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(CASE REPORT)

From endothelial dysfunction to advanced cardiovascular disease: A case report of cardiometabolic progression

Michael Mario Vélez Lora [1,*], Joaquín Alberto Chois Antequera [1], Mauro Alonso Moreno Artuz [1] and María Mónica González Chinchilla [2]

[1] *Internal Medicine Resident, Universidad Libre. Barranquilla, Colombia.*
[2] *Physician, Universidad Metropolitana. Barranquilla, Colombia.*

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Abstract

Background: Endothelial dysfunction represents one of the earliest pathophysiological events in the development of atherosclerotic cardiovascular disease. Metabolic abnormalities such as dyslipidemia, prediabetes, and overweight contribute to oxidative stress, vascular inflammation, and progressive impairment of nitric oxide bioavailability.

Case presentation: A 60-year-old male with cardiometabolic risk factors presented with progressive dyspnea and was diagnosed with severe dilated cardiomyopathy and multivessel coronary artery disease.

Conclusion: This case highlights the continuum from endothelial dysfunction to advanced cardiovascular disease.

Keywords: Cardiovascular risk; Endothelium; Atherosclerosis; High blood pressure; Prediabetes; Dyslipidemia; Inflammation

1. Introduction

Endothelial dysfunction represents the silent starting point of atherosclerotic disease, where metabolic factors such as dyslipidemia, overweight, and prediabetes converge to trigger a cascade of oxidative stress, inflammation, and nitric oxide dysfunction that, over time, promotes the formation and progression of arterial plaque. Discussing this process is fundamental because it allows us to understand that cardiovascular risk does not begin with a heart attack or stroke, but years earlier, in an already damaged endothelium that loses its protective capacity. Recognizing this early phase facilitates timely intervention through intensive control of risk factors, preventing progression to major complications. Therefore, addressing the transition from endothelial dysfunction to clinical cardiovascular disease not only improves our pathophysiological understanding but also highlights the importance of prevention as a key tool for reducing morbidity and mortality in patients with high-risk metabolic phenotypes.

2. Clinical case presentation

A 60-year-old male patient with a history of overweight, dyslipidemia, and prediabetes presented with a 40-day history of progressive dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and lower extremity edema. Hyperglycemia was noted, suggesting impaired glucose metabolism and contributing to a cardiometabolic phenotype. Physical examination revealed grade III jugular venous distension, basal crackles, and grade III peripheral edema. Laboratory tests showed proBNP at 2721 pg/mL, polycythemia, elevated creatinine, and an LDL level of 165 mg/dL. X-ray with grade II cardiomegaly, with echocardiogram report: dilated cardiomyopathy with severely reduced LVEF 18%, bilateral atrial

* Corresponding author: Michael Mario Vélez Lora

dilation and type I diastolic dysfunction. Acute heart failure was diagnosed, so management with loop diuretics was indicated, controlling with urinary sodium and diuresis volume. The patient improved with treatment at 96 hours and outpatient management was indicated with foundational therapy, lipoprotein a, Apo A and Apo B levels were requested in order to determine if he would benefit from recombinant messenger RNA (siRNA) therapy.

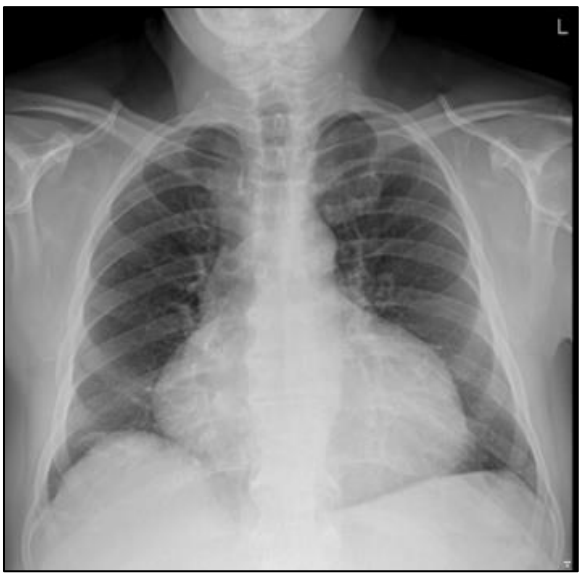

Figure 1 Chest radiograph shows evident cardiomegaly, with increased cardiothoracic ratio, accompanied by hilar and perihilar vascular redistribution consistent with mild pulmonary congestion

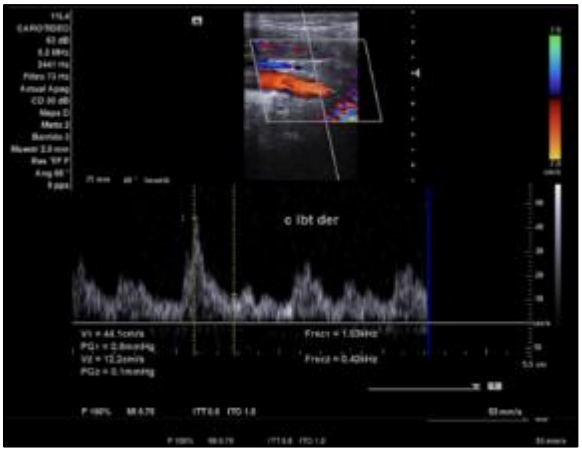

Figure 2 Doppler of neck vessels showed a calcified plaque in the right internal carotid artery of 1.8 mm, with velocities of 44/12 cm/s corresponding to a stenosis of 35%, compatible with mild arteriosclerotic disease of the extracranial carotid tree

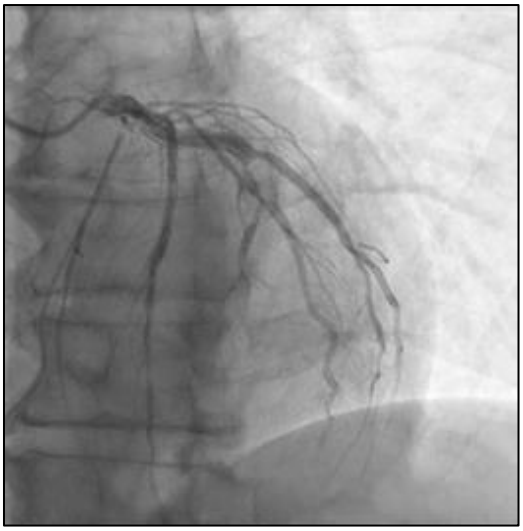

Figure 3 Coronary arteriography revealed severe multivessel coronary artery disease, with critical lesions in multiple territories, while the first and second diagonal branches showed single critical stenosis. The left anterior descending artery showed 70–80% stenosis in the mid-third, 90% stenosis in the circumflex artery, and 80–90% stenosis in the distal segment. The third obtuse marginal branch showed a 70% lesion at the bifurcation. The right coronary artery, the dominant vessel, showed a 90% proximal lesion, multiple 70–80% stenoses in the mid-third, and a 90% critical stenosis in its mid-course, as well as an 80% distal lesion. These findings confirm severe multivessel coronary artery disease

3. Discussion

The transition from endothelial dysfunction to overt cardiovascular disease represents a pathophysiological continuum that, in this patient, culminated in severely decompensated heart failure (1,2). Endothelial dysfunction is defined as an alteration of vascular homeostasis characterized by vasoconstriction and a prothrombotic state, primarily due to reduced nitric oxide bioavailability (3,4). Worldwide, cardiovascular diseases are the leading cause of morbidity and mortality; in Colombia, the nutritional transition has increased the prevalence of cardiometabolic phenotypes, where overweight and prediabetes accelerate premature vascular aging (5,6).

The pathophysiology in this case is explained by the synergistic impact of chronic hyperglycemia and excess LDL (165 mg/dL), which act as chronic irritants, activating a state of systemic oxidative stress (7). The alteration in shear stress, along with the accumulation of reactive oxygen species, leads to disruption of the endothelial barrier, exposing the collagen-rich subendothelium (8). This phenomenon facilitates the vascular deposition of calcified lipid molecules and pro-inflammatory mediators, accelerating atherosclerosis and increasing arterial stiffness (9). Consequently, the sustained increase in filling pressures and afterload resistance triggers pathological activation of the renin-angiotensin-aldosterone system (RAAS) and a compensatory release of endogenous amines (10). This chronic neurohormonality activates cardiovascular remodeling receptors that induce stretching of myocardial fibers and biventricular dilation. Although there is an initial increase in brain natriuretic peptides as a compensatory mechanism, the predominant activity of enzymes such as neprilysin degrades these peptides, perpetuating the cycle of heart failure and systemic congestion (11,12). This, in turn, through mechanisms of low cardiac pump efficiency, generates concentricity and subsequent eccentricity, leading to dilated cardiomyopathy with a severely reduced ejection fraction and subsequent clinical presentation of pulmonary edema and/or congestion in the vascular economy (12).

This clinical case presentation has become significantly more frequent in emergency departments given the increased recognition of heart failure by physicians. However, when comparing this case report with local studies in the Colombian Caribbean region, it is evident that patients with intermediate metabolic risks are often underdiagnosed, presenting with severe structural failure such as the dilated cardiomyopathy with an LVEF of 18% observed in this individual (13). This case aligns strictly with international guidelines describing heart failure of metabolic phenotype, where elevated proBNP (2721 pg/mL) reflects extreme wall stress and a failure of counterregulatory mechanisms (14). In conclusion, endothelial damage is the critical initiating event that links metabolic factors to end-stage organ failure (15). Preventive management and rigorous control of prediabetes and dyslipidemia in early stages is the only strategy capable of slowing the progression towards irreversible deterioration of the ejection fraction in the Colombian population.

4. Conclusion

In conclusion, this clinical case demonstrates that the endothelial dysfunction. This is not an isolated finding, but rather the critical initiating event that connects metabolic risk factors—such as prediabetes and dyslipidemia (LDL 165 mg/dL)—with end-stage organ failure and adverse cardiac remodeling. The torpid progression to dilated cardiomyopathy with an LVEF of 18% highlights the aggressiveness of the cardiometabolic phenotype when the compensatory mechanisms of natriuretic peptides are overwhelmed by neurohormonal activation of the RAAS axis and neprilysin activity. The relevance of this report to the Colombian medical literature lies in emphasizing that management should not be purely reactive to edema or dyspnea, but rather preventive and early. Rigorous control of metabolic alterations in stages of endothelial dysfunction is the only real window of opportunity to prevent irreversible structural changes and reduce the high burden of morbidity from decompensated heart failure in our population.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References

[1] Libby P. The changing landscape of atherosclerosis. Nature. 2021;592(7855):524-533.

[2] Mach F, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias. Eur Heart J. 2020;41(1):111-188.

[3] Puri R, et al. Pathophysiology of the vascular endothelium and the plaque. J Am Coll Cardiol. 2018;72(20):2485-2491.

[4] Fishermen FLJ, et al. 2021 ESC Guidelines on cardiovascular disease prevention. Eur Heart J. 2021;42(34):3227-3337.

[5] Ministry of Health.Health Situation Analysis (ASIS) Colombia 2023. Bogotá; 2023.

[6] Cuerda A, et al. Pre-diabetes and cardiovascular risk: an updated review. Endocrinol Diabetes Metab. 2021;4(3):e00249.

[7] Bonetti PO, et al. Endothelial dysfunction: a marker of atherosclerotic risk. Arterioscler Thromb Vasc Biol. 2020;23(2):168-175.

[8] Ruiz-López AJ, et al.Cardiovascular risk factors in Colombian adults.Rev Colomb Cardiol. 2022;29(2):145-154.

[9] Heidenreich PA, et al. 2022 AHA/ACC/HFSA Guideline for Heart Failure. Circulation2022;145(18):e895-e1032.

[10] Zuchi C, et al. Role of endothelial dysfunction in heart failure. Heart Fail Rev. 2020;25(1):21-30.

[11] Gómez-Arbeláez D, et al.Metabolic profile in the Colombian Caribbean region.Acta Med Colomb. 2021;46(3):12-19.

[12] Daiber A, et al. Oxidative stress and inflammation in heart failure. Antioxid Redox Signal. 2020;32(15):1110-1125.

[13] McDonagh TA, et al. 2021 ESC Guidelines for acute and chronic heart failure. Eur Heart J. 2021;42(36):3599-3726.

[14] Yancy CW, et al. Impact of obesity and prediabetes on heart failure. JACC Heart Fail. 2022;10(5):341-352.

[15] Saba S, et al. Global epidemiology of heart failure. Curr Probl Cardiol. 2022;47(11):101340.