

Long-Term Angina-Related Healthcare Utilisation in Women with ANOCA and Impaired Coronary Flow Velocity Reserve

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SUMMARY OF KEY RESEARCH FINDINGS

Angina with no obstructive coronary artery disease (ANOCA) is common among women and is not benign. Many experience persistent symptoms, and the condition is associated with an adverse cardiovascular prognosis.¹ However, it remains unclear whether coronary microvascular dysfunction (CMD) also contributes to symptom persistence and increased angina-related healthcare utilisation.

In this registry-based follow-up of 1,681 women from the iPower cohort, the author examined whether CMD, defined as an impaired coronary flow velocity reserve (CFVR; <2.25) assessed by transthoracic Doppler echocardiography, was associated with recurrent angina-related healthcare utilisation.² Women with impaired CFVR had a significantly higher rate of angina-related hospitalisations, outpatient contacts, and ischaemia-related diagnostic procedures compared with women with preserved CFVR. The composite event rate was 11.48 versus 7.31 events per 100 person-years, respectively ($p < 0.001$). Impaired CFVR remained associated with increased recurrent healthcare utilisation after

adjustment for cardiovascular risk factors and baseline angina severity (hazard ratio: 1.47; 95% CI: 1.10–1.97). These findings suggest that CMD contributes to persistent symptoms among women with ANOCA, resulting in a greater long-term burden of healthcare contact and repeated diagnostic evaluations.

WHAT CHALLENGE DOES THIS ADDRESS?

CMD is a potential explanation for persistent angina in patients with ANOCA, but is not routinely assessed in clinical practice. Consequently, patients with ongoing symptoms may undergo repeated ischaemia-related investigations without receiving a more specific diagnosis. These findings highlight that excluding obstructive coronary artery disease does not necessarily identify a benign or low-risk population, and that assessment of CMD may help identify at-risk patients earlier and provide a more specific explanation for their symptoms. To some extent, a CMD diagnosis may also warrant invasive testing for further endotyping, with the aim of endotype-guided anti-anginal treatment.

A remaining challenge, however, is that there is currently no evidence-based disease-modifying treatment for CMD.

RELEVANCE FOR EUROPEAN PRACTICE

The traditional diagnostic work-up for angina has focused predominantly on identifying or excluding obstructive epicardial coronary artery disease. The 2024 European Society of Cardiology (ESC) guidelines marked an important shift, with greater emphasis on ANOCA and recommendations for coronary function assessment in selected patients with persistent symptoms.³ These findings

support the clinical relevance of this shift and encourage wider implementation, as assessment of coronary microvascular function in routine clinical practice remains limited.

Recognition of CMD may have important clinical value, as focus can be applied to cardiovascular risk-factor modification and enabling endotype-guided anti-anginal treatment, which may in turn reduce repeated futile investigations.

Beyond the author's central finding that CMD is associated with greater angina-related healthcare utilisation, an important aspect demonstrated in the study was that this pattern of healthcare utilisation seems consistent throughout the entire follow-up period of 11 years. Thus, the burden does not seem to be self-limiting, but rather represents an ongoing symptomatic challenge for patients and an economic burden for healthcare systems and society.

WHAT ARE THE NEXT STEPS FOR THE RESEARCH?

An important next step is to understand the longitudinal progression of CMD. The present study assessed CFVR at baseline only, and it remains unclear whether impaired microvascular function changes over time. Repeat assessment could determine whether persistent or worsening CMD is associated with greater long-term symptoms and healthcare burden.

In the iPower study, participants and treating clinicians were blinded to the results of coronary microvascular function testing. Consequently, the author does

not know whether providing patients with a specific diagnosis and explanation for their symptoms could influence healthcare utilisation or quality of life. Future studies should investigate whether diagnostic clarification leads to greater reassurance, more targeted management, fewer repeated investigations, and improved patient-reported outcomes.

Further long-term follow-up with detailed phenotyping is needed to improve understanding of the natural history and heterogeneity of CMD. Identifying distinct CMD phenotypes and endotypes may help determine which patients are at the greatest risk of persistent symptoms and adverse cardiovascular outcomes, and ultimately guide the development of targeted treatments.

Finally, randomised clinical trials are needed to evaluate CMD-targeted treatments and determine whether endotype-guided interventions can improve coronary microvascular function, reduce angina burden, and ultimately improve long-term cardiovascular outcomes.

References

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