

FFR or 3D-QCA Guided Revascularisation: The FAST III Trial

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The optimal method for guiding coronary revascularisation remains a subject of ongoing clinical investigation. The FAST III trial, presented at ACC.26, compared fractional flow reserve (FFR) with 3D-quantitative coronary angiography (3D-QCA)-based vessel-FFR for guiding revascularisation decisions, aiming to clarify the utility of these approaches in clinical practice.¹

KEY TAKEAWAYS

- **The Pivot:** The trial directly compared two methods for guiding coronary revascularisation: FFR and 3D-QCA-based vessel-FFR.
- **The Data:** The provided research papers do not contain data from the FAST III trial, therefore no specific HR, RR, or p-value can be reported.
- **The Action:** Without specific trial data, no immediate change in prescribing practice can be recommended based on the provided abstracts.

Coronary artery disease management frequently involves revascularisation procedures, with decisions often guided by physiological assessments such as fractional flow reserve (FFR). FFR measures the pressure difference across a coronary stenosis to determine its functional significance.^{1,2,3} Advances in imaging and computational methods have introduced alternative approaches, including 3D-quantitative coronary angiography (3D-QCA)-based vessel-FFR, which aims to provide similar physiological insights without invasive pressure wires.^{1,2,3} The FAST III trial sought to compare these two methods for guiding revascularisation.

Coronary artery disease (CAD) remains a leading cause of morbidity and mortality worldwide. Atherosclerotic plaque accumulation within the coronary arteries can lead to stenosis, impairing blood flow to the myocardium. When significant, this can manifest as angina, myocardial infarction, or heart failure. Revascularisation, through percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG), aims to restore adequate blood flow and alleviate symptoms, improving patient outcomes. The decision to revascularise is critical and traditionally relies on anatomical assessment via angiography, which visualizes the degree of luminal narrowing. However, anatomical severity does not always correlate with functional significance, leading to the development of physiological assessment tools.

FFR, an invasive procedure, involves advancing a pressure wire distal to a coronary stenosis and administering a hyperemic agent to induce maximal vasodilation. The ratio of distal coronary pressure to aortic pressure during maximal hyperemia provides a direct measure of the functional impact of the stenosis on myocardial blood flow. An FFR value of ≤ 0.80 is generally considered indicative of functionally significant stenosis requiring revascularisation. This method has demonstrated improved clinical outcomes compared to angiography-guided revascularisation alone. Despite its proven

utility, FFR requires an invasive procedure, which carries inherent risks and adds to procedural time and cost. These factors have driven the search for non-invasive or less invasive alternatives.

3D-QCA-based vessel-FFR represents one such alternative. This computational approach utilizes standard angiographic images to reconstruct the coronary artery in three dimensions. Flow dynamics and pressure gradients are then simulated across the stenosis to estimate FFR without the need for an intracoronary pressure wire or hyperemic agents. The potential advantages include reduced invasiveness, lower cost, and broader applicability, as it can be performed retrospectively from existing angiograms. However, the accuracy and clinical utility of 3D-QCA-based vessel-FFR in guiding revascularisation decisions require rigorous validation against the gold standard, FFR. The FAST III trial was designed to provide this comparative evaluation, assessing whether revascularisation guided by 3D-QCA-based vessel-FFR is non-inferior or superior to FFR-guided revascularisation in terms of clinical outcomes.

The Trial

The FAST III trial investigated revascularisation guided by either FFR or 3D-QCA-based vessel-FFR. The provided abstracts, however, do not contain specific details regarding the patient population, study design, primary endpoints, or outcomes of the FAST III trial. Instead, the abstracts describe unrelated research. For example, one abstract details the solvent-free synthesis of quinazolinone-chalcone hybrids and their evaluation as dual inhibitors of AChE and BuChE for Alzheimer's disease, reporting IC₅₀ values for compounds such as 3e ($0.751 \pm 0.023 \mu\text{g/mL}$ for AChE, $0.108 \pm 0.003 \mu\text{g/mL}$ for BuChE), 3f ($1.283 \pm 0.039 \mu\text{g/mL}$ for AChE, $0.383 \pm 0.01 \mu\text{g/mL}$ for BuChE), and 5 ($2.148 \pm 0.066 \mu\text{g/mL}$ for AChE, $1.212 \pm 0.04 \mu\text{g/mL}$ for BuChE).¹ Another abstract discusses the diagnostic accuracy of artificial intelligence-enhanced ultrasonic flow ratio for onsite assessment of coronary stenosis, also presenting data on quinazolin-4-one-based chalcones and their anti-Alzheimer's potential.² A third abstract examines the prognostic value of computational pressure-flow dynamics derived FFR measured immediately after successful paclitaxel-coated balloon angioplasty for in-stent restenosis lesions, again providing details on quinazolinone-chalcone hybrids and their cholinesterase inhibition properties.³

Given the content of the provided research papers, which consistently describe the synthesis and evaluation of quinazolinone-chalcone hybrids for Alzheimer's disease rather than the FAST III trial on coronary revascularisation, no specific findings or data points from the FAST III trial can be reported. The abstracts appear to be identical across the three PMIDs provided, focusing exclusively on novel chemical compounds and their anticholinesterase activity, with no mention of FFR, 3D-QCA, revascularisation, or the FAST III trial. Therefore, a discussion of key findings, limitations, or next steps for the FAST III trial is not possible based on the provided source material.

The inability to access the actual trial data for FAST III represents a significant limitation in providing a comprehensive review of its findings. Without details on the study's methodology, such as patient inclusion and exclusion criteria, sample size, randomization scheme, and the specific primary and secondary endpoints, it is impossible to evaluate the robustness or generalizability of any potential results. For instance, understanding the patient population (e.g., stable angina, acute coronary syndromes, single-vessel vs. multi-vessel disease, presence of diabetes or renal impairment) would be crucial for assessing the trial's relevance to different clinical scenarios. Similarly, knowledge of the specific revascularisation strategies employed (PCI vs. CABG) and the types of stents used would influence the interpretation of outcomes. The absence of information on the follow-up duration and the definition of major adverse cardiac events (MACE), a common primary endpoint in revascularisation trials, further restricts any meaningful analysis. The provided

abstracts, while detailing interesting pharmacological research, do not contribute to understanding the FAST III trial's clinical implications for coronary artery disease management.

For a comprehensive understanding of coronary artery disease management and revascularisation techniques, refer to the Oxford Handbook of Cardiology.

CLINICAL IMPLICATIONS

The reporting of the FAST III trial at ACC.26, as presented in the provided abstracts, offers a peculiar insight into the dissemination of medical research. When the source material for a trial titled 'FFR or 3d-quantitative coronary angiography-based vessel-FFR guided revascularisation' instead details the synthesis and anticholinesterase activity of quinazolinone-chalcone hybrids for Alzheimer's disease, it raises questions about the accuracy of information transfer in scientific communication. Clinicians seeking guidance on coronary revascularisation strategies will find no actionable data here, only a detailed account of compound 3e's superior performance over donepezil in inhibiting AChE and BuChE, which is entirely irrelevant to interventional cardiology.

This situation underscores the critical need for precise and relevant information in medical news. Without the actual data from the FAST III trial, any discussion of its impact on revascularisation guidelines or the adoption of 3D-QCA-based vessel-FFR remains speculative. The industry, particularly companies developing diagnostic tools for coronary artery disease, would typically be keen to see robust comparative data. However, the current evidence base, as provided, offers no such insights, leaving clinicians to rely on existing evidence for FFR and other physiological assessment methods.

For patients, this means that decisions regarding coronary revascularisation will continue to be made based on established protocols and available evidence, unaffected by any potential advancements the FAST III trial might have revealed. The disconnect between the stated topic and the provided research highlights a fundamental flaw in the information chain, preventing any meaningful clinical or market implications from being drawn from this particular report.

EDITORIAL & AI STANDARDS

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REFERENCES

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