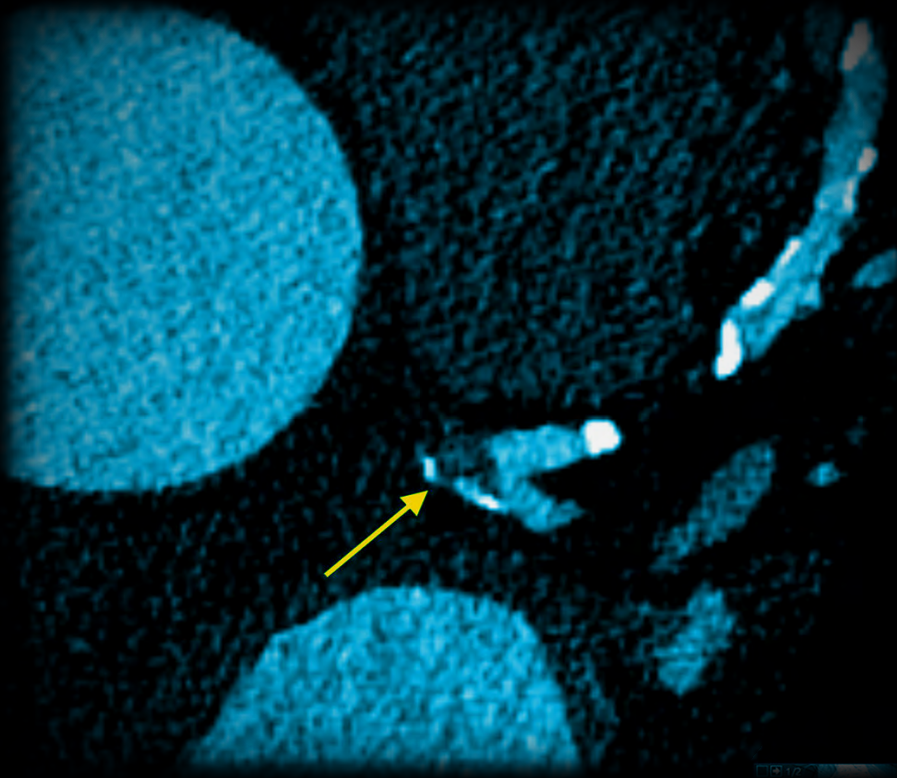


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The Journal of Practical Medical Imaging and Management



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to Photon Counting:
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AI-Guided Coronary Plaque Analysis from Coronary CTA: An Emerging Paradigm for Personalized Preventative Cardiology

Azfar Basunia, MD; Dinesh Jagasia, MD;
Faisal Jamal, MD

AI-guided quantitative plaque analysis (AI-QPA) is transforming coronary CT angiography (CCTA) from a tool focused on detecting stenosis to one capable of characterizing plaque biology and predicting cardiovascular risk. By automating plaque segmentation and quantification, AI improves efficiency, reproducibility, and diagnostic accuracy while enabling identification of high-risk plaque features linked to adverse outcomes. Emerging evidence shows AI-QPA can enhance risk stratification, guide preventive therapies, and support more personalized management of coronary artery disease. As adoption grows, these tools are poised to shift cardiovascular care toward earlier intervention and precision prevention, though challenges around standardization, validation, and cost remain.

From Energy Integration to Photon Counting: Redefining the Design and Performance of Clinical CT

Henrique Carvalho, BS; Peter B. Noël, PhD; Saurabh Jha, MD; Faisal Jamal, MD

This review traces the evolution of computed tomography from early energy-integrating detector systems to the emergence of photon-counting CT (PCCT). Highlighting how PCCT overcomes key limitations of conventional CT by providing higher spatial resolution, reduced noise, and intrinsic spectral imaging in every scan, the authors outline the clinical impact of these advances across cardiovascular, pulmonary, musculoskeletal, and oncologic imaging.

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Nina S. Vincoff, MD, is the editor-in-chief of *Applied Radiology*. She is Executive Medical Director of the Radiology Care Division at Prisma Health and Clinical Associate Professor of Radiology at the University of South Carolina School of Medicine Greenville. She can be reached at nina@appliedradiology.com

The Evolving Role of Cardiac Imaging in Care Delivery

Nina S. Vincoff, MD

Not long ago, the evaluation of coronary artery disease was defined by cardiac angiography. The pathway from symptom to diagnosis typically began in the emergency department and led directly to the catheterization laboratory.

Today, cardiac imaging spans the full continuum of care. In asymptomatic patients, coronary artery calcium scoring identifies risk before symptoms develop, informing preventive strategies and medical management.

For symptomatic patients, imaging options have expanded from echocardiography and nuclear imaging to include coronary CTA (CCTA) and cardiac MR. These modalities provide detailed characterization of cardiovascular pathophysiology, often eliminating the need for invasive procedures.

In this issue, 2 review articles highlight advances in CT technology, including photon counting, and the emergence of artificial intelligence-guided coronary plaque analysis. These developments are helping to drive a broader transformation now underway in cardiac care.

Today, in some settings, select low-risk symptomatic patients in the emergency department are being triaged for coronary artery disease using CCTA. A normal study allows safe discharge without admission, reducing length of stay and avoiding unnecessary hospitalization.

The implications extend beyond the emergency department. Primary care patients with risk factors and equivocal symptoms may be referred as outpatients directly for CCTA rather than cardiology consultation. Imaging becomes the decision point, determining who requires further testing or intervention and who can be safely managed without escalation.

These care algorithms depend not only on advanced imaging capabilities, but on interpretation that is accurate, consistent, and clinically meaningful. We have the tools. Whether these care pathways succeed will rely on the availability of qualified cardiothoracic radiologists.

At a time when cardiac imaging is becoming more central to triage and care coordination, there is a growing mismatch between capability and workforce. Radiology faces a well-recognized physician shortage, and the number of radiologists with dedicated cardiothoracic expertise remains limited relative to the expanding demand for these services.

The stage is set for a change in the way cardiac care is delivered. Radiology must meet this moment by providing the expertise to match the technology and guide cardiac care delivery into the future.

AI-Guided Coronary Plaque Analysis from Coronary CTA: An Emerging Paradigm for Personalized Preventive Cardiology

Azfar Basunia, MD; Dinesh Jagasia, MD; Faisal Jamal, MD

Abstract

Coronary artery disease (CAD) remains the leading cause of death worldwide and is driven by atherosclerotic plaque formation. Due to advances in CT technology, coronary CTA (CCTA) has emerged as a leading noninvasive imaging technique to analyze the coronary artery lumen and atherosclerotic plaque. CCTA can characterize plaque types (calcified, noncalcified, and low-attenuation [lipid-rich]) components, which carry different risks. Total plaque burden measured on CCTA, especially the volume of noncalcified plaque, has emerged as a strong predictor of acute coronary syndrome (ACS), independent of traditional risk factors and calcium score. Contemporary CCTA reporting requires manual plaque segmentation, which can be time-intensive and show suboptimal inter- and intraobserver reproducibility. Artificial intelligence-guided quantitative plaque analysis (AI-QPA) algorithms have emerged to address these challenges and increase analytic throughput. In multiple studies over the past few years, AI-QPA has demonstrated superiority over conventional myocardial perfusion imaging and achieved excellent agreement with expert human readers and invasive imaging. Furthermore, the therapeutic basis of lipid-lowering medications was demonstrated using AI-QPA, ushering in an era of personalized preventative cardiology. This review briefly delves into the common AI-QPA workflow, the inner workings, and validation for the 3 most common commercially available AI-QPA platforms: Cleerly, HeartFlow, and PlaqueIQ (Elucid).

Keywords: artificial intelligence, AI, plaque analysis, coronary CTA, CCTA

Introduction

Coronary artery disease (CAD) remains the leading cause of death worldwide. For decades, clinical assessment of CAD has revolved around detecting luminal stenoses that impede blood flow. However,

growing evidence shows that the nature of atherosclerotic plaque, not just the degree of stenosis, critically determines a patient's risk of myocardial infarction (MI). Many MIs arise from plaques that are not severely obstructive but are biologically "vulnerable," that is, characterized by

large lipid cores, thin fibrous caps, inflammation, and positive remodeling.¹⁻³ Identifying such high-risk plaques before they cause an acute event is a major goal of preventive cardiology.

Coronary CTA for Robust CAD Diagnosis

Coronary CTA (CCTA) has emerged as a noninvasive imaging technique that enables visualization of the coronary lumen, arterial wall, and plaque within these structures. Initially, CCTA was used primarily for the detection of obstructive CAD to guide revascularization therapy. A normal CCTA confers a

Affiliations: Department of Radiology, Hospital of University of Pennsylvania, Philadelphia, Pennsylvania (Basunia, Jamal). Department of Cardiology, Hospital of University of Pennsylvania, Philadelphia, Pennsylvania (Jagasia).

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Declaration on the use of AI: The authors of this manuscript declare that in the writing process of this work, no generative artificial intelligence (AI) or AI-assisted technologies were used to generate content, ideas, or theories. We utilized AI solely for the purpose of enhancing readability and refining language. This use was under strict human oversight and control. After the application of AI technologies, the authors carefully reviewed and edited the manuscript to ensure its accuracy and coherence.

very high negative predictive value for ruling out significant CAD and carries excellent prognosis.^{4,5} Trials like PROMISE, SCOT-HEART, and CREDESCENCE had established CCTA as at least equivalent, if not superior, to functional stress testing for initial evaluation of chest pain.⁶⁻⁸ Trials like DISCHARGE showed lower rates of procedure-related complications using CCTA in women and patients <65 years of age, but a similar incidence of major adverse cardiac events (MACEs), defined as cardiovascular death, a nonfatal MI, or a nonfatal stroke, between CCTA and invasive coronary angiography among patients with stable chest pain and intermediate CAD risk.⁹⁻¹¹ In its 2019 guidelines, the European Society of Cardiology designated CCTA as first-line diagnostic testing for suspected CAD, equivalent to functional stress testing (class I recommendation) and an alternative to invasive coronary angiography when functional testing is nondiagnostic (class II recommendation).¹² Complementing this, the 2021 American College of Cardiology/American Heart Association guidelines recommend CCTA for evaluation of chest pain in intermediate-risk patients to exclude plaque and obstructive CAD (class I recommendation).¹³

Plaque Formation Pathophysiology

Coronary plaque formation is a complex, multifactorial process driven by endothelial dysfunction, lipid accumulation, and chronic inflammation.¹⁴ The inciting event is typically endothelial injury, followed by subendothelial low-density lipoprotein deposition and oxidation. This leads to a downstream cascade of macrophage recruitment and transformation into foam cells (responsible for low attenuation in plaques). Proliferation of smooth muscle cells, synthesis of extracellular matrix, and further migration and apoptosis of white blood cells lead to plaque progression, fibrous cap formation, and chronic inflammation. Inflammatory cells secrete proteases that degrade the fibrous

cap and increase the risk of plaque rupture. Pro-inflammatory cytokines promote osteogenic differentiation of smooth muscle cells and plaque mineralization, which have important prognostic implications. Early microcalcifications (<15 µm) are associated with plaque instability and increased risk of rupture, while macrocalcifications appear as the inflammation resolves and are typically associated with plaque stability.^{15,16}

Current Risk Stratification Clinical Tropes and Diagnostic Dilemmas

The coronary artery calcium (CAC) score is widely used for risk stratification of asymptomatic patients. In the acute setting at a single institution, a negative coronary calcium score (CAC = 0) showed high negative predictive value (99.3%) for obstructive CAD in low- or intermediate-risk patients.¹⁷ However, the direct causal relationship between CAC and MACE is unclear. In fact, heavily calcified plaque measuring >10,000 Hounsfield units (HU) carries a lower risk of acute coronary syndrome on a per-patient and per-lesion basis, likely corresponding to histopathologic macrocalcifications related to resolving inflammation and plaque stability.¹⁸ In the multicenter PROMISE trial, 25% of patients who suffered MACE had a CAC score of 0.¹⁹ Since the CAC burden and score increases with age, CAC score may underpredict the risk of MI in younger patients (especially those with noncalcified plaques) and overpredict the risk of MI in older patients.

Rationale for Plaque Quantification by CCTA: A Shifting Clinical Management Paradigm

Over the last decade, successive improvements in image quality and the advent of powerful software have dramatically enhanced CCTA's plaque visualization capabilities. It is now feasible to quantify plaque volumes (in mm³) and differentiate plaque subcomponents by CT density (eg, calcified versus

lipid-rich) on standard CCTA scans. CCTA could noninvasively characterize plaque, defined as tissues 1 mm² within or adjacent to the coronary artery lumen in 2 independent planes that are distinct from the lumen and epicardial fat.²⁰ When compared to the intravascular US (IVUS), the gold standard for plaque detection and characterization, a meta-analysis of 42 studies with 1360 patients found high sensitivity (93%) and specificity (92%) for CCTA plaque detection and no statistically significant differences in plaque volumes or area.²¹ Furthermore, low-attenuation plaques (LAPs), characterized by ≤30 HU, correspond well to lipid-rich plaques and thin-capped fibroatheromas on IVUS.^{22,23} The Society of Cardiovascular Computed Tomography (SCCT) and the North American Society for Cardiovascular Imaging acknowledge that LAP burden is a strong independent predictor of incident MI and future cardiac events beyond stenosis severity alone.²⁴

A substantial body of evidence indicates that CCTA-derived plaque metrics are independently associated with clinical outcomes and offer additional prognostic value beyond traditional risk factors.^{1,3,20,25-30} The total burden of plaque found on CCTA is a strong predictor of events. A recent study using artificial intelligence (AI) quantification in ~1600 patients reported that in those with total plaque volume (TPV) in the highest quartile or ≥238.5 mm³ (an outcome-optimized threshold), the risk of MI was 5-fold higher.²⁹ This association remained after controlling for stenosis severity and clinical risk score.

Numerous studies identify LAP as an important prognostic marker. In a post hoc analysis of 1769 patients from the SCOT-HEART trial, MACE was 3 times more frequent among those with LAP or positive vessel remodeling; in fact, an LAP volume increase of 4% was associated with a 5-fold increase in the risk of future MI and showed stronger predictive value than luminal stenosis grading.^{25,30} A secondary analysis of the PROMISE trial showed that the presence of any high-risk plaque feature (eg, LAP, spotty

calcification, positive remodeling, napkin ring sign) was associated with a 70% higher risk of MACE, especially in women and younger patients.²⁰ In a multicenter study of 252 patients who underwent CCTA and invasive fractional flow reserve (FFR), high-risk plaque features increased the risk of ischemia by 3- to 5-fold.²⁸

In the ROMCAT-II trial, the presence of high-risk plaque features remained a significant predictor of acute coronary syndrome even after adjusting for 50% vessel stenosis, 70% vessel stenosis, and clinical risk factors (eg, age, sex, and number of cardiovascular risk factors).²⁶

Plaque analysis using CCTA has also demonstrated the therapeutic basis of risk-modifying medications, paving the way for personalized therapeutic pathways and noninvasive imaging follow-up. In multiple large studies (such as the multinational PARADIGM study with 2252 patients), CCTA showed that lipid-lowering drugs such as statins were associated with slower progression of overall coronary plaque volume, increased plaque calcifications, and reduction in high-risk plaque features.^{31,32} In the randomized, placebo-controlled EVAPORATE trial, patients on icosapent ethyl had 17% and 9% reduction in LAP and TPV compared with a 109% and 11% increase in LAP and TPV after 18 months.³³

The Coronary Artery Disease—Reporting and Data System (CAD-RADS) was developed as a standardized reporting system to improve assessment consistency, facilitate clinical decision-making, and guide management after CCTA.³⁴ CAD-RADS categorizes patients according to the maximal degree of coronary stenosis, ranging from 0 (no CAD) to 5 (total occlusion). It also incorporates plaque burden modifiers (P1-P4) and ischemia testing results (I+, I-, I±). Higher CAD-RADS categories (3-5) and the presence of high-risk plaque features are associated with increased risk of MACE. Accordingly, the American College of Cardiology recommends integrating all available diagnostic information for patient risk stratification.³⁵ However, variability in

the visual assessment of coronary stenosis and plaque burden remains a challenge as interobserver differences can affect CAD-RADS categorization. Less experienced CCTA readers have a higher rate of overestimation compared with expert readers, potentially increasing the risk of unnecessary invasive procedures.³⁶

AI and Plaque Characterization: Improving Imaging Workflows and Altering Therapeutic Management

AI-based algorithms substantially reduce interobserver variability and improve risk stratification compared with traditional visual CAD-RADS scoring. AI-based algorithms substantially reduce interobserver variability and improve risk stratification compared with traditional visual CAD-RADS scoring. AI-driven CAD-RADS assessment demonstrates excellent agreement with expert readers (weighted $\kappa = 0.73$ -0.97) and provides a high negative predictive value for ruling out obstructive disease, thereby improving workflow efficiency and reducing reporting times by up to 40%.³⁷⁻³⁹ In a post hoc analysis of the CREDENCE trial, AI-guided quantitative plaque analysis (AI-QPA) showed superior diagnostic performance compared with myocardial perfusion imaging for clinically significant stenosis ($\geq 50\%$ or FFR < 0.8) as determined through invasive coronary angiography.⁴⁰

AI-QPA showed excellent agreement with IVUS and near-infrared spectroscopy-IVUS (NIR-IVUS) for TPV, vessel and lumen roadmap, calcified plaque, noncalcified plaque, and LAP volume calculations.^{41,42} For long-term risk stratification, AI-QPA staging that incorporates high-risk plaque features and percent atheroma volume showed greater prognostic discrimination for MACE compared with manual CAD-RADS and clinical risk scores, with improved net reclassification and higher area under the curve (AUC) over 10-year follow-up.⁴³ With growing adoption of CCTA, age- and sex-specific quantitative plaque normograms based on an international cohort of 11,808 patients were calculated with AI-QPA,

where manual plaque segmentation would have been cumbersome and impractical.⁴⁴

AI-QPA results directly impact clinical decision-making, leading to more personalized and effective management strategies for patients with suspected or known CAD. In the DECODE study, clinicians reclassified the preventative therapy plans of 66% of the patients after AI-QPA results were made available in addition to the CCTA results; when separated by CAC, preventative therapy upstaging ranged from 47% (CAC = 0) to 96% (CAC > 400).⁴⁵ In the multicenter CERTAIN study, compared with conventional CCTA analysis, AI-QPA and AI-assisted FFR analysis increased physicians' confidence in the care pathway, increased the initiation of aspirin and statins by 28.1% and 23%, respectively, and decreased the need for downstream invasive and noninvasive cardiac testing by 37%.⁴⁶

Important Drawbacks of AI-QPA

AI-QPA is suboptimal below a certain plaque volume. For instance, plaque volume thresholds below 2.3 mm³ had steep negative effects on diagnostic accuracy (42% for <2.3 mm³ vs 94% for >2.3 mm³) and specificity (27% for <2.3 mm³ vs 94% for >2.3 mm³) and minimal effect on sensitivity (93% for <2.3 mm³ vs 100% for >2.3 mm³) compared with NIR-IVUS.⁴¹

Differences in AI-QPA output persist between the make and versions of software due to training data heterogeneity and proprietary algorithms affecting approach to vessel, plaque boundary identification, attenuation-based classification, and integration of clinical data.^{47,48} Even variability in hardware and scanning parameters can produce discordant CCTA and AI-QPA results.⁴⁹ For instance, decreasing tube current from 140 kVp to 100 kVp can result in overestimation of TPV from overestimation of calcified plaque (secondary to blooming artifact), and underestimation of noncalcified plaque.⁵⁰ Nonetheless, the SCCT emphasizes

that standardization of annotation, segmentation, and validation protocols are essential for reliable clinical adoption. Their consensus document recommends rigorous external validation against gold standards (IVUS, invasive angiography) and independent cohorts, and highlights the need for harmonized data annotation and transfer learning to address scanner and population variability.⁵¹

AI Plaque Analysis Workflow

Overall plaque analysis workflow is similar among the 3 popular commercial vendors in the United States (Figure 1), all of whom have received clearance from the U.S. Food and Drug Administration. After a physician determines known or suspected CAD, CCTA is obtained and images are uploaded to the vendor's server for AI-QPA. The AI algorithm's work is double-checked for quality control by an "expert reader" who can be a physician or an experienced nonphysician reader. A report is then routed back to the interpreting physician (radiologist or cardiologist). All 3 vendors offer online platforms to manually view/interrogate the coronary tree and AI segmentation in greater detail (Figure 2).

A brief discussion of the derivation and/or the performance of the 3 common vendors is provided next.

AI-QPA Platform: Cleerly

The Cleerly AI-QPA platform (Cleerly Labs, Denver, Colorado) performs automated coronary CTA analysis using an array of validated convolutional neural networks.^{39,52,53} The AI pipeline generates artery centerlines, contours lumen and outer wall across all available phases, selects the 2 highest-quality phases per vessel, and applies automated anatomical labeling with proximal, mid, and distal segmentation. Diameter stenosis is computed from interpolated reference diameter at the site of stenosis. Against quantitative coronary angiography in the CLARIFY study, per-patient performance (sensitivity, specificity, positive predictive

value, negative predictive value, and accuracy) for $\geq 50\%$ stenosis was 94%, 68%, 81%, 90%, and 84%; for $\geq 70\%$ stenosis, per-patient performance was 94%, 82%, 69%, 97%, and 86%.⁵² On a per-vessel analysis, Cleerly's software performed similar to level 3 cardiac CT readers but superior to level 2 cardiac CT readers (AUC = 0.86 vs 0.69, $P < .001$).⁵³ Inner and outer wall boundaries are segmented and plaques are detected as tissue $> 1 \text{ mm}^3$ within the wall using proximal and distal reference slices. Plaques are classified by HU thresholds; below $< 30 \text{ HU}$ = LAP, between 30 HU and 350 = noncalcified plaque, and $> 350 \text{ HU}$ = calcified plaque. Analyses include all epicardial vessels $\geq 1.5 \text{ mm}$. Quantitative metrics such as external elastic lamina area, lumen area, plaque area, plaque burden, and length-normalized percent atheroma volume correlate well (correlation coefficient > 0.74) with IVUS and NIR-IVUS in the INVICTUS study groups.^{41,54}

AI-QPA Platform: HeartFlow

The HeartFlow AI-QPA deep learning algorithm (HeartFlow Inc, Mountain View, California) automatically segments the vessel outer wall and lumen to quantify plaque volume.^{42,55} The lumen model was trained on clinical CCTA cases from HeartFlow's FFR CCTA (FFR_{CT}) datasets; the outer-wall model was trained on AutoPlaque annotated cases from Cedars-Sinai and HeartFlow's FFR_{CT} cases that included at least one vessel with $> 30\%$ diameter stenosis, supporting generalizability to symptomatic patients.

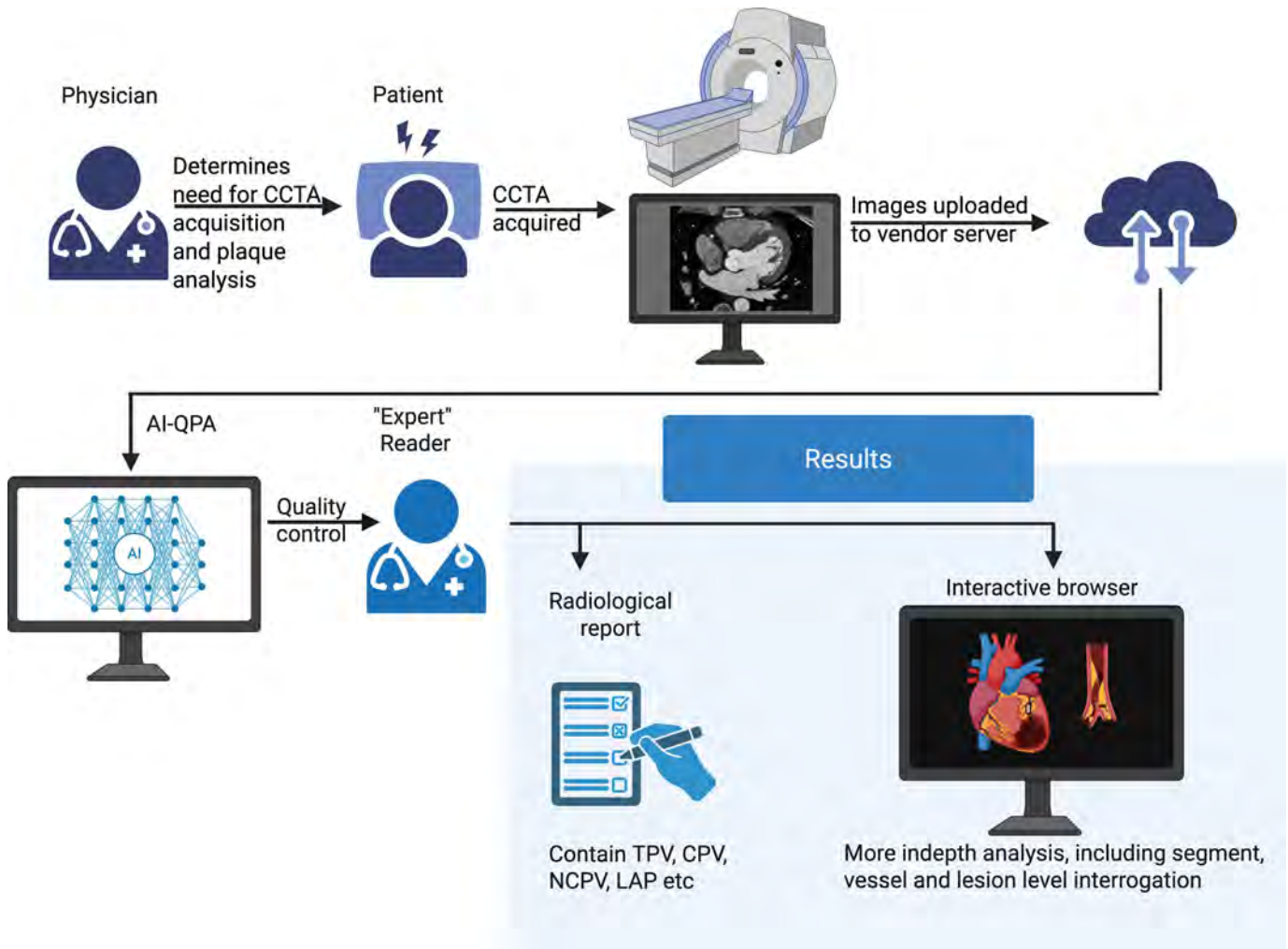
AI and structured human quality control from trained CT analysts are used to generate patient-specific 3D reconstructions of the aorta and coronary arteries from the highest-quality CCTA phase. Deep learning models segment lumen and outer wall on cross-sections orthogonal to vessel centerlines. The AI algorithm extracts the centerline of vessels $\geq 1.8 \text{ mm}$ in diameter. Analysts verify and, if needed, adjust boundaries using a standardized protocol. After final

segmentation, plaque volume is quantified by HU thresholds: LAP between -30 and 30 HU , noncalcified plaque at 30 to less than the calcified threshold, and calcified plaque defined adaptively as greater than 350 HU or mean luminal attenuation plus 1 SD. Quantitative metrics such as TPV and calcified and noncalcified plaque volumes correlate well (> 0.87) with IVUS in the REVEALPLAQUE study.⁴²

AI-QPA Platform: PlaqueIQ

PlaqueIQ (Elucid, Boston, Massachusetts) uses convolutional neural networks trained on carotid endarterectomy histopathologic images of atherosclerotic plaques that were co-registered to CTA images for establishing ground truth to classify individual plaque components.^{56,57} CCTA voxel Hounsfield values can overlap between different histopathologic tissue types such as lipid-rich necrotic core (LRNC), intraplaque hemorrhage, and other components, making them difficult to distinguish. To overcome this, the software uses spatial distribution of tissue properties instead of fixed HU ranges to interpret adjacent voxels. Regions defined by biological features are translated into color-coded segments to reduce data complexity. Key plaque characteristics such as LRNC, stenosis, remodeling ratio, dilation, and ulceration are identified using semantic segmentation to reduce variability caused by differences in scanner hardware, reconstruction settings, and contrast administration.⁵⁷ PlaqueIQ performs well compared with histology-based identification of LRNC, calcified and noncalcified plaque (correlation coefficient > 0.87). Quantitative metrics such as TPV and calcified and noncalcified plaque volumes correlate well (> 0.74) compared with HU threshold-based plaque identification.⁵⁸ However, only modest correlation (0.54) is noted between LAP from HU-thresholding platforms and LRNC from PlaqueIQ due to significantly higher volume estimates of LAP compared with LRNC.⁵⁸

Figure 1. Plaque analysis workflow among the popular 3 vendors in the United States. AI-QPA, artificial intelligence-guided quantitative plaque analysis; CCTA, coronary CTA; CPV, calcified plaque volume; LAP, low-attenuation plaque; NCPV, noncalcified plaque volume; TPV, total plaque volume. Created in BioRender. Basunia, A.



Conclusion and Future Directions

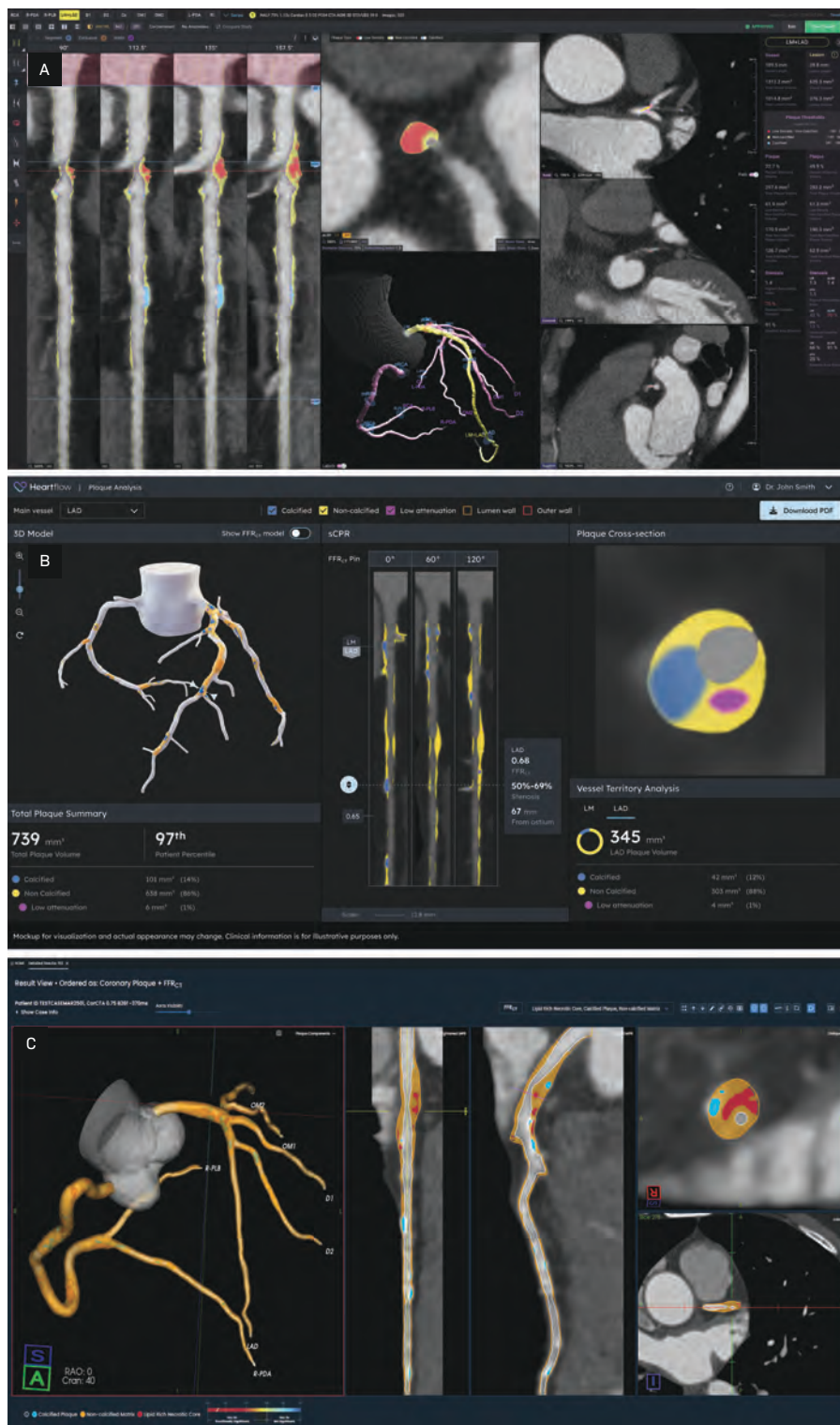
Recent guidelines from US and international cardiology societies have supported a paradigm shift away from stenosis-based to plaque biology-based assessment of CAD.⁵⁹ Over the past decade, robust evidence has established that quantitative plaque analysis by CCTA provides powerful prognostic insights on risk stratification and supports early clinical intervention and risk-factor modification. Contemporary CCTA reporting requires manual plaque segmentation, which can be time-intensive and show suboptimal inter- and intraobserver reproducibility.

Technological innovations in AI algorithms for plaque quantification enable these assessments to be performed rapidly in clinical practice. Furthermore, myocardial ischemia is driven by plaque and vascular morphology and as much as 50% of patients with CAD have ischemia in at least one vascular territory.⁶⁰ All 3 vendors are actively developing or offering tools to estimate the probability of coronary vessel-specific ischemia by calculating FFR from CCTA data (FFR_{CT}) through plaque-based analysis (Elucid and Cleerly) or computational fluid dynamics (HeartFlow).⁶¹⁻⁶³ In fact, the EMERALD-II study has shown increased predictability of ACS from combined AI-QPA and FFR_{CT}

compared with standard CCTA analysis (AUC 0.84 vs 0.78; $P < .001$).⁶⁴

Limited reimbursement has been a significant impediment to broad US adoption of AI-QPA. Per-case pricing for AI-QPA typically ranges from several hundred to several thousand dollars, which substantially exceeds the Medicare reimbursement for CCTA, generally in the low hundreds of dollars.⁴⁸ However, the momentum is shifting; the U.S. Centers for Medicare & Medicaid Services has assigned AI-QPA a Category I CPT code effective calendar year 2026, establishing a pathway for federal and state insurance program coverage.⁶⁵ In parallel, multiple private insurers have announced

Figure 2. Interactive browser with analytic tools for in-depth vessel- and plaque-level analysis from each vendor. (A) Cleerly, (B) HeartFlow, and (C) Elucid. Images are reproduced with permission from Cleerly, Inc, HeartFlow, and Elucid.



coverage beginning October 1, 2025, which will likely promote further widespread clinical adoption.⁶⁶

Increasing insurance reimbursement and widespread clinical adoption will further promote industry-academic partnerships, drive innovation, and transform clinical practice. Many prospective randomized clinical trials aim to challenge the current diagnostic and treatment norms. For example, the TRANSFORM trial from Cleerly aims to compare the performance of Cleerly's AI-aided CAD score to the current atherosclerotic cardiovascular disease risk scores for identifying asymptomatic individuals at increased risk for cardiovascular events⁶⁷; the DECIDE Registry from HeartFlow aims to assess treatment changes in clinically stable patients who undergo CCTA with AI-QPA versus CCTA alone.⁶⁸

As AI-QPA adoption scales, harmonization of vendor- and site-specific acquisition and reconstruction protocols is imperative to support accurate and reproducible CCTA analyses for longitudinal comparisons. Scan to rescan variability and limits of agreement should be rigorously characterized and incorporated into analytic pipelines before attributing any change to plaque biology.

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From Energy Integration to Photon Counting: Redefining the Design and Performance of Clinical CT

Henrique Carvalho, BS; Peter B. Noël, PhD; Saurabh Jha, MD; Faisal Jamal, MD

Abstract

Computed Tomography (CT) has advanced continuously since the 1970s, evolving from static brain imaging to high-speed, high-resolution volumetric and spectral systems. This review outlines key milestones leading to photon-counting CT (PCCT) and how it addresses the fundamental limitations of energy-integrating detector (EID) technology. While innovations such as helical scanning, multidetector arrays, dual-source systems, and iterative reconstruction (IR) have improved image quality and reduced dose, EID-CT remains constrained by electronic noise, limited spatial resolution, and limited accessibility of spectral information. PCCT introduces direct-conversion detectors that count and classify individual x-ray photons by energy, delivering improved spatial and contrast resolution, intrinsic spectral data, and noise-efficient imaging. PCCT unites ultra-high-resolution, enhanced contrast-to-noise ratio, and multienergy capability in every scan. Early clinical results demonstrate substantial benefits in cardiovascular, pulmonary, musculoskeletal, and oncologic imaging. As PCCT enters routine practice, it marks a paradigm shift toward data-rich, spectrally resolved imaging with greater diagnostic precision and efficiency.

Keywords: photon counting CT, spectral CT, temporal resolution, iodine maps, dual source CT

Introduction

The history of CT is a story of continuous innovation driven by the pursuit of improved diagnostic precision. The journey began in the early 1970s with the pioneering work of Godfrey Hounsfield and Allan Cormack, whose breakthroughs laid the foundation for a technology that would become a cornerstone of modern medical imaging.¹

From those beginnings, CT technology advanced rapidly. Early systems with

narrow fan-beam geometries evolved into wide fan-beam and multidetector-row configurations, enabling greater z-axis coverage and thinner image slices. The introduction of helical (spiral) scanning further improved temporal resolution and scanning efficiency, while refinements in detector design and gantry mechanics enhanced spatial resolution. Concurrently, developments such as automated tube current modulation and IR techniques reduced radiation dose without sacrificing diagnostic accuracy. These

innovations established the framework for modern CT performance and paved the way for emerging technologies.²

Despite these advances, conventional energy-integrating CT still has important limitations. Spatial resolution can be insufficient for coronary CTA in patients with heavy calcification or stents, where blooming and beam-hardening artifacts may obscure fine detail and overestimate stenosis.³ Quantitative accuracy and low-contrast lesion detection may also suffer because Hounsfield Units vary with patient size and surrounding tissue attenuation, and image quality can worsen in larger patients because of reduced dose efficiency and greater electronic noise.⁴

To address some of these challenges, dual-energy CT (DECT) was introduced, allowing material characterization

Affiliations: Herbert Wertheim College of Medicine, Florida International University, Miami, Florida (Carvalho). Perelman School of Medicine at the University of Pennsylvania, Philadelphia, Pennsylvania (Noël, Jha, Jamal).

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through spectral imaging. DECT systems employed methods such as dual x-ray tubes operating at different kilovolt peak (kVp) settings, rapid kVp switching, split-filter techniques, and dual-layer spectral detectors. While DECT represented an important step forward, it continued to rely on EID technology and thus retained some of its inherent constraints.^{4,5}

Recently, PCCT has entered clinical practice as a new approach to x-ray detection. By counting individual photons and recording their energies directly, it offers better spatial and contrast resolution, lower electronic noise, and routine multienergy data from every scan. Together, these features provide a stronger basis for diagnostic performance across a wide range of clinical applications.⁶

Foundations and Early Generations

CT's theoretical basis traces to Johann Radon (1917), who developed the mathematical principles showing that a function can be reconstructed from its line integrals, laying the groundwork for tomographic imaging.⁷ In the 1960s, William Oldendorf built a prototype with a rotating x-ray source and detector that demonstrated cross-sectional imaging.⁸ In parallel, Allan Cormack developed the reconstruction algorithms needed for clinical use.⁹ Building on these foundations, engineer Godfrey Hounsfield designed the first practical CT scanner and, together with James Ambrose, performed the first clinical brain scan in 1971.¹⁰ This marked a turning point in diagnostic medicine and earned Cormack and Hounsfield the 1979 Nobel Prize. This first-generation scanner employed a pencil beam and single detector, requiring several minutes to acquire each slice. Second-generation fan-beam systems (e.g., ACTA, 1973) improved speed, while third-generation rotate-rotate scanners (e.g., Siemens SOMATOM, 1977) achieved whole-body imaging in seconds.

Volumetric Advances

Slip-ring technology eliminated cable constraints, enabling continuous gantry rotation and helical CT in the early 1990s. A slip-ring is an electromechanical device that uses brushes to transmit power and data between a stationary surface and a rotating one, eliminating the need for direct wiring, paving the way for helical (or spiral) CT. In a helical scan, the patient table moves through the continuously rotating gantry, allowing the x-ray beam to trace a spiral trajectory around the body.¹¹ This enabled volumetric imaging within a single breath-hold and facilitated the transition from xenon gas detectors to solid-state technology. These advances produced faster scans, improved image quality, and reduced artifacts.¹¹⁻¹³

Electron Beam CT

Electron beam CT (EBCT) was developed in the 1980s for imaging rapidly moving organs, particularly the heart.¹⁴ Instead of rotating a physical x-ray tube, EBCT employed a stationary anode ring and deflected an electron beam to generate x-rays at multiple points, enabling slice acquisitions in approximately 50-100 ms.^{14,15} This ultrafast temporal resolution made it possible to “freeze” cardiac motion, enabling coronary artery calcium (CAC) scoring and early noninvasive imaging of coronary anatomy.¹⁶ Although EBCT offered superior temporal resolution, it was hampered by lower spatial resolution, limited versatility for body imaging, and high operational cost. Its clinical use declined as ECG-gated multidetector CT (MDCT) emerged.¹⁴

Multidetector, Dual-Source, and Spectral Systems

The late 1990s introduced MDCT, beginning with the Elscint Twin Flash Spiral CT's 2-slice scanner and rapidly

advancing to 16-, 32-, and ultimately 64-slice systems by 2004.¹⁷ The defining feature of MDCT was its capability to capture multiple slices per gantry rotation, significantly expanding volume coverage along the z-axis. Wide-detector arrays enabled near-whole-organ coverage in a single rotation and supported dynamic perfusion imaging of organs such as the brain, heart, and liver.¹⁸ Developments in area-detector technology, exemplified by Canon's Aquilion ONE (introduced in 2007), expanded z-axis coverage to encompass entire organs within one rotation.^{17,19} Contemporary platforms with 256-512 detector rows provide up to 160 mm of coverage, enabling single-beat whole-heart imaging and comprehensive trauma scans in one rotation.²⁰

In 2005, dual-source CT (DSCT) introduced two x-ray tubes and detectors mounted roughly 90° apart, improving temporal resolution relative to single-source MDCT and enabling robust cardiac imaging even at high or irregular heart rates. DSCT also benefits trauma and obese patients by allowing rapid acquisition without sacrificing image quality and offers an alternative to wide-detector scanners.^{21,22} DSCT achieved notable results through shorter acquisition times per view, offering improved resilience to arrhythmias, providing an alternative solution to wide-area coverage, with equal or greater clinical benefits when temporal resolution was the limiting factor.²³

Beyond dual-source systems, rapid kVp-switching and split-filter techniques achieve dual-energy imaging with a single x-ray tube. However, spectral separation is constrained by generator performance and system design, and implementations typically support only a limited set of predefined acquisition protocols. Additionally, rapid kVp switching imposes restrictions on tube current modulation, which limit dose efficiency in large patients.^{4,5}

Dual-layer detector CT uses a single x-ray spectrum combined with a stacked EID design. The top scintillator layer

absorbs lower-energy photons, while higher-energy photons penetrate to the lower layer. Because spectral separation occurs at the detector rather than the source, dual-layer CT intrinsically acquires spectral data for every scan without requiring protocol selection. However, spectral separation is constrained by the physics of scintillator absorption, resulting in greater overlap between the energy spectra than in source-based dual-energy. This overlap is mitigated using advanced reconstruction algorithms and deep-learning techniques.^{4,24} This transformed CT from a purely anatomic modality into one that also integrates tissue characterization and functional insights.^{25,26} Despite these innovations, performance remained constrained by the fundamental limitations of energy-integrating detectors.^{27,28}

Iterative Reconstruction

Filtered back projection (FBP) has been the standard analytic method for CT reconstruction, applying a filter to projection data and back-projecting the result to form images. The filtering step corrects the blurring inherent in simple back-projection, and the overall process is efficient. However, FBP is prone to increased noise and streak artifacts with reduced radiation dose or undersampled projection data. These shortcomings prompted the development of more advanced reconstruction methods.²⁹⁻³¹

IR algorithms address the limitations of FBP for applications requiring dose reduction. IR methods start with an initial image estimate and iteratively refine it by comparing it to the measured data. This process allows selective suppression of noise and artifact reduction, enabling radiation reduction while maintaining or improving image quality. However, IR can introduce unnatural image textures and is computationally more demanding than FBP, which has limited its universal adoption.^{27,32,33}

In both DECT and PCCT, IR improves image quality and quantitative reliability under challenging noise and spectral conditions by more accurately modeling polychromatic spectra, detector response, and statistical noise. This facilitates more robust material decomposition and helps translate spectral data into clinically usable images, such as low-noise virtual monoenergetic images (VMIs) and quantitative maps at doses comparable to conventional CT.^{27,33}

Deep learning image reconstruction (DLIR) utilizes convolutional neural networks to produce images with reduced noise and preserved anatomical detail, while maintaining a noise texture comparable to that of FBP. DLIR enables more radiation dose reduction without compromising diagnostic accuracy or low-contrast detectability. However, DLIR performance depends on the quality of training data and continued validation across a broad range of clinical applications.³⁴⁻³⁶

Introduction of Photon-Counting Detectors

PCCT has evolved through decades of innovation and translational research. In the early 2000s, General Electric (GE) conducted the first human scan using a prototype system, while Philips introduced the first rotating-gantry PCCT designed for small animal imaging, generating preclinical data.^{28,37,38} By the 2010s, both Siemens and Philips had developed advanced prototypes capable of whole-body imaging and spectral acquisition, enabling preclinical and early human research. This progress culminated in 2021 with Siemens' introduction of the first full-field-of-view clinical PCCT system, soon followed by platforms from other vendors (GE, Canon, United Imaging). The transition from research to clinical practice enabled large-scale studies confirming PCCT's advantages in spatial resolution, spectral imaging, and artifact reduction.^{28,39-41}

The difference between conventional and PCCT lies in the detector design. Traditional EID-CT systems use an indirect, two-step process. First, x-ray photons strike a scintillator that converts their energy into visible light photons. These photons travel to a photodiode, converting them into a cumulative electrical signal (see Figure 1). One drawback is that the light conversion results in loss of the spectral identity of individual x-ray photons. Furthermore, the light produced by the scintillator tends to scatter, necessitating physical septa-light-blocking dividers that isolate individual detector pixels and prevent optical crosstalk. The space occupied by these septa represents a "dead area" insensitive to x-rays, reducing the detector's geometric dose efficiency, particularly as pixel size decreases.^{38,42}

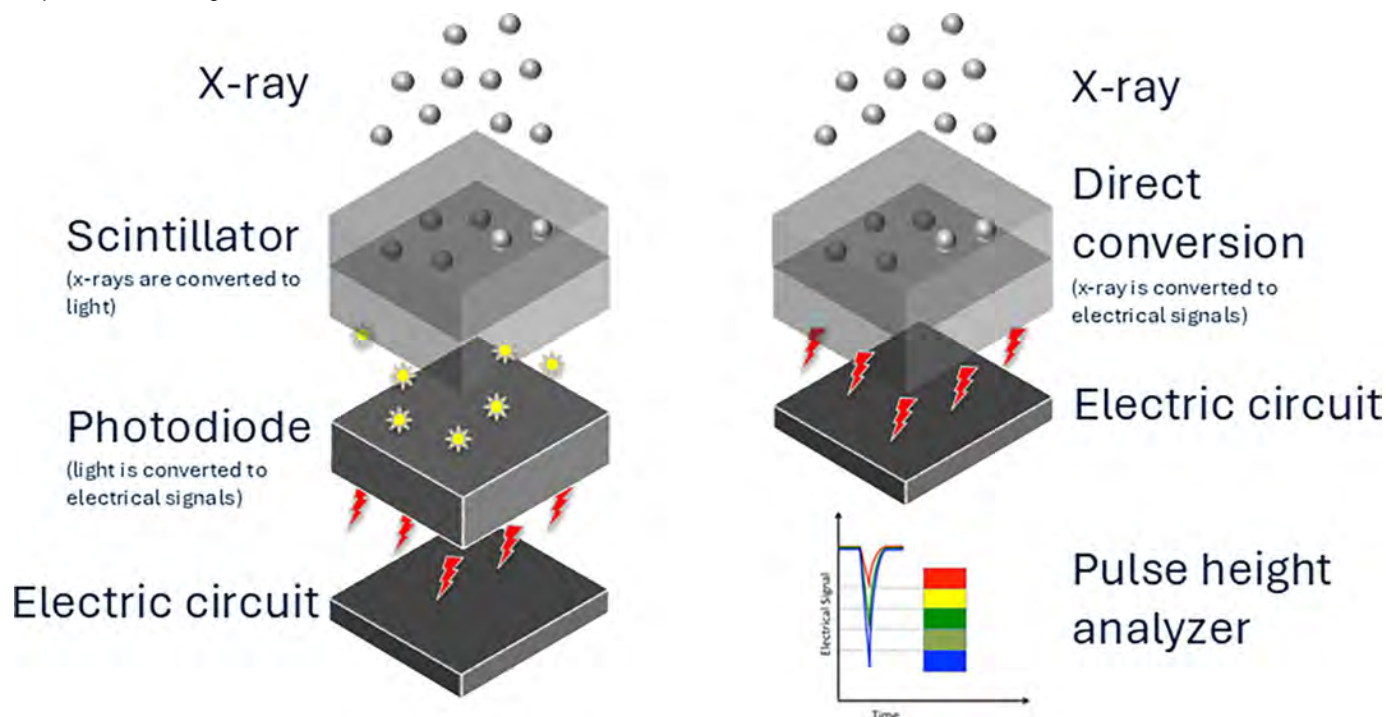
In contrast (see Figure 1), PCCT uses a direct, single-step conversion process. The detector is made of high-atomic-number semiconductor materials such as cadmium telluride (CdTe), cadmium zinc telluride (CZT), or silicon (Si), which are efficient absorbers of x-rays.^{38,42-44} When an x-ray photon strikes the semiconductor, it generates electron-hole pairs directly within the detector material. An electric field separates these pairs, driving the electrons toward a positively charged, pixelated anode, creating a short electrical pulse. The height of this voltage pulse is directly proportional to the energy of the absorbed photon. An application-specific integrated circuit (ASIC) captures each pulse, categorizing them into discrete energy bins according to preselected thresholds (see Figure 2). This direct detection and energy-sorting mechanism is the foundation for all the key advantages of PCD-CT.^{38,42,44,45}

Key Technical Advancements and Their Synergistic Effects

High Spatial Resolution

PCDs eliminate the need for physical septa because the signal is generated electronically rather than optically,

Figure 1. Comparison of energy-integrating detector (EID) and photon-counting detector architectures. Left: conventional EID-CT uses a 2-step, indirect conversion process in which x-ray photons are first absorbed by a scintillator that converts them into visible light. The resulting light photons are then detected by a photodiode and converted into electrical signals. This indirect process causes light scatter and loss of spectral information. Right: In photon-counting CT (PCCT), x-ray photons are directly converted into electrical signals within a semiconductor layer, eliminating the scintillator. Each photon is individually counted and its energy measured by a pulse height analyzer, enabling direct energy discrimination and multienergy data acquisition from a single scan.



allowing smaller detector pixels without sacrificing geometric detection efficiency. The result is a substantial increase in spatial resolution, enabling ultra-high-resolution (UHR) imaging with a single slice thickness as thin as 0.2 mm. For comparison, typical EID-CT detector pixel sizes range from 0.5 to 0.625 mm, achieving measured in-plane resolutions of 20–25 line pairs per centimeter (lp/cm) (10% MTF) with sharp kernels. As stated, PCCT detectors use subpixel binning ($0.151 \times 0.176 \text{ mm}^2$ at isocenter unbinned; $0.302 \times 0.352 \text{ mm}^2$ binned), enabling UHR modes with limiting resolutions of 40 lp/cm (125 μm) or finer. Reconstruction kernels affect realized resolution: standard kernels yield equivalent MTF curves across detector sizes (~ 10 lp/cm), while ultra-sharp kernels unlock PCCT's full potential, achieving 16–18 lp/cm (10% MTF) versus ~ 9 –10 lp/cm for EID-CT.^{39,46,47} This improved spatial resolution is

essential for visualizing fine anatomic structures and can be achieved without the dose penalty often associated with high-resolution modes on conventional CT systems.⁴⁸

Noise Elimination and Enhanced Contrast

Another advantage of PCCT is its ability to eliminate electronic noise.⁴⁹ The lowest energy threshold in the detector's counting process can be set to a level above the energy of pulses caused by electronic noise.⁴⁵ By filtering out low-energy photons, PCD-CT's signal contains primarily statistical quantum noise, resulting in lower-noise images, especially under low x-ray flux conditions. This is beneficial for low-dose CT protocols and for imaging obese patients.⁴⁵ Clinical studies report noise reductions of approximately 40% in PCCT compared with EID-CT at matched radiation

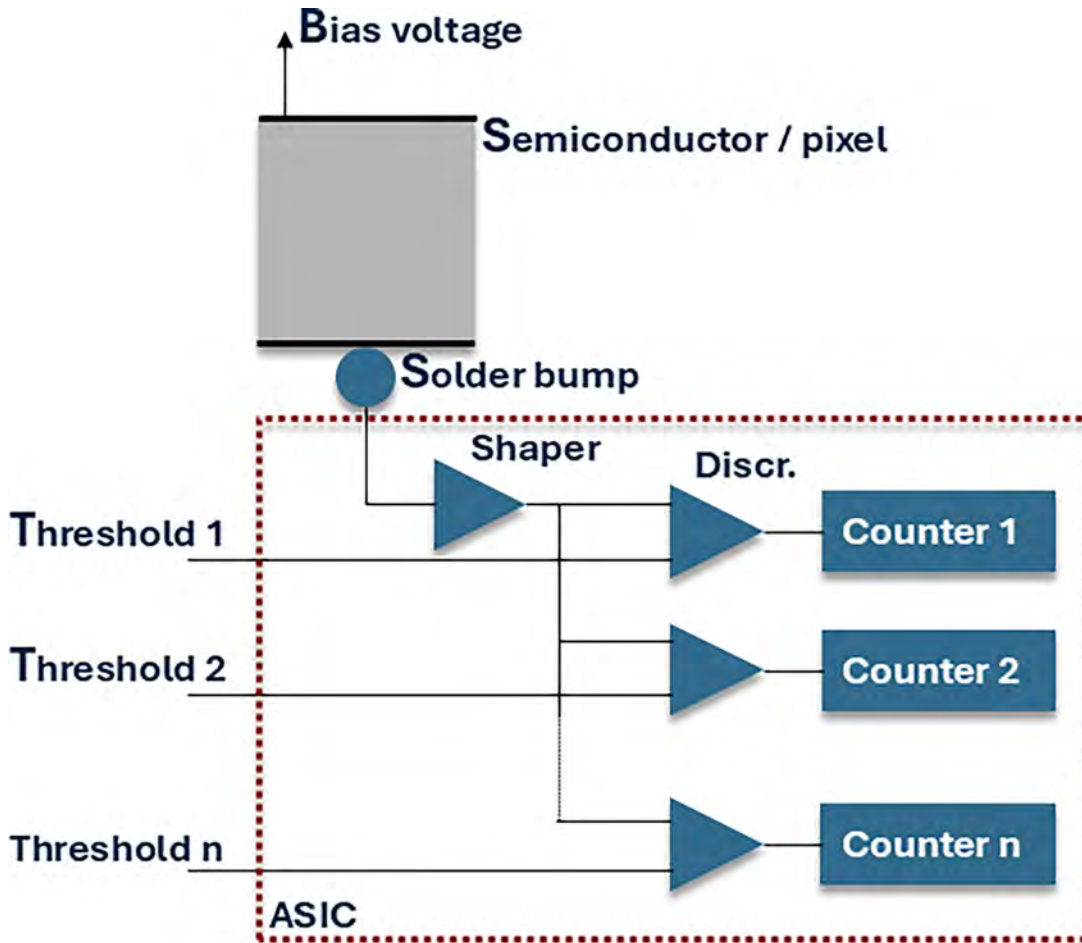
dose,⁵⁰ or radiation dose reductions of approximately 20–30% while maintaining equivalent noise levels.^{28,51}

Furthermore, the direct conversion approach gives equal weight to all detected photons. This is a change from EID-CT, where lower-energy photons contribute less to the final signal. Since low-energy x-rays are more likely to be attenuated by materials like iodine, this equal weighting enhances the contribution of these photons, resulting in a higher CNR and improved soft-tissue contrast.⁴⁵

Multienergy and Spectral Imaging

Another feature of PCCT is its inherent ability to acquire multi-energy spectral information. The detector's ASIC sorts photons into energy bins based on their deposited energy. This capability is inherently acquired, ensuring that spectral information is acquired for every scan without the need for a protocol

Figure 2. Schematic of a photon-counting detector pixel architecture. Each pixel consists of a semiconductor sensor connected to an application-specific integrated circuit (ASIC) via a solder bump. When x-ray photons interact with the semiconductor, they generate electrical pulses that are shaped and processed by the ASIC. The signal passes through multiple energy thresholds, each linked to a discriminator and a corresponding counter. This configuration allows the detector to sort and count individual photons according to their energy levels, enabling simultaneous multi-energy data acquisition for spectral CT imaging.



or prospective selection, as is often the case with EID-based rapid kVp switching, split-filter methods, and dual-source systems. This allows for retrospective reconstruction of advanced image types, including VMIs, virtual noncontrast (VNC) images, iodine maps, and other material-specific reconstructions.⁵²

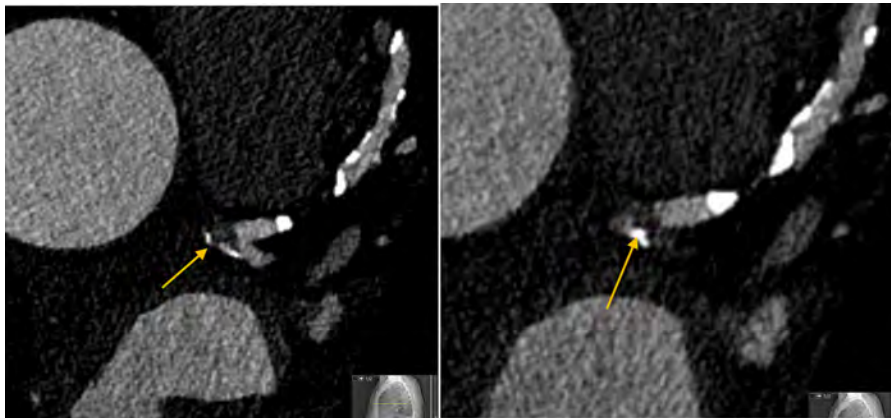
Quantitative assessment of spectral CT performance relies on spectral separation, iodine-related contrast-to-noise ratio (CNR), iodine quantification bias, and the effectiveness of iodine suppression in VNC images. In clinical PCCT, VNC quality is evaluated by comparing

attenuation in VNC reconstructions with true noncontrast images; for example, aortic valve calcium studies with dual-source PCCT have reported that the fraction of cardiac voxels above 130 HU is reduced from roughly 80% in contrast-enhanced CTA to about 0.2–0.6% in PCCT-derived VNC series, closely matching values of approximately 0.5–1% in true noncontrast scans and indicating near-complete suppression of iodine-related signal. Phantom studies of PCCT further show iodine quantification errors on the order of a few milligrams per milliliter (mg/mL) and percentage

biases within low single digits across a range of iodine concentrations and dose levels, supporting its use as a quantitatively robust platform for VNC, iodine maps, and virtual monoenergetic imaging.^{53–56}

For iodine maps, performance is characterized by iodine concentration error (mg/mL), percentage bias, and measurement precision across repeated acquisitions. Phantom experiments with clinical PCCT systems demonstrated small absolute iodine errors and low percentage bias, often within low single digits over clinically relevant concentration ranges and down to reduced dose

Figure 3. Photon-counting CT reduces blooming artifact from coronary calcium. Photon-counting detector CT with ultra-high-resolution acquisition (left, 0.2 mm ultra-high-resolution [UHR], Bv56u kernel) depicts distal left main bifurcation plaque with sharply defined calcified components and preserved visualization of the true lumen extending into the proximal left anterior descending and left circumflex arteries, minimizing calcium blooming. In contrast, a standard-resolution photon-counting detector CT acquisition without UHR mode (right) demonstrates more pronounced blooming of the same calcified plaque, obscuring the residual lumen and leading to overestimation of stenosis severity and potential up-classification of Coronary Artery Disease-Reporting and Data System (CAD-RADS).



levels, indicating accurate and dose-stable iodine quantification. VMIs are evaluated using iodine CNR, noise, and HU accuracy as functions of energy, and PCCT studies show that low-keV VMIs (around 40–60 keV) can provide increased iodine CNR while maintaining noise characteristics compatible with quantitative use, supporting PCCT as a robust platform for quantitative spectral imaging.^{55–58}

Clinical Applications

Technical enhancements of PCCT translate into clinical benefits across a wide spectrum of radiological subspecialties. In cardiovascular imaging, high spatial and temporal resolution and advanced artifact reduction techniques address the “triple threat” of motion, calcification, and stents, leading to more accurate stenosis assessment and a reduction in obscuring artifacts⁵⁹ (Figure 3). Reduction in stenosis degree often leads to reclassification rate to a lower CAD-RADS with PCD-CT, one study demonstrating a 49% rate of reclassification.⁶⁰ In ILD, ultra-high-resolution PCCT improved depiction of fine fibrotic changes, increasing the median visible bronchial generation from the 9th to the 10th order and raising the

proportion of clearly delineated bronchial walls from 23% to 57%, while reducing CTDIvol and DLP by approximately 27% and 32%, respectively, and reclassifying 4/112 patients from non-fibrotic to fibrotic ILD.⁶¹ In oncology, PCCT enhances detection of skull base infiltration, laryngeal cartilage involvement, and small pulmonary nodules through superior spatial resolution. Spectral capabilities provide higher iodine CNR in low-keV VMIs for improved lesion conspicuity and achieve 94% sensitivity for hepatic steatosis detection using VNC images. High-resolution imaging of trabecular bone microstructure improves visualization and characterization of osseous metastases.⁶² PCCT is poised to usher in new diagnostic paradigms, from quantitative data extraction during routine screening to functional imaging enabled by next-generation contrast agents.⁶³

Overcoming Technical Hurdles

PCCT's clinical adoption has required continued efforts to address several intrinsic physical challenges. Key issues include charge sharing, when a photon's charge cloud spreads across adjacent pixels and is registered at lower energies, and K-escape, when detector interactions generate secondary fluorescence that

can be absorbed by neighboring pixels. Both can reduce energy discrimination accuracy, but modern systems mitigate these effects through smaller pixel sizes and coincidence logic that combines simultaneous signals into a single count.^{28,64}

Pulse pile-up is another limitation, occurring when individual photon pulses overlap at very high x-ray flux and are registered as one event, which underestimates photon count and overestimates photon energy. It can be partially corrected with calibration-based algorithms, but is best limited by high-speed readout electronics and detector pixel optimization.^{28,64}

From a clinical standpoint, most charge-sharing and pile-up corrections are handled by scanner hardware and reconstruction software. Only extreme high-flux protocols, such as very high tube current cardiac imaging, may require additional protocol optimization, while routine practice is usually managed by automated flux control, optimized pixel sizing, and calibration-based corrections. These limitations therefore rarely affect day-to-day radiologist workflow, though they remain important for understanding scanner behavior at the extremes of dose and temporal resolution.

Conclusion

Over more than five decades, CT has evolved from rudimentary brain imaging to high-speed, high-resolution volumetric and spectral systems. Each innovation, from helical scanning and multidetector CT to dual-source geometries and iterative reconstruction, incrementally improved diagnostic confidence. PCCT represents a transformative step forward in computed tomography, moving beyond the limitations of conventional energy-integrating detector systems. By directly counting individual x-ray photons, this technology delivers a host of synergistic benefits, including unprecedented spatial resolution, the virtual elimination of electronic noise, and the intrinsic spectral data from every scan.

These technical advantages are directly translating today into improved diagnostic capabilities across a range of clinical applications, from cardiovascular and musculoskeletal imaging to oncology and pediatric radiology. The shift to PCCT is poised to enhance diagnostic confidence, streamline workflows, and create new opportunities for earlier disease detection and improved management. This transition, from protocol-driven to data-rich imaging, marks the next era in the ongoing evolution of CT technology.

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AI-Enabled Medical Imaging for a Changing Cardiovascular Disease Landscape

How AI-integrated imaging can redefine cardiovascular disease assessment through continuous phenotyping and personalized models

Partho P. Sengupta, MD, MBBS, FACC

Cardiovascular disease (CVD) remains the leading cause of mortality worldwide, yet its underlying risk profile is undergoing a profound transformation. Historically dominated by hospitalizations for acute symptomatic events like myocardial infarction, stroke, arrhythmias or decompensated heart failure, contemporary cardiovascular disease is increasingly driven by chronic, lifestyle-related exposures—obesity, sedentary behavior, metabolic dysfunction, and psychosocial stress. These risks accumulate gradually, interact nonlinearly, and manifest clinically over decades. Consequently, the central challenge in modern cardiovascular care is no longer limited to diagnosis, but extends to the continuous identification, visualization, and modification of risk across the lifespan.

Traditional paradigms of cardiovascular assessment are poorly

aligned with this evolving landscape. Clinical evaluation relies heavily on episodic measurements and threshold-based classifications, often detecting disease only after structural or functional impairment has occurred. Medical imaging, while central to cardiovascular care, has largely mirrored this approach—focusing on discrete parameters such as ejection fraction or valvular gradients. These representations, although clinically useful, fail to capture the dynamic and multidimensional nature of cardiovascular physiology.

AI Integration in Cardiovascular Imaging

Artificial intelligence (AI), particularly when integrated with imaging, offers an opportunity to

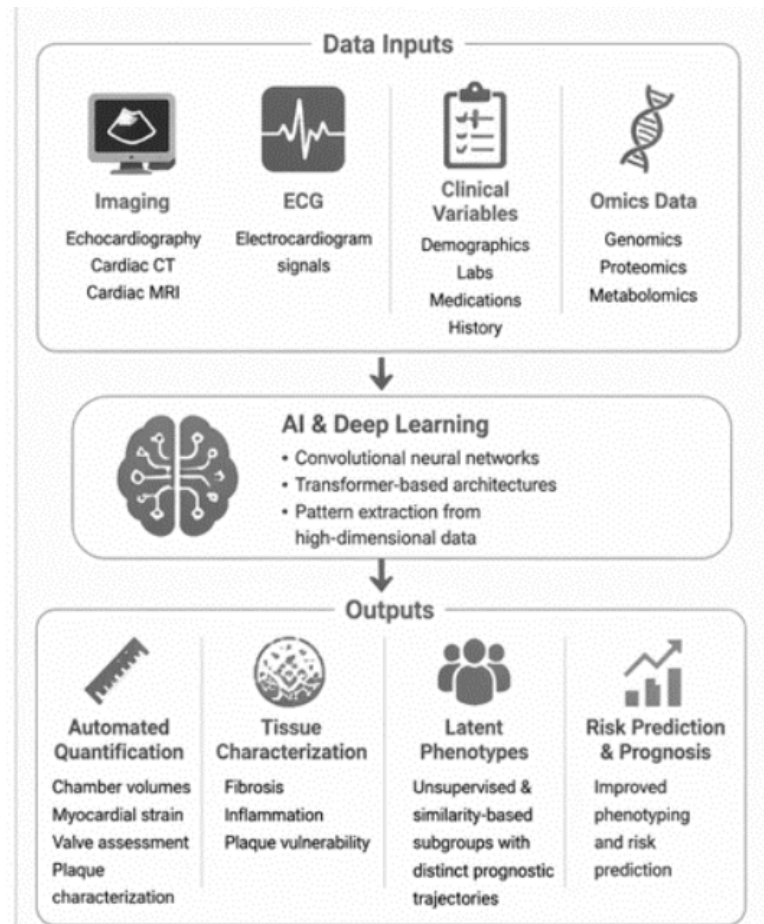
fundamentally redefine this paradigm. Over the past decade, AI has enabled automation and predictive analytics across imaging modalities, improving efficiency and reducing variability in tasks such as segmentation, quantification, and disease detection.¹ Across modalities such as echocardiography, cardiac CT, and cardiac MRI, deep learning—particularly convolutional neural networks and emerging transformer-based architectures—now enables automated chamber quantification, myocardial strain analysis, valve assessment, and plaque characterization with accuracy approaching expert readers. A major shift is toward integrated, multimodal AI, combining imaging with ECG, clinical variables, and even omics data to improve phenotyping and risk prediction. In echocardiography, AI is

Affiliations: Dr. Sengupta is a tenured Professor of Medicine, Chief of the Division of Cardiology at Rutgers Robert Wood Johnson Medical School, and Chief of the Cardiovascular Service Line at Robert Wood Johnson University Hospital.

“Cardiovascular risk builds continuously from lifestyle exposures, but episodic measurements detect disease too late, overlooking the dynamic and multidimensional nature of cardiovascular health.”

Figure 1. AI-Enabled Cardiovascular Phenotyping Framework

Multimodal AI integrates diverse data to extract patterns and enable superior phenotyping and risk prediction.



moving beyond static measurements to time-resolved waveform and video analysis, capturing cardiac mechanics (e.g., deformation, relaxation, and flow dynamics) and enabling detection of subclinical disease such as early diastolic dysfunction. In cardiac CT and MRI, AI enhances tissue characterization (e.g., fibrosis, inflammation, and plaque vulnerability) and enables rapid, reproducible quantification.

However, the clinical impact of these advances has been incremental rather than transformative, with limited evidence of improved patient

outcomes or cost-effectiveness. A key limitation lies in the prevailing approach which is that AI has primarily been used to optimize existing measurements rather than to rethink how cardiovascular disease is represented.

From Parameter-Based Assessment to Continuous Phenotyping

A more transformative opportunity lies in shifting from parameter-based assessment to continuous, data-driven phenotyping.² Cardiovascular

function is inherently dynamic, governed by complex interactions between myocardial mechanics, hemodynamics, and systemic physiology. Imaging modalities—particularly those capturing temporal signals such as echocardiographic motion or Doppler waveforms—encode rich, high-dimensional information about these processes. AI excels at extracting patterns from such data, enabling the identification of latent phenotypes that extend beyond conventional classifications. Indeed, unsupervised and similarity-based approaches have demonstrated the ability to define clinically meaningful subgroups with distinct prognostic trajectories, often outperforming traditional frameworks.

Digital Twins and Autoresearch in Cardiovascular Care

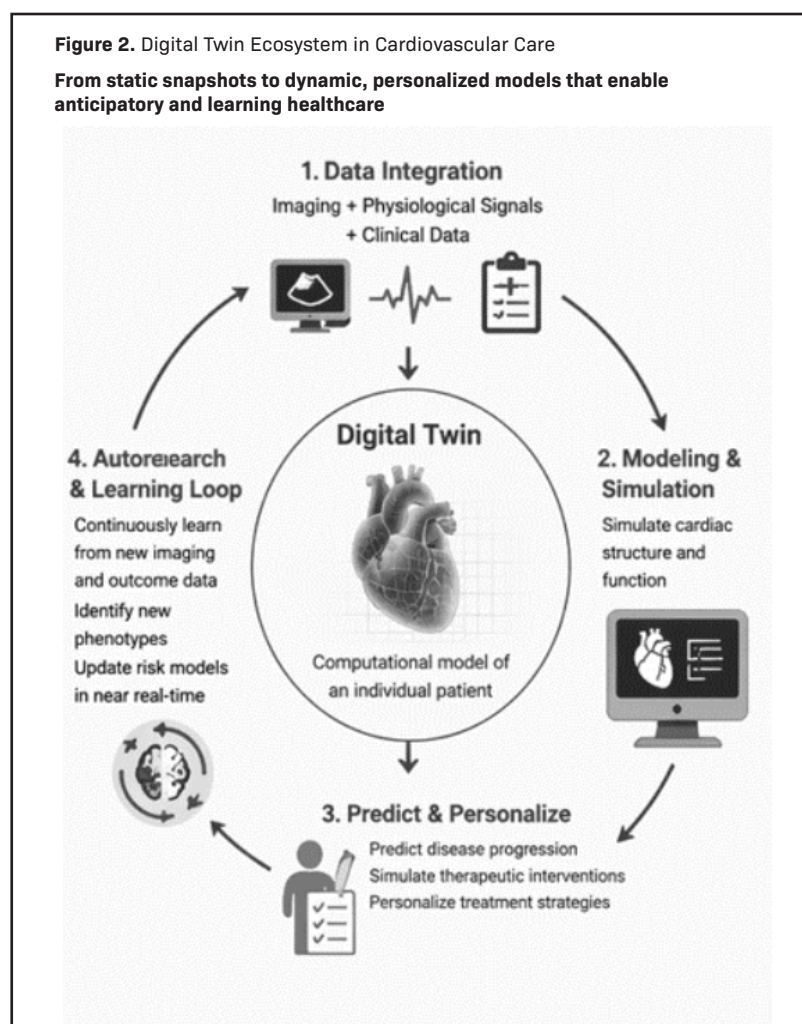
Within this evolving landscape, the concept of the digital twin has emerged as a powerful paradigm.³ A cardiovascular digital twin represents a computational model of an individual patient that integrates imaging, physiological signals, and clinical data to simulate cardiac structure and function. Rather than providing a static snapshot, digital twins enable continuous representation of disease states and their evolution over time. AI-enabled imaging serves as a critical foundation for constructing these twins, providing the high-resolution, multidimensional data required to model cardiac dynamics. Such models have the potential to predict disease progression, simulate therapeutic interventions, and personalize treatment strategies—transforming care from reactive to anticipatory.

“The future of cardiac care lies in making cardiovascular risk visible, dynamic and personalized through high resolution imaging and AI.”

Closely related is the emerging concept of automated AI and autoresearch, wherein AI systems not only analyze data but iteratively generate, test, and refine hypotheses within clinical datasets. In cardiovascular imaging, autoresearch frameworks can continuously learn from incoming imaging and outcome data, identifying new phenotypes, uncovering previously unrecognized associations, and updating risk models in near real-time. This represents a shift from static research paradigms toward a learning health system, in which knowledge generation is embedded within routine clinical care. By integrating autoresearch with digital twin models, it may be possible to move beyond population-level inference toward individualized, continuously evolving representations of disease.

Multimodal AI and Global Accessibility

The convergence of these concepts is further amplified by advances in multimodal AI. Modern systems increasingly integrate imaging data with electronic health records, genomics, and physiological signals such as the electrocardiogram (ECG). The PRIME 2.0 framework highlights this transition toward multimodal and generative AI, emphasizing the need for rigorous evaluation and integration across diverse data types.¹ In this context, the relationship between electrical and mechanical cardiac function becomes particularly relevant. AI models capable of



translating between modalities—for example, reconstructing mechanical function from ECG signals—offer the potential to extend imaging insights to scalable, low-cost platforms.⁴

This capability is critical for addressing the global burden of cardiovascular disease. Advanced imaging modalities, while informative, are resource-intensive and not universally accessible.⁵ By contrast,

widely available tools such as ECG or wearable sensors can serve as entry points for AI-driven phenotyping when linked to imaging-derived representations. In this framework, imaging informs the creation of high-fidelity digital twins, while scalable modalities enable their deployment across populations—bridging the gap between precision medicine and public health.

Augmented Intelligence and Implementation Challenges

Importantly, these advances must be viewed through the lens of augmented intelligence. AI is not a replacement for clinical expertise, but a tool to enhance it—integrating complex data, reducing cognitive burden, and supporting more informed decision-making. The goal is to enable clinicians to move beyond isolated measurements toward a more holistic understanding of cardiovascular disease, while preserving the nuance and contextual judgment that define clinical practice.

Significant challenges remain.⁶ Cardiovascular imaging data are inherently heterogeneous, with variability in acquisition, annotation, and quality that complicates model development and generalization. Ethical considerations—including bias, fairness, and data privacy—must be addressed to ensure equitable deployment.

Moreover, prospective validation and real-world implementation studies are essential to demonstrate meaningful clinical impact beyond technical performance.

Conclusion

In conclusion, AI-enabled medical imaging is poised to play a central role in addressing the changing cardiovascular disease landscape. Its true potential lies not only in automating existing workflows, but also in redefining disease representation—through continuous phenotyping, digital twin modeling, and autoresearch-driven discovery. By making cardiovascular risk more visible, dynamic, and personalized, these approaches can shift care from reactive treatment to proactive prevention. In doing so, they offer the possibility of not only reducing mortality but also extending healthy life expectancy in an increasingly complex and interconnected world.

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The Growing Demand for Lung CT Screening

Kerri Reeves, BA

Kerri Reeves is a freelance writer from Ambler, PA, and a frequent Applied Radiology contributor.

The demand for low-dose CT (LDCT) screening scans has increased steadily since 2021, when the US Preventive Services Task Force (USPSTF) broadened lung cancer screening eligibility criteria, lowering the recommended age from 55 to 50 years and pack-year smoking history from 30 to 20 years.¹ Still, screening volumes have significant room to grow due to education and access disparities across various demographics.

“We estimate that about 15.3 million people in the United States are eligible for screening, and roughly [only] 16% of them get screened. We’d like to get a lot more,” says Lary Robinson, MD, director of the Lung Cancer Early Detection Center for Moffitt Cancer Center in Tampa, Florida, who points out that lung cancer is the leading cause of cancer deaths in the United States². “An estimated 80,000 lives could be saved per year if every eligible person were screened, because you’d find the cancer [which has no symptoms] at a curable and early stage.”

Treating early stage lung cancer, identified through screening, yields both better clinical outcomes and lower costs, and LDCT is the only proven way to find early stage lung cancer and improve cure rates, according to Dr. Robinson. Despite screening program growth across the United States, including the incorporation of mobile CT units, limited public awareness of lung cancer screening—and the nuanced eligibility guidelines that determine insurance coverage—has hindered widespread uptake of task force recommendations.

Screening Education and Eligibility

Overcoming awareness barriers requires both primary care providers and public education of lung screening as an effective, early detection tool for at-risk people. Researchers who studied perceived barriers to LDCT screenings found that

40% of patients were simply unaware of lung cancer screening³—no one ever talked to them about it, Dr. Robinson says.

“There’s a real problem with awareness, so we’re working on trying to educate people,” he explains, describing lung cancer screening as “the new kid on the block” for both potential screening patients and primary care providers who must get on board with recommending it.

Specialty organizations, including the American College of Chest Physicians, the American Lung Association, the American Thoracic Society, the President’s Cancer Panel, and the American College of Radiology, provide a variety of frameworks, resources, and recommendations in tandem with initiatives to promote lung screening benefits and shared decision-making for patients. While campaigns are moving the needle in the right direction, it is taking time to translate knowledge into action.

“Where we are with lung cancer screening is where we were with mammography screening 20 to 30 years ago,” says Tina M. Sowers, FACHE, administrator of the thoracic service line at The James, The Ohio State University Comprehensive Cancer Center in Columbus, noting that mammography was not widely accepted or discussed much prior to a highly successful awareness campaign. “Like mammography, the more education we get out, the more awareness there is for people, the more we will see the demand for lung CT grow.”

Today, about 75-80% of eligible women are up to date with breast cancer screening,⁴ a stark contrast to the 16% of eligible people who have gone for their lung screen. Insurance eligibility requirements founded on USPSTF recommendations are much simpler for mammograms, says Dr. Robinson, while LDCT requirements for specific groups leave many people uncertain about how their specific

The mobile lung cancer screening unit from The James, The Ohio State University Comprehensive Cancer Center in Columbus, improves access throughout Ohio. Image/courtesy Tina Sowers.



smoking history factors into the determination for a covered exam.

“With current reimbursement policy, if someone has stopped smoking for over 15 years, they are no longer eligible for screening,” says Douglas Wood, MD, chair of surgery at the University of Washington in Seattle and chair of the Lung Cancer Screening Guidelines for the National Comprehensive Cancer Network (NCCN). He adds that barriers to better screening uptake include complex eligibility requirements and restrictions that some argue are unjustified. “This limitation is not justified by the data we know about lung cancer risk, and it also paradoxically gives individuals an incentive to resume smoking if they want to qualify to receive lung cancer screening in the future. That is just poor policy.”

Dr. Wood also notes that NCCN guidelines have simplified the inclusion criteria for screening, using smoking duration [20 years] as an alternative to pack-years. “Recent research shows that

smoking duration is easier to calculate, more accurately identifies individuals at risk of getting lung cancer, and lowers disparities of access to lung cancer screening due to a clearer understanding of inclusion guidelines.”

For people who don’t meet smoking duration or pack-year history criteria but have concerns for their health or non-smoking-related familial risks, they can seek the exam out-of-pocket, says Dr. Robinson, whose institution provides screening LDCTs at cost for \$189 to ensure “people can get screened if they want to,” he says.

Meeting Demand with Mobile Screening

In response to access barriers, mobile screening programs—complemented by educational campaigns that spread awareness of early detection to people who have smoked cigarettes—are

popping up nationwide to bring cancer prevention efforts to underserved areas.

Sowers said her institution has recently invested in mobile lung CT screening to increase rural access for eligible adults. As awareness of screening for lung cancer grows, it becomes critical to “meet people where they are,” she says, referring both to logistical considerations and an effective communication approach.

“Our goal is to ... make it easy and convenient for [patients] to have their lung cancer screening,” Sowers says, noting that Ohio’s mobile unit is closing gaps in nine rural counties that do not have any health care facilities, giving them “the ability to serve populations where the incidence of lung cancer is higher.”

The James has successfully partnered with community cancer prevention coalitions and other community groups to reach more screening candidates on their terms. Sowers says her team approaches education about screenings and smoking history in a judgment-free, culturally sensitive way. Dr. Robinson agrees that a positive provider outlook will continue to help reduce the stigma around patients being “at fault” for their risk due to smoking. He reports that Moffitt’s mobile unit has experienced high on-site demand in part due to shifting perceptions resulting from awareness campaigns.

“[People are] less likely to feel intimidated about going to get a screening CT scan,” he reports.

Regardless of screening setting, health system best practices include providers notifying patients of the findings, with processes in place that will support them in receiving timely, appropriate care. Ensuring that LDCT screenings and subsequent care pathways fit into the real lives of the communities served is critical to maintaining a program that successfully meets the needs of patients.

“The most important thing is for patients not to ignore the recommendations if they have a positive screen and see an experienced lung nodule physician,” concludes Dr. Robinson. “And if a solitary single lung nodule turns out to be cancer, it probably has been found at an early, and likely curable, stage.”

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Sylvian Fissure Lipoma with Associated Focal Cortical Dysplasia and Venous Malformation

Endri Gjonbrataj, MD; Adrian Hoti, MD; Altin Stafa, MD; Arben Rroji, MD

Case Summary

An asymptomatic adolescent who developed a scalp hematoma following a fall underwent a head CT to rule out parenchymal injury. When an incidental finding was noted, the patient was referred for further evaluation with an MRI.

Imaging Findings

Head CT (Figure 1) revealed a fat-attenuation lobulated mass overlying the right Sylvian fissure, surrounded by abnormally thick underlying cortex. Brain MRI (Figure 2) demonstrated a well-defined lobulated extra-axial right Sylvian fissure mass with high signal intensity in T1 and T2, surrounded by chemical shift artifact. The lesion was encased by a gray matter-lined cortical cleft. The signal dropped out in fat saturation sequences. No enhancement was noted except for the venous component traversing through the lesion.

Diagnosis

Sylvian fissure lipoma with associated focal cortical dysplasia and venous malformation.

The differential diagnosis between intracranial fat-containing masses should be made with intracranial dermoid (scattered fat droplets in the subarachnoid space if ruptured), intracranial teratoma, and lipomatous transformation of neoplasms such as primitive neuroectodermal tumor, ependymoma, or glioma. Thrombosed berry aneurysms (usually have a calcified rim or hemosiderin staining best seen in gradient echo and SWI sequences), while white epidermoids are rare and will show restricted diffusion. Intracranial lipomas are congenital lesions, usually asymptomatic; therefore, they are mostly diagnosed incidentally or after brain imaging due to related symptoms such as headache, epilepsy, psychomotor retardation, or cranial nerve deficits.¹

Discussion

Intracranial lipomas are congenital lesions and usually asymptomatic; therefore, they are usually diagnosed incidentally or after brain imaging due to issues such as headache, epilepsy, psychomotor retardation, or cranial nerve deficits.¹

Figure 1. Axial CT image demonstrates a well-defined extra-axial mass of fat attenuation with lobulated margins centered in the right Sylvian fissure. The surrounding cortex appears abnormally thickened (arrows).

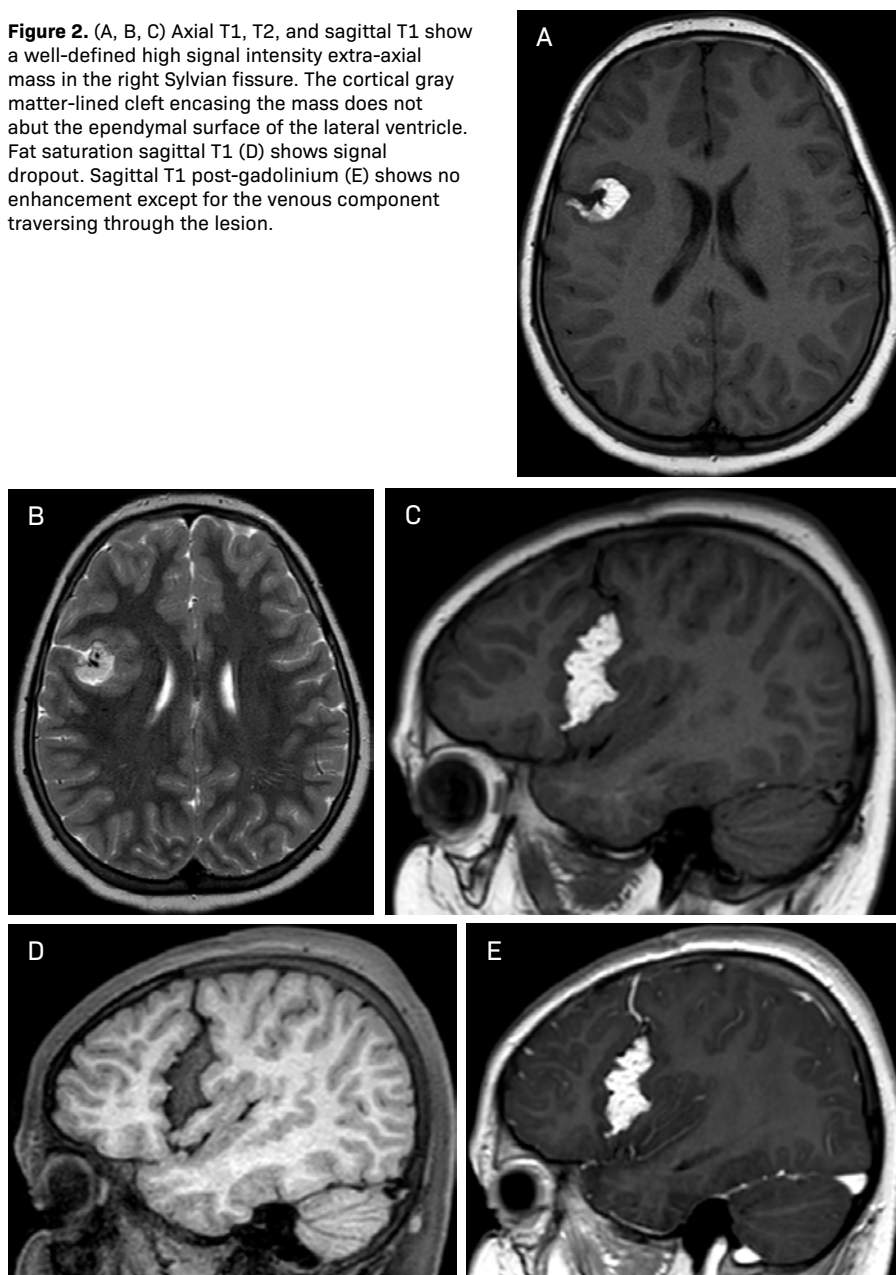


Intracranial lipomas are rare lesions with an incidence of 0.1-1.7% of all intracranial tumors, mostly located in the midline.² Regarding their location, intracranial lipomas can be found anywhere; nevertheless, the most frequently reported sites are: 50% pericallosal with associated agenesis or dysgenesis of the corpus callosum in half of cases and 25% in the quadrigeminal plate cistern with associated underdevelopment of the inferior colliculus. Suprasellar cistern lipomas are encountered in 15% of cases, while 10% are found in the cerebellopontine angle, with the facial and vestibulocochlear nerves often coursing through the lesion. Sylvian fissure lipomas are rare, accounting for

Affiliations: Department of Radiology, Catholic Hospital Our Lady of Good Council, Tirana, Albania (Gjonbrataj, Hoti); Department of Neuroradiology, Ca' Foncello Hospital, Treviso, Italy (Stafa); Department of Neuroradiology, Mother Teresa University Hospital Center, Tirana, Albania (Rroji).

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Figure 2. (A, B, C) Axial T1, T2, and sagittal T1 show a well-defined high signal intensity extra-axial mass in the right Sylvian fissure. The cortical gray matter-lined cleft encasing the mass does not abut the ependymal surface of the lateral ventricle. Fat saturation sagittal T1 (D) shows signal dropout. Sagittal T1 post-gadolinium (E) shows no enhancement except for the venous component traversing through the lesion.



only 5% of cases, and choroid plexus lipomas are even rarer.³

Among the various theories proposed to explain the development of intracranial lipomas, the most widely accepted is the *persistence and maldifferentiation of the meninx primitiva*, a precursor of the subarachnoid space. This theory accounts for the typical subarachnoid location,

associated parenchymal anomalies, and traversing vascular structures observed in such cases.^{4,5}

Various adjacent brain malformations are reported in 55% of cases with intracranial lipoma, with 36% of them showing traversing nerves and intracranial vessels through the lesion, best seen on high-resolution sequences.⁶

The most common associated malformations of the CNS in such a setting are agenesis or dysgenesis of the corpus callosum, absence of the septum pellucidum, encephalocele, myelomeningocele, vermis hypoplasia, cranium bifidum, spina bifida, and cortical dysplasia, as in this case.⁷

Imaging findings on both CT and MRI of intracranial lipomas reveal a mass of fat attenuation and signal intensity. Lipomas do not enhance. They have a lobulated appearance surrounded by a chemical shift artifact. They appear hyperintense in T1 and T2, show low signal intensity in the fat-saturation sequences, and produce blooming due to susceptibility artifact.⁸

In most cases, intracranial lipomas are asymptomatic, and no treatment is required even when associated with symptomatic malformations such as callosal dysgenesis. Local tumor growth is thought to be directly related to the onset or worsening of symptoms.

The main goal of surgery is adequate decompression. Because of their vascular characteristics and adhesion to surrounding parenchyma, resection attempts have sometimes led to little benefit and high morbidity; therefore, in most cases, only partial removal is carried out.^{1,9,10}

Intracranial lipomas are slow-growing tumors with a favorable outcome. If the lesion is diagnosed incidentally, the patient should be scheduled for follow-up. Seizures or hydrocephalus must be treated if present.^{1,9}

Conclusion

Sylvian fissure lipomas are rare congenital lesions, asymptomatic in most cases, and therefore discovered incidentally. Association with other coexisting abnormalities, such as focal cortical dysplasia or venous malformations, is even rarer, as in this case. Despite limitations to completely remove such lesions, decompressive surgery is the

mainstay of treatment in case of symptoms related to local tumor growth. The patient was scheduled for a follow-up assessment.

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Stomach-Containing Paraumbilical Hernia with Obstruction

Wayne Ngo, DO; Maria Francesca Ysabelle Martinez, DO; Miguel Salinas, MD; Mukul Maheshwari, MD; Reid Cline, DO

Case Summary

An elderly patient presented to the emergency department with abdominal pain, nausea, and vomiting. The patient had a known, large ventral abdominal paraumbilical hernia that was diagnosed 1 month prior on CT (Figure 1). This initial CT showed a large hernia sac containing small and large bowel loops without signs of bowel obstruction or strangulation. 1 month later, she returned with worsened abdominal pain, nausea, and vomiting. A repeat abdominal and pelvis CT examination was performed. This showed interval herniation of the distal stomach and proximal bowel into the patient's known large paraumbilical hernia, with resultant gastric outlet obstruction. After receiving intravenous fluid and nasogastric tube placement, the patient underwent surgery. The operative report described a large, ventral abdominal hernia with viable stomach, proximal duodenum (D1), cecum, ascending and transverse colon, and multiple loops of small bowel. There was no evidence of strangulation. The patient successfully recovered after surgery.

Imaging Findings

Prior comparison of abdomen and pelvis CT with intravenous contrast (axial and sagittal reconstructed images) (Figure 1) showed a large paraumbilical hernia containing small and large bowel loops as well as the appendix. There was no evidence of bowel obstruction or strangulation. Repeat abdomen and pelvis CT with intravenous contrast (axial and sagittal images) obtained 1 month later (Figure 2) showed interval herniation of distal stomach and proximal duodenum (D1 segment) into the known large paraumbilical hernia with resultant gastric outlet obstruction. There was no evidence of strangulation. Serial axial images from cranial to caudal (Figure 3) again show interval herniation of distal stomach and the proximal duodenum into the large paraumbilical hernia.

Diagnosis

Stomach within paraumbilical hernia.
Differential diagnoses could include incisional hernia, epigastric hernia, rectus

abdominis diastasis, and abdominal wall neoplasm.

Discussion

Abdominal hernias are abnormal protrusions of peritoneal lined organs through the abdominal wall. They are commonly encountered and have an estimated prevalence of 25% in adults.¹ Although abdominal hernias can be asymptomatic, surgical treatment may be required due to the risk of complications such as incarceration and strangulation.^{1,2}

In this case report, we specifically focus on midline abdominal hernias, which include epigastric, paraumbilical, umbilical, and hypogastric hernia types. Among these types, umbilical/paraumbilical hernias are the most frequently encountered and account for about 13.9% of all hernias. They occur in and around the umbilicus as a result of a defect in the linea alba and/or abdominal rectus muscle diastasis.² Patients typically present with an abdominal bulge that can be asymptomatic or tender to palpation. Ventral abdominal hernia contents are typically a combination of peritoneal fat, omentum, and small bowel.³ Larger-sized ventral abdominal hernias may contain large bowel as well. Stomach herniation into a pre-existing ventral abdominal hernia, however, is rare and has only been described a few times in the past century.⁴⁻⁸

Affiliations: University of Arkansas for Medical Sciences, Little Rock, Arkansas (Ngo, Cline); Baylor College of Medicine, Houston, Texas (Ysabelle Martinez, Salinas, Maheshwari).

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Figure 1. Abdomen and pelvis CT with intravenous contrast at initial presentation. (A) Axial slice at the level of the hips demonstrating a large midline ventral abdominal wall hernia sac containing large and small bowel with associated mesentery. (B) Parasagittal slice demonstrating a large complex paraumbilical hernia with dominant wide-necked hernia sac containing large and small bowel without evidence of bowel obstruction or strangulation. No herniation of the stomach in the hernia sac.

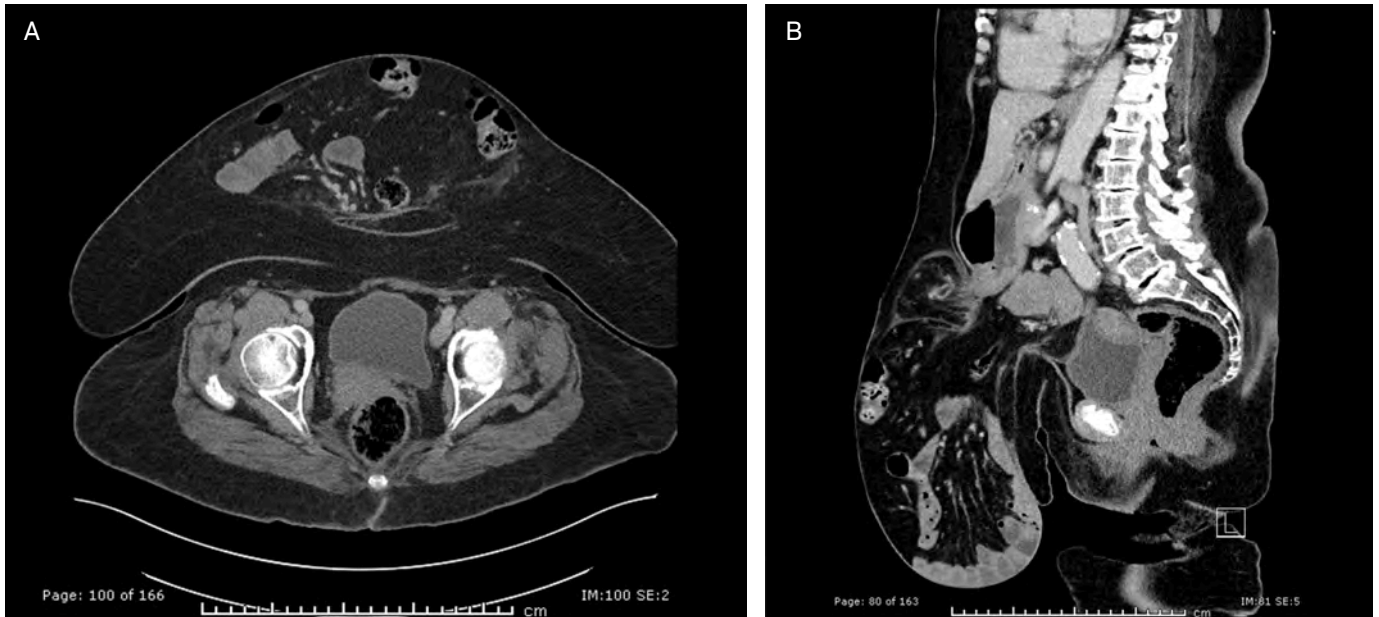


Figure 2. Abdomen and pelvis CT with intravenous contrast obtained 1 month later. (A) Axial slice showing interval herniation of the distal stomach into the sac along with previously seen colon and small bowel. (B) Parasagittal slice demonstrating a significantly distended stomach which narrows as it passes through the hernia neck. The proximal duodenum is also seen in the hernia sac (white arrow). There was no evidence of bowel strangulation. However, the patient had a resultant gastric outlet obstruction at this time, as evidenced by her fluid-distended stomach.

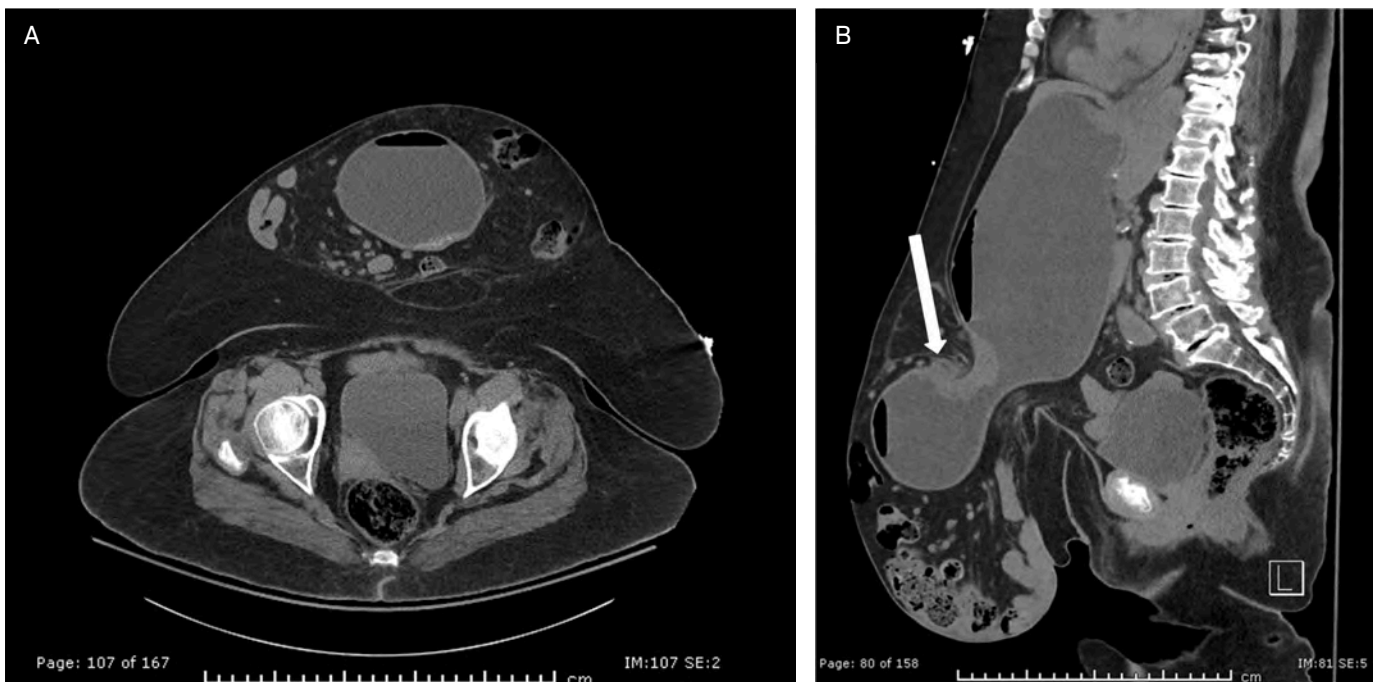


Figure 3. Additional contrast-enhanced abdomen and pelvis CT images acquired during the 1-month follow-up. (A) A more superior axial slice at the level of the kidneys demonstrating a fluid-distended stomach as a result of gastric outlet obstruction. (B) Axial slice at the level of the hernia neck showing herniation of the proximal duodenum (white arrow) and distal stomach with narrowing through the hernia neck.



Few similar previous case reports were found in a literature review. Thomas et al was the only other study to identify the stomach in a paraumbilical hernia on CT.⁸ In their study, a 91-year-old woman with a long-standing paraumbilical hernia presented with repeated vomiting and dehydration. Endoscopy showed external gastric compression, and CT revealed a hernia sac containing the distal stomach, pylorus, and proximal duodenum. Sampaio et al used a barium swallow to visualize herniation of the gastric antrum and duodenal bulb in a 59-year-old woman.⁷ Orr et al. used a similar technique to demonstrate stomach incarceration in a 49-year-old female with chronic vomiting.⁴ Bryk et al used barium swallow to show gastric outlet obstruction in a 63-year-old woman with large umbilical hernia.⁵ In another published case involving a 62-year-old woman, US demonstrated a herniated stomach appearing as a cystic mass with internal echoes protruding through the linea alba.⁶

The clinical findings in our case are consistent with those described in prior

reports. The most frequently observed symptom is vomiting, which carries a risk of dehydration and electrolyte disturbances. These hernias tend to occur in multiparous women in middle to later adulthood. Pregnancy is considered a risk factor due to the increased intra-abdominal pressure, which can stretch and weaken the abdominal wall muscles.^{8,9} Although the stomach is secured by multiple ligaments, these attachments may become increasingly lax with age.^{5,8} This laxity may contribute to the development of stomach herniation later in life, as seen in our patient.

The initial diagnosis of paraumbilical hernias is made clinically and can be assisted by plain radiography, barium studies, ultrasonography, or CT.^{2,9,10} Although previous cases have used barium studies and endoscopy, CT is currently the preferred imaging modality because it provides higher accuracy for identifying hernia contents and complications such as obstruction or incarceration.² US may also be useful because it allows real-time assessment of motion to identify

herniated organs. In our case, CT provided high anatomical resolution, enabling rapid identification of the hernia contents and assessment for bowel obstruction or strangulation.

Definitive treatment of paraumbilical/umbilical hernias typically requires surgical repair with sutures or mesh reinforcement.¹ Prophylactic surgical treatment is often recommended to prevent complications even in asymptomatic patients.⁹ Our patient successfully underwent ventral abdominal surgical repair and abdominoplasty with complex repair. A clinical implication from our case is that prompt surgical treatment at the initial encounter may have prevented her gastric herniation.

Conclusion

Gastric herniation into an existing paraumbilical/umbilical hernia is a rare complication. Patients may present with vomiting and gastric outlet obstruction. CT should be ordered to quickly identify

herniated content and other complications such as bowel obstruction, strangulation, and incarceration. Multiparous women in middle to late adulthood are at higher risk of stomach herniation due to weakened abdominal muscles from pregnancy and age-related laxity of stomach ligaments. Patients fitting this demographic should be educated about their risk of herniation and referred to surgical services for prophylactic surgical treatment.

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Abdominal Pseudocyst in a Patient with a VP Shunt

Mark Potesta; Emma Guld; Daniel Jacobson, MD

Case Summary

A middle-aged patient, with a history of long-standing hydrocephalus and ventriculoperitoneal (VP) shunt secondary to a ruptured posterior inferior cerebellar artery (PICA) aneurysm at the age of 2, presented with increasing upper extremity weakness that worsened over the course of 6 months.

A significant increase in ventricular size is observed during image comparison from a year prior.

A shunt tap revealed an opening pressure of 7 mmHg without indications of a cerebrospinal fluid (CSF) infection. A CT scan of the abdomen revealed a pseudocyst above the liver, with a catheter in situ.

The recent deterioration in the patient's condition, characterized by progressive upper extremity weakness, notable ventricular enlargement, shunt pressure measurements, and CT findings, indicated a need for surgical intervention. Surgery was performed to address both the abdominal catheter and valve concerns identified.

Image Findings

An axial CT without contrast reveals the presence of a large fluid-density mass

in the right upper quadrant measuring $10.9 \times 11.5 \times 5.6$ cm. This demonstrates a mass effect on the liver, with deformity of the hepatic contour. The VP shunt catheter can be seen as the dense curvilinear structure within the collection (Figure 1).

A coronal CT without contrast confirms these findings and shows the mass referenced above deforming the adjacent contour of the liver (Figure 2).

Diagnosis

A diagnosis of abdominal pseudocyst (APC) was made and confirmed through imaging. Differential diagnosis of abdominal cystic mass includes pancreatic pseudocyst, lymphocele, cystic lymphangioma, mesenteric cyst, benign cystic teratoma, seroma, cystic spindle-cell tumor, and cystic mesothelioma.¹

Discussion

The patient's complex medical history, stemming from a ruptured PICA aneurysm during childhood, necessitated the use of a VP shunt for his hydrocephalus. APC is a rare long-term complication of childhood hydrocephalus, with only about 2.3% of pediatric patients who have received a

VP shunt being affected by an APC later in life.²

It is crucial for the physician to account for the patient's medical history when considering an APC, as this diagnosis occurs exclusively in the setting of a VP shunt. Consequently, it will not appear on a common list of differential diagnoses without careful consideration of the patient's medical history. Other causes of abdominal cystic masses, such as a pancreatic pseudocyst, lymphocele, cystic lymphangioma, mesenteric cyst, benign cystic teratoma, seroma, cystic spindle-cell tumor, or cystic mesothelioma, may be more strongly considered in the absence of a history of VP shunt placement.¹

APC typically manifests as well-demarcated, near water-density abdominal masses. CT imaging shows the abdominal fluid collection in close proximity to the catheter tip of the VP shunt. Associated mass effects on neighboring structures may be seen. Less commonly, APC may reside within intra-abdominal organs, adding a layer of complexity to its diagnosis and management. The variability in APC presentation underscores the importance of comprehensive imaging analysis and clinical assessment for accurate and appropriate treatment.³

The etiology of an APC is not well known. The most widely accepted mechanism is that APCs arise from an inflammatory process.³ Other proposed mechanisms related to APCs include chronic infection, foreign body reaction, and particle-like protein in CSF.³ Abdominal adhesions also potentially play

Affiliations: Lake Erie College of Osteopathic Medicine, Bradenton Campus, Bradenton, Florida (Potesta, Guld); Buffalo General Hospital, Buffalo, New York (Jacobson).

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Figure 1. (A, B) Axial CT without contrast shows a large mass in the right upper quadrant measuring $10.9 \times 11.5 \times 5.6$ cm, appearing as a loculated collection that deforms the contour of the liver containing the distal tip of a VP shunt catheter.

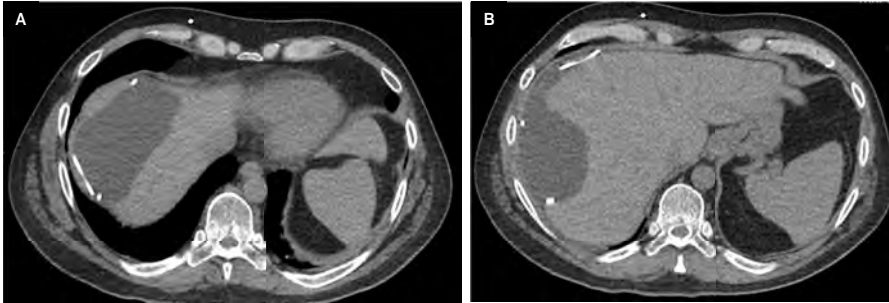
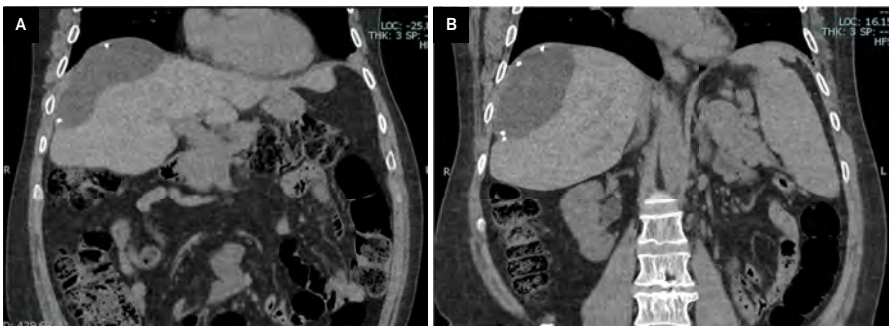


Figure 2. (A, B) Coronal CT without contrast shows a well-demarcated abdominal mass at the superior lateral portion of the liver, deforming its contour.



a role in the pathogenesis of APCs.⁴ Numerous dense adhesions were found and subsequently incised in the case of this patient. These proposed mechanisms need further exploration to reach a comprehensive understanding.

An untreated APC poses multifaceted complications. These complications include inadequate CSF drainage, associated hydrocephalus, and symptomatic presentations, including abdominal discomfort and pain. Complications such as abscess formation, cellulitis, and rare instances of gastric outlet obstruction are documented in cases of infected pseudocysts. When working up the etiology of an APC, CSF studies can lead to an accurate diagnosis. A shunt tap

or lumbar puncture enables the clinician to rule out catheter- or hardware-related infections. The ramifications of an untreated APC vary but often result in recurrent shunt failures and infections.⁴

Various interventions are used when treating an APC, depending on the clinical scenario. However, surgical resection with excision of the fibrous wall is the preferred treatment. These interventions attempt to minimize symptoms and prevent complications such as abscess formation and infection. In some cases, complete removal of shunt hardware via endoscopic third ventriculostomy may be considered.^{4,5} The choice of intervention depends on many factors, such as the severity of the patient's symptoms, the

size and location of the pseudocyst in the abdominal cavity, and the patient's overall health.

Conclusion

APC is a rare complication of an indwelling VP shunt catheters. The characteristic features of an APC on contrast-enhanced CT scans reveal well-defined abdominal fluid collection, often with mass effect, in a patient with abdominal pain and/or neurologic changes. This case demonstrates the rare occurrence of APC causing VP shunt malfunction in the presence of multiple, dense abdominal adhesions. Prompt diagnosis allows the physician to initiate interventional therapies, minimize the risk of infection/abscess formation, and allow for proper management.

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Congenital Zika Virus Infection and Chorioretinal Coloboma

Oswaldo A. Guevara Tirado, MS; Livja Mertiri, MD; Thierry A. G. M Huisman, MD

Case Summary

The patient is a child with clinical presentation and findings consistent with congenital Zika virus (ZKV) infection, who immigrated to the United States at the age of 2 years. The patient has a history of prematurity, seizure disorder, intellectual disability, cerebral palsy, microcephaly, dysphagia, slow transit constipation, and severe malnutrition. The patient presented to our institution due to a significant increase in seizure frequency, progressing from one seizure per day to approximately 6-8 tonic-clonic seizures per day, each lasting about 2.5-3 minutes. The patient has also had myoclonic jerks during the day. Treatment with valproic acid was initiated at the age of 1, and upon arrival in the United States, levetiracetam was subsequently prescribed. Neuroimaging was performed for seizure evaluation. Ophthalmologic consultation was performed because of "misaligned eyes." The findings on eye examination suggested a left-sided macular coloboma as well as a mild bilateral optic nerve atrophy.

Imaging Findings

An MRI of the brain (Figure 1) performed at 5 years of age demonstrated severe microcephaly with a markedly diffuse supratentorial simplified gyral pattern. There was incomplete/impaired myelination throughout the parieto-occipital and temporal lobes and subcortical white matter of the frontal lobes bilaterally. Severe global supratentorial white matter volume reduction was seen along with diffuse thinning and apparent foreshortening of the small corpus callosum, predominantly posteriorly. The bilateral lateral and third ventricles were markedly enlarged, while the fourth ventricle was moderately enlarged. The cerebellum appeared diffusely hypoplastic. Furthermore, there was a significantly enlarged mega cisterna magna, which appeared to communicate with the fourth ventricle while rotating and pushing the vermis upward, resulting in a moderately elevated tectum-vermian angle of 24 degrees. The pons was severely hypoplastic, and the pontine belly is completely missing with an apparent flat ventral pontine surface. In

addition, scattered peripheral parenchymal calcifications were seen (Figure 2). Lastly, there was a mid-sized coloboma along the posterolateral left eye globe in the region of the macula measuring approximately 0.6 cm in diameter (Figure 3). Both optic nerves appeared thinned with a maximal cross-sectional diameter of 1.4 mm.

Diagnosis

Congenital ZKV infection.

Discussion

Zika virus is a single-stranded RNA arbovirus of the *Flaviviridae* family that is generally transmitted by the *Aedes aegypti* mosquito. In adults, ZKV infection is typically asymptomatic, but when symptoms occur, patients report flu-like symptoms such as fever, headache, conjunctivitis, rash, and joint pain lasting days to weeks.¹⁻⁵ Vertical transmission to a fetus is reported and confirmed to occur when a mother is exposed to ZKV during the pregnancy. Children infected in utero, especially within the first or second trimester, tend to have a much more serious presentation. In early intrauterine infections, more first- and early second-trimester developmental processes, like neuronal migration, neuronal differentiation, sulcation/gyration, and myelination processes, may be

Affiliations: Ponce Health Sciences University School of Medicine, Ponce, Puerto Rico (Guevara Tirado); Edward B. Singleton Department of Radiology, Texas Children's Hospital and Baylor College of Medicine, Houston, Texas (Mertiri, Huisman).

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Figure 1. Sagittal T1 brain MRI (A) in a child with congenital ZKV infection shows severe microcephaly, hypoplastic pons (arrow) with a flat ventral surface, and an enlarged mega cisterna magna, which appears to communicate with the fourth ventricle (arrowhead). Axial (B, C) and coronal (D) T2 brain MRI demonstrate a hypoplastic cerebellum, as well as a markedly diffuse simplified gyral pattern with abnormal myelination and ventriculomegaly.

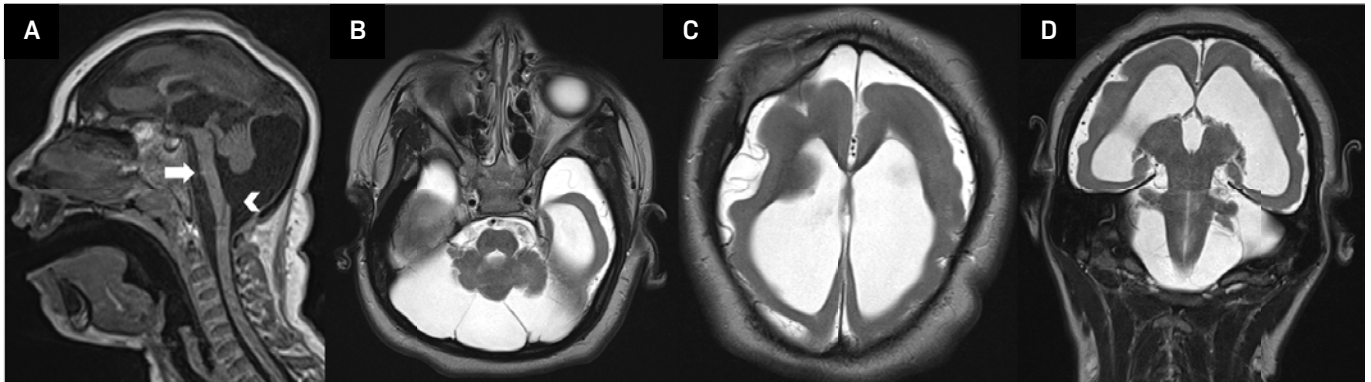
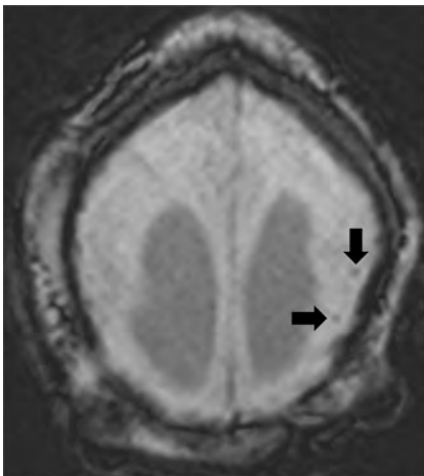


Figure 2. Axial susceptibility weighted imaging brain MRI demonstrating markedly diffuse simplified gyral pattern and scattered peripheral parenchymal calcifications.



impacted, eventually resulting in more severe brain malformations. Manifestations of congenital infection include severe microcephaly, joint contractures, hearing impairments, hypertonia, and ocular findings like chorioretinal atrophy, retinal pigment epithelium mottling, optic nerve hypoplasia or atrophy, iris coloboma, microphthalmia, and lens subluxation.¹⁻⁶ This constellation of abnormal central nervous system (CNS) and optic findings is known as congenital Zika syndrome (CZS).

The difference in presentation between adults and children is likely due to the ZKV's tropism for undifferentiated neurons over differentiated neurons.¹⁻³

Figure 3. Axial T2 brain MRI demonstrating a chorioretinal coloboma (arrow) located in the posterolateral aspect of the left orbit as well as mild bilateral optic nerve atrophy (right optic nerve not shown).



Intrauterine infection, especially in early pregnancy, can have devastating effects on the fetus, resulting in an overall decrease in neurons and consequently a number of brain abnormalities.^{1,3} Studies have also revealed that cells lining the blood-retinal barrier, retinal endothelium, and retinal pigment epithelium have an increased susceptibility to ZKV-induced cell death.^{1,3} As a result, extensive research has been conducted to investigate ocular manifestations (in addition to the CNS findings) in individuals with ZKV infection. These studies have demonstrated that ocular abnormalities, especially macular atrophy, occur so frequently in CZS that

they may serve as valuable diagnostic indicators for ZKV infection, even in the absence of microcephaly.¹⁻³ Recent efforts have also given clinicians ways to differentiate ZKV from other similarly presenting pathologies like rubella and toxoplasmosis.¹ Chorioretinal colobomas however are a much more rare presentation with only a few cases reported in the current literature.^{1,3,5,6} To understand how chorioretinal colobomas occur in CZS, it is essential to discuss what colobomas are, as well as how and when they form.

Colobomas are focal discontinuities or areas of missing tissue in the eye's structure.⁷ They occur when the embryonic choroidal fissure fails to completely close during weeks 5-7 of fetal life.^{7,8} They can present anteriorly in the iris or ciliary body, or more rarely, posteriorly in the choroid, retina, or optic nerve.⁷ Those diagnosed with posterior colobomas tend to be older in age.^{7,8} Patients with chorioretinal colobomas have a significantly increased risk for retinal detachment, with an incidence reported as high as 42%.⁸ Given the emergency of this complication, prompt diagnosis and patient education of red-flag symptoms are key. This is especially important in patients with diseases such as CHARGE (Coloboma, Heart defects, Atresia choanae, Retardation of growth, Genital abnormalities, and

Ear abnormalities) syndrome, VATER (Vertebrae, Anus, Trachea, Esophagus, and Renal) syndrome, MPPC (Microcornea, Posterior megalolenticular, Persistent fetal vasculature, and Chorioretinal coloboma) syndrome, and DiGeorge syndrome where chorioretinal colobomas can occur.^{7,8}

ZKV's neurotropism for undifferentiated neurons, in conjunction with our knowledge of posterior coloboma formation and presentation, allows us to form a reasonable hypothesis for why chorioretinal colobomas are so rare in ZKV patients. First, to develop a coloboma, fetal exposure to ZKV must occur early in the first trimester as colobomas typically form within weeks 5-7.⁷ Second, to find a chorioretinal or optic nerve coloboma, a patient must undergo a thorough ophthalmologic evaluation, likely with neuroimaging included. Given this knowledge, it is crucial for newborns suspected of early intrauterine ZKV infection to be promptly screened for orbit abnormalities via comprehensive ophthalmologic and neuroimaging evaluations. In cases of severe microcephaly, radiologists specifically should place special emphasis on studying the eyes in addition to the brain to

prevent these ophthalmologic findings from being missed.

Conclusion

Congenital ZKV syndrome is characterized by a series of CNS and optic findings, with chorioretinal colobomas being one of the more rare ocular presentations. This is likely due to the combination of the timing of coloboma formation and the neurotropism ZKV exhibits. As a result, interdisciplinary care of infants with suspected early intrauterine ZKV infection is essential for minimization of complications like retinal detachment, optimal management, and long-term patient care. Specifically, ophthalmologists and radiologists should ensure appropriate communication and perform careful and thorough evaluations of the orbit in patients suspected of congenital ZKV syndrome.

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CHARGE Syndrome

Julia Wieland; Daniel Wieland; Theresa Estiphan, MD; Brynn Weakley; Devri Weakley, MD; Tara Mangum, MD; Jasmine Knoll, MD; Cory M. Pfeifer, MD

Case Summary

A neonate was transferred to our neonatal intensive care unit (NICU) with respiratory failure. The child had been born at 39 weeks' gestation with a nuchal cord and required intubation at birth. The mother's reported pregnancy history was unremarkable, and no antenatal imaging was available. Upon transfer to the NICU, the patient was found to have a large patent ductus arteriosus, hyperphosphatemia, hypocalcemia, conductive hearing loss, and dysmorphic facial features. Hematologic workup revealed severe combined immunodeficiency. Physical exam was positive for dysmorphic facial features, including ear anomalies and micropenis.

Chromosomal microarray (CMA) analysis was unrevealing, but whole-exome sequencing confirmed a de novo chromodomain helicase DNA-binding protein 7 (CHD7) splice-site variant (c.2498+5 G>A).

The patient was discharged home tracheostomy- and feeding tube-dependent after an 8.5-month hospitalization on prophylactic antimicrobials (acyclovir, azithromycin, fluconazole, and trimethoprim-sulfamethoxazole) and infusion of

intravenous immunoglobulins. The patient returned to the hospital at 10 months of age, after having increased diarrhea and vomiting, followed by left-arm rigidity and right-eye deviation.

As of the end of the first year of life, the patient was awaiting a thymic transplant. The patient has been readmitted multiple times for infections.

Image Findings

Chest x-ray at initial presentation revealed an absent thymic shadow. Brain MRI at 22 days of age showed frontal-predominant low parenchymal volume (Figure 1A), which persisted at 10 months (Figure 1B). CT revealed lack of the clivus (Figure 2). Though dedicated imaging of the temporal bones was not performed, semicircular canals were not seen on CT of the head. There was no choanal atresia, and globe contours were normal.

MRI performed at 10 months of age showed multifocal diffusion restriction corresponding to multiple vascular territories (Figure 3). magnetic resonance angiography and magnetic resonance venography showed no focal stenosis in any of the major branches of the Circle of Willis and no dural sinus thrombosis.

Diagnosis

CHARGE syndrome.

The differential diagnosis includes benign enlargement of the extra-axial spaces, which typically presents with macrocephaly; normal variation of the thymic shadow, although the final diagnosis supported athymia; and isolated clival hypoplasia.

Discussion

CHARGE syndrome is a rare, autosomal-dominant genetic disorder that affects multiple areas of the body.¹ The name "CHARGE" is an acronym for several common features of the disorder, including coloboma, heart defects, atresia of the choanae (also known as choanal atresia), growth retardation, genital abnormalities, and ear abnormalities. In approximately 60%-65% of cases, the syndrome is caused by heterozygous loss-of-function mutations in the CHD7 gene, a chromatin remodeler gene essential for normal development.^{2,3}

The prevalence of CHARGE syndrome is approximately 1 in 10,000 births, and it is not typically inherited, with most cases being sporadic.⁴ Individuals with CHARGE syndrome may also have distinctive facial features, cognitive function ranging from normal intelligence to major learning disabilities, and various other health problems affecting the kidneys, immune system, spine, and limbs. CHARGE was originally based on the clinical presentation of 4-6 cardinal signs. It has since been updated by Blake et al, who focused on

Affiliations: Creighton University School of Medicine, Phoenix, Arizona (J Wieland); University of Arizona College of Medicine, Phoenix, Arizona (D Wieland); Department of Radiology, Phoenix Children's, Phoenix, Arizona (Estiphan, Mangum, Knoll, Pfeifer); University of Texas San Antonio Health Sciences Center, San Antonio, Texas (B Weakley); UT Southwestern, Dallas, Texas (D Weakley).

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Figure 1. Axial T2-weighted MRI of the brain obtained at 22 days of age (A) and 10 months (B) demonstrates low parenchymal volume, most pronounced in the frontal lobes, with expanded frontal extra-axial cerebrospinal fluid (arrows). Synthetic MRI at 10 months of age revealed brain parenchymal fraction of 10.3 standard deviations below normal for age, CSF volume 6.7 standard deviations above normal for age, and intracranial volume 3.9 standard deviations below normal for age.

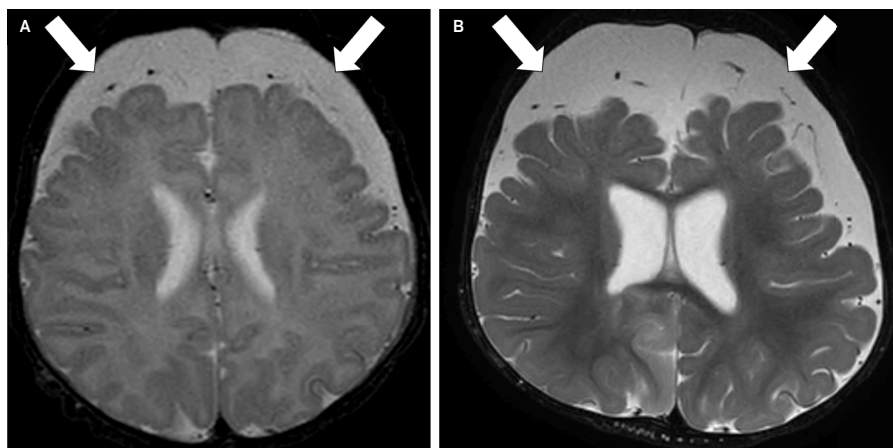


Figure 2. Sagittal CT of the head acquired at 10 months of age uncovered the lack of a clivus (arrow).



brainstem and cranial nerve anomalies, and Verloes, who focused on the 3C triad (coloboma, choanal atresia, abnormal semicircular canals), which is highly predictive of a CHD7 mutation.^{4,5} CMA

is initially performed for infants with multiple anomalies.⁶ However, if CMA is nondiagnostic, then CHD7 genetic testing is recommended in potentially affected individuals.

The CHD7 gene plays a crucial role in brain development, and heterozygous mutations in this gene have been characterized by brain structural anomalies and associated with various neurological conditions like CHARGE syndrome.⁷ An ATP-dependent chromatin remodeler, CHD7 is essential for the development of migratory, multipotent neural crest cells by controlling gene expression. Studies have shown that CHD7 is indispensable for mammalian brain development and controls a core neuronal differentiation program during cerebellar development.⁸ One study utilizing MRI and high-angular resolution diffusion MRI tractography demonstrated statistically significant reduction in volume of the bilateral putamen, brainstem, bilateral cerebellum, bilateral globus pallidus, and left subthalamic nucleus in patients with CHARGE syndrome compared with their gender- and age-matched controls.⁹

Thymic aplasia and hypoplasia are observed in patients with CHD7 mutations.¹⁰ In mouse embryos, Randall et al reported that approximately 11% of mice with the heterozygous CHD7 mutation had either thymic hypoplasia or an ectopic thymus, which was correlated with downregulation of the FOXN1 gene.¹⁰

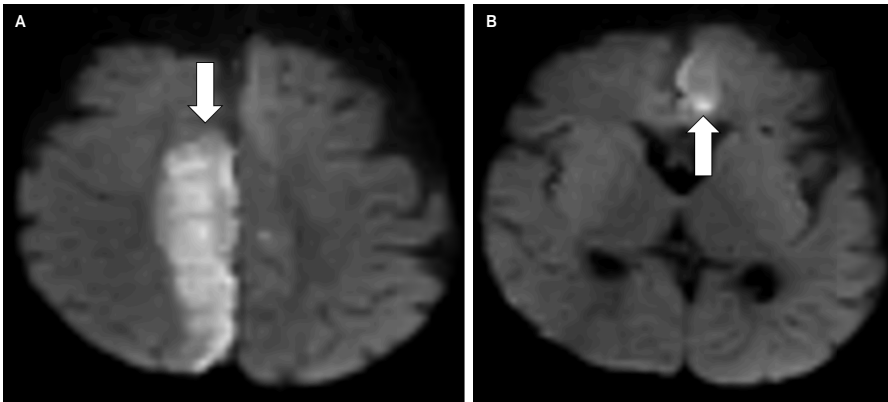
As part of his stroke workup, our patient was found to have a patent foramen ovale with shunting that puts him at risk of thromboembolic events, especially given the multifocal nature of the stroke. This could have been precipitated by diarrhea and/or vomiting-induced dehydration. The patient has been referred to the cardiology service for device closure.

There is no known increased stroke risk in CHARGE syndrome. The patient's stroke could have been precipitated by diarrhea and/or vomiting induced dehydration, which was not specific to the patient's diagnosis of CHARGE.

Conclusion

This case highlights a unique case of CHARGE with a CHD7 variant in which the diagnosis was not immediately evident

Figure 3. Axial diffusion-weighted MRI of the brain exhibits acute ischemia (arrows) in the posterior right paramedian frontal and parietal lobes (A) and the anterior left frontal lobe (B).



on imaging of the head as there was no choanal atresia and the globes appeared normal. Absence of a thymic shadow early in life may aid in suggesting this disorder. Clival hypoplasia may be a previously unreported but related finding. Although low parenchymal volume is nonspecific, frontal predominance may provide an additional diagnostic clue.

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If you can't add to the discussion, don't subtract by talking.

—Lois Wyse

Just Call Me the Narrator

C. Douglas Phillips, MD, FACR



Dr Phillips is a professor of Radiology, director of Head and Neck Imaging, at Weill Cornell Medical College, New York Presbyterian Hospital, New York, New York. He is a member of the *Applied Radiology* Editorial Advisory Board.

I have a few clinical colleagues who will invariably call me to discuss an exam that I've read (or occasionally one that a colleague has read).

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Yes. That's what I think it is also; that is the first line in my impression. Do you see it?

Ever want to reach through the phone and pull them to you so you can read the report together? Point at your words as they ask questions?

I appreciate my role in the health care team. I would like to think I do a good job. I also like to think that my reports are pretty good.

My feeling is that some people prefer the verbal input. I'm not even sure some of them read a report at all—maybe their staff does.

What did they gain? Audio.

What did I gain? Hmmmmmm. I don't think I added to the discussion. I just hope I didn't subtract with all that talking.

I know one thing for sure—that's one less exam I will turn over today.

Keep doing that good work, my fellow narrators. Mahalo.