

## VIEWPOINT

**Coronary Artery Microfistulas: Historical Description and Contemporary Clinical Relevance**Stans Murad Netto,<sup>1</sup> Rachel Vianna O'Neill de Souza,<sup>1</sup> Victor Murad,<sup>1</sup> Evandro Tinoco Mesquita<sup>1</sup> *Universidade Federal Fluminense,<sup>1</sup> Niterói, RJ – Brazil***Introduction**

Coronary microfistula was initially described as an anatomical finding in the late 1960s by Murad and colleagues, and it was later recognized as a morphofunctional abnormality of the coronary arteries capable of causing clinical findings<sup>1</sup> (Figure 1). This often asymptomatic entity is still poorly recognized in clinical practice, although it can be readily identified in the catheterization laboratory, cineangiography studies, and more recently by coronary CT angiography.

Initially, this condition was not taken into consideration, and it was justified as a possible “error” in cineangiography technique. Different studies have demonstrated its existence and its potential as a nosological entity responsible for myocardial ischemia, cardiac chamber overload, arrhythmias, and even heart failure.<sup>2,3</sup>

Congenital abnormalities in the coronary arteries have been observed in 0.6% to 1.5% of individuals undergoing cardiac catheterization, with the most common type being fistulas between the coronary arteries and the interior of the cardiac chambers. Coronary microfistulas are currently referred to as coronary-cavitary fistulas, most commonly connected to the right atrium and then to the left atrium.<sup>4</sup>

Coronary microfistulas, or cardiac malformations, are defects that arise during embryogenesis and may be associated with congenital heart diseases such as tetralogy of Fallot, persistent ductus arteriosus, pulmonary atresia, atrial septal defect, and ventricular septal defect. The presence of coronary microfistulas has been identified among other conditions, such as non-compacted cardiomyopathy and hypertrophic cardiomyopathy.<sup>1,4</sup>

**Keywords**

CF; Myocardial Ischemia; Angina Pectoris; Coronary Vessels.

This perspective aims to review historical findings and the ingenious strategies of researchers to prove the pathophysiological and clinical contributions resulting from alterations in normal coronary flow, compromising cardiac perfusion and causing myocardial ischemia.

**Historical descriptions and controversy**

The discovery of a new entity requires close observation and correct pathophysiological reasoning, which are fundamental attributes for an astute medical scientist. We can clearly identify these attributes when we review the history of the discovery of coronary microfistulas, which were observed during a cardiac catheterization examination by the hemodynamicist physician Stans Murad Netto and his colleagues in the 1960s.

In the history of science, it is common for innovative discoveries to be made by chance, where an accidental finding becomes something new, referred to as serendipity. An example was the discovery of penicillin by Scottish physician and bacteriologist Alexander Fleming in 1928, which revolutionized the treatment of infectious diseases and gave rise to modern antibiotic therapy.

Our view is that, in the last five decades, the concept of coronary microfistulas, as a clinical entity, has gone through several distinct stages, as commonly observed with the introduction of new concepts. Thus, for didactic purposes, we have established the following three phases:

Phase 1. Description of the phenomenon and proof of concept – The description during a routine coronary angiography, with filling of the right atrial cavity, led to the consideration of the existence of micro-communications between the coronary artery and the right atrium. These “invisible communications” were then termed coronary microfistulas. After the identification of this finding, its demonstration in scientific forums had a moment of non-acceptance when the brilliant pathologist Professor

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Thomas James questioned whether the contrast had been injected with excessive pressure inside the coronary artery, which was promptly refuted by Dr. Stans Murad Netto. Subsequently, young patients with coronary syndrome, chest pain, and normal coronary arteries in different parts of the world were also observed to have microfistulas.

Phase 2. The pathophysiological connection between microfistulas and myocardial ischemia – The presence of myocardial ischemia associated with coronary microfistula was meticulously documented by identifying anaerobic metabolism when injecting mixed blood into the right coronary artery and simultaneously aspirating from the left ventricle, showing the presence of a shunt within the left ventricular cavity. Additionally, cases of patients with myocardial ischemia in scintigraphy and stress tests, presenting angina symptoms, started to be described in daily clinical practice with the presence of microfistulas during coronary angiography. Murad also identified the presence of these coronary microfistulas in other situations, such as congenital heart disease, as described in a case of aortic septal defect, where microfistulas were observed in the left ventricular cavity from both coronary arteries (Figure 2).

Phase 3. The convergence of coronary microfistulas and clinical conditions – Starting in the 1980s, coronary microfistula became well established not only as an etiology for chronic myocardial ischemia syndrome in the absence of coronary obstruction, but also as a cause of other cardiovascular conditions, such as chamber overload, arrhythmias resulting in heart murmurs, and right and/or left heart failure. In some series, microfistulas from coronary arteries, as demonstrated by Salah et al., showed a relationship with T-wave inversion in precordial leads in 40% of cases. Some cases were associated with hypertrophic cardiomyopathy, making coronary microfistula a differential diagnosis for left ventricular wall.

## Conclusion

Currently, coronary microfistula, although often an asymptomatic condition, requires greater recognition and further research to understand its molecular and genetic mechanisms, as well as new approaches for closing these microfistulas.

This anatomical entity, described in the 1960s as a condition that was poorly recognized by doctors, can present as different clinical forms of myocardial ischemia,



Figure 1 – Presence of microfistulas in the left coronary artery filling the left ventricle.

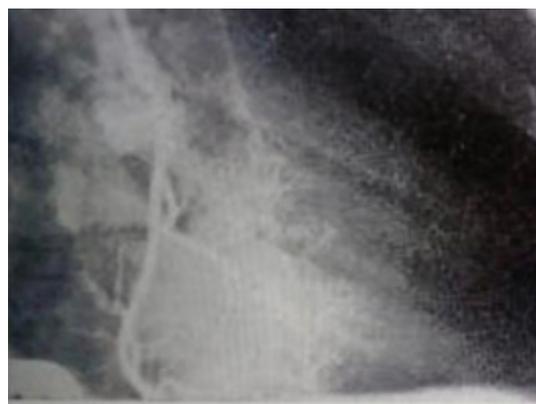


Figure 2 – Microfistulas communicating the right coronary artery with the interior of the left ventricular cavity

chamber overload, cardiac arrhythmia, and heart failure. Over five decades, various stages have been overcome to prove its role in the context of cardiology. Its discovery was a result of serendipity, a fortunate accident. Just like in the history of science, professionals committed to the scientific method and trained in cardiovascular pathophysiology have transformed these findings into important discoveries for the advancement of clinical cardiac science.

## Author Contributions

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