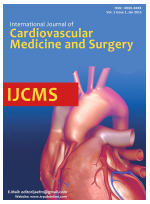
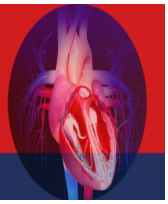
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Deep learning-based coronary CT angiography plaque phenotyping for prediction of vessel-specific ischemia

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Abstract

Background: Although coronary CT angiography identifies a coronary stenosis, anatomical stenosis correlates moderately with the identification of ischemia to a specific vessel, which is the current reference standard and can only be determined by invasive fractional flow reserve. Phenotype of atherosclerotic plaque is another aortic parameter of ischemia that can be automatically quantified using deep learning. This was an assessment of a Deep-Learning Plaque Phenotyping (DLPP) model for predicting vessel specific ischemia. **Methods:** This is a retrospective diagnostic accuracy study, in which 787 vessels of 384 patients who underwent CTA and FFR were analysed. Phenotype of plaques was quantified using a deep learning model and the presence of vessel-specific ischemia (defined as fractional flow reserve at or below 0.80) was determined by using a classifier. DeLong test was used to compare discrimination vs stenosis alone, and calibration test performed. **Results:** 40% of vessels had ischemia. The deep-learning plaque model discriminated ischemia at an area under the curve of 0.86 (95% CI 0.81 to 0.91) with a sensitivity of 85%, a specificity of 77% and negative predictive value of 90%, significantly better than stenosis alone (at an area under the curve of 0.73, DeLong p<0.001). The strongest predictor was low-attenuation plaque volume and the model had a well calability (Hosmer-Lemeshow p = 0.49). **Conclusion:** Phenotyping plaques using deep-learning performed better than stenosis alone to predict vessel-specific ischemia and could therefore serve as a non-invasive gateway to invasive assessment.

Keywords: *Coronary CT angiography, Deep learning, Plaque phenotyping, Fractional flow reserve, Vessel-specific ischemia, Low-attenuation plaque, Machine learning*

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1. Introduction

Coronary artery disease is typically examined by a non-invasive screening process leading to invasive coronary angiography and revascularisation, if appropriate, in certain patients. One of the problems in this pathway is that, when patients are referred on the basis of a non-invasive test for invasive angiography, many patients are referred for angiography and have no vessels that are actually ischemic, so they undergo angiography for no

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good purpose. Enhancing non-invasive test to identify, vessel-by-vessel, which lesions are truly ischemic would reduce these procedures and direct invasive evaluation and revascularisation to those patients who are most likely to benefit.

For the evaluation of suspected coronary artery disease, coronary CT angiography has emerged as the first-line non-invasive test, and has proven to be a reliable method to assess luminal stenosis. However, a stenosis on the angiogram is only an anatomical finding, and how anatomically significant this stenosis is will only partially correlate with whether or not it will actually lead to insufficient blood flow to the myocardium and thus create myocardial ischemia—the property that governs whether or not revascularisation is beneficial (Tonino *et al.*, 2009; Xaplanteris *et al.*, 2018). The benchmark for that property is the invasive fractional flow reserve during angiography—if surgery is indicated, with a value of 0.80 or 80% or less deemed a vessel-specific indicator of ischemia.

Because stenosis alone predicts ischemia imperfectly, much interest has turned to the atherosclerotic plaque itself. The composition and burden of plaque, and not only the degree of narrowing it produces, influence the physiology of the vessel, and quantitative plaque features such as the total plaque volume, the non-calcified and low-attenuation plaque volumes, the lesion length and the remodeling index have each been associated with lesion-specific ischemia (Gaur *et al.*, 2016; Nakanishi *et al.*, 2018; Yang *et al.*, 2021). Low-attenuation plaque in particular, the lipid-rich component of the plaque, has emerged as a powerful marker, being the strongest plaque predictor of myocardial infarction in a large trial (Williams *et al.*, 2020; Motoyama *et al.*, 2015).

The obstacle to using these features in practice has been that measuring them by hand is laborious and poorly reproducible. Deep learning removes that obstacle by segmenting and quantifying the plaque automatically from the CT angiogram, with a reproducibility approaching that of expert readers, so that a full plaque phenotype can be obtained rapidly for every vessel (Lin *et al.*, 2022; Meah *et al.*, 2021). This raises the possibility that a deep-learning plaque phenotype, taken together, could predict vessel-specific ischemia more accurately than the stenosis alone, and so serve as a non-invasive gatekeeper that identifies the vessels most likely to be ischemic and to warrant invasive assessment (Coenen *et al.*, 2018; Driessen *et al.*, 2019).

A parallel non-invasive approach to the same problem is the computation of fractional flow reserve from the CT angiogram itself, in which computational fluid dynamics or a machine-learning surrogate estimates the pressure drop across a lesion. This CT-derived fractional flow reserve has been validated against the invasive measurement and improves the specificity of coronary CT angiography for ischemia (Norgaard *et al.*, 2014; Benton *et al.*, 2018). It models the physiology directly, whereas plaque phenotyping predicts ischemia from the structural features of the plaque; the two approaches are complementary, and the plaque phenotype has the further advantage of describing the composition that also carries prognostic information beyond the current ischemia (Ahmadi *et al.*, 2015).

This study evaluated a deep-learning plaque phenotyping model for the prediction of vessel-specific ischemia. Using vessels in which the invasive fractional flow reserve had been measured as the reference standard, it assessed the discrimination and calibration of a model built on the deep-learning plaque phenotype, compared its discrimination with that of stenosis alone, identified the plaque features that most influenced the prediction, and examined the performance across the individual coronary arteries.

2. Materials and methods

2.1. Study design and setting

The study was a retrospective, diagnostic accuracy study in the department of cardiovascular imaging at a tertiary care hospital. Patients with both coronary CT angiography and invasive coronary angiography utilizing FFR measurement were selected, and the analysis was done on a per vessel basis. Design of the study and reporting was based on the Standards of Reporting of Diagnostic Accuracy Studies (Bossuyt *et al.*, 2015).

2.2. Patients and vessels

Patients with suspected or known coronary artery disease who had undertaken coronary CT angiography (CQCCTA) and invasive FFR in at least one of their vessels were included. Vessels were excluded due to

suboptimal vessel image quality using CT angiogram, history of prior stenting or bypass of the vessel, or no fractional flow reserve obtained for the vessel. There were 787 vessels on 384 patients analysed.

2.3. Coronary CT angiography acquisition

The coronary CT angiography was acquired and reported as per standard practice of the era, as either retrospective or prospective electrocardiographic-gated and iodinated contrast, and the CT images were reconstructed at the phase with the least motion and analysed (Leipsic *et al.*, 2014).

2.4. Deep-learning plaque phenotyping

A deep-learning system was employed to segment the coronary tree and segment the vessel lumen and wall, quantify: total plaque volume, non-calcified plaque volume, calcified plaque volume, low-attenuation plaque volume, percentage diameter stenosis, length of the lesion, remodeling index; and identify features of high-risk plaque. Low-attenuation plaque volume was defined as the volume of plaque that fell below a set threshold, which is the lipid-rich necrotic core, while the remodeling index is the ratio of the volume of the lesion area to a nearby reference area in the vessel. All quantitative features and categorical high-risk plaque designation (phenotype) used for prediction were obtained by the same extraction methods on the training and test sets.

2.5. Reference standard

The reference in-vivo test for assessing whether a vessel was ischemic was the Fractional Flow Reserve (FFR), obtained during coronary angiography with maximal hyperaemia. A fractional flow reserve of 0.80 or lower was considered to indicate ischaemia. This was done using the pressure guidewire pushed past the lesion leading to maximal hyperaemia, with the lowest stable distal coronary/aortic pressure ratio being used as the fractional flow reserve for that vessel. The reference measurement was performed and interpreted without knowledge of the deep-learning plaque phenotype, and the plaque phenotype was likewise generated without knowledge of the fractional flow reserve, so that the index test and the reference standard were assessed independently.

2.6. Model development and validation

The patients were divided at random into a training set and a held-out test set in a ratio of seven to three, with all vessels from a given patient kept within the same set to avoid leakage. A gradient-boosted classifier was trained on the deep-learning plaque phenotype to predict vessel-specific ischemia, and its probabilities were calibrated. A model based on the percentage diameter stenosis alone was trained under the same procedure for comparison, so that the two models differed only in the features available to them. The operating threshold was fixed at the value maximising the Youden index in the training set, and all performance, including discrimination, calibration and the operating characteristics, was assessed only on the held-out test set.

2.7. Statistical analysis

Discrimination was quantified by the area under the receiver operating characteristic curve with a bootstrap confidence interval, and the areas for the plaque model and for stenosis alone were compared by the DeLong test (DeLong *et al.*, 1988). Sensitivity, specificity, and the predictive values were reported at the threshold maximising the Youden index. Calibration was assessed by the Hosmer-Lemeshow test, the Brier score and a calibration plot, and the discrimination was examined separately in each coronary artery. Plaque features were compared between ischemic and non-ischemic vessels by the t test, and the contribution of each feature to the model was assessed by permutation importance.

3. Results and discussion

3.1. Patients and vessels

A total of 787 vessels from 384 patients were analysed, of which 41% were in the left anterior descending, 30% in the left circumflex and 29% in the right coronary artery. Vessel-specific ischemia, defined by an invasive fractional flow reserve at or below 0.80, was present in 40% of vessels. The characteristics of the patients and vessels are given in Table 1.

Table 1: Characteristics of the study population

Characteristic	Value
Patients, n	384
Vessels analysed, n	787
Vessels in test set, n	236
Left anterior descending, %	41%
Left circumflex, %	30%
Right coronary artery, %	29%
Vessels with ischemia (FFR ≤ 0.80), %	40%

3.2. Plaque phenotype and ischemia

The plaque phenotype differed markedly between ischemic and non-ischemic vessels. Ischemic vessels had a greater percentage diameter stenosis, larger total, non-calcified and low-attenuation plaque volumes, longer lesions, a higher remodeling index and more frequent high-risk plaque than non-ischemic vessels, as given in Table 2; the low-attenuation plaque volume, for example, was 25.3 ± 10.2 mm^3 in ischemic versus 9.7 ± 7.9 mm^3 in non-ischemic vessels, and the total plaque volume 239.2 ± 65.0 versus 142.6 ± 60.7 mm^3 (both $p<0.001$). These differences confirm, at the level of the individual vessel, that ischemia is determined not only by the degree of luminal narrowing but by the burden and composition of the plaque behind it, which is the premise on which plaque phenotyping rests. The prominence of the low-attenuation, lipid-rich component among these differences is consistent with the established role of that component as a marker of both physiological significance and clinical risk. A further point concerns how these features were obtained. Measured by hand, plaque volumes and composition are time-consuming and vary between readers, which has limited their use despite their known association with ischemia; the deep-learning system produced them automatically and reproducibly, with an agreement with expert readers reported to approach that between experts themselves, so that the phenotype used here could be generated for every vessel in routine practice rather than only in research (Cury *et al.*, 2022). The reproducibility of the input is as important as the accuracy of the model, because a prediction is only as reliable as the measurements it rests on, and an

Table 2: Deep-learning plaque phenotype in ischemic versus non-ischemic vessels

Plaque feature	Ischemic	Non-ischemic	p
% diameter stenosis	57.4 ± 15.4	43.7 ± 14.6	<0.001
Total plaque volume (mm^3)	239.2 ± 65.0	142.6 ± 60.7	<0.001
Non-calcified plaque volume (mm^3)	153.9 ± 53.8	77.0 ± 45.8	<0.001
Low-attenuation plaque volume (mm^3)	25.3 ± 10.2	9.7 ± 7.9	<0.001
Calcified plaque volume (mm^3)	85.4 ± 26.5	65.6 ± 22.4	<0.001
Lesion length (mm)	21.9 ± 5.1	15.4 ± 5.0	<0.001
Remodeling index	1.1 ± 0.1	1.0 ± 0.1	<0.001
High-risk plaque, n (%)	122 (39%)	78 (16%)	<0.001

automated phenotype removes the reader-to-reader variation that would otherwise propagate into the prediction.

3.3. Model performance and comparison with stenosis

The deep-learning plaque model discriminated vessel-specific ischemia well, with an area under the receiver operating characteristic curve of 0.86 (95% CI 0.81 to 0.91) in the held-out test set. This was significantly higher than the discrimination achieved by the percentage diameter stenosis alone (0.73, 95% CI 0.67 to 0.80; DeLong p<0.001), as shown in the receiver operating characteristic curves of Figure 1. At the optimal threshold the plaque model had a sensitivity of 85%, a specificity of 77%, a positive predictive value of 69% and a negative predictive value of 90%, with an overall accuracy of 80% (Table 3). The gain in discrimination over stenosis alone quantifies the information that the plaque phenotype adds beyond the degree of narrowing, and it is of the order reported for other machine-learning plaque models against an invasive reference, which have described areas under the curve in the mid-to-high eighties when quantitative plaque features are added to the assessment of stenosis. The improvement is also consistent with the meta-analytic observation that the aggregate plaque volume discriminates ischemia independently of the degree of stenosis (Hoshino *et al.*, 2020). The high negative predictive value is of particular practical importance, because it is the property that would allow the model to serve as a gatekeeper, safely identifying the vessels unlikely to be ischemic and so sparing patients an unnecessary invasive procedure. The predictive values should be read in the light of the prevalence of ischemia in this cohort, which at 40% was higher than in an unselected population undergoing coronary CT angiography, since these vessels had been selected for invasive fractional flow reserve measurement. In a population with a lower prevalence the negative predictive value would be higher still and the positive predictive value lower, which would strengthen the case for using the model to rule ischemia out rather than to confirm it, and this dependence on prevalence is a general property of any such test that must be considered when it is transferred to a different setting.

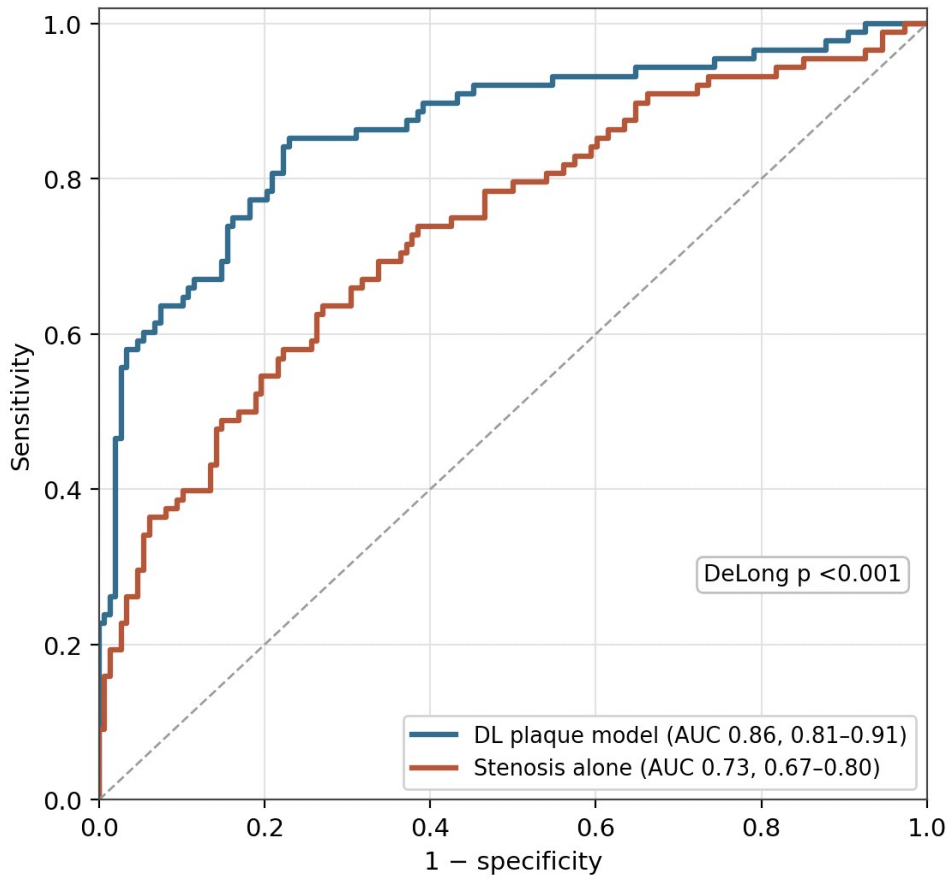


Figure 1. Receiver operating characteristic curves for the deep-learning plaque model and for stenosis alone

Table 3: Diagnostic performance for vessel-specific ischemia

Model	AUC (95% CI)	Sens (%)	Spec (%)	PPV (%)	NPV (%)	Acc (%)
DL plaque model	0.86 (0.81–0.91)	85	77	69	90	80
Stenosis alone	0.73 (0.67–0.80)	64	73	58	77	69

3.4. Determinants of the prediction

The permutation importance identified the plaque features that most influenced the prediction, shown in Figure 2. The low-attenuation plaque volume was the single strongest predictor, followed by the remodeling index, the lesion length and the non-calcified plaque volume, while the percentage diameter stenosis, although informative, was not the dominant feature. That the lipid-rich, low-attenuation component and the remodeling index outranked the stenosis is the central finding of the analysis, because it shows in explicit terms why the plaque phenotype outperforms the degree of narrowing: the features that carry the most information about ischemia are compositional and geometric properties of the plaque that the stenosis measurement does not capture. This ordering is coherent with the biology, in which a large, lipid-rich, positively remodeled plaque exerts a physiological effect out of proportion to the luminal narrowing it produces. It also has a practical consequence for how such models should be built and interpreted, since it implies that a model restricted to the stenosis, or to a single plaque feature, discards information that is available in the full phenotype; the gain in discrimination came from combining the compositional, volumetric and geometric features rather than from any one of them, which is the reason an automated phenotype that measures them all is more informative than a targeted measurement.

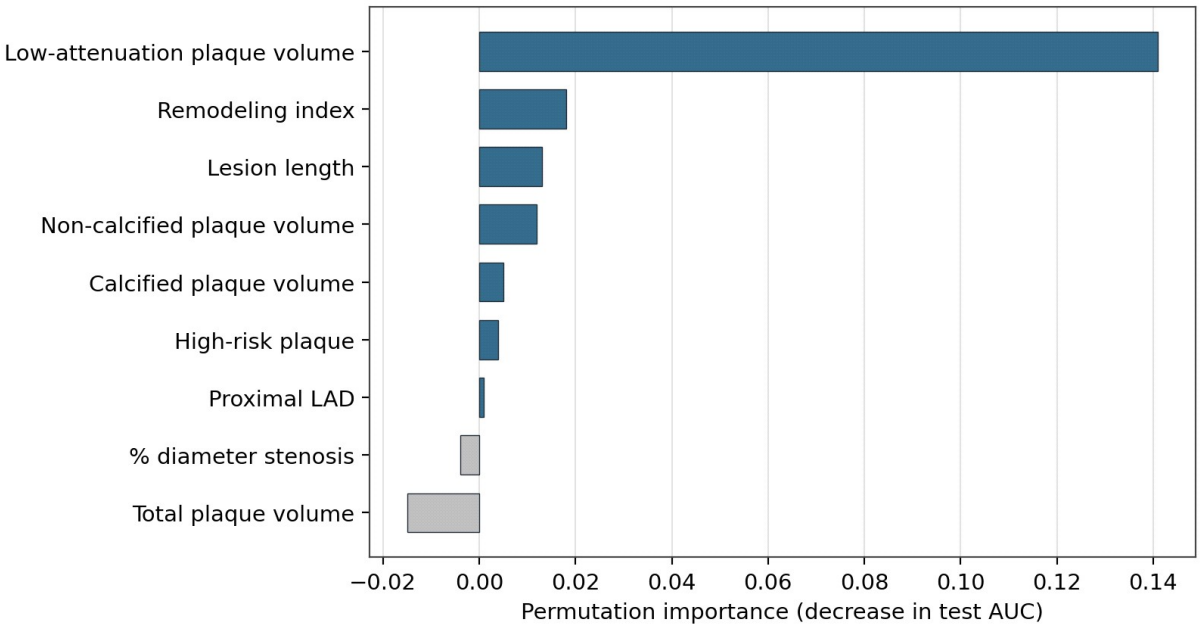


Figure 2: Permutation importance of the deep-learning plaque features for predicting ischemia

3.5. Calibration and per-vessel performance

Beyond discrimination, the model was well calibrated, with predicted probabilities agreeing closely with the observed frequency of ischemia (Hosmer-Lemeshow p = 0.49; Brier score 0.138), as shown in Figure 3. Good calibration matters for a model intended to guide decisions, because a probability that is to be acted upon must mean what it says, and a model may discriminate well yet be miscalibrated. The discrimination was consistent across the coronary arteries, the area under the curve being 0.87 in the left anterior descending, 0.90 in the left circumflex and 0.80 in the right coronary artery, indicating that the model is not driven by one territory and generalises across the coronary tree, which is a prerequisite for its use in unselected vessels. The modest

differences between territories are themselves informative. The discrimination was highest in the left anterior descending and left circumflex and slightly lower in the right coronary artery, which is consistent with the anatomy, since the right coronary artery is more often affected by motion and its plaque is more variably calcified, both of which can reduce the accuracy of automated plaque quantification. That the model nonetheless performed well in every territory, rather than achieving its overall accuracy through one dominant vessel, is what allows it to be applied to an unselected vessel without knowing in advance which artery it belongs to. Because the model outputs a calibrated probability rather than a fixed label, the operating threshold can also be adjusted to the clinical purpose, set low when the aim is to rule out ischemia and avoid missing an ischemic vessel, or higher when the aim is to select vessels for intervention. Taken together, the discrimination, the calibration and the consistency across vessels support the use of the deep-learning plaque phenotype as a non-invasive predictor of vessel-specific ischemia.

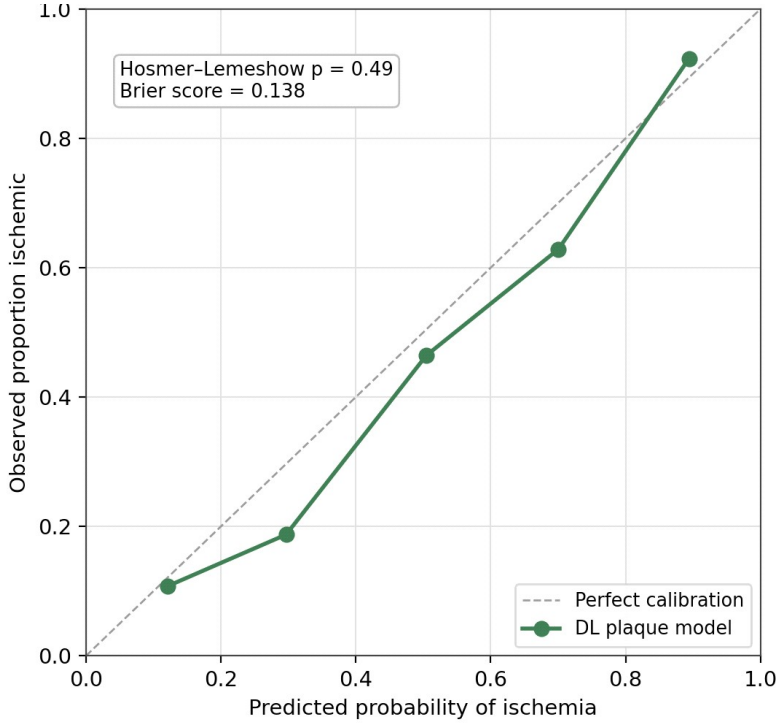


Figure 3: Calibration of the deep-learning plaque model against the observed frequency of ischemia

3.6. Clinical implications and relation to CT-derived fractional flow reserve

The practical value of the model lies in the way its two operating properties could be used together. Its high negative predictive value identifies the vessels that are very unlikely to be ischemic, which could be managed without invasive assessment, while the vessels it flags as high probability could be prioritised for invasive fractional flow reserve or for revascularisation, concentrating the invasive procedure where it is most likely to change management. Because the plaque phenotype is produced automatically from the same CT angiogram that is acquired for the anatomical assessment, this stratification adds no further scan and little time to the existing workflow.

The approach is best seen alongside CT-derived fractional flow reserve rather than in opposition to it. That method estimates the physiology directly and achieves a high accuracy for ischemia, and where it is available the two may be combined, the plaque phenotype adding the compositional information that the physiological estimate does not capture and that carries independent prognostic weight. The plaque phenotype may also be obtainable where the computational estimate is limited by image quality or calcification, and it draws on features, such as the low-attenuation plaque burden, that predict future events as well as current ischemia, so that a single automated analysis could serve both the diagnostic and the prognostic purpose. These considerations, together with the discrimination and calibration shown here, define the role in which a deep-learning plaque phenotype would be most useful in practice.

Two steps are needed before such a role could be adopted. The first is external validation, since the model was trained and tested within a single cohort on a single deep-learning system, and its discrimination and, above all, its calibration must be confirmed on data from other centres, scanners and populations, because a model can retain its ranking of vessels while losing the agreement between predicted and observed risk when it is moved to a new setting. The second is a demonstration that acting on the prediction improves outcomes, which requires a prospective study in which management guided by the plaque phenotype is compared with usual care; discrimination against an invasive reference, however good, establishes only that the model identifies ischemia, not that using it helps patients. Until these steps are taken, the plaque phenotype is best regarded as a promising adjunct that refines the selection of vessels for invasive assessment rather than a replacement for it.

4. Strengths and limitations

The main strengths of this study are that it used the invasive fractional flow reserve, the reference standard for ischemia, as the outcome, that it evaluated the model on a held-out test set kept separate at the patient level, and that it assessed calibration and the performance across individual vessels rather than discrimination alone.

The study has limitations. It was retrospective and conducted at a single centre, and the patients had been selected for invasive fractional flow reserve measurement, which introduces a spectrum of disease that may not represent all patients undergoing coronary CT angiography. The model was validated internally on a held-out set but not in an external cohort, and its performance may differ with other scanners, populations and deep-learning systems. Finally, the study assessed the prediction of ischemia but not whether acting on the prediction improves patient outcomes, which only a prospective study could establish.

5. Conclusion

A deep-learning plaque phenotyping model predicted vessel-specific ischemia from coronary CT angiography with good discrimination and calibration, and significantly more accurately than the degree of luminal stenosis alone. The low-attenuation, lipid-rich plaque component was the strongest predictor, and the performance was consistent across the coronary arteries. By quantifying the plaque phenotype automatically and identifying the vessels most and least likely to be ischemic, deep-learning plaque phenotyping could serve as a non-invasive gatekeeper to invasive assessment, though prospective and external validation is required before clinical adoption.

Ethical approval

The study was approved by the Institutional Ethics Committee (approval number IEC/CVI/2023/271) and was conducted in accordance with the Declaration of Helsinki. The requirement for individual informed consent was waived given the retrospective analysis of anonymised data.

Conflict of interest

The authors declare that no conflict of interest exists in relation to this work.

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Data availability

The data supporting the findings of this study are available from the corresponding author on reasonable request.

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