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Validity of Angiography-Derived Microvascular Resistance in Hypertrophic Cardiomyopathy: A Head-to- Head Comparison With Pressure-Wire IMR

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Abstract

Purpose: Angiography-derived indices of microvascular resistance offer wire-free assessment from routine angiograms, but their validity in hypertrophic cardiomyopathy (HCM) remains uncertain. We evaluated correlation, agreement, diagnostic performance, and reproducibility of angiography-derived IMR versus invasive pressure-wire IMR in HCM.
Methods: Consecutive HCM patients without obstructive coronary artery disease underwent invasive left anterior descending artery IMR during adenosine hyperaemia. Angiography-derived IMR was computed offline using three-dimensional reconstruction, QFR, TIMI frame count, and pressure assumptions. Analyses included Spearman correlation, Bland–Altman agreement, ROC analysis, and intraclass correlation coefficients.

Results: Thirty patients were included. Invasive IMR was ≥25 mmHg × s in 20 patients (67%). Angiography-derived IMR correlated moderately with invasive IMR (Spearman r = 0.53, 95% CI 0.21–0.75; P = 0.003). Bland–Altman analysis showed a mean bias of 4.1 mmHg × s, with 95% limits of agreement from −28.4 to 36.6 mmHg × s. Angiography-derived IMR identified invasive IMR ≥25 mmHg × s with an AUC of 0.83 (95% CI 0.65–0.96). The optimal cutoff was 26, yielding 80% sensitivity (95% CI 56–94%) and 80% specificity (95% CI 44–97%). Intra-observer reproducibility was high and inter-observer reproducibility moderate.

Conclusion: In HCM, angiography-derived IMR showed moderate correlation and acceptable cohort-level discrimination but wide agreement limits, indicating limited individual-level interchangeability. HCM-specific refinement is required before replacing invasive microvascular assessment.

Introduction

Coronary microvascular dysfunction (CMD)-related ischaemia plays a major role in the pathophysiology and prognosis of hypertrophic cardiomyopathy (HCM). Myocardial ischaemia in HCM has been associated with replacement fibrosis, adverse remodelling, heart failure progression, ventricular arrhythmias, and worse clinical outcomes [1, 2]. Despite this prognostic relevance, microvascular assessment is not routinely performed in daily practice, partly because current cardiomyopathy guidelines do not provide a standardized invasive or non-invasive testing pathway for this purpose [3].

Microvascular dysfunction can be assessed invasively using the index of microcirculatory resistance (IMR), a pressure-wire based parameter intended to reflect coronary microvascular resistance during maximal hyperaemia [4]. In contrast to coronary flow reserve (CFR), which integrates both epicardial and microvascular components of coronary circulation, IMR is considered more specific to the microcirculation. Invasive IMR assessment in HCM is feasible, but previous studies remain limited and have suggested that abnormal IMR may be associated with more advanced myocardial abnormalities [5, 6]. However, invasive assessment is time-consuming, costly, and requires pressure-temperature guidewires and pharmacologically induced hyperaemia. Over the past few years, functional coronary angiography methods have been developed to estimate microvascular resistance without a pressure

wire. Angiography-derived IMR combines vessel geometry, pressure assumptions, quantitative flow ratio (QFR), and contrast-flow velocity modelling [7]. These methods have shown promising results in coronary artery disease populations, but their applicability to HCM is uncertain because HCM is characterized by specific microvascular remodelling, abnormal vasodilator reserve, increased myocardial mass, and usually non-obstructive epicardial coronary arteries. Accordingly, the aim of the present study was to evaluate the correlation, agreement, diagnostic performance, and reproducibility of angiography-derived IMR compared with invasive pressure-wire IMR in patients with HCM.

Methods

Study population

We identified consecutive patients with HCM from a single-centre registry between 17 June 2019 and 2 February 2025, who underwent clinically indicated invasive coronary angiography for chest pain and/or positive myocardial perfusion scintigraphy. The reference diagnosis of HCM was adjudicated by a multidisciplinary cardiomyopathy team including cardiologists, radiologists, nuclear medicine physicians, internists, and geneticists. Final adjudication used all available follow-up information, including clinical data, laboratory investigations, repeat imaging when appropriate, family history, and genetic testing, in accordance with contemporary guideline recommendations [3].

Patients were not eligible in the presence of haemodynamic instability, acute or chronic myocarditis, hypertensive cardiomyopathy, severe valvular heart disease, infiltrative myocardial disease, or obstructive coronary artery disease. Obstructive coronary artery disease was defined as a coronary stenosis ≥ 50% and/or FFR < 0.80. Patients with previous PCI or CABG were also excluded, as were patients with contraindications to invasive coronary angiography, particularly severe renal impairment defined as MDRD-estimated glomerular filtration rate < 30 mL/min/1.73 m².

The study was registered at ClinicalTrials.gov (NCT03479580), conducted in accordance with the Declaration of Helsinki, and approved by the West I Committee for the Protection of Persons (No. 2017T3-14). All patients provided informed consent.

Pressure-wire IMR

Coronary angiography was performed via radial access using 5-French catheters. IMR was measured in the left anterior descending artery using a pressure-temperature sensor guidewire during maximal hyperaemia induced by intravenous adenosine (140 µg/kg/min), according to the principle described [4]. After calibration and equalization, the wire was advanced to the distal segment of the target vessel. Intracoronary nitrates (100 or 200 µg) were administered before measurement. Resting and hyperaemic mean transit times were obtained from triplicate 3-mL room-temperature saline injections. Hyperaemic mean aortic pressure, distal coronary pressure, and hyperaemic mean transit time were recorded, and IMR was calculated as distal coronary pressure multiplied by hyperaemic mean transit time. An IMR ≥ 25

mmHg × s was considered indicative of coronary microvascular dysfunction, consistent with the threshold commonly used in INOCA [8].

Angiography-derived IMR

Angiography-derived IMR was computed offline from routine coronary angiograms after intracoronary nitrate administration using QAngio XA 3D software (QFR version 2.1 Research Edition, Medis Medical Imaging, Leiden, the Netherlands). Two projections at least 25° apart were selected, and end-diastolic frames with optimal contrast opacification and minimal overlap or foreshortening were retained. Analyses were performed blinded to clinical characteristics and invasive IMR results.

The workflow included vessel contouring, three-dimensional reconstruction, TIMI frame count analysis, QFR computation, and incorporation of resting mean aortic pressure. Angio-IMR was calculated using the formula: Angio-IMR = estimated hyperaemic aortic pressure × QFR × vessel length / estimated hyperaemic flow velocity. Resting mean aortic pressure was used as input, hyperaemic aortic pressure was estimated as resting aortic pressure reduced by 10%, and hyperaemic flow velocity was derived from resting frame count using a quadratic model, as previously described for functional angiography approaches [7]. All image processing was performed blinded to clinical outcomes. All measurements were performed by two investigators (LB and GB), both of whom had completed formal training in angiography-derived IMR analysis.

Statistical analysis

Continuous variables are presented as mean ± standard deviation when normally distributed and as median [interquartile range] otherwise. Categorical variables are reported as counts and percentages. Normality was assessed visually and using the Shapiro-Wilk test. Associations between invasive IMR and angiography-derived IMR were assessed using Spearman correlation coefficients. Agreement was evaluated by Bland-Altman analysis. ROC curve analysis was used to assess the ability of angiography-derived IMR to identify invasive IMR ≥ 25 mmHg × s, and the optimal cutoff was determined using the Youden index. Furthermore, to assess the variability of the measures, patients were evaluated twice by the same observer for intra-observer variability, and by two different observers for interobserver variability. Variability was quantified computing the intraclass correlation coefficient (ICC). Inter-observer reliability for measurement of angiography-derived IMR was assessed by using two-way random single-measure ICC analysis. Intra-observer reliability was assessed by using one-way random two-measures ICC analysis. A two-tailed P value < 0.05 was considered statistically significant.

Results

Study population

Thirty patients were included. Mean age was 60.8 ± 10.8 years, 22 patients (73%) were men, and mean body mass index was 28.6 ± 5.2 kg/m². Left ventricular ejection fraction was preserved overall, NT-proBNP was frequently elevated, and late gadolinium enhancement was present in 22/28 patients (79%). Baseline clinical, biological, echocardiographic, CMR, and invasive physiological characteristics are summarized in Table 1.

Table 1
Baseline characteristics of the study population

Variable	Overall cohort (n = 30)
Age, years	60.8 ± 10.8
Male sex	22/30 (73%)
Body mass index, kg/m²	28.6 ± 5.2
Current or former smoking	3/30 (10%)
Diabetes mellitus	9/30 (30%)
Hypertension	14/30 (47%)
Dyslipidaemia	8/30 (27%)
Family history of HCM	9/30 (30%)
Previous stroke	2/30 (7%)
NYHA class I	12/30 (40%)
NYHA class II	16/30 (53%)
NYHA class III	2/30 (7%)
Heart rate, bpm	63.3 ± 12.5
NT-proBNP, ng/L	690 [214–997]
Creatinine, µmol/L	85.1 ± 19.4
Echocardiographic LVEF, %	64.7 ± 6.8
Maximal LV wall thickness by echocardiography, mm	18.5 ± 4.6
LV outflow tract gradient, mmHg	0 [0–30]
TAPSE, mm	23.2 ± 4.5
E/e' lateral	8.6 [6.8–11.6]
Systolic pulmonary artery pressure, mmHg	26.7 ± 10.7
Left atrial volume index by echocardiography, mL/m²	41 [29–58]
CMR LVEF, %	61.3 ± 9.0
CMR LV end-diastolic volume index, mL/m²	68.9 ± 17.5

Values are mean ± standard deviation, median [interquartile range], or n/N (%). CMR, cardiac magnetic resonance; CFR, coronary flow reserve; FFR, fractional flow reserve; HCM, hypertrophic cardiomyopathy; IMR, index of microcirculatory resistance; LV, left ventricular; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; TAPSE, tricuspid annular plane systolic excursion.

Variable	Overall cohort (n = 30)
CMR LV mass index, g/m²	74.8 ± 22.8
CMR maximal LV wall thickness, mm	18.9 ± 4.1
CMR left atrial volume index, mL/m²	54.0 ± 20.6
No late gadolinium enhancement	6/28 (21%)
Late gadolinium enhancement < 15%	13/28 (46%)
Late gadolinium enhancement ≥ 15%	9/28 (32%)
Systolic blood pressure, mmHg	139.9 ± 24.6
Diastolic blood pressure, mmHg	77.8 ± 13.6
Resting mean blood pressure, mmHg	103.4 ± 18.3
Hyperaemic mean blood pressure, mmHg	97.3 ± 21.3
FFR	0.93 [0.90–0.95]
CFR	1.9 [1.6–2.7]
Invasive IMR, mmHg × s	27.0 [23.2–35.8]
Invasive IMR ≥ 25 mmHg × s	20/30 (67%)
Angiography-derived IMR	28.9 ± 10.7
Angiography-derived IMR ≥ 25	19/30 (63%)

Values are mean ± standard deviation, median [interquartile range], or n/N (%). CMR, cardiac magnetic resonance; CFR, coronary flow reserve; FFR, fractional flow reserve; HCM, hypertrophic cardiomyopathy; IMR, index of microcirculatory resistance; LV, left ventricular; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; TAPSE, tricuspid annular plane systolic excursion.

Invasive and angiography-derived IMR

Invasive IMR was abnormal (≥ 25 mmHg × s) in 20 of 30 patients (67%). Median invasive IMR was 27.0 [23.2–35.8] mmHg × s. Angiography-derived IMR was ≥ 25 in 19 patients (63%), with a mean value of 28.9 ± 10.7 mmHg × s. Invasive IMR and angiography-derived IMR showed a moderate positive correlation (Spearman r = 0.53, 95% CI 0.21–0.75; P = 0.003) (Fig. 1).

On Bland–Altman analysis, the mean difference between invasive IMR and angiography-derived IMR was 4.1 mmHg × s, with 95% limits of agreement ranging from − 28.4 to 36.6 mmHg × s, indicating wide individual-level variability (Fig. 2).

The ability of angiography-derived IMR to discriminate invasive IMR ≥ 25 mmHg × s was good at the cohort level, with an AUC of 0.83 (95% CI 0.65–0.96). The optimal Youden cutoff was 26, yielding 80% sensitivity (95% CI 56–94%) and 80% specificity (95% CI 44–97%) (Fig. 3).

Reproducibility

Angiography-derived IMR showed high intra-observer reproducibility in the final dataset (ICC 0.93; bootstrap 95% CI 0.76–1.00). Inter-observer reproducibility was more moderate (ICC 0.56; bootstrap 95% CI 0.29–0.75).

Discussion

The main finding of this study is that, in patients with HCM, angiography-derived IMR showed a statistically significant but only moderate correlation with invasive pressure-wire IMR. Although ROC analysis suggested acceptable cohort-level discrimination of invasive IMR ≥ 25 mmHg × s, Bland–Altman analysis demonstrated wide limits of agreement. Thus, the currently available angiography-derived formula may be useful as a population-level signal but does not yet appear sufficiently reliable to replace invasive IMR for individual patient assessment in HCM.

This limited agreement is physiologically plausible. Current angio-IMR formulas were developed and validated predominantly in coronary artery disease populations [7]. They rely on several assumptions, including estimation of hyperaemic aortic pressure from resting aortic pressure, derivation of coronary flow from angiographic contrast transit, and extrapolation of hyperaemic flow velocity from resting contrast-flow analysis. These assumptions may be less robust in HCM, where vasodilator reserve is often impaired and the haemodynamic response to adenosine may differ from that observed in patients with stable coronary artery disease.

A first source of error is the estimation of hyperaemic pressure. In several angio-IMR models, hyperaemic aortic pressure is approximated as resting pressure reduced by 10% [9]. This correction may be valid at a population level in selected coronary artery disease cohorts, but it may not apply uniformly to patients with HCM, who frequently have altered diastolic function, variable loading conditions, dynamic obstruction in some cases, and abnormal microvascular vasodilator response. In our HCM cohort, the observed decrease in mean arterial pressure from rest to hyperaemia was only 5.7%, rather than the fixed 10% reduction assumed in several angio-IMR models, suggesting that this correction may introduce error in this population.

A second limitation relates to QFR and contrast-flow modelling. Although angiography-derived fractional flow indices have shown good diagnostic performance compared with pressure-derived FFR in selected coronary artery disease populations [10], this performance relies on modelling assumptions that may not be transferable to HCM. In the FAVOR developmental framework, contrast-flow QFR estimated hyperaemic flow velocity from resting contrast-flow velocity using a quadratic function derived from

training datasets of patients with coronary artery disease [11]. Therefore, hyperaemic flow is not directly measured but mathematically inferred from resting angiographic contrast transit. This is particularly relevant in HCM, where microvascular remodelling, extravascular compressive forces, impaired arteriolar vasodilation, abnormal diastolic perfusion, and increased myocardial mass may dissociate resting contrast-flow dynamics from true hyperaemic microvascular conductance. Thus, an equation calibrated in patients with epicardial coronary artery disease may not accurately capture the hyperaemic flow response in a disease primarily characterized by microvascular dysfunction.

In addition, independent evaluations of angiography-derived fractional flow indices have reported lower correlations than initial validation studies [12]. Resting TIMI frame count is also an imperfect surrogate for true coronary flow velocity, and slow contrast flow is not equivalent to microvascular dysfunction [13]. These limitations are important for angio-IMR because the calculation of microvascular resistance may amplify small errors in both pressure and flow estimation. Therefore, the limited agreement observed in our study may reflect not only measurement variability but also a physiological mismatch between algorithmic assumptions developed in epicardial coronary artery disease and the specific microvascular haemodynamics of HCM.

Overall, even in standard coronary artery disease populations, the validity of angiography-derived IMR has recently been challenged. Independent core-laboratory analyses have reported only modest correlations with invasive IMR, substantially lower than those observed in initial validation studies, raising concerns about the robustness and generalizability of these models beyond their original development cohorts [14, 15]. In HCM, where epicardial vessels are often angiographically normal but microvascular reserve is impaired, these modelling limitations may be amplified.

The interpretation of invasive IMR itself may also require caution in HCM. IMR is influenced by the amount of subtended myocardium, and prior work suggests that a larger myocardial territory may be associated with lower measured IMR for a given degree of microvascular disease [16]. Because the LAD in HCM may subtend an enlarged hypertrophied myocardial mass, a universal IMR threshold of 25 mmHg × s may not optimally classify microvascular dysfunction in this disease.

These findings do not negate the clinical potential of angiography-derived microvascular indexes in HCM. Recent prognostic data suggest that elevated angiography-derived microvascular resistance is associated with adverse outcomes in HCM, including major cardiovascular events and cardiac death [17]. However, prognostic enrichment does not necessarily imply interchangeability with invasive IMR. The present results instead suggest that angiography-derived indexes should be refined specifically for HCM before being used as direct substitutes for invasive measurements.

Future work should aim to develop HCM-specific angiography-derived models. Such models should incorporate disease-specific physiology, including impaired vasodilator reserve, altered hyperaemic pressure response, abnormal diastolic perfusion, and myocardial mass subtended by the analyzed vessel. Microvascular resistance reserve may also be particularly relevant in HCM because it has been proposed as a more specific index of microvascular function, less dependent on autoregulation and

subtended myocardial mass [18]. Artificial intelligence-based approaches, similar to recent fully automated CT-FFR techniques, could also help adapt microvascular modelling to disease-specific phenotypes [19].

Limitations

This study has several limitations. First, it was a single-centre study with a limited sample size, reflecting the relative rarity of HCM patients undergoing both invasive microvascular testing and analyzable coronary angiography. Second, the HCM population was phenotypically heterogeneous. Third, angiography-derived IMR was calculated using a formula developed in non-HCM populations. Fourth, the study was not powered to assess clinical outcomes. Finally, the use of an IMR threshold of 25 mmHg × s, although standard in INOCA, may not be optimal in HCM and requires dedicated validation.

Conclusion

In patients with HCM, angiography-derived IMR is moderately correlated with invasive pressure-wire IMR but shows wide limits of agreement. These findings suggest that currently available angiography-derived formulas require HCM-specific refinement before they can replace invasive microvascular assessment in individual patients. Nevertheless, these tools remain promising for broader, less invasive assessment of coronary microvascular dysfunction and risk stratification in HCM.

Declarations

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Competing interests
Dr. Barone-Rochette has received research grants from Merck Sharp and Dohme, Abbott Vascular, and Pfizer, and consulting fees from Bayer, General Electric, Boehringer Ingelheim, Medis Medical Imaging, Abbott Vascular, Novo Nordisk, Sanofi, Novartis, and Amgen. The remaining authors have no relevant financial or non-financial interests to disclose.

Ethics approval
This study was conducted in accordance with the Declaration of Helsinki and was approved by the West I Committee for the Protection of Persons (No. 2017T3-14).

Consent to participate
All patients provided informed consent.

Consent for publication
Not applicable.

Data availability

The data underlying this article will be shared on reasonable request to the corresponding author.

Clinical trial registration

ClinicalTrials.gov identifier: NCT03479580.

Author contributions

Gilles Barone-Rochette conceived and designed the study. Louise Brunel, Nicolas Pioch, Benoit Caullery, Ikram El Marzouki, and Gilles Barone-Rochette contributed to patient inclusion and/or data acquisition. Louise Brunel, Nicolas Pioch, and Gilles Barone-Rochette performed invasive IMR and angiography-derived IMR measurements. Louise Brunel and Gilles Barone-Rochette performed the statistical analyses. Louise Brunel drafted the first version of the manuscript. All authors contributed to data interpretation and critically revised the manuscript for important intellectual content. Gilles Barone-Rochette supervised the study. All authors read and approved the final manuscript.

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Figures

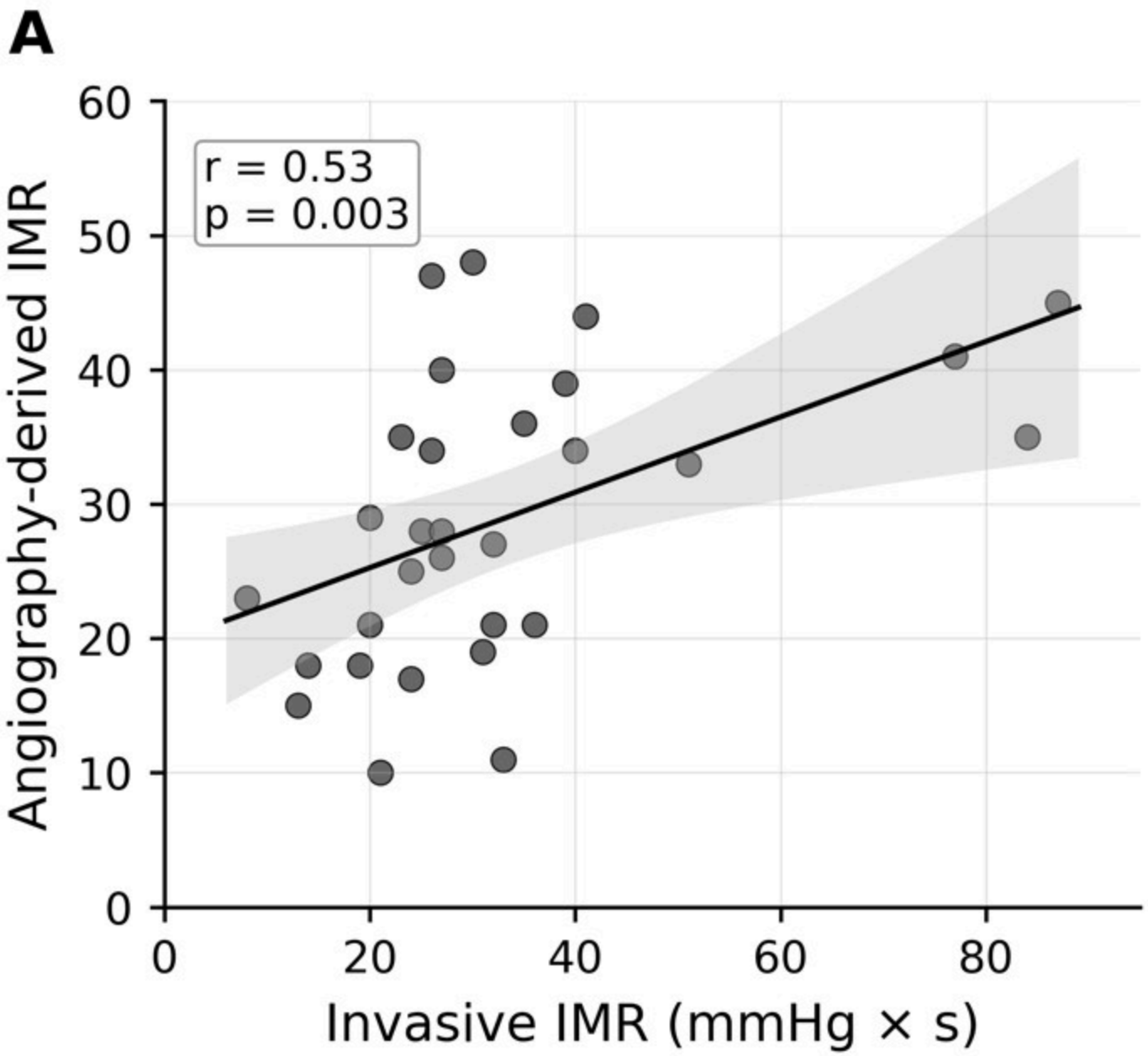


Figure 1

Relationship between invasive pressure-wire IMR and angiography-derived IMR in patients with hypertrophic cardiomyopathy.

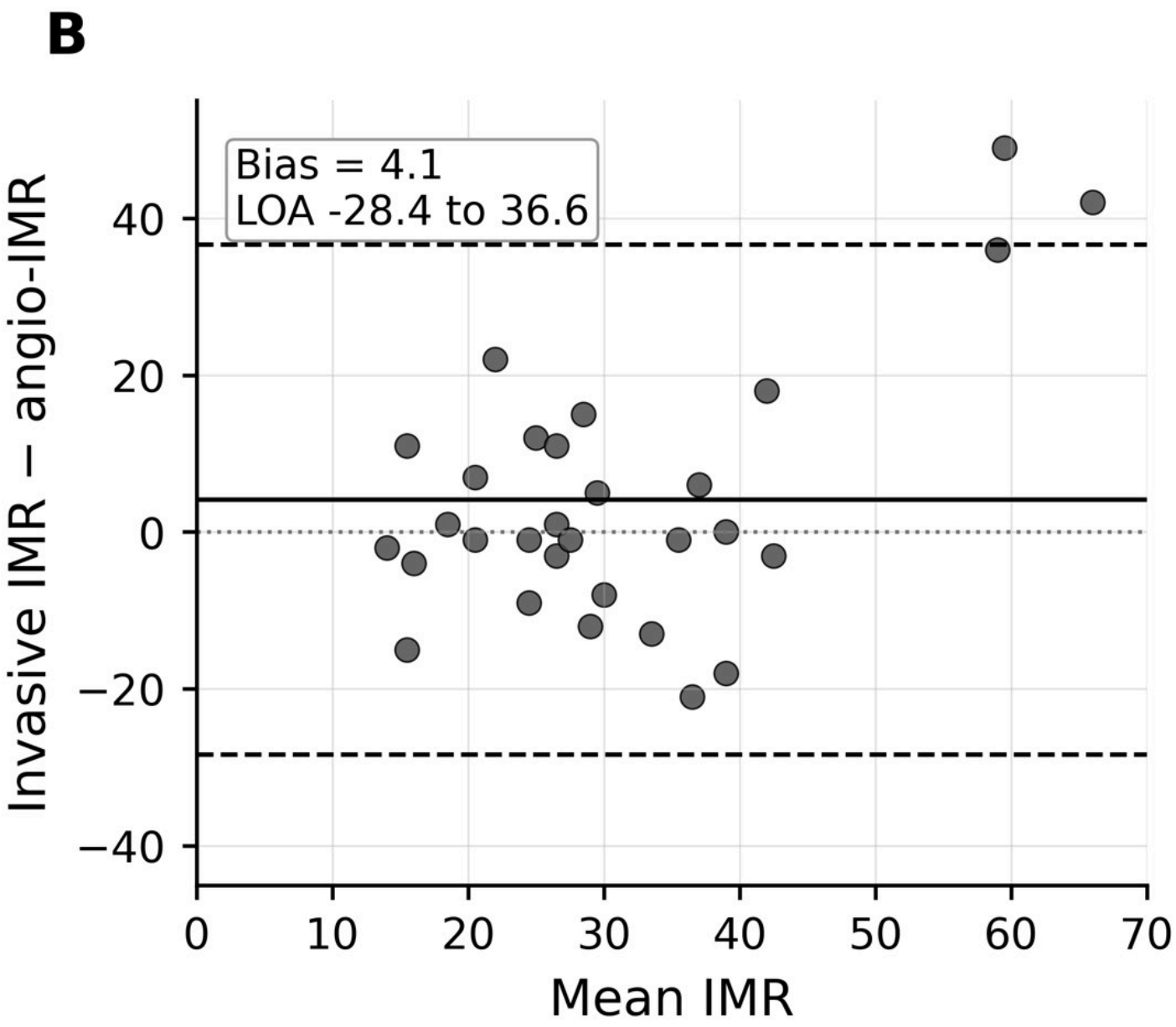


Figure 2

Bland-Altman analysis comparing invasive pressure-wire IMR and angiography-derived IMR.

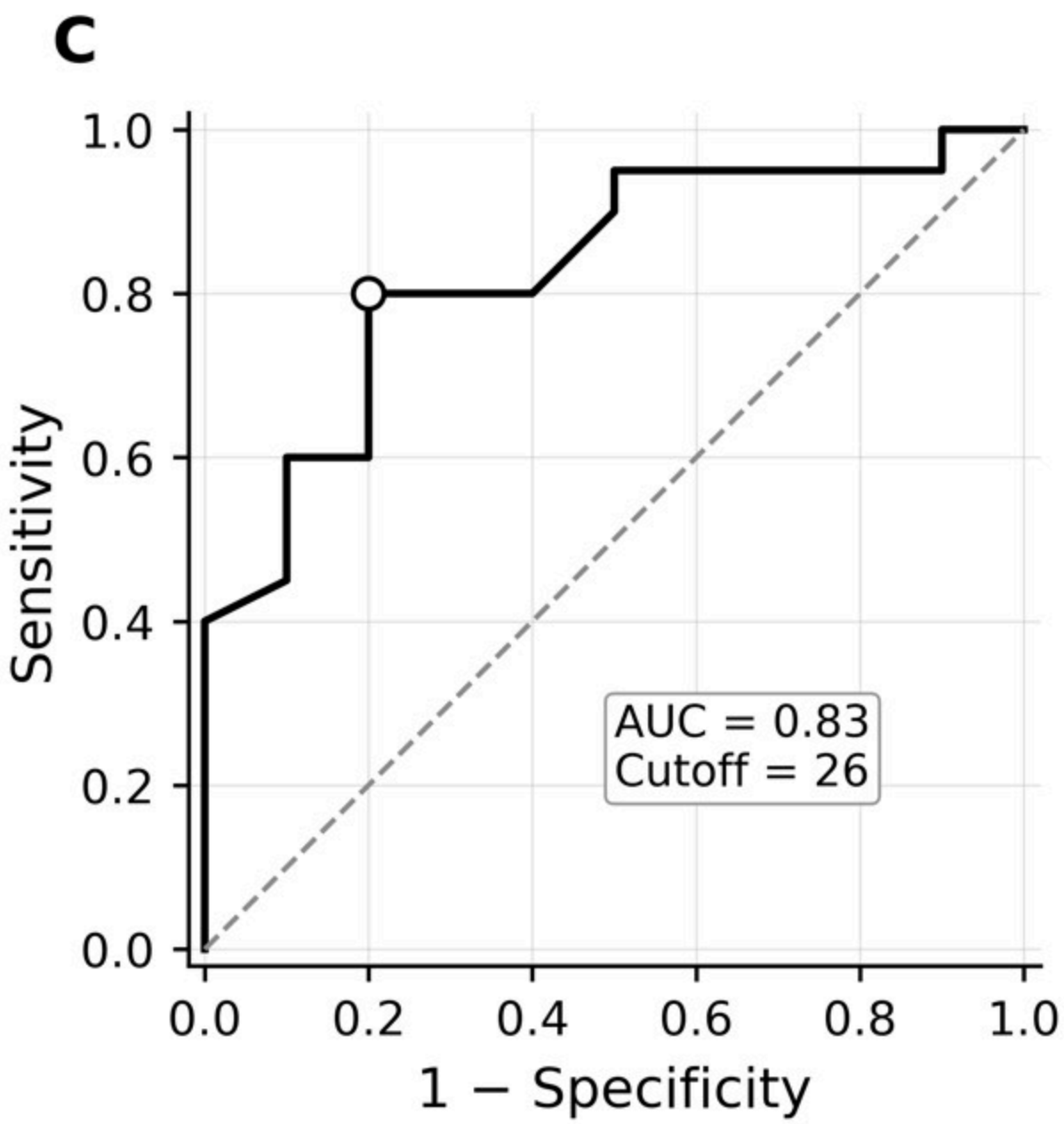


Figure 3

ROC curve for angiography-derived IMR to identify invasive IMR ≥25 mmHg × s.

Supplementary Files

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