumption, and baseline renal dysfunction with serum creatinine around 2 mg/dL presented with gradually progressive breathlessness over several years, recently worsening to dyspnea even at rest. A non-invasive coronary CT performed approximately six months earlier was reportedly normal. During the current evaluation, laboratory investigations showed serum creatinine of 1.92 mg/dL and hyperuricemia. Two-dimensional echocardiography revealed regional wall motion abnormality with mild left ventricular systolic dysfunction, left ventricular ejection fraction around 45%, and mild mitral regurgitation. In view of worsening symptoms and suspected ischemic myocardial involvement, coronary angiography with possible stenting was planned. Considering his renal dysfunction, contrast volume was minimized. Surprisingly, invasive coronary angiography showed normal epicardial coronary arteries, and no stent was placed. The patient was subsequently managed conservatively with optimized antihypertensive therapy, heart-failure-directed medical treatment as tolerated, renal monitoring, lifestyle modification, and alcohol cessation counseling, following which he showed symptomatic improvement.

Conclusion: This case highlights the diagnostic discordance between dyspnea, echocardiographic RWMA, and normal coronary anatomy. In hypertensive patients with chronic alcohol use and renal dysfunction, normal coronary angiography should prompt consideration of hypertensive heart disease, alcohol-related myocardial dysfunction, coronary microvascular dysfunction, and ischemia with non-obstructive coronary arteries rather than premature dismissal of symptoms.

Keywords: Hypertension; dyspnea; regional wall motion abnormality; normal coronary angiography; INOCA; alcoholic cardiomyopathy; chronic kidney disease.

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INTRODUCTION

Dyspnea in a middle-aged hypertensive patient is a common but clinically important presentation, as it may reflect progressive hypertensive heart disease, left ventricular dysfunction, occult ischemia, or non-ischemic cardiomyopathy. Long-standing systemic hypertension produces structural and functional myocardial changes, including left ventricular hypertrophy, interstitial fibrosis, impaired relaxation, increased myocardial oxygen demand, and eventual systolic dysfunction. These changes may produce heart failure symptoms even in the absence of obstructive epicardial coronary artery disease.[1] More recently, coronary microvascular dysfunction has also been recognized as an important link between hypertension and myocardial ischemia, as hypertensive vascular remodeling can impair coronary flow reserve and produce ischemia despite angiographically normal coronary arteries.[2]

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Regional wall motion abnormality on two-dimensional echocardiography often raises suspicion of obstructive coronary artery disease and frequently prompts invasive coronary evaluation, particularly when symptoms progress or left ventricular systolic function is reduced. However, the presence of ischemic symptoms or imaging abnormalities with normal or non-obstructive coronary arteries is increasingly recognized as a distinct clinical entity. The EAPCI consensus document on ischemia with non-obstructive coronary arteries notes that a substantial proportion of patients undergoing invasive angiography for angina or objective evidence of ischemia do not have obstructive coronary artery disease, and many may have underlying coronary microvascular dysfunction or vasospastic mechanisms.[3] Similarly, patients with chest pain and non-obstructive coronary artery disease have been shown to have a high prevalence of coronary microvascular abnormalities, supporting the concept that a normal angiogram does not necessarily exclude clinically significant myocardial ischemia.[4]

Chronic alcohol consumption adds another layer of diagnostic complexity in such patients. Alcohol-induced cardiomyopathy is an acquired form of dilated cardiomyopathy caused by prolonged heavy alcohol intake and may present with exertional dyspnea, reduced functional capacity, and left ventricular systolic dysfunction. The condition is clinically relevant because it can mimic ischemic cardiomyopathy, particularly when regional or global ventricular dysfunction is detected on echocardiography, and its recognition is important because abstinence and optimized medical therapy may improve outcomes in selected patients.[5]

We present the case of a 55-year-old hypertensive male graphic designer with chronic alcohol use and baseline renal dysfunction who developed progressive breathlessness worsening to dyspnea at rest. Despite a reportedly normal non-invasive coronary CT performed six months earlier, repeat echocardiography demonstrated regional wall motion abnormality and mild left ventricular systolic dysfunction, prompting coronary angiography with a plan for possible stenting. Surprisingly, invasive coronary angiography revealed normal epicardial coronaries, and the patient improved with conservative medical therapy. This case highlights the diagnostic discordance between symptoms, echocardiographic abnormality, and coronary anatomy, and emphasizes the need to consider hypertensive heart disease, alcohol-related myocardial dysfunction, and ischemia with non-obstructive coronary arteries in patients with apparent ischemic cardiomyopathy but normal coronary angiography.

Case Presentation

A 55-year-old male, working as a graphic designer, with a history of long-standing systemic hypertension, chronic alcohol consumption, and baseline renal dysfunction with serum creatinine around 2 mg/dL, presented with progressive breathlessness. He had been experiencing exertional dyspnea for several years, which had gradually worsened over time. Initially, the breathlessness was present only during routine activities, but in the recent period it progressed to dyspnea on minimal exertion and eventually breathlessness even at rest. There was no clear history suggestive of an acute febrile illness or infective respiratory trigger. In view of his cardiovascular risk profile and chronic symptoms, he had undergone a non-invasive coronary CT evaluation approximately six months earlier, which was reportedly normal and did not suggest significant obstructive coronary artery disease.

During the current evaluation, the patient's worsening rest dyspnea raised concern for cardiac decompensation or an ischemic equivalent. He was evaluated with laboratory investigations and echocardiography. His blood reports showed hemoglobin of 14.7 g/dL, total leukocyte count of 14,200/mm^3 with neutrophilic predominance, platelet count of 3.01 lakh/mm^3, random blood glucose of 128 mg/dL, serum sodium of 141.1 mEq/L, serum potassium of 4.65 mEq/L, serum urea of 46 mg/dL, serum creatinine of 1.92 mg/dL, and serum uric acid of 9.2 mg/dL. Liver enzymes were within normal limits, and HIV, HBsAg, and anti-HCV tests were non-reactive. The renal profile was clinically important because any decision for invasive coronary angiography required careful consideration of contrast exposure and the risk of worsening renal function.

Two-dimensional echocardiography revealed mild left ventricular systolic dysfunction with an ejection fraction of approximately 45%. The left ventricular end-diastolic dimension was around 56 mm and end-systolic dimension around 46 mm, with mild increase in wall thickness. Regional wall motion abnormality was documented, along with mild mitral regurgitation. The final echocardiographic impression was coronary artery disease with RWMA, mild LV dysfunction, LVEF around 45%, and mild MR. These findings created a diagnostic dilemma because, despite a previously normal non-invasive coronary CT, the patient now had worsening rest dyspnea and echocardiographic evidence suggestive of possible ischemic myocardial involvement.

In view of the progressive symptoms, high cardiovascular risk profile, and echocardiographic regional wall motion abnormality, a decision was taken to proceed with invasive coronary angiography, with the possibility of percutaneous coronary intervention and stenting if a significant culprit coronary lesion was identified. Given the patient's baseline renal dysfunction, contrast use was kept limited. Coronary angiography was performed through radial access, with approximately 30 mL of contrast. The aortic pressure during the procedure was documented as approximately 120/80 mmHg. Surprisingly, the angiogram revealed normal epicardial coronary arteries. The left main coronary artery was normal. The left anterior descending artery was normal and gave rise to major diagonal branches without significant stenosis. The left circumflex artery and obtuse marginal branches were normal. The right coronary artery was also normal. The final angiographic impression was normal coronary angiography, and therefore no stent was placed.

Following the angiographic finding of normal coronaries, the clinical diagnosis was reconsidered. The working impression shifted from obstructive coronary artery disease to a non-obstructive myocardial process causing dyspnea and mild LV dysfunction. In this context, possible contributors included hypertensive heart disease, alcohol-related myocardial dysfunction, coronary microvascular dysfunction, demand ischemia, or ischemia with non-obstructive coronary arteries. The patient was therefore managed conservatively with optimized medical therapy rather than revascularization. Treatment focused on strict blood pressure control, heart-failure-directed therapy as tolerated, cautious use of diuretics based on volume status, renal function monitoring, avoidance of nephrotoxic agents, lifestyle modification, and counseling for alcohol cessation. After initiation

and optimization of conservative medical therapy, the patient showed symptomatic improvement, with reduction in breathlessness and better functional tolerance.

This case represents a clinically important example of discordance between symptoms, echocardiographic findings, and coronary anatomy. The patient had progressive breathlessness at rest and regional wall motion abnormality, both of which strongly suggested ischemic heart disease and led to planned angiography with possible stenting. However, invasive coronary angiography demonstrated normal epicardial coronaries, preventing unnecessary stent placement and redirecting management toward conservative optimization. The case highlights that regional wall motion abnormality should not always be equated with obstructive coronary artery disease. In hypertensive patients with chronic alcohol exposure and renal dysfunction, non-ischemic cardiomyopathy, hypertensive myocardial remodeling, and coronary microvascular dysfunction should be considered when coronary angiography is normal.

DISCUSSION

This case highlights an important diagnostic dilemma in cardiovascular medicine: progressive breathlessness with echocardiographic regional wall motion abnormality does not always indicate obstructive epicardial coronary artery disease. The patient had several features that reasonably raised suspicion for ischemic heart disease, including long-standing hypertension, worsening dyspnea at rest, mild left ventricular systolic dysfunction, and regional wall motion abnormality. In routine practice, such a combination often pushes the clinician toward coronary angiography with possible revascularization. However, the finding of normal epicardial coronaries on invasive angiography shifted the diagnostic frame from "stentable coronary artery disease" to a broader myocardial and microvascular pathology.

The initial decision to proceed with coronary angiography was clinically defensible. Dyspnea at rest may function as an anginal equivalent, particularly in a hypertensive patient with left ventricular dysfunction. Moreover, emergency and cardiology literature emphasizes that suspected myocardial ischemia requires rapid and structured evaluation but also acknowledges that ECG and initial testing may have limitations and must be interpreted along with history, biomarkers, echocardiography, and risk profile. In an emergency medicine study on patients presenting with myocardial infarction, ECG was described as a fundamental tool, but the authors also noted that clinicians must use additional clinical information and diagnostic tests to improve specificity because false-positive or incomplete interpretations may lead to unnecessary interventions.[6-8] This principle applies well to the present case, where the echocardiographic finding of RWMA created a strong ischemic suspicion, but definitive coronary anatomy clarified that there was no obstructive lesion requiring stenting.

The normal coronary angiogram is the central teaching point of this case. A normal angiogram should not be interpreted as absence of disease; rather, it should redirect clinical reasoning toward ischemia with non-obstructive coronary arteries, coronary microvascular dysfunction, vasospasm, hypertensive myocardial remodeling, myocarditis, stress cardiomyopathy, alcohol-related cardiomyopathy, or other non-ischemic causes of left ventricular dysfunction. The INOCA concept is now well recognized, and expert consensus documents describe that many symptomatic patients undergoing angiography for angina or objective ischemia do not have obstructive coronary artery disease; in such patients, coronary microvascular dysfunction and vasospastic disease are important mechanisms.[9]

Long-standing hypertension provides a strong pathophysiological explanation for the patient's symptoms.[10-14] Chronic hypertension increases afterload, causes left ventricular hypertrophy and myocardial fibrosis, impairs diastolic relaxation, and eventually may lead to systolic dysfunction. Hypertensive heart disease may also produce ischemia despite normal epicardial coronaries because coronary microvascular remodeling reduces coronary flow reserve.[15] Camici *et al.* described coronary microvascular dysfunction as an important mechanism in left ventricular hypertrophy and heart failure, while Taqueti *et al.* linked coronary microvascular ischemia, cardiomyocyte injury, and myocardial stiffness to future heart failure risk even in the absence of obstructive coronary artery disease.[16,17] In the present patient, mild LV dysfunction, breathlessness, and normal coronaries therefore fit well with a hypertensive myocardial disease phenotype rather than fixed epicardial coronary obstruction.

This study on hypertensive crisis further supports the relevance of cardiac and renal target-organ involvement in hypertensive patients. Hypertensive emergencies were described as severe blood pressure elevation with target-organ damage, and cardiac manifestations included acute left ventricular failure, unstable angina, myocardial infarction, and ECG changes such as LVH, strain pattern, and myocardial ischemia. Dyspnea among presenting complaints and documented renal dysfunction in a subset of patients, reinforcing that hypertensive disease frequently affects the heart and kidneys together. This is particularly relevant here because the patient had baseline renal dysfunction with creatinine around 2 mg/dL and current serum creatinine around 1.92 mg/dL, indicating that the myocardium and kidneys were both vulnerable organs.

Alcohol use is another important contributor in this case. In addition to alcohol, chronic occupational stress and prolonged screen-based work may have contributed indirectly to this patient's cardiometabolic deterioration. As a graphic designer, the patient was likely exposed to extended computer use, sedentary behavior, irregular sleep, and psychological stress, all of which can worsen sympathetic activation, blood pressure variability, endothelial dysfunction, and long-term cardiovascular risk. Internet overuse and pathological internet use have increasingly been recognized as emerging behavioral health concerns, with one Indian study highlighting internet addiction as a prevalent public health issue requiring further attention.[18,19] Regular yoga, breathing exercises, structured sleep, and stress-reduction practices may have attenuated autonomic overactivity and improved blood pressure control, as meta-analytic evidence suggests yoga-based interventions can reduce systolic and diastolic blood pressure in hypertensive individuals.[20,21] Similarly, a balanced cardioprotective diet, particularly a low-sodium DASH-style diet rich in fruits, vegetables, fiber, potassium, and polyphenols, could have reduced vascular inflammation, improved endothelial function, and slowed the progression of hypertensive heart disease.[22-25] Therefore, while lifestyle modification cannot be claimed to have definitively prevented the present presentation, earlier adoption of alcohol cessation, stress control, yoga, regular physical activity, sleep hygiene, and a heart-healthy diet may have reduced the cumulative burden

of hypertension-mediated cardiac and renal injury. Chronic alcohol consumption can cause a non-ischemic cardiomyopathy characterized by ventricular dysfunction in the absence of obstructive coronary artery disease. Alcohol-related myocardial dysfunction can mimic ischemic cardiomyopathy when the patient presents with dyspnea and reduced ejection fraction. Importantly, the diagnosis is clinically meaningful because improvement is possible with abstinence and optimized heart failure therapy. Nicolás et al. showed improvement in left ventricular ejection fraction over follow-up among patients with alcoholic cardiomyopathy who abstained or controlled alcohol intake, and contemporary reviews continue to emphasize alcohol cessation as the cornerstone of therapy. In the present patient, the improvement after conservative medical therapy supports the possibility of a functional, hypertensive, microvascular, or alcohol-related myocardial process rather than an irreversible obstructive coronary lesion.[26]

The echocardiographic RWMA deserves careful interpretation. RWMA is commonly associated with obstructive CAD, but it is not pathognomonic. It can occur due to transient ischemia, microvascular dysfunction, prior silent myocardial injury, myocarditis, stress cardiomyopathy, conduction abnormalities, loading-condition changes, or technical limitations of echocardiography. The Fourth Universal Definition of Myocardial Infarction emphasizes that myocardial infarction requires acute myocardial injury with a rise and/or fall in cardiac troponin along with evidence of ischemia; therefore, this case should not be labeled as MINOCA unless serial troponin elevation and clinical criteria for MI are documented. A better working formulation would be: "mild LV systolic dysfunction with RWMA and normal epicardial coronaries, likely due to hypertensive/alcohol-related cardiomyopathy with possible coronary microvascular dysfunction."

The renal dimension adds to the clinical value of the case. Coronary angiography in a patient with chronic kidney disease requires a careful risk-benefit decision because contrast-associated acute kidney injury is a recognized complication after coronary angiography and PCI. Mehran and Nikolsky defined contrast-induced nephropathy using absolute or relative creatinine rise after contrast exposure and highlighted patient-level risk factors, including pre-existing renal dysfunction. In the present case, limiting contrast volume to approximately 30 mL was an important procedural decision. Thus, the angiogram had diagnostic utility while minimizing renal risk, and it prevented an unnecessary stent in a patient who already had baseline renal vulnerability.[26,27]

The patient's hyperuricemia is also noteworthy. His serum uric acid was 9.2 mg/dL, which may be interpreted as part of a broader cardiometabolic risk profile rather than an isolated biochemical abnormality. Indian data on serum uric acid and metabolic parameters reported that uric acid correlated significantly with BMI, waist-hip ratio, sagittal abdominal diameter, and fasting plasma glucose, and discussed its association with inflammatory and oxidative pathways.[28] Anthropometry and blood biomarkers found serum uric acid and insulin resistance to be significantly increased in diabetic individuals and suggested uric acid as a potential early diagnostic marker of metabolic risk.[10,29] Although this patient was not frankly hyperglycemic, hyperuricemia along with obesity may still strengthen the argument for aggressive cardiometabolic risk reduction.

The conservative improvement after a normal angiogram is clinically important. Once a stentable lesion was excluded, management appropriately shifted toward blood pressure optimization, heart failure-directed therapy as tolerated, renal monitoring, alcohol cessation, and long-term risk-factor control. A statin review has emphasized that statins have benefits beyond LDL reduction, including endothelial function improvement, anti-inflammatory actions, antithrombotic effects, and plaque stabilization.[30,31] In a patient with high cardiovascular risk, statin therapy may be justified for risk reduction if lipid profile or ASCVD risk supports it, even though the angiogram did not show obstructive disease. However, antiplatelet therapy should not be automatically continued indefinitely solely because angiography was planned; it should be individualized based on confirmed ACS, atherosclerotic burden, stroke risk, bleeding risk, and cardiology advice.

The novelty of this report lies in the sequence of diagnostic contradiction and therapeutic restraint. A patient with progressive rest dyspnea and RWMA appeared to be moving toward coronary stenting, but invasive coronary angiography showed normal epicardial vessels. This outcome prevented unnecessary intervention and redirected the diagnosis toward hypertensive heart disease, alcohol-related myocardial dysfunction, and possible microvascular ischemia. The case therefore reinforces an important clinical message: in symptomatic hypertensive patients, RWMA should trigger ischemic evaluation, but normal coronaries should prompt deeper assessment rather than dismissal of symptoms.

CONCLUSION

In conclusion, this case illustrates that progressive breathlessness, mild LV dysfunction, and RWMA in a hypertensive alcoholic patient do not necessarily indicate obstructive coronary artery disease. Normal invasive coronary angiography should broaden the differential diagnosis to hypertensive myocardial remodeling, alcohol-related cardiomyopathy, coronary microvascular dysfunction, and other non-ischemic myocardial disorders. The patient's improvement with conservative therapy supports the value of individualized medical management and highlights the importance of avoiding reflex stenting when coronary anatomy is normal.

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