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Perspective Article

Fixing the Narrowing vs Saving Lives: Vulnerable Plaque, Lifestyle Medicine, and the Future of Interventional Cardiology

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Abstract

Interventional cardiology has traditionally focused on fixing angiographic stenosis and documenting ischaemia. Yet, most MI and sudden deaths arise from morphologically vulnerable but often non-flow-limiting plaques. During the COVID-19 pandemic, this tension between "fixing the narrowing" and truly preventing MI and death became starkly visible in daily practice. This paper explains the ethical, clinical, and spiritual motivations behind our decision to prioritise vulnerability-guided complete revascularization (VGCR) during the pandemic, despite controversy and systemic resistance, and presents the outcomes of this approach. We describe a single-centre experience in which 1,750 elderly COVID-19 patients with coronary artery disease and angiographically identified vulnerable plaques (VPs) underwent VGCR between early 2020 and mid-2023, combined with a whole food plant-based diet and comprehensive risk-factor optimisation. Despite advanced age and high baseline risk, zero mortality was observed over three years of follow-up in this cohort. Rather than treating anatomy and ischaemia in isolation, this approach targets the biological substrate of plaque rupture and erosion while simultaneously modifying systemic drivers of vulnerability, such as inflammation, endothelial dysfunction, and metabolic derangement. We argue that current guidelines, training pathways, and ethical frameworks in interventional cardiology should evolve from a narrow focus on stenosis severity and FFR towards a broader mandate: preventing MI and death by detecting and stabilising VPs as early as possible, including during global crises such as COVID-19. Our experience suggests that VGCR, when coupled with aggressive lifestyle and medical therapy, may offer a powerful—yet underused—strategy to protect high-risk patients from fatal coronary events.

Introduction

Interventional cardiology was built on a simple visual promise: find the tightest stenosis, open it, yet patients will not be prevented from having myocardial infarctions (MI) or live longer, as the ISCHEMIA trial has already demonstrated [1-3]. For decades, pathology, imaging, and longitudinal studies have quietly shown a different reality. Most MI and sudden deaths arise not from the tightest, most ischaemia-producing lesions, but from morphologically vulnerable, often non-flow-limiting plaques that rupture or erode under the right biological stress [4-7]. The COVID-19 pandemic turned this long-standing tension into an unavoidable, real-time clinical question.

When SARS-CoV-2 reached Indonesia, patients with coronary artery disease (CAD) suddenly faced a "perfect storm" of plaque-destabilising forces: systemic inflammation, endothelial injury, hypercoagulability, microthrombosis, and major disruptions in routine cardiovascular care [8-11]. In many centres, the institutional response was to scale back activity, defer non-urgent procedures, and adhere strictly to pre-existing stenosis- and ischaemia-centric guidelines [12-14]. In practice, this meant that lesions below traditional thresholds and non-culprit plaques were often left untreated, even in highly vulnerable patients living through an intensely pro-thrombotic, plaque-vulnerable environment.

In our centre, the central question was framed differently: Is the role of interventional cardiology merely to fix angiographic stenosis, or to prevent MI and death—especially in a pandemic that amplifies plaque vulnerability? If VPs are the true substrate of most fatal coronary events, then ignoring them during COVID-19 might be biologically convenient but clinically unsafe. Rather than retreating to the narrowest interpretation of "appropriate care", we chose to remain in the catheterization laboratory (Cath lab), actively searching for and treating VPs while simultaneously addressing

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systemic drivers of vulnerability.

Between early 2020 and mid-2023, our hospital implemented a vulnerability-guided strategy that combined advanced coronary imaging, complete revascularization (CR) of high-risk plaques (HRPs), and an intensive whole-food plant-based diet (WFPBD) and lifestyle programme for elderly COVID-19 patients with CAD [15]. Elderly patients (age ≥60 years) with confirmed COVID-19 infection and angiographically visible VPs on invasive angiography, combined with coronary computed tomography angiography (CTCA) findings, were offered vulnerability-guided complete revascularization (VGCR), together with standardised WFPBD counselling and intensive risk-factor optimisation. Consecutive patients who accepted this programme between early 2020 and mid-2023 (n=1,750) were entered into a prospective registry with scheduled clinical follow-up and active tracing of all-cause mortality via hospital records and direct contact with patients or families. In this cohort of patients with angiographically identified VPs who underwent CR, we observed no mortality over a 3-year follow-up period, despite their advanced age and high baseline risk. This experience did not arise in a vacuum; it was the outcome of long-standing convictions about plaque biology, lifestyle medicine, ethics, national responsibility, and personal faith.

This perspective article does not present a randomised trial, but a carefully documented single-centre cohort used as a lens to examine what interventional cardiology is ultimately for. It explains the ethical, clinical, and spiritual motivations behind our decision to prioritise VGCR during the pandemic and contrasts this approach with the dominant stenosis- and ischaemia-driven paradigm. By sharing both our data and our inner reasoning, we aim to stimulate a broader conversation on how interventional cardiology can move from simply fixing the narrowing to truly preventing MI and death during pandemics and in everyday practice.

Loving the Patient as Yourself

At the simplest level, my choice was rooted in a basic moral intuition: a patient is not a case, but a neighbour.

I have always believed that "love your neighbour as yourself" is not a poetic slogan; it is a practical command [16-19]. During COVID-19, each patient who arrived at our hospital doors carried not only a virus, but also fear, family responsibilities, and a fragile sense of hope. To treat them as I would wish to be treated—if I were on the stretcher—meant being present, not absent; engaged, not hidden; creative, not passive.

This love of neighbour is not sentimentality. It translates into staying up at night reviewing CTCA images, re-thinking protocols, and asking: *If this were my own heart, or my own parent, what would I want my doctor to do?* Once that question is taken seriously, withdrawing from the front line became, for me, very difficult to justify.

Taking the Physician's Oath Seriously—Especially When It Hurts

We all recite some version of a physician's oath. We promise to care, to serve, to protect life, and to accept personal risk. But an oath is not tested when the hospital is calm, and the income is stable. It is tested when stepping into the ward or the Cath lab, which genuinely feels dangerous.

To me, abandoning high-risk/ vulnerable patients in a pandemic (vulnerable environment) would have been like a spouse who is faithful only when the partner is prosperous, healthy, and successful, but disappears when sickness and poverty arrive. The true meaning of the marital vow is revealed in crisis. I believe the same is true for the physician's vow.

COVID-19 was the moment when we could either demonstrate that our oath was real or reveal that it was merely ceremonial [20-23]. That realization weighed heavily on my conscience.

From Theory to Duty: VP, Lifestyle, and My Own Heart

Long before COVID, my clinical and research journey had led me to a deep interest in VP, WFPBD, and lifestyle medicine [24-26]. I had studied:

- how thin-cap fibroatheromas and lipid-rich plaques rupture
- how nitric oxide (NO) supports endothelial health
- how oxidative stress, chronic inflammation, microbiota, TMAO, and telomere shortening all interact with atherosclerosis biology.

I had not only applied these insights to my patients; I had used them on myself. I had seen what happens when LDL is driven to ultra-low levels, when diet is radically improved, and when inflammation and metabolic stress are actively controlled. I had already experienced, in real people, that plaque can regress, stabilize, and become less dangerous [27-30]. Similarly, these approaches have been shown to decrease restenosis and stent thrombosis [31].

When the pandemic struck, I could not simply "forget" this body of knowledge. If I believed it could reduce heart attacks and sudden deaths in high-risk patients, then failing to use it when the risk was felt to be highest, in my own moral framework, would feel like a form of neglect.

The Dominant Role of VP Compared with Stenosis

The interaction between VP and stenosis severity is summarised in the table.1 highlight a central message of this paper: biological vulnerability matters far more than the angiographic percentage of narrowing in predicting MI [32-35].

When both VP and significant stenosis are present (VP+ / stenosis+), the risk of MI is maximal, rising to approximately 31-fold above that of individuals with no angiographic CAD. This is intuitive: a flow-limiting lesion that is also biologically unstable represents the "perfect storm" for plaque rupture or erosion, thrombus formation, and acute coronary occlusion. In such lesions, both haemodynamic stress and plaque biology work in the same direction to precipitate events.

However, perhaps the most important—and counterintuitive—quadrant is where VP is present, but stenosis is non-significant (VP+ / stenosis−). Here, the MI risk remains exceptionally high, at about 25-fold above baseline, only modestly lower than in the VP+ / stenosis+ state. This means that the presence of a VP alone, even without a tight narrowing, is enough to confer a very high risk. For clinicians trained to focus primarily on diameter stenosis and ischaemia testing, this is a crucial wake-up call: leaving a "mild" but vulnerable lesion untreated may expose patients to nearly the same risk as a severe, unstable stenosis.

Table 1. *Relative Risk of MI According to Vulnerable Plaque Status and Stenosis Severity*

Vulnerable plaque (VP)	Stenosis severity	Approx. relative risk of MI*	Mechanistic interpretation
Present (VP+)	Significant stenosis (+)	31×	Highest risk: unstable substrate and flow-limiting lesion
Present (VP+)	Non-significant stenosis (−)	25×	Very high risk driven mainly by plaque vulnerability
Absent (VP−)	Significant stenosis (+)	4.3×	Risk modestly increased; lesion is tight but biologically stable
Absent (VP−)	Non-significant stenosis (−)	2×	Low risk: no vulnerable morphology and no major narrowing

*Relative to individuals with no angiographic CAD (no vulnerable plaque and no significant stenosis).

In contrast, when VP is absent, but stenosis is significant (VP− / stenosis+), the relative risk of MI is much lower—around 4.3-fold. The lesion is tight, but if the plaque is morphologically stable (thick fibrous cap, low lipid content, minor inflammation), the likelihood of acute rupture or erosion is substantially reduced. These lesions can certainly cause angina and ischaemia and may require treatment for symptom relief or to prevent progressive obstruction; however, they are not the primary drivers of sudden, unexpected MI in the way VPs are.

Finally, in patients with no VP detected and non-significant stenosis (VP-/stenosis−), the MI risk is only about 2-fold higher than in individuals with completely normal coronaries. This group likely reflects early or stable atherosclerosis with relatively preserved endothelial function and no highly unstable plaque substrate. Although the risk is not zero and still warrants risk-factor modification, it is dramatically lower than in the VP-positive groups.

Taken together, these four profiles convey several key messages:

1. VP is the main "engine" of MI risk. Whether there is a tight narrowing, VP+ lesions carry a very high risk and require proactive strategies for detection and stabilisation.
2. Stenosis without vulnerability is mainly a problem of flow, not rupture. It may cause angina and ischaemia, but its contribution to sudden MI is much smaller unless biological instability is also present.
3. Current paradigms that prioritise stenosis severity and fractional flow reserve (FFR) alone will systematically underestimate risk in patients with non-flow-limiting but VPs.
4. A vulnerability-guided approach reorders our priorities: first, identify and stabilise VP; second, address haemodynamically significant disease, particularly when both vulnerability and stenosis coexist.

In practical terms, this framework justifies a shift from a purely lumen-centric model ("How tight is the stenosis?") to a plaque- and biology-centric model ("How vulnerable is this plaque, and what is the global burden of vulnerability in this patient?"). It supports the use of advanced imaging (e.g., CTCA, IVUS, OCT) and aggressive systemic therapy (lipid-lowering, inflammation control, WFPBD, lifestyle medicine) aimed at modifying vulnerability rather than just relieving obstruction [36-40]. Ultimately, the table formalises what our COVID-19 experience made impossible to ignore: to prevent MI and death, we must treat the VP, not just the narrow lumen.

Formed to Innovate—and to Resist a Harmful Status Quo

My training taught me not only how to perform procedures, but also how to question paradigms. I was educated in systems where data and pathophysiology, not seniority or tradition, had the final word. I learned that medicine advances when someone is willing to ask: What if our current assumptions are wrong—or at least incomplete?

During COVID-19, the dominant paradigm in many places remained stenosis- and ischaemia-centric:

- treat only lesions with ≥70% narrowing,
- intervene when the fractional flow reserve (FFR) is positive,
- assume that "non-obstructive" lesions plus optimal medical therapy (OMT) are enough.

But decades of data on VP and my own experience told a different story—especially in a hyperinflammatory, hypercoagulable pandemic environment [36, 41-46]. I felt obliged to act on the biological reality I saw, not just on the cultural comfort of an outdated algorithm.

When we first committed to a vulnerability-guided strategy in early 2020, the only primary prospective evidence we had for the prognostic importance of plaque vulnerability was the original PROSPECT study [47-51]. It showed that non-culprit thin cap fibroatheromas and other high-risk features predicted future events, but it did not yet offer a clear interventional roadmap; treating such lesions was considered speculative, even controversial. Only later did PROSPECT ABSORB [36,52,53], PREVENT [54-57], and DEBuT-LRP [36,37,56-59] emerge, providing additional evidence that identifying and, in selected cases, intervening on HRPs could reduce events. In retrospect, these studies did not initiate our approach; they validated and strengthened the direction we had already taken in the Cath lab. What some perceived as disobedience to the status quo was, in fact, an early alignment with where the science of VP was already heading.

From "Stepchild" to Front-Line Contributor

Professionally, I have often felt like a "stepchild" in my own cardiology community. This was not a neutral or random experience. In my view, it stemmed from a pattern of discrimination [60-63], like-and-dislike, nepotism, and, at times, jealousy toward those whose clinical outcomes and innovations speak for themselves. I was denied additional certification, rarely invited to national or international scientific stages, and frequently misinterpreted or dismissed whenever I tried to present data on VP and lifestyle medicine.

Paradoxically, this marginalisation became a source of inner strength during COVID-19. If I could not rely on institutional favour, I could at least rely on my conscience and my data. And when many senior figures chose to self-isolate and minimise exposure, my team and I felt a deep responsibility:

If the "stepchild" is the one still standing at the bedside and in the Cath lab, then let the stepchild do the work that needs to be done.

In a way, it is like making a film. A devoted biological child caring for loving parents is beautiful, but not unusual; it is what everyone expects. The story that truly deserves to be filmed is when a stepchild—overlooked, doubted, even mistreated—chooses to care for his stepparents with extraordinary devotion. That reversal of expectation is what makes the narrative powerful.

In the same way, the desire to transform discrimination into contribution—to show that the one treated as an outsider could be the one who protects patients in a time of crisis—became a powerful motivator for me.

An Indonesian Cardiologist, for Indonesian Patients

Another motivation was national: I wanted to prove that there are Indonesian cardiologists whose thinking and practice are not inferior to "world-class" centres abroad.

For years, many of our patients have flown overseas for cardiac care [64-67], sometimes spending their life savings. I believe that if we can apply cutting-edge concepts—VP assessment, advanced imaging, WFPBD, metabolic control, and selected preventive interventions—we can give our own people a standard of care that they no longer need to seek elsewhere.

COVID-19 was an opportunity to show that Indonesia is not condemned to be a passive consumer of foreign guidelines, but can be a generator of innovative, context-specific solutions.

The Moral Weight of Knowing How to Do Good

At a deeper level, my decision was grounded in a simple ethical rule:

If I know how to help, and I consciously refuse to help, I am not neutral—I am complicit.

By the time COVID-19 peaked, I already knew that:

- specific plaque morphologies are far more likely to rupture,
- WFPBD and lifestyle interventions can dramatically improve risk profiles,
- NO, inflammation, and microbiota are modifiable,
- and imaging can identify HRPs before they kill.

Knowing all this, the idea of sitting at home, or practicing only the safest, most conservative version of cardiology, became morally impossible for me. I would have felt I was turning my back not only on my patients, but on the very knowledge I had been given.

Thinking Beyond the Pandemic: Legacy for Nation and Profession

I did not see COVID-19 as a temporary storm to survive and then forget. I saw it as a defining chapter—for my career, my hospital, and my country's cardiology community [68]. I wanted to emerge from it with more than just memories. I wanted to emerge with:

- documented evidence that a vulnerability-guided, lifestyle-anchored approach can protect high-risk patients, even in a pandemic,
- a narrative that shows future generations of doctors what it means to stand firm when it would have been easier to step back,
- and a practical template for how to respond when—not if—the next pandemic or crisis arrives.

The concept of legacy mattered to me: not fame, but a demonstrable record that in one hospital, in one difficult era, people tried to do something more than the minimum.

A Living Example for Younger Doctors—and for My Own Family

Every consultant knows that our trainees watch more than we realize. They notice whether we show up when patients are afraid, whether we scrub in when the risk is higher, whether we hide behind guidelines or think beyond them. They will shape their own practice not only based on what we say, but on what we do.

I wanted my younger colleagues, my children and grandchildren, my students and friends, to see that it is possible to combine high-level science with high-level courage and compassion. A doctor can be technically excellent and yet still willing to sacrifice comfort, privacy, and personal safety when patients need it.

In that sense, my time in the COVID-19 Cath lab was not only about today's patients. It was about the kind of doctors—and people—I hope the next generation will become.

Leaving a Digital Trail That Points Beyond Myself

One reason I have been deliberate about publishing our experience, writing, and documenting outcomes is that I see the digital world as a kind of "modern archive." Long after we are gone, our articles, data, and reflections will still be accessible [69-71].

I hope that when others read them, they will not only see my name or my hospital, but also see evidence that scientific talent and clinical skill can be used in a way that points beyond ego, to something larger: truth, compassion, and, in my own belief, the God who entrusted those talents in the first place.

Building a Playbook for Future Pandemics

From a research perspective, every decision we made during COVID-19 was also a hypothesis. We hypothesized that:

- detecting VP early,
- combining interventional strategies with WFPBD and aggressive risk factor modification,
- and treating moderate lesions in a highly pro-thrombotic

milieu,

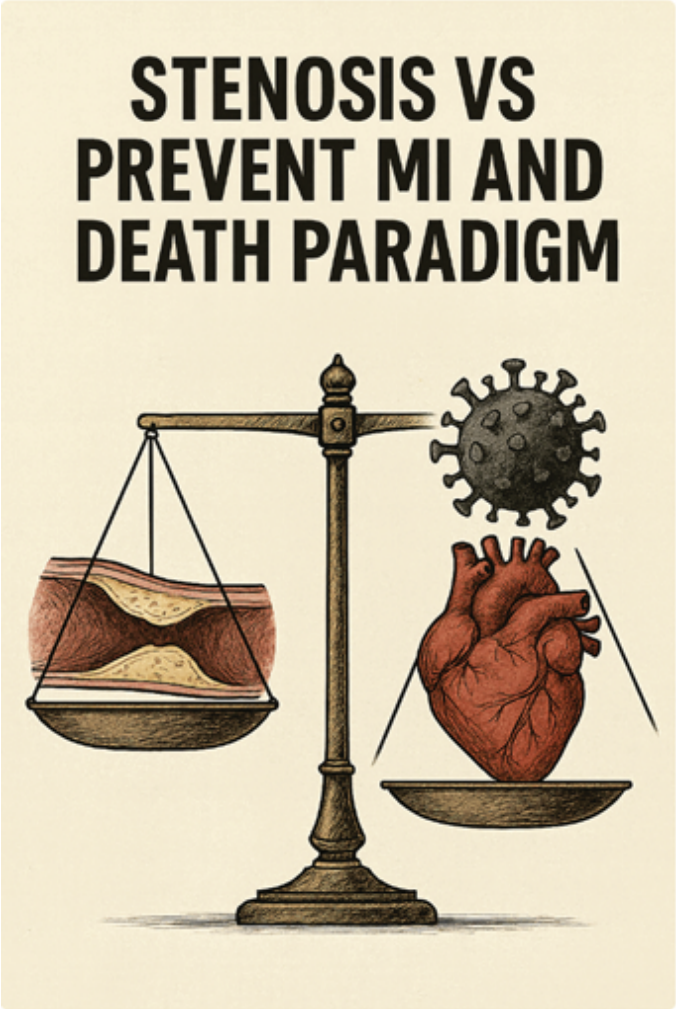
could lower MI and death rates in ways standard care could not.

We tested this hypothesis not in a controlled trial, but in real life, under the pressure of a pandemic. By collecting data, structuring observations, and publishing our results, we have begun to create a playbook that may be useful when the next global health crisis hits [72-75]—whether driven by another virus or by some other destabilizing factor.

A Long View: Beyond Earthly Metrics

Finally, I must be honest about a spiritual dimension. For me, medicine is not only a profession; it is also an act of worship. Every time a patient avoids an infarct, every time a family member can take a loved one home instead of arranging a funeral, I experience that as something sacred.

I believe there is such a thing as "treasure in heaven"—a form of reward that does not appear on curriculum vitae (CV), h-index, or award plaques. During COVID-19, I wanted my decisions to matter not only in the eyes of journals and colleagues, but also in that unseen ledger.



Picture 1. *Stenosis vs Preventing MI and Death: Competing Paradigms in Interventional Cardiology*

Limitations

This perspective is based on a single-center, non-randomized experience from a hospital with a strong pre-existing culture of lifestyle medicine and VP-focused intervention. Our cohort reflects patients who agreed to intensive dietary and risk-factor modification, which may limit generalizability to centers without similar infrastructure or patient engagement. The apparent zero-mortality outcome in 1,750 elderly COVID-19 patients should therefore be interpreted as hypothesis-generating and illustrative, not as definitive proof of a specific protocol. Nonetheless, the biological rationale and convergent evidence from pathology, imaging, and preventive cardiology suggest that a vulnerability-guided, lifestyle-anchored strategy deserves systematic evaluation in future trials.

Conclusion

When the Storm Exposes What We Really Believe

The COVID-19 pandemic forced every cardiologist to answer a brutal question:

Will I practice the safest, narrowest version of cardiology, or will I confront the biology of VP directly in a pro-thrombotic, pro-inflammatory world, preventing MI and saving lives? (Picture 1)

For me, staying in the Cath lab and aggressively preventing MI and sudden death in high-risk patients was not an act of reckless heroism. It was the inevitable consequence of twelve convictions that refused to be silenced: love of neighbour, seriousness about the physician's oath, lived experience with VP and lifestyle medicine, a training that honours innovation over inertia, a determination to turn discrimination into contribution, a commitment to Indonesian patients, a moral duty to act on what I know, a desire for legacy, a wish to model courage for younger doctors and my own family, the intention to leave a meaningful digital record, a research drive to prepare for the next pandemic, and a spiritual hope in rewards that no citation index can measure.

COVID-19 exposed a hard truth: guidelines are written for average conditions, not for extraordinary storms. In a storm, it is not only our algorithms that are tested, but our character. If we, as cardiologists, genuinely believe that VP can be detected, stabilized, and in some cases mechanically "disarmed"—and that lifestyle and metabolic interventions can radically shift risk—then the next crisis will demand more than tidy compliance. It will demand clinicians who are willing to stand precisely where science, ethics, and courage intersect, even when that stand is uncomfortable, unpopular, or personally costly.

In that space, preventive cardiology stops being a lecture slide or a slogan. It becomes a way of life—for our patients, for our profession, and for some of us, before God. The real question is not whether VP-guided, lifestyle-anchored prevention works. The real question is: when the next storm comes, will we be willing to live what we say we believe?.

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